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Pandora's Canister: A Preliminary examination of the Safety Assessment SR-Site for the SKB proposed KBS-3 Nuclear Waste Repository at Forsmark Sweden and associated activities relating to the disposal of spent nuclear fuel

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SSM2011-3522 for repository application
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Contents

1. Introduction

1.1 Scope

I have reviewed environmental impact documentation which consists of several thousand pages in three DVDs containing data, graphs, tables and text relating to the proposal to build a high level nuclear waste repository beneath the Baltic Sea at the Forsmark nuclear site in Sweden. In what follows I refer to pages in the Final report TR-11-01, FLIK1-FLIK16 and supporting documents. The purpose of my following report is two-fold. First, since the Swedish Environmental Court and SSM have asked in this round for a review of the data from SKB to highlight and failures of the group to provide sufficient data or analysis to fulfill its obligations under Swedish Law I will conclude with a list of questions regarding missing data or analyses. Second, I will make some general points about the process which may be of utility to those who are charged with allowing the project to go ahead. I am even grateful to Swedish NGO MILKAS for some financial support for this work.

In what follows I will refer to the SKB's Environmental Impact Report as EIR. I have already drawn attention to a number of concerns about the safety of this project which were based on the initial proposals which were provided before the full documentation became available (Busby 2011). In addition I have drawn attention at meetings with the Swedish Radiological Protection *Competent Authority* SSM and in reports and letters to SSM and also to the Swedish Justice Minister to the failures of the various competent authorities in Sweden to address concerns relating to the assessment of radiation risk in Sweden. SSM has not replied to these letters and the Justice Minister had written to say that such concerns are outside her department's remit. This is particularly of concern since there is no scientific reviewer whose independent knowledge–base and authority will be available to address the question of the radiation risk model on which the predictions of the health effects under various site-failure scenarios are based. This report examines the full environmental and safety case made by the proposers SKB in the documentation and critically reviews the arguments and data contained therein. In the time that I have had available I have not been able to examine the SKB calculations and computer codes and inputs whose results are presented in the various reports. This would take a considerable effort, but in my opinion this is necessary, for reasons I shall elaborate. However, I am able to state, from what I have read, that there is sufficient evidence that the safety case is not made.

1.2 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 from the University of Kent. I was elected to the Royal Institute of Chemistry in 1971 and the Royal Society for Chemistry in 1974. I am a scientific reviewer for, among others: *The Lancet, The European Journal of*

Cancer, The Journal of Paediatric Radiology, The International Journal of Radiation Biology, Science of the Total Environment and *Science and Public Policy* and some other journals.

I have studied the health effects of low dose radiation for 20 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 (www.cerrie.org) and the Ministry of Defence Depleted Uranium Oversight Board www.duob.org). I was Science Policy leader and the Senior Rapporteur on Ionising Radiation for the EU *Policy Information Network for Child Health and Environment* (PINCHE). I also have officially advised British government and other expert or investigative committees e.g. *The Committee on Radioactive Waste Management* (CORWM), the *US Congressional Committee on Veterans Affairs*, The *Royal Society, The House of Commons Enquiry into the health of A-Bomb veterans* and the European Parliament. I have been an official expert witness for the Canadian Parliament on the health effects of Uranium. I was until recently a fellow of the University of Liverpool in the Faculty of Medicine and I am currently Visiting Professor in the Faculty of Health (Department of Molecular Biosciences) in the University of Ulster in Northern Ireland where I have been supervising research on uranium photoelectron enhancement effects. I am Guest Researcher at the German Federal Agricultural Laboratories in Braunschweig near Hanover. I was recently appointed Visiting Researcher at Jacobs University, Bremen, Germany where I will be researching the effects of radionuclide contamination of the environment. 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 both its 2003 report and its latest reports *ECRR2010 Recommendations of the European Committee on Radiation Risk: The Health Effects of Low Doses of Ionising Radiation* and *Fukushima—What to Expect* (2012). The 2003 ECRR risk model has now been translated into French, Russian, Japanese, Spanish and Czech and has been used for radiation protection purpose scoping by many organisations including (2006) the UK Committee on Radioactive Waste Management (CORWM). I was invited in 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 (see below). I am expert witness in the on-going case of the Nuclear Test Veterans vs. MoD in the Royal Courts of Justice and have successfully overturned Ministry of Defence decisions in more than 6 Pensions Appeals relating to Nuclear Test Veterans. I have been retained on this issue for a further 16 cases which are currently awaiting trial. My current CV is attached.

My particular area of expertise is the health effects of internally deposited radionuclides, particularly Uranium. 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 and based on the Japanese A-Bomb studies and other external high dose exposures. The radionuclide dose coefficients published by the International Commission of Radiological Protection (ICRP) and employed in calculations made by this organisation and those that follow its methodology are unsound since they depend on inappropriate averaging of

energy in tissue, as I shall elaborate. 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 *Health Physics* 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 ICRP approach is fundamental to the approach of the discipline of *Health Physics* which is a black boxed version of it applied by technicians. I will argue that it is this approach which is the fundamental, though not the only flaw in the Forsmark safety case.

2 The Forsmark project

There are things we know that we know. There are known unknowns. That is to say there are things that we now know we don't know. But there are also unknown unknowns. There are things we do not know we don't know.

Donald Rumsfeld

2.1 The proposals

The problem of disposal of high level radioactive waste from civilian nuclear reactors has not been solved. Very large amounts of radioactivity are involved. A number of proposals have been made but to date none of these has been found to be ethically acceptable. This is because disposal always carries a risk (and ultimately in the long time scale, *a certainty*) of contamination of the environment with radioactive substances. This itself carries a finite risk of causing serious illness and death to humans and unquantifiable but serious effect on biota. For this reason, the enormously expensive rock storage project in the USA, Yucca Mountain, intended to deal with the historic high level waste from the US civilian reactors, 42,000MT of material in 1995 (Eisenbud and Gesell 1997) equivalent to about 1.5×10^{21}Bq of radioactivity, was cancelled. In the UK, the NIREX proposal for deep disposal was also ruled out in the 1990s following an enquiry in which it was shown that the cumulative uncertainties in the computer modelling of environmental risk were too great to have any faith in the results (Western 2009). This is a matter which is also applicable to the Forsmark project.

Forsmark is another deep disposal idea. A brief overview of the proposal is necessary, though it is well described with diagrams and cartoons in the available documentation. In the Swedish version the fuel elements from the ten operating Swedish reactors on three sites are removed from their current locations on site (either from the reactors themselves or from the cooling ponds where the initial spent fuel is reducing in activity through decay of the shorter half-life nuclides) and shipped by boat to the Oskarshamn nuclear site. Here they are placed in cooling ponds in a facility named CLAB where they remain until the activity has decayed to an acceptable level for them to be encapsulated in copper coated cast iron cylinders in a separate facility on the same site named CLING. The cylinders consist of 5cm of pure copper metal with cast iron inserts into which the fuel element carriages direct from the reactor fit, and after being inserted are dried and are filled with Argon gas and sealed by welding on a 5cm copper lid. They are then transported by road to a dock where they are taken by boat to the Forsmark site, some 180km north of the Oskarshamn site. There they are taken by road to the repository. The repository essentially consists of a system of tunnels located 500m below the Baltic sea on the coast. The cylinders are packed into cylindrical cavities drilled into the granite rock and they are then surrounded by Bentonite clay. This Bentonite represents a secondary containment should the primary containment (the 5cm copper) fail.

2.2 The quantities of radioactivity involved

12,000 tons of spent nuclear fuel by 2045 will be encapsulated in 6000 copper containers. Table 1 below is reduced from The Forsmark document *flik16 p647* which lists the components of an assumed spent fuel inventory in 2045. Not all the nuclides are listed in Table 2.1. The total quantity of material is enormous and can be compared with the releases from Chernobyl and Fukushima. It may be difficult for the lay person to grasp numbers so large. If we take the nuclide Sr-90 with an inventory of 1.6 E+19Bq, this compares with the releases from Chernobyl of 8 E+15 (http://www.bsrrw.org/wp-content/uploads/2012/03/fukushima-chernobyl-comparison-report-11.03.2011.pdf). In other words, a complete failure of the repository would release to the Baltic Sea 2000 times more Strontium 90 than was released by Chernobyl. For Caesium-137, Chernobyl released 3.8 E+16Bq and thus the failure of Forsmark would release the equivalent of 80 Chernobyl accidents. The quantities of Plutonium released by Chernobyl were significantly less, owing to its low volatility, but a similar approach suggests that the Plutonium releases from a Forsmark catastrophe inside the half life of Pu-239 of 24,000 years would be the equivalent of hundreds of billions of Chernobyl accidents in terms of Plutonium contamination. And how can anyone guarantee that there will be no failure in this period of time?

Table 2.1. Nuclide inventory at the repository in 2045 and corrected to encapsulation time, according to Table 5.4 on page168 of flik16.pdf. Note these are not all the nuclides but are those which the report considers to be mainly relevant for dose construction purposes in the case of a failure. The full list is not available in the documentation but is in a separate Data Report on the internet. I use the E-notation. Thus 1.7×10^{18} is written 1.7E+18. I have added the half lives and notes.

Nuclide	Half Life	Activity	Notes
Am241	432y	1.7E+18	α decays to Np-237 α ($T_{1/2}$= 2.14million y) Mp 1176; bp 2607
C-14	5730y	5.2E+14	Biological constituent
Cl-36	301000y	2.3E+12	
Cs-137	30y	2.3E+19	Bp 670
I-129	15.7 million y	1.4E+13	
Nb-94	20300y	9.3E+14	
Pu-238	87.7y	1.3E+18	α decays to U-234; adds to the U-234 chain
Pu-239	24,100y	1.4E+17	α decays to Y–235; adds to the U-235 chain Mp 639 bp 3228
Pu-240	6560y	2.5E+17	α undergoes spontaneous fission; decays to U-236 which α decays to Th232 and the Thorium alpha series (see below).
Pu-241	14.4y	1.1E+19	Quickly produces Am-241 α see above).
Sr-90	28.8y	1.6E+19	Bp 1372
U-234	245500y	6.0E+14	α with 6 alpha decay daughters including Ra-226 and Rn222 gas.

U-238	4.47 x 10^9y	1.3E+14	α ; represents about 10,000 tons mp 1132, bp 3818
TOTAL		5.3e+19	
*TOTAL α		1.4e+19	α is ionized Helium gas

* Assumes Pu241 has decayed to Am241 but does not include Np237 decay $T_{1/2}$= 2.14Million years

The table from which these data were taken is a fundamental one to the understanding of the project as it reveals the contents of the repository. It should be noted that the total content in terms of activity is far greater than this since a large number of decay chain nuclides and other nuclides are not listed.

So the first point to be registered is that the consequences of a failure of even part of the process would be grave. Chernobyl, one single Chernobyl, has caused devastation over large areas of Belarus, Ukraine and the Russian Republic. The health of the population of Belarus has deteriorated alarmingly to the point where there is now no replacement: more are dying than are being born, 4 out of 5 children are sick (Bandashevsky in ECRR2012). The effects have caused increases in cancer and leukemia in many countries in Europe including Sweden (Tondel 2004). It has caused the Baltic Sea to become the most radioactive sea in the world, with sediment concentrations of Cs-137 as high as 100kBqm^{-2} (HELCOM 2009).

This is a very high impact, low probability risk, just like Chernobyl, Three Mile Island, and Fukushima but considerably worse. There is another point. Note that Table 1 shows that the total alpha activity is more than 1.4 x 10^{19} Bq. Since each alpha decay produces one atom of Helium, this represents an increasing amount of Helium inside each sealed canister, a process which I return to below.

2.3 Oskarshamn: CLAB and CLING

The environmental impact report is modeled on the basis of a choice of radionuclides that SKB believes contribute the most significant exposures to humans and the environment in the event of a failure of the repository in the long term. However, the process involves shipping the fuel elements to the Oskarshamn site for cooling and encapsulation. This will result already in releases of activity to the environment of the CLAB and CLING facilities. The significant biologically important gaseous nuclide Tritium is missing from the list and there are expected to be releases of this and other gaseous nuclides to the environment of Oskarshamn. There are dangers inherent in the process leading up to the placement of the sealed canisters in the repository. These dangers are not examined in the EIR and I would wish to see a more in-depth safety analysis of this period in the sequence between the removal of the spent fuel and the placement of the canisters in the repository.

2.4 The safety case requirement of SSM

SSM require that it can be shown that the annual risk of harmful effects after closure of the repository at Forsmark does not exceed 10^{-6} per year. SSM state that this risk corresponds to a dose of 1.4×10^{-5} Sv (14 μSv per year). This calculation is based on the cancer risk model of the International Commission on Radiological Protection, ICRP. I assume that this requirement covers the entire period from removal of the spent fuel from the reactors to the closure of the repository as well as the period after the repository is closed.

3 Problems with the Radiation Risk Model

3.1 The ICRP risk model

The principal problem with the EIR is that the SSM requirement that it aims to follow itself is unsafe. If it is a decision by society to permit a fatal cancer risk from exposure to radiation of 1×10^{-6} per year this is equivalent to deciding to allow the nuclear industry to kill 12 people every year in Sweden, arguably a contravention of both International and National Human Rights agreements which ironically were mainly formulated in Sweden. However, the problem is that the conversion of this accepted level of risk into a radiation absorbed dose of 14 μSv per year relies entirely on the radiation risk model of the ICRP which has been shown unequivocally to be incorrect for the kinds of exposures delivered by the Forsmark modeled releases and has even been admitted to be incorrect by the editor of the most recent version of the model Dr J Valentin.
(http://www.euradcom.org/2009/lesvostranscript.htm)
The problem relates to the concept of *absorbed dose* itself. For internal exposures this quantity cannot be used to model the biological effects of radiation because of the completely different nature of internal radiation releases form internalised radionuclides.

3.2 The health effects of ionising radiation

In order to understand the argument that I will advance regarding the ICRP risk model it is necessary to have some basic understanding of the biological mechanism of action of radiation. The areas of radiobiology and radiation epidemiology, in which I have worked for 20 years have been undergoing a scientific revolution as a result of new laboratory discoveries and also epidemiological analyses of those who have been exposed to low doses of internal radiation e.g from Nuclear power station releases and from Chernobyl fallout.

Briefly, ionising radiation causes its harmful effects because it is genotoxic. It damages DNA at the cell level, and in the last fifteen years new evidence has become available from scientific laboratories that show that the subtle effects of low doses have significant long term genetic consequences both at the somatic (cell/ body) levels and also for germ cells (heritable damage). This process is termed 'genomic instability'.

It is the genetic damage induced in the DNA that results in increased risk of cancer, lymphoma and leukaemia. Increases in cancer, lymphoma and non Hodgkin Lymphoma have been associated with prior radiation exposure to internal radiation since the discovery of a ten-fold excess of Leukemia and Non Hodgkin Lymphoma at the Sellafield reprocessing plant in 1983. Since then, increases in childhood cancer have been studied and reported near many nuclear sites which release largely the same radioactive

material that will be released by failures of containment in the Forsmark process (ECRR2010). Most recently, in 2007, the German Childhood Cancer Registry (Kinderkrebsregister) published a study which showed that there was a statistically significant excess risk of childhood cancer and leukaemia in children aged 0-4 living within 5 km of German Nuclear sites between 1980 and 2005. This is the largest study of its type ever carried out and shows an effect which can only have resulted from inhalation and ingestion of radioactive nuclides or particles released from the plants (Spix et al 2007). Other examples of the failure of the ICRP model to predict or explain the effects of internal exposures include the discovery of infant leukemia in those who were in the womb as reported in five countries in Europe (Busby and Scott Cato 2000, Busby 2009) and also the correlation between cancer in Northern Sweden and Cs-137 contamination from Chernobyl reported by Tondel et al (2004). Taken together these show an error in the ICRP risk model for internal exposures to the kind of fission product mix potentially released by the Forsmark project of between 200 and 600-fold. That is to say that for a given internal dose, there will be between 200 and 600 times more cancers induced than are predicted by the ICRP risk model. For example, Tondel et al 2004 found a statistically significant 11% increase in cancer for each 100kBq/m^2 of Cs-137 contamination. Such a level of contamination will provide about 3mSv over a year and the ICRP would predict no excess cancers at these doses. Yet the cancer level increased in these areas after the Chernobyl radiation, and in proportion to the level deposited in the communities studied. This defines an error in the risk ICRP model of 600-fold. This would mean, in practice, that to conform to the SSM requirements of a risk of 10^{-5} per year, instead of 14μSv annual dose limit, the limit for the spent fuel radionuclide mix must be 23nSv per annum. The exact value can be calculated from the dose coefficients of the risk model of the ECRR which allow for the enhancement of hazard from individual radionuclides according to their affinity for DNA and other considerations (ECRR2010).

These increased levels of risk that are seen in epidemiological studies i.e empirical data from observation, have been routinely dismissed in the past by official radiation risk agencies on the basis that the absorbed doses are too low. In the case of the Sellafield children, the doses have been estimated to be about 0.4mSv at maximum; and for the other nuclear sites where cancer clusters have been confirmed (AWE Aldermaston, Harwell, Dounreay, Hinkley Point, La Hague, Kruemmel) the doses are much less than this. The question is always raised of comparison with variations in Natural Background Radiation. However the scientific concept of 'Absorbed Dose' is one which cannot be equally applied to all kinds of radiation exposure. The reason for this is that absorbed dose is a large scale averaging concept. Scientifically, Absorbed Dose is Absorbed Energy in Joules divided by the Mass of Tissue into which the energy is diluted. For external radiation, radiation from outside the body (which would be registered by a film badge) the quantity is valid. Dose from gamma rays from an A-Bomb, or even from gamma rays from fallout on the ground, can be compared since all the cells in the body get the same energy: all the DNA gets the same damage. But for a particle of radioactive fallout inhaled and translocated from the lung to the lymphatic system the dose to tissue or DNA local to the particle can be enormous.

3.3 External and internal radiation, the ECRR risk model

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 ECRR2010 report. A more accessible explanation of the basic science is given in my books *Wings of Death* 1995 and *Wolves of Water* 2007.

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 (e.g. heart disease, diabetes, arthritis, stroke, premature ageing, congenital malformation, fertility loss) (ECRR2010). 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 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 note here that the primary cause of mutation is the reactive ion or free radical produced by radiolysis and so it is the concentration of these species 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, Grays, 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 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 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 fallout dust particles. The ICRP are aware of these arguments and have acknowledged them by adding a weighting factor of 20 to alpha emitters because of the high ionization density in the cell caused by an alpha track. But ICRP stopped short of the logical extension of this, adding weighting factors for high ionization density near DNA to those nuclides which impart high ionization density to DNA by virtue of chemical affinity or otherwise e.g. Auger emitters. Although it was apparently considered in the 1970s it was shelved because it would have put too great a restraint on the nuclear industry (Jensen 2010).

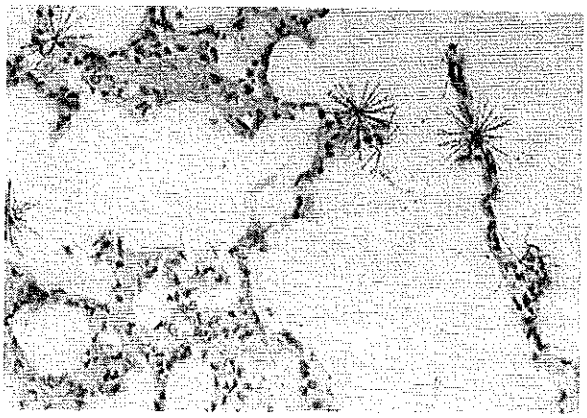
Averaging the energy into large tissue masses in whole body or in organs, dilutes the ionisation density and makes it seem as if the whole body doses are very low, perhaps well below natural background doses. But since cancer always starts in a single cell (as we know from mosaic studies of tumours) 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.

The European Committee on Radiation Risk (ECRR) an independent group of scientists was founded in 1998 to address this issue and in 2003 had developed and published a radiation risk model which employed weighting factors to accommodate hazard enhancements due to various kinds of internal exposures according to their ability to produce higher ionization at the nuclear DNA than the same dose delivered randomly and externally. It is a major plank of the ECRR deliberations and now in the mainstream of argument in the radiation risk community. For example in 2001-2004 the UK government set up the Committee Examining radiation Risk Form Internal Emitters CERRIE (www.cerrie.org). Although the final reports of this committee (which included members of the nuclear industry and regulators) disagreed about the levels of error in the ICRP approach, even the regulators and the nuclear industry members on the committee agreed that the uncertainty could be as high as 10-fold whilst others pointed to reports that showed that the errors were as high as 500-fold. Dr Jack Valentin, editor of the ICRP model talking in Stockholm in 2009 agreed that there would be situations where the uncertainty could be two orders of magnitude. He also stated that since he was no longer employed he could agree that ICRP and UNSCEAR were wrong in not addressing the evidence from Chernobyl that this was so. Chapters 5 and 6 of ECRR 2010 (the latest version of the model) 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. The CERRIE Majority Report (2004) stated (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) concluding 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 2005 the French official radiation risk agency, *Institut de Radioprotection et de Surete Nucliare (IRSN)*, agree that the ICRP dose averaging approach is insecure. They pointed 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.*

Fig 1 Alpha star photomicrograph showing radiation tracks emanating from hot particle in rat lung; track length has the distance of about five cells.



[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

sometimes empirical and pragmatic procedures must be applied.

But ICRP did nothing in their 2007 report to change any of the dose coefficients for isotopes that caused such exposures or to apply such *empirical and pragmatic procedures.*

In 2009 the 3rd International Conference of the ECRR in Lesvos Greece focused on Chernobyl research. Many papers were given showing the serious harm caused by exposures to the fission spectrum radionuclides released from the accident. The scientists who attended concluded with the "Lesvos Declaration" which called for the abandonment of the ICRP risk model which they stated would fail to protect members of the public in the event of another similar release. The Lesvos Declaration is attached as Appendix B. Sadly, the events at Fukushima have shown that the ICRP model, with its emphasis on external radiation and simplistic Absorbed Dose modeling is still being employed to protect the Japanese public. The results of this failure to learn from experience will be visited on the Japanese who failed to evacuate the contaminated areas.

3.4. Uranium

There is another way in which the ICRP external dose risk model is incorrect. It involves Uranium. Uranium is a major component of the Forsmark repository and represents about 10,000 tons of the spent fuel. Much has been learned about the health effects of Uranium exposure in the last ten years owing to concerns about deployment of Uranium weapons. I have been involved in this issue and have made some fundamental contributions.

Uranium has two singular properties which make it highly mutagenic. First it binds strongly to DNA, a fact that has been known since the 1960s when it came into use as a DNA imaging stain for electron microscopy. Second, it has the highest atomic number of any natural element, Z=92, and since the absorption of Gamma and X-rays by elements is proportional to the fourth power of the atomic number, Uranium in the body will absorb more than 50,000 times the background gamma radiation than tissue water. Thus it acts as an antenna, on the DNA, for gamma background radiation; the energy of which it redirects into the DNA as photoelectrons, identical with short range beta particles. It is this unlucky combination of properties that account for the serious health effects emerging in areas where the material is used as a weapon (Iraq, Balkans). My discovery of this Uranium photoelectron enhancement effect was developed in 2003-2005 but came to the attention of the wider scientific community after it was published in a conference proceedings from Germany (Busby and Schnug 2008) and was the main 2-page news item in the *New Scientist* in September 2008. There are now many reports of the dangers of Uranium exposure and I was the editor of the new ECRR report on Uranium which is a free resource on www.euradcom.org and is also part of the larger ECRR2010 report.

4. Problems with the Partition models

A brief account of the time period beyond one million years
Section 14.5; SKB Forsmark EIR

4.1 Philosophical and scientific problems: Modeling

The historical triumph of the scientific method in discovering truth in the natural world was a result of its empirical basis. Thus earlier attempts to model the Universe (by e.g the Church) were perceived to be incorrect when their predictions were compared with measurements. As science moved forward, experiment and mathematical descriptions of the results went hand in hand, resulting in valuable theoretical understanding of the nature of the physical world. For simple phenomena, mathematical approaches could predict and explain many observations, and in the last century were increasingly able to predict quite complex phenomena. However, the ability of mathematical reasoning to explain or predict phenomena becomes increasingly difficult or rather *uncertain* as the complexity increases. The usual approach is then to make a choice about what can be ignored and to carry on with a simplified system which can then be dealt with mathematically. The results of any such prediction can then be compared with the system being considered and the approach refined or abandoned according to the closeness of fit of the predictions. Whilst it is quite possible to employ such an approach to model an unknown system, to obtain some idea about what might happen at some future time as the system evolves, the results cannot be compared with the model predictions until the end of the experiment. This is the essential problem with the global warming modeling: we have to wait and see what the outcome is and then congratulate the modelers, or not. And it might be prudent to use such modeling as a guide to practice, to policy, and this is what is happening. Thus we use the Precautionary Principle. It cannot do us harm to assume that global warming is caused by anthropogenic CO_2 because a mathematical model supports this. But to use mathematical models to justify a sealed radionuclide repository containing thousands of Chernobyl accidents, a repository which cannot be opened and dealt with if the model is wrong, seems the height of folly. The Precautionary Principle here would make us refuse to be reassured by the results of a mathematical model.

The SKB Forsmark EIR is entirely based on mathematical modeling. Thus we have complex mathematical models for almost every aspect of the process and these models are used to predict that the project will be safe in the time frame of up to 1 million years.

A detailed risk analysis is required for the first thousand years after closure. Also, for the period up to approximately one hundred thousand years, the reporting is required to be based on a quantitative risk analysis. For the period beyond one hundred thousand years, the General Guidance states that a strict quantitative comparison of calculated risk in

relation to the criterion for individual risk in the regulations is not meaningful. Rather, it should be demonstrated that releases from both engineered and geological barriers are limited and delayed as far as reasonably possible using calculated risk as one of several indicators.

It is clearly impossible to model such time scales in a meaningful way. And of course, such a model cannot be compared by experiment with its prediction as we would have to wait a long time. For that reason however carefully the modelers approach the problem, the result must be considered unsafe. The result of an error in the models could be catastrophic: this is an area of low probability high impact risk. The impact, in the case of the failure of the process, the failure of the model, would be the contamination of the Baltic Sea and its coastal population with Strontium-90 equivalent to the releases from 2000 Chernobyl accidents. This would effectively destroy all life in the Baltic and make the coasts of Sweden, Denmark, Finland, Germany, Poland, Russian Republic and the Baltic States uninhabitable. So let me take a closer look at the models.

4.2 The computer models used to make the safety case.

The process involves what are termed *partition models*. The idea is to first determine what radionuclides are released to the environment following various different failure scenarios, termed FEPs (Features, Events, Processes). This is followed by modeling the intake or exposure to these environmental contaminations. This is followed by the conversion of the intakes to health problems in the exposed populations. The FEPs model all the *Events* that the SKB modelers can think of, presumably in some kind of corporate scientific brainstorming session. Of course, there may be events that the modelers have not thought of, the point of the quotation by Donald Rumsfeld. A number of these unknown unknowns have been at the origin of the Chernobyl and Fukushima catastrophes. I discuss one scenario that seems to have been overlooked by the SKB modelers below, the *Helium Explosion* .

The partition model consists of a system of ordinary differential equations representing radionuclide concentrations in a model compartment. An example of one such calculation is the rate of loss of a radionuclide from a corroded and failed canister and its flow toward the surface human environment. The COMP 23 model has 6 "B" compartments, 3 "C" compartments and a "D" and an "E" compartment (EIS Flik 16 Fig 13.2). These are 11 compartments. It is a statistical feature of sequential compartmental processing that errors associated with each rate constant k_i describing a flow from one compartment to the next are multiplied (not added) together to obtain the overall error in the final compartment. In this case the initial compartment is the concentration or activity of the radionuclide (i) in the failed canister and the final compartment is the concentration in the environment. If we assume that there is a mere 50% error in the value chosen or guessed at for each rate process in the 11 compartments the final error is about 85-fold. For a 25% error the accumulation is 11-fold. This accumulation of small errors in a complex system can make the final result so uncertain as to be useless. A number of computer models are listed in the report and are employed for various predictive purposes. They include MIKE-SHE, FARF21, COMP23, ERICA and the appropriately

named *Pandora* which calculated a quantity termed the LDF or *Landscape Dose Conversion Factor.* This *Pandora* (Eckstrom 2011 referred to in Flik 16) assesses potential doses to humans by multiplying different release rates (obtained from the COMP23 code and similar) by the LDF. Since the radionuclide model for the biosphere *relies on 140 input parameters of which a third represent radionuclide or element specific properties* (EIR Flik 16 p 87) we can imagine what the cumulative uncertainties are in the final outcome, or on the other hand how the model can be manipulated by its inputs to provide any result required. With regard to this latter point it should be noted that these models are software code written by employees of SKB and as far as I can tell are not peer-reviewed or examined. They are Black Boxes which take in data and emit results. I have not been able in the time to open any of these black boxes to see how they achieve their results but I would suggest that I am commissioned to do this before any major decisions are made regarding their accuracy.

4.3 The variation in and feedback between inputs to modeling parameters

I have argued above that the cumulative uncertainty in an 11 compartment model with a 50% individual transfer rate coefficient would result in an 86-fold uncertainty in the final value calculated. The individual data parameterisation errors can easily be 50%. The models used to provide the answer to the question: *is the process safe*? rely upon data and assumptions. Much of this data is known for *precisely defined laboratory conditions*, but is largely unknown for the chemical and physical environment for which the modeling is being carried out. A very good example is nuclide solubility. This is a key data input for the models. It is necessary to know the solubility of a wide range of radionuclides in the form of their compounds or complex ions in order to determine their rate of movement through a failed canister, through the buffer, the rock, the water in the repository and so forth. But the solubility of these radionuclides in the chemical form they are likely to be in at the temperature of the various compartments, in the presence of other elements as ions or complex ions, in the presence of adsorbates of many different types and materials is just plain unknown. So the modelers will use the laboratory-determined solubility or some version of this which they make an *expert judgement* on. The value chosen is unlikely to be correct, and this will introduce an error. The correct value *cannot be determined* without carrying out the experiment of building a pilot repository and making measurements using remote sensing equipment. The solubility of a chemical compound is enormously dependent upon the temperature of the solvent. The spent fuel is extremely hot, owing to the radioactive decay processes and the absorption of beta, gamma and alpha radiations by the material of the canister. It is stated that the surface temperature of the canister should not be appreciably more than 100 degrees, but to predict what the solvent temperature might be (and it will of course vary with distance from the canister, and be different for different canister geometries) is in my expert opinion impossible. The solubility can change by a *factor* of several times over the temperature range 0 to 100 degrees even in pure water. And example is the beta-emitting parent of Po-210 the Radon daughter Pb-210 Chloride which has a laboratory solubility in pure water of 0.67g/l at 0

degrees C but 3.34g/l at 100 degrees. Here we are not dealing with a 50% error in the data but 500 % over the range of temperature where a choice has to be made.

Then there is the effect of the radiation field and its effect on solubility for the various different radionuclides. Ionising radiation will result in radiolysis of water and the formation of hydrogen peroxide which will react with various radionuclides to form compounds with different solubilities. I have seen hardly any proper assessment of the effects of the radiation field on the components of the canister and the spent fuel. The high radiation field will certainly affect the copper. Over long periods of time metals become embrittled by radiation. Can we be sure that over hundreds of thousands of years this process will not cause the Copper to disintegrate altogether? Very few copper artifacts older than 2000 years are found in archeology. Or more serious, the metals which make up the cradles and tubes in which the fuel pellets are held at some critical distance apart from each other so as to reduce the possibility of a critical even, an explosion, like the one that happened at Chelyabinsk (Kyshtym) in 1959 and made a large part of Russia uninhabitable (Medvedev, 1979: Nuclear Disaster in the Urals).

The interaction of the radionuclides with each other and other material in the sealed canister over very long time scales does not appear to have been considered. The study of chemistry and chemical reactions has been carried out over time scales in which experimental results can be obtained. The working lifespan of a chemist may be 50 years. Few experiments are carried out over a period longer than a week. The late Prof George Porter, whose research field was fast reactions, was interested also in slow reactions, at least as an idea. We know that chemical reactions take place as a result of molecular collisions. There is no reason to suppose that very slow reactions, where the collision-reaction probability function is low, may still take place over very long period of time where the collision numbers are very large. It follows that we really have no idea what will happen in the spent fuel container, containing many radioactive elements, metals and non metals and gases, surrounded at high temperatures by radiation induced electrons and oxidizing molecular fragments, ions and radicals over very long time scales of hundreds, thousands and even hundreds of thousands of year. Over such time scales the idea that outcomes can be accurately modeled is ludicrous.

4.4 Features, Events, Processes; the Helium explosion

The EIS methodology consists essentially in thinking up everything that can go wrong (the Features, Events, Processes) and then using mathematical modeling (e.g Pandora) or *expert judgment* to show that it can't go wrong. The problem with this approach in a high impact low probability risk scenario is Donald Rumsfeld's problem: there are unknown unknowns. I have identified one problem which I now discuss which does not seem to have been examined sufficiently.

From Table 2.1 we can see that there is a high level of α activity in the canisters. The total amount is actually significantly greater than the value given because the table does not include the decay daughter sequences of the various nuclides listed. Some of these are given in Table 4.1

Table 4.1 Some series alpha emitters not listed in Table 1 (intermediate betas not listed).

U-238	***U-235***	**Am241**	**Pu-238**	**Pu-239**	**Pu-240**	***Th-232***
U234	Pa231	Np237	U-234	U-235	U-236	Th-228
Th230	Ra223	U-233	See **U-234**	See ***U-235***	Th-232	Ra-224
Ra226	Ra219	Th-229			See ***Th-232***	Rn-220
Rn222	Po215	Ac-225				Po-216
Po214	At215	Fr-221				Po-212
Po210	Bi211	At-217				
	Po211	Po-213				
		Bi-209				

It is clearly possible, though I have not had the time, to calculate the exact alpha flux with time on the basis of summing the various decays of all the nuclides and their alpha emitting progeny. However I can use a conservative approach by employing the total repository value of 1.4×10^{19} Bq to make some interesting schoolboy calculations. In this I ignore the decays of the parent in the series since as Table 4.1 shows there are a whole series of alpha emitters downstream of the shorter half-life parent alpha-emitter nuclides.

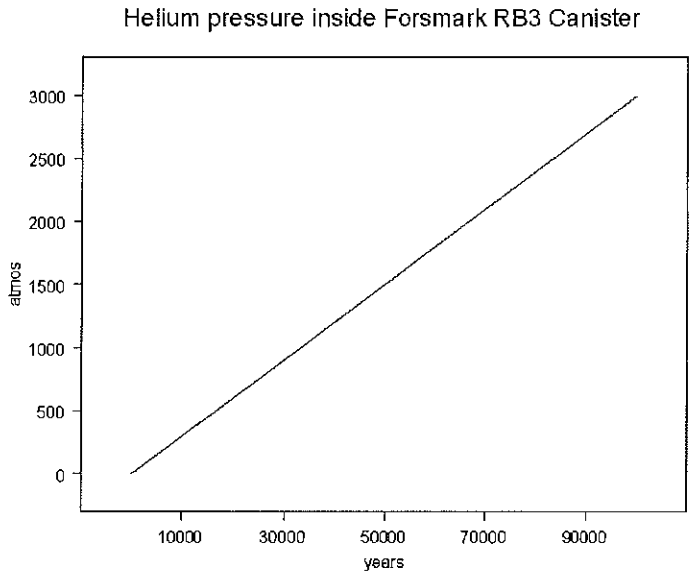
If there are 6000 canisters, the content of one canister by simple division is 2.3×10^{15} Bq of alpha emitters, and since an alpha particle is a charged Helium ion, this means there are the same number of Helium gas atoms being produced every second. In 100,000 years (the kind of time frame we are being invited to consider) this is 7.25×10^{27} atoms. One Mole of an element contains 6×10^{23} atoms and so there will be 12088 Moles of Helium produced in the canister in 100,000 years. Since Gay Lussac's law tells us that at STP (ambient temperature and pressure) one Mole of a gas occupies 22.4 litres, we can say that the volume V of the Helium in a canister at STP would be 270789 litres i.e without any consideration of heating expansion. Let us turn to the canister. The BWR canister is a cylinder of diameter 100cm and length 483 cm. It is full up with an iron cylinder containing 12 square cross section channels of 16cm x 16cm. Thus the total available volume without the spent fuel assemblies is 1483 litres. If I assume that this space is filled up by the assemblies to 90% capacity, the remaining volume will be 148.3litres. But we have 270289 litres of Helium. Using Boyles Law, which can be written $P_1V_1 = P_2V_2$ the pressure in the canister is now 1829 atmospheres (185.3MPa). This ignores Temperature effects which we can happily also model. Such a model is far simpler than "Pandora". I assume a temperature in the canister of 200 degrees greater than the initial Argon fill temperature. This brings the internal pressure up to about 3000 atmospheres (304MPa). I think we can assume that the canister would have violently exploded long before the 100,000 years are up since the design was made to withstand

isostatic pressure of 45MPa from the outside (geophysical effects), not the inside (Flik 16 Section 12.7.1). I have not included the large amounts of Radon which will be created from the Ra-226 but these will be second order.

Then let me turn to the Bentonite backfill, the secondary containment. The sudden explosive release of about 300,000 litres of hot Helium gas will probably blast most of the Bentonite plug out of the channel like a cannon shell and create its own channel to the surface. This will carry with it large amounts of contamination and allow in water. The new water channel will then dissolve the radionuclide components of the canister and carry them to the surface where they will contaminate the Baltic Sea above the repository. The presence of Iron and Copper together in moderately saline water will create an electrochemical couple which will quickly dissolve the canister, adding to the general chemical and physical reaction complex. Once the water is in, then we may see criticality (see 5.2 of this report).

The Helium production is a Feature. Its explosion is certainly an Event. And the Process can be easily envisaged without more modeling than the application of simple chemistry and physics. But it was not envisaged by the SKB modelers. What is worrying (besides the Helium explosion and the destruction of the Baltic sea and the death of the coastal populations of course) is perhaps there are other FEPs which are Rumsfeld's unknown unknowns, which have not been thought of. I have spent about a week on this and certainly have not had time to examine the whole project in sufficient depth, but was able quite quickly to see one simple flaw that seems to have been overlooked. What is unknown here, of course, is the capacity of the welded copper lid to sustain internal pressures to blow it off. It is likely to be far lower than the 45MPa design load for external pressure. This information is missing from the EIR. The Helium generation is approximately linear with time. The graph in Fig 4.1 here shows the internal pressure in atmospheres in a canister calculated on the basis of the assumptions above. The pressure after only 1000 years will be roughly 30 atmospheres (450lbs in^{-2}, 3.04MPa) which is about 31kg cm^{-2}. The outward force on the copper lid of area 7855cm^2 1000 years after the repository was sealed will be 243505kg or 243 tonnes. If I assume that the canister has the same ability to resist internal pressure as a steam boiler, then it will explode at a pressure of about 25 atmospheres, 830 years after the repository is sealed in 2045 i.e. in the year 2875. Clearly this prediction can be refined when the data is supplied. But it is not as if the scientists at SKB missed this problem. I note that in Section F-15 it states: *Helium production is neglected since the amount of Helium produced will not increase the pressure inside enough to affect its mechanical stability.* This is such a fundamental issue that I had my own calculations checked by Dr D E Caddy of the University of London (Queen Mary College). However, my assumption of 90% space filling may be incorrect, the exact proportion is not given in the data provided, and this value will alter the time period for the explosion to occur. But it will occur as the volume of helium is very much greater than the available volume in the canister even with no allowance for contained fuel.

Fig 4.1 Internal pressure (atmospheres) in a Forsmark RB3 canister over time due to Helium gas produced by alpha particles.



5. Other concerns

5.1 Missing exposure routes

The models for exposures are black-boxed and their assumptions, inputs and data are not presented in the documentation. Two exposure routes are particularly relevant and I will raise this as an issue here. The first is the inhalation of airborne particulates and gases. This is relevant to the process and specifically to the population of the community of Oskarshamn, the location of the CLAB and CLING facilities. Since nuclear sites are now known through many research reports to cause child leukemia in those living within 5 km, and since my own researches have shown (see Busby 2007, CERRIE 2004b) that these effects extend to female breast cancer, it would seem that the potential releases from the CLING/CLAB site should be modeled and tabulated both for routine releases and for various FEPs involving the earlier stages of the overall Forsmark project. The procedures leading up to the point where the intact sealed canisters are put into the repository seem to represent a much greater and immediate danger than the repository itself. This is because there are very many handling and moving stages involved. I opportunities for error in all these movements represents a grave risk of accidental release of large amounts of radioactivity. The amounts of gaseous high activity material (noble gases, Iodines, Chlorines, Tritium) involved in this early stage of the project is very large.

The second exposure route that must be examined closely is sea-to-land transfer of radionuclides in Baltic Sea sediments. This is already a serious health hazard which is entirely ignored by SSM. As a result of Chernobyl, weapons fallout and also historic releases from the Swedish (and other) nuclear plants, and Studsvik the Baltic is now the most radioactive sea in the world. There is already evidence that coastal populations of the contaminated Irish Sea have a significantly higher risk of cancer (CERRIE 2004b, Busby 2007) and there is some evidence that this is also true of the Baltic (Busby 2010, Hakkulinen 2010) though research funding for a INTERREG IV study by the ECRR Baltic Sea group at the Karolinska Institute was refused in 2011.

Inhalation is an overlooked area of radiation risk, and this is particularly relevant for the alpha emitters like Uranium and Plutonium which are present in the environment as nano particulates. The ICRP dose coefficient for inhaled U and Pu are more than 400 times greater than for ingestion (ICRP72). The ECRR adds significant enhancements to Uranium exposures and evidence from those battlefields where Uranium weapons have been deployed shows alarming increases in cancer and birth defects (Busby et al 2010, Alaani et al 2011).

For these reasons I would need to see the details of the calculations in the models which resulted in the doses calculated in the EIR for the various scenarios envisaged.

5.2 Criticality

The EIR states (flik 16 p254):

As long as the containment is intact, the possibility of criticality is ruled out. Therefore, no safety function related to criticality is formulated for an intact canister. See further Section 8.4.
But we have already seen one scenario, the Helium explosion, and several FEPs already allow for the failure of the canister. So it seems unusual that the modelers do not include a safety function for this possibility. The section 8.4 looks in more detail:

Uncertainties in the determining parameters such as the position of the assemblies in the canister, the manufacturing tolerances and the size of fuel compartments in the insert, and temperature, variation with enrichment, were taken into account. The calculations were performed for fresh fuel with an initial enrichment of 5% U-235. For a loaded and sealed canister filled with argon the keff value is less than 0.4 and the system is strongly subcritical.If it is assumed that the canister is leaking and that the canister storage positions and the fuel assemblies are water filled, the reactivity will increase. With all fuel element locations occupied in a canister deposited in the repository, surrounded by 35 cm bentonite and filled with water, the following results are found:
BWR: keff = 0.9959 ± 0.0002
PWR: keff = 1.0888 ± 0.0002
It can therefore be concluded that the reactivity criteria could not be met for a failed canister with the pessimistic assumption that the fuel is fresh.

This is not reassuring. The margin is very fine. For the PWR the neutron multiplication factor is actually positive. And this is with the fuel separated by a centre to centre distance of 21cm (BWR) and 37cm (PWR). If there were damage to the mechanical support system (which is assumed to remain intact for a million years) then the fuel pellets would fall to the bottom of the canister in a heap. The neutron flux would then increase, the temperature would increase and criticality would be extremely likely. Fresh fuel contains U-235 in this scenario, but spent fuel contains Plutonium and Neptunium. It is not at all clear why the calculation was not made for the actual spent fuel in the canister. Furthermore, no calculation seems to have been made for MOX fuel which has a Plutonium component and for which the parameters are quite different. It was the MOX reactor 3 at Fukushima which exploded violently in what many to believe to have been a nuclear criticality. The Chernobyl explosion was demonstrably a criticality: this was shown by the analysis of the Xenon isotope ratios as measured by Russian scientists at St Petersburg. The spent fuel tank at Kyshtym in the Chelyabinsk facility exploded in 1959 causing contamination of hundreds of square kilometers of land which had to be made into an evacuation area (Medvedev 1978). Calculations made by various scientists at the time showed mathematically by modeling that this was impossible. Yet it happened. Clearly there are complex issues here which are beyond calculation. I will make one suggestion of a mechanism which is overlooked by the

modelers. The melting point and boiling point of Plutonium is lower than that of Uranium. If the temperature becomes high (i.e. meltdown) the Plutonium will fractionally distill off from the mixture and condense in a cooler part of the canister. The critical mass for Plutonium is much lower than Uranium.

5.3 Bias and Spin: The equivalent doses calculated for chosen scenarios

In presenting the results, and indeed in the entire set of documents, the approach has been to make is seem that the process is harmless or even environmentally friendly. I have referred elsewhere to earlier reports on the issue from SKB, how the reports contain high quality whole page photographs of wildlife, sunlit streams and beautiful scenery. We see the same approach throughout the documentation, with one entire page showing a close-up photo of a flowering wild plant. What we do not see, and which is more relevant to radiation, is a photo of a child with no hair who is undergoing treatment for leukemia. I was tempted to make a whole page of this report into such a picture, but have desisted.

The point however is a relevant one: the report is written in such a way as to influence those reading it to agree with its (quite differently presented) apparently scientifically obtained conclusions. More serious is the way in which various aspects of the scientific case are presented. For example, the graph on page 715 of Flik 16 Fig 13.61 shows the near-field equivalent doses to the local population and compares these with natural background in Sweden. For a lay person not familiar with 7-decade logarithmic axes it might seem as if these doses were not much greater than the natural background in Sweden. But such a display squeezes the axis at the top of the graph. What this graph shows is that if the canisters fail but the Bentonite remains intact, the radiation doses are about 3mSv above background for a period of 10,000 years. For the more serious Case D (where both the backfill and the canisters are breached, one consequence of the Helium Explosion scenario) the dose starts at 300mSv and falls to 100mSv over a period of 100 years (Fig 13.62). Using the same ICRP model approach, the dose from Chernobyl to the population of Belarus was stated to be 2mSv and in the 30km zone was stated to be 10mSv. Of course, these doses are only an indication of the fact that there would be enormous contamination of the local area, and as I have stated above, would have to be properly reassessed using the ECRR2010 model to provide a true picture of the human health consequences.

There are then the misleading arguments about the fate of the material in the repository. It is argued that at the end of a million years, the content will revert to "natural Uranium". That may be so, but what is not stated is that this would be an enormous amount of concentrated uranium which has been trucked and shipped to this point at Forsmark and which would not otherwise be there.

5.4 A specious argument: natural Uranium

The specious argument employed by the SKB reports misses the real point which is that the overall process takes Uranium from all over the world, extracts it from rocks where its concentration is very low and its weak gamma radioactivity shielded from the public, refines and concentrates it, ships it to Sweden, turns it into nuclear fuel which then becomes contaminated with fission and activation products, and buries it in the ground at one position underneath the Baltic sea. Even if it does return to U-238 and Lead after millions of years, this U-238 and lead is highly concentrated and would not be located at Forsmark or dissolved in the Baltic sea under natural conditions. The point is also made by SKB that being near the Baltic is an advantage: that dilution will reduce the doses to the public. This is also an incorrect and specious argument. The fact is that dilution merely contaminates more people with smaller amounts of radioactivity, and if the ICRP assumption of linear no threshold effects of exposure is correct then there will be no different between contaminating ten people with 100 units and contamination 100 people with 10 units. The cancer yield is the same.

5.5 Best Available Technique

The Best Available technique arguments in the documentation are not extended to the alternative method of Recoverable Dry Storage.

6. Conclusions

I conclude that the documentation provided shows that the safety case is not made. The Radiation Risk model of the ICRP which is employed by SKB is unsafe for exposures to internal radionuclides. The model of the ECRR should be employed. The documentation is biased and spun in favour of the project and omits a significant number of calculations and information necessary to properly evaluate the whole process. In particular, the activities between the removal of the fuel from the reactors and the placement of the sealed canisters in the repository are not properly discussed or evaluated. Finally I have shown that the project must fail because the alpha particle derived Helium gas produced in the reference scenario will cause the sealed canisters to explode in the 1000 year time frame. This will effectively destroy all life in the Baltic Sea and make the coasts uninhabitable.
I list a series of questions and ask for missing analyses and data.

7. Recommendations

In view of the closeness of the SSM to the ICRP and the apparent lack of any truly independent scientific expert evaluation of the Forsmark process, together with the refusal of SSM to allow such expertise e.g the unsafe methodology presented by SSM for the selection of experts for reviewing the documentation, I would suggest that one or more representatives proposed by the ECRR be appointed for two years as an external reviewer of the process. This would be most important for those likely to suffer the immediate effects of the operation, namely the population of Oskarhamn where the CLAB and CLING facilities will be located.

8. Missing Analyses and Questions

The following missing analyses and questions follow from the considerations in my report:

1. Data and a full analysis of the both potential and design releases and consequent risks associated with the processes up to and including the encapsulation of the canisters.
2. The full analysis of Helium gas evolution in the canisters with time and the resistance of the canisters to internal pressure with a further analysis of outcome of the canister gas explosions for radioactivity release to the environment.
3. Scoping calculations using the risk model of the ECRR. Details of the extent to which the SKB examined the accuracy and safety of the ICRP risk model by literature searches of available radiation risk research documentation relating to internal exposure situations that might be relevant e.g Chernobyl, nuclear site child leukemias.
4. Details of criticality calculations for various missing FEPs including MOX spent fuel, the meltdown of the spent fuel due to mechanical failure, collapse of the supports and juxtapositioning of fuel element rods.
5. Details of all inputs and codes for all the calculations made using the Pandora and ERICA models.
6. A simple list of all inputs and the uncertainties in each input to the codes.
7. Calculation of the temperature time diagram for the spent fuel elements in the intact sealed canisters.
8. Calculation of the gas temperature with time and the canister surface temperature with time.
9. Tables of solubilities of all modeled radionuclides in the form they are in aqueous media at the expected pH and ionic strength at the range of temperatures expected near the surface of the canister.
10. Adsorption isotherms for all relevant radionuclide species on the Bentonite suspensions.
11. Discussion of the effect of high radiation fields on
(a) the metallic integrity of the mechanical support systems and the canister over 100,000 years
(b) the radiolysis of water at the surface of the canister and the production of peroxides and other oxidizing species that would attack copper
(c) the solubility of copper which is highly charged due to photoelectron induction by gamma radiation in aqueous media
(d) the effect of the electrochemical couple Fe/Cu on the integrity of a canister which has been damaged and has allowed moderate ionic strength electrolyte access to the Fe/Cu interface.

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11. Appendix A

Ionising radiation and health

A.1 Early history.

I will condense much of the historical evidence from my book *Wings of Death* 1995. 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. It soon emerged that the invisible rays were harmful. 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 were to hear a presentation describing more than 50 cases of `radiation poisoning'. From the beginning attempts were made to minimize or dismiss risks. For example, 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 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 radioactive elements from which these radiations were originating were separated, identified and researched in the following twenty years. Researchers who worked with these substances were to pay the price: the most famous of these, Marie Curie, died of leukaemia in 1934 with both hands destroyed. But by 1920 deaths from cancers and leukemias amongst the radiation researchers made protection guidance necessary and 1927 the International Congress of Radiology, a consortium of national groups adopted some guidelines at a meeting in Stockholm. These were, however, relatively arbitrary, and did not relate to the most important question, both then and now: how much radiation is dangerous?

A.2 The development of dose limits.

The earliest methods of measuring biological effects were extreme: one indication was hair falling out as an indication of excessive dose. A more usual objective indicator was the Erythemal (or skin burn) Dose (ED), the amount of radiation which caused reddening of the skin. This was a very crude measure and the amount of radiation needed to have this effect varied over a range of 1000 for different individuals and different dose regimes: these were primitive concepts of dose (Eisenbud and Gesell 1997). Although this system of measurement remains in the present assessment of skin cancer risk following exposure to ultraviolet radiation, ionising radiation is vastly more energetic and penetrating and causes effects deep within tissues.

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 Rolf Sievert of Sweden, made the fundamental move that has influenced the perception of radiation hazard ever since when he suggested tying the safe dose to Natural Background Radiation (NBR). 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 ionizing radiation. He decided arbitrarily that humans could tolerate 1/10th of this erythemal 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 some spurious scientific validity to the choice of the first radiation protection standard; yet at least these choices were based upon comparison of gross illness in humans with prior radiation exposure. At this time, the later concept of Absorbed Dose had not been developed; health risks were described in terms of exposures measured in terms of ionization of air. And what they did not anticipate, and could not consider, was the very long development period for the cancers which later became associated with radiation exposures. The only logical underpinning of the first dose limit was Sievert's idea to tie exposure to natural radiation. This use of 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.

During the first twenty years of the radiation age physical science developed many methods for measuring radiation quantity. Until the 1920s radiation was measured by measuring its ionisation, using an electroscope. It was only in the 1930s when this crude method was refined by the development of the early Geiger counter, a device which also measures ionisation but is more sensitive than the electroscope. All of these devices 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 hypothetical water-throw unit could encompass either a cupful of boiling water in the face or a bath of water at room temperature: more information is needed before the health consequence can be assessed. Another comparison which I often employ is that of a person warming themselves by a fire, and then reaching into the fire and swallowing a red hot coal: the same amount of energy is transferred. As I will show, this issue is fundamental to the arguments about risk.

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 (Failla 1932). 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. One milliGray (mGy) is one thousandth of a Gray. One Gray replaces the old Rad (Radiation Absorbed Dose). Rads, which were the units employed at the time of the tests were taken to be approximately equal to 1 Roentgen although strictly, a Roentgen is an 'exposure' and not a 'dose', and the conversion of Roentgen to Rad depends upon the energy of the ionising radiation (which can vary by a large amount). One Gray is 100 rads. It is the energy of 1 Joule absorbed by 1kilogram of tissue.

The 1934 decision of a limit of 0.1R per day is equivalent to an annual dose of 365mGy. These units have confused many who try to understand these issues, and I briefly explain them and relate them to one another in Table A.1. It should be noted that 365mGy is approximately 180 times the annual natural background dose (about 2mSv, if we include radon) and so the idea that the limits were somehow tied to the natural background is already questionable.

Table A.1 The main radiation units explained and compared

Unit	Written	Definition and usage
Roentgen	R	Exposure: The quantity of radiation which causes a defined number of ions in dry air
Rep	R	Radiation equivalent physical (93ergs/g or 0.0093J/kg) before and almost equal to the rad below, no longer used but sometimes encountered in early reports.
Rad	R	Absorbed dose (0.01J/kg). 1/100th Joule per kilogram
Rem	R	Absorbed Dose Equivalent. Developed to recognise the greater biological effect of alpha particles and neutrons (for alpha absorption e.g. radon gas, 1 rad = 20rem)
Gray	Gy	Absorbed Dose; Modern (Systeme Internationale SI) unit. 1 Joule per kilogram = 100rad; natural background gamma annual doses in UK is about 0.8mGy per year.
Sievert	Sv	Absorbed Dose Equivalent; Modern (SI) unit. 1 Sv = 100rem; 1 mSv = 100mrem or 0.1rem. Natural background in UK is about 2mSv per year (200mrem) half of which is from radon gas exposure for which the alpha multiplier of 20 is used.
Curie	Ci	Quantity of radioactive material in terms of radium. 1 Ci is a very large amount of radioactivity. Although it is a mass, a physical amount, radioactivity is described in terms of its activity, not its weight, since you can have a large weight of low activity (e.g. 350 tons of depleted uranium in Iraq) or a

		small mass of higher activity (e.g. 1.5kg of plutonium near Sellafield) which have the same radiation i.e. the same number of decays or ability to cause damage.
Becquerel	Bq	Modern unit for quantity of radioactive material; in terms of its activity 1 Bq is the amount of material giving 1decay per second, a very small amount of radioactive material
Milli	m	$1/1000^{th}$. 1 mSv is $1/1000^{th}$ or 0.001Sievert.
Micro	μ	1/millionth or 1 x 10^{-6} times the unit quantity

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. Even at the time, the genetic effects of radiation had been reported in the scientific literature by many researchers (e.g. Muller, 1929, 1930, Paterson 1932, Hanson 1928, see also Lea 1946 for further references).

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)

With the discovery of the neutron and its ability to penetrate the nucleus and bring about nuclear transformations and new radioactive substances, new sources of radiation were slowly appearing. By the late 1930s, with the discoveries by Fermi of the nuclear transformations and then by Hahn and Meitner that Uranium could be split, research had begun in earnest on atomic physics and the various transmutations that would lead to runaway fission. World War 2 was midwife to this principle of nuclear fission: completely novel substances appeared on earth for the first time in evolution. These included strontium-90, caesium-137, iodine-131, plutonium-239 all radioactive substances with chemical affinity for various living organelles.

At this time, the benchmarks for exposures were still 0.1R (1mGy) per day from whole body external radiation and 0.1μCi (3.7kBq) as the maximum body burden for Radium-226. This latter concept, MPBB had arisen out of the discovery made in the late 1920s and forced by media attention and public alarm on the scientific community, of the extreme dangers of exposures to the naturally occurring radioactive material (NORM) internal emitter Radium-226, used to produce luminous dials. This story is instructive of the ways in which science is forced by the media and the public to alter its position.

Following the fissioning of uranium in an atomic pile by Fermi in Chicago, it became clear that an atomic bomb could be made. Factories were enlarged to separate U-235, the fissile isotope of natural uranium and the Manhattan Project was set up to use this U-235 and make Plutonium for the bomb. This happened in secret and in near total ignorance of the effect of plutonium and the other fission products on health. Plutonium was known to be an alpha emitter so, for expediency, the standards for Radium were extended to Plutonium, modified by animal experiments comparing the effects of the two

substances. These safety standards were unlikely to reflect the long-term effects since the animals studied were of shorter lifespan than humans and in addition had different metabolic systems but they did have the huge philosophical advantage of being rooted in reality; the men and women who drove the inquiry into Radium's effects followed the essentially scientific principle of looking for a relationship between cause and effect.

By 1944 everything had changed. Plutonium was being produced in significant amounts and any potential it might have to kill its own workforce now affected a top-level policy funded by a bottomless budget with the imperative of building the bomb before Stalin (or Hitler) could. This was wartime: the aim of making a bomb took precedence over health and set the stage for the same approach and the same paramountcy of successful bomb development over health which was to occur in the 1050s Cold War bomb tests. More crucially for the scientific principles of radiological safety, physicians were no longer in charge, but physicists, a change which continued also into the Cold War period. Indeed, in 1959, when evidence began to emerge of the effects of atmospheric dispersion of fission products in infant mortality and leukaemia rates, this change was crystallized in the 1959 agreement between the World Health Organization (WHO) and the International Atomic Energy Agency (IAEA) in which the former UN agency (doctors and medics) is forced to leave radiation and health investigations to the latter (physicists), whose remit is the development of nuclear energy. This conflict of interest agreement is still in force although calls for its review have been made by the European Parliament following the extreme lack of research and falsification of data carried out after Chernobyl.

A main agent of change was a British physicist, Herbert Parker, head of radiation protection at the Manhattan Project. His earlier career had made him familiar with X-rays and a kind of therapy that used Radium as an external source, confining it in tubes and placing it carefully to irradiate cancerous tissues, a medical application which, for once in those days, did not involve Radium becoming intimately mingled with the patient's bones. Parker had a physics-based view; radiation was a single phenomenon, whether it came from an X-ray machine or a speck of Plutonium. As with light, where the physicist isn't too interested in whether the source is a candle or a light bulb or the sun, Parker was concerned with how much energy the radiation delivered to the tissue of interest. The language here was of *ergs*, from the Greek for *work*. It is defined in *dynes*, the Greek for *force*; the units are physical, movement, velocity, grammes of mass, centimetres of length, seconds of time. In this world there's no call for a doctorly bedside manner; Parker was one of the first to call himself a *Health Physicist*.

Using his physicist's approach, Parker shifted the focus from investigating the effects of specific substances onto a new concept, *absorbed dose,* which would apply to radiation from any source and all sources, providing a way to assess workers' total exposure to all the novel nuclides they were now being generated in the Manhattan Project. He defined a unit of dose in ergs per gramme of tissue and called it the *Roentgen Equivalent Physical*, or *rep*. Its very name reveals the thinking; Roentgen was the discoverer of X-rays (for a long time they were called *Roentgen rays*). The source of X-rays is always outside the body, so we can see the understanding of dose, and hence risk, was now to be based on an external paradigm (Cantrill and Parker 1945).

The first limit for Plutonium in the body based on Parker's dose model was set at 0.01 reps per day, making the rep the equivalent of the Roentgen. Now, instead of the

empirical scientific inquiry based on actual tissue damage and instead of the tentative subjectivity of the 1941 Standards Bureau Committee's decision on a Radium level, the new model gave an impression of mathematical precision, certainty and universal applicability.

Any risk model needs two types of data, for exposure and for effect. Unfortunately, there were no reliable data even for X-rays despite 50 years' experience. There was too much variability in the machines and the conditions in which they were used, doses were largely unknowable, and many of the long-term effects had yet to emerge. But after 1945 the people of Hiroshima and Nagasaki (those who hadn't been vaporized by the Atom bombs that fell on them on 6th and 9th August) provided the authorities with a fresh opportunity. Funded and controlled by the USA, data on the survivors' health was gathered (as it still is) in what have become known as the Life Span Studies or *LSS*.

There have been many criticisms of the LSS as a method of assessing harm even from external radiation (ECRR2003, IRSN 2005, ECRR2010, Sawada 2009) and I will return to this topic. As far as studying internal radioactivity is concerned the flaw is fatal; the control population providing the base-line of expected rates of disease, to be compared with disease in the exposed population, was recruited from the bombed cities themselves. They had either been outside the city when the bomb fell, or in some other way were shielded from the flash of the explosion. The exposed population consisted of people who had been in the open and so received a large dose of external gamma rays. Both groups ingested and inhaled just as much fallout, uranium, plutonium or residual radioactive material as each other, so the LSS are totally silent on internal radiation. The only difference was in the external irradiation. LSS nevertheless is the basis of radiation protection standards all over the world to this day for both external and internal. The LSS were not begun until 1950. This was another flaw, since five years of epidemiological data would be missing from the study and in addition, those selected into the study would have been healthy survivors: many of the victims of radiation would have died in the five years before the study began (Stewart and Kneale, 2000)

Long before then America's Atomic Energy Commission (AEC) urgently needed to regulate the growing nuclear industry. The AEC pressed the National Council for Radiation Protection (NCRP) to develop safety standards. An especial concern was the quantity of novel elements which, being alpha emitters, would present internal radiation hazards. Separate sub-committees addressed internal and external radiation. The external sub-committee completed its work quite quickly but the other was slowed down by the many complexities of internal contamination. The problem is that while physicists can tell you the ergs from any radioactive decay, they don't have much clue about where internal radioactivity goes inside the body, how long it stays there or what biological damage it's doing. Impatient with the delays, NCRP's Executive closed down the internal committee in 1951, and stretched the report of the external committee to cover internal radiation. This was a key mistake; it had no scientific basis, but it was the cornerstone for what has happened since, and the origin of an enormous public health scandal which continues to this day. This is the systematic poisoning of life on earth by novel radioactive isotopes generated by nuclear fission of uranium.

After the war, American influence revived the international radiation protection community from its dormancy to be reborn as the International Commission on

Radiological Protection. ICRP's first act was to adopt the NCRP report. The first formal recommendations in 1951 were for maximum permissible doses from X-rays and gamma rays of 0.5 R at the surface of the body in any one week. This represents a dose of 260mSv a year, a reduction on the 1934 limits. The ICRP took a critical step for science: it adopted the Maximum Permissible Body Burden (MPBB), defined now as the quantity of radionuclide in the body which would deliver a radiation absorbed dose equivalent at the radiation limit defined for external radiation.

The die was cast: this is the source of the error which has been promulgated to this day, the source of all the discrepancies between predictions of the model and the many examples of cancer and leukaemia in those exposed to internal radiation, including the A-Bomb Test veterans. It is here at this point in time that the error which flowed from Parker's physically defined rep was fixed for all time into the risk model.

In 1953, the ICRP met in Copenhagen and agreed recommendations which were published in December 1954. The committee agreed *no radiation level higher than the natural background can be regarded as absolutely safe* and that the problem was therefore *to choose a practical level that, in the light of present knowledge, involves negligible risk.* For internal radiation, the concept of the critical organ was introduced: this was a development that conceded that different internal radionuclides might concentrate in different organs, and so absorbed doses must be calculated on the organ mass, rather than the whole body mass. This concession shows that the problem of anisotropy of dose from internal radionuclides (which I will discuss below) had been conceded. However the ICRP stopped at the organ level: the idea that such local high dose effects might occur at a more microscopic level, at the cellular DNA, was not accommodated, and is still not accommodated.

But we should recall that this was perhaps forgivable: 1953 was the year when the DNA structure was first described by Watson and Crick. The location of the radiation effects in the cell nucleus, the critical involvement of the DNA as target for radiation induced effects would have to wait for twenty years or more, until the 1980s. Even so, no one made the obvious connection: the point that if ionisation at the DNA was the critical target, external exposure and internal exposure could not be described in the same way with the averaging tools of absorbed dose. It waited until 2003 when the European Committee on Radiation Risk (see below) published its new risk model for these effects to be considered.

The 1954 report reduced the dose limits to 300mrem (3mSv) per week, or 156mSv per year). In this report, the roentgen equivalent man or rem was introduced: radiation from external and internal radiation could be summed as if it were the same exposure. Although seemingly a rational development, as I have made clear, this decision was to become the basis of the most serious mistake ever made in the area of radiation risk. Although the report noted: *much uncertainty still remains regarding the behaviour of radioactive materials inside the body* it nevertheless went on to apply the same 300mrem average dose at the organ level when calculating maximum permissible body burdens of radioisotopes. The Chairman of Committee 2 of the ICRP, dealing with internal exposure was Karl Z Morgan, who was later was to become a massive critic of the ICRP and the nuclear industry. He was very concerned about the lack of knowledge of internal isotopes and their concentration in tissues. The Ra-226 MPBB at the time was 0.1microCurie (3.7kBq). This was reduced by Morgan's Committee a factor of 5 to allow

for possible non-uniformity of deposition. For other radionuclides, the dose limit was set on the basis of the external limit as applied to the organ where the isotope was likely to be concentrated.

But by 1956, concerns began to be raised in the media about genetic effects. Muller had written an influential paper on the effects of radiation on Drosophila, the fruit fly (Muller 1950); other scientists (Ralph Lapp, Linus Pauling) were arguing from first principles that incorporated radionuclides were going to cause genetic damage. Pauling, a double Nobel Prize winner (and later the Russian Sakharov) drew attention to the harmful effects of Carbon-14, produced in abundance in the tests, and Strontium-90, a long lived (228 year half life) bone seeking isotope from the Calcium Group 2 of the Periodic Table (Busby 1995). Nevertheless, the requirements of military research for bombs caused pressure on the regulators. Limits were slightly relaxed, allowing the period of averaging of dose to be extended to 13 weeks, so long as *the total dose to any organ accumulated during a period of 13 consecutive weeks does not exceed ten times the basic permissible dose.* This introduced the concept of the integrated dose: but note that this new dose limit permitted an annual dose of up to an enormous 1560mSv. Pressure built up: research results leaked out. Fallout Strontium began to show up in childrens' milk. The doses were again revised in 1958 when ICRP considered the exposure of individuals in a number of categories. For the highest risk category, ICRP recommended a new weekly limit of 0.1rem (1mSv) or 52mSv in a year with a proviso that not more than 3 rems (30mSv) were delivered in 13 weeks.

By 1958, books were appearing that argued that radiation was a much more serious hazard than had been believed: that the health effects were essentially genetic mutation driven (e.g. Pauling 1958, Alexander 1957, Wallace and Dobzhansky 1959). The British Medical Research Council were cautiously concerned (MRC 1956). In 1957, in Oxford, Alice Stewart looked for the cause in the sudden increase in a new childhood disease, leukaemia and found that a significant cause of the increased levels was obstetric X-raying. She had identified the sensitivity of the foetus to radiation, finding that a foetal dose of as little as 10mSv caused a 40% increase in childhood cancer 0-14. Her findings were attacked by those who had contributed to the MRC reports which had concluded that the fallout at the level it was at the time could not be a cause of concern (e.g. Richard Doll) and her career was affected. But she was later shown to have been correct (Wakeford and Little 2003). Her conclusions meant that the levels of Strontium fallout in milk would have significant effects on childhood cancer and this issue ultimately resulted in the Kennedy /Kruschev Test Ban of 1963. Therefore by 1964, despite the continued use of such high dose limits, there began to be serious concerns, particularly about internal irradiation. The British physicist W. Mayneord (an ex- member of the ICRP) was to write:

my worry about the numerical values of ICRP is the weakness of the biological and medical foundations coupled with a most impressive numerical façade. . . we give a false impression of certainty; comforting to administrators but not so comforting to live with as scientists. (Radiation and Health, Nuffield Hospital Trust 1964).

Other members (e.g. Ed Radford, Carl Z Morgan, John Gofman) were to resign or be sacked and were to attack the ICRP and its dose limits for the rest of their lives.

By 1977 more evidence was coming in from the Japanese A-Bomb Lifespan Studies (LSS) that the long term effects of external irradiation were significantly greater

than had been believed and so ICRP decided that it had to reduce the integrated annual doses to members of the public to 5mSv. By 1985, after the discovery of the Sellafield child leukemia cluster, this was modified to 1mSv. In 1990, more evidence had appeared that radiation was much more dangerous than had been thought: evidence was appearing from radiation biology, from epidemiology, from animal studies. The effects were seen to be consequences of genetic damage and it was decided that there could be no threshold for such effects. The 1990s saw more and more evidence of the subtle effects of low doses of radiation. However, the 1mSv level could not be reduced since by then too many industries or other radiation related operations depended upon this limit. So the limit was held at 1mSv, although the British NRPB made a limit from a single source of 0.3mSv in a year, and EURATOM reduced this single source limit to 0.15mSv in 1996/29 Directive, which became EU law in 2001. The principle of ALARA, *as low as reasonably achievable* for exposures was introduced. Even this was tempered in practice by *social and economic considerations*. So this is the position that is presently embedded in legislation. All the major risk agencies now concede that there is no safe dose of radiation, and that genetic or genomic effects can occur at the lowest possible dose.

It is instructive to see the dose limits plotted over the period of the last century. It is clear from Table A.2 and the plots (Figs A.1 and A.2) that the exponential reduction in the perception of hazard shown by the plot has bottomed out only for the reason that the nuclear and other industries, and the military, cannot operate with radiation discharges at the present levels if the true hazard from exposure were reflected in legal constraints on exposures.

But these dose limit reductions by 2007 still did nothing to address the real problem with radiation risk, that of internal chronic exposure. The increasing quantities of novel radionuclides and technologically enhanced natural substances like Radium and Uranium in the environment has resulted in everyone one earth being exposed though inhalation and ingestion of contaminated material from an increasingly contaminated environment. 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. Internal isotope exposure is the overlooked hazard of the nuclear age; it is necessary here to back-track and return to the discovery and parallel development during the infant X-ray age of the phenomenon of radioactivity.

Table A.2 Statutory annual radiation dose limits to members of the public over the radiation age 1920-present (mSv)

Year	Statutory Annual Dose Limit mSv (public)	Note
1927	1000	Based on erythemal (skin reddening) X-ray dose
1934	365	Following Radium dial painters incident
1951	260	A-Bomb development. Japan Lifespan Study begins
1954	156	Weapons fallout period begins. DNA structure found
1958	52	Weapons fallout peaks 1959-1964. Muller
1966	5	Sr-90 in milk, in bone. Kennedy test ban 1963
1977	5	
1985	1	Nuclear site child leukemias; Chernobyl in 1986
1991	1	The 1990s saw discovery of genomic instability following single alpha tracks in cells
2003	1	ECRR introduces 0.5mSv limit; adjusts internal doses
2007	1	ICRP holds its 1985 1mSv limit despite huge evidence of harm from internal exposures at lower doses

Fig A.1 Statutory (ICRP and predecessors) annual radiation dose limits to members of the public over the radiation age 1920-present (mSv) (exponential trend fitted to data points)

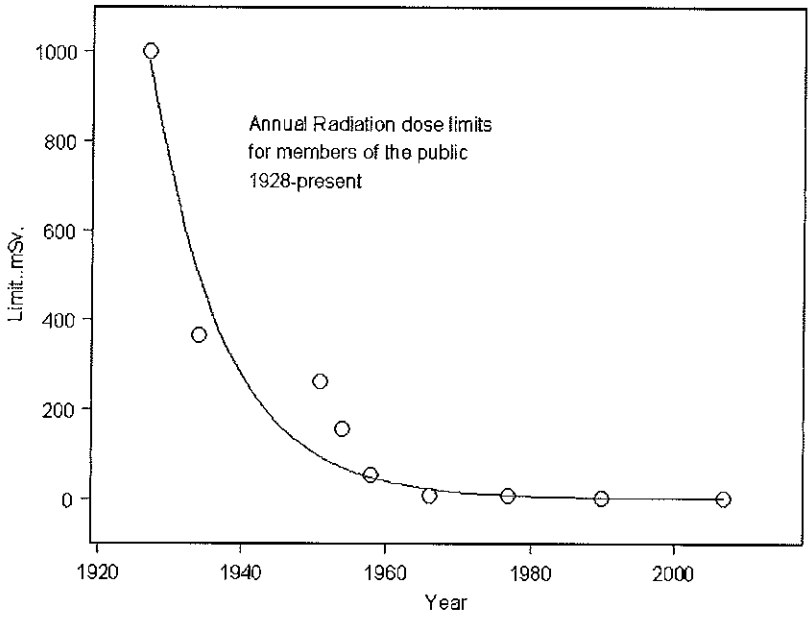
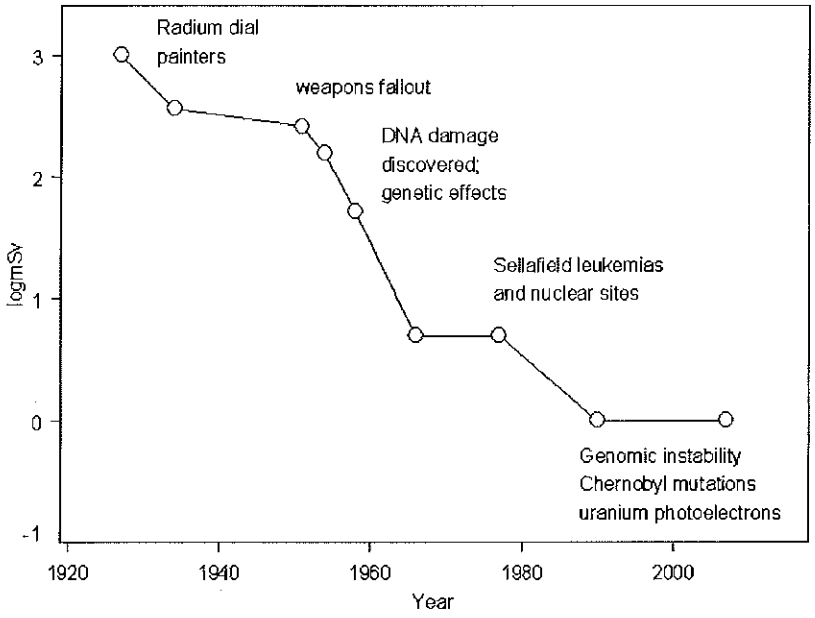


Fig A.2. Log plot of statutory annual radiation dose limits to members of the public over the radiation age 1920-present (log(mSv))with some radiation exposure events influencing reduction of dose limit. Note that new discoveries in radiobiology and Chernobyl effects since 1985 cannot reduce the limits further as the industry cannot take this.



A.3 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 lifetime work to chemically isolate Radium, processing tonnes of radioactive ore, resulted in the isolation of one gram. She and her husband Pierre shared the Nobel Prize but she died in 1934 of leukaemia, her hands terribly scarred from having handled the radioactive materials. Her daughter Irene who worked with radiation was also to die of leukemia. Roentgen himself died of bone cancer.

In the period following her discovery, 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 worrying. In 1901 he borrowed from the Curies a phial containing a minute quantity of a Radium salt. He carried the tube in his waistcoat pocket for six hours and noticed that he had burned his skin through several layers of clothing. The doctor that he consulted pointed out that the lesion was similar to 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.

A.4 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 kept 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, 2009, Yablokov et al 2009).

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 (Martland 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 believed that Radium poisoning was the cause. The company continued to argue that this was impossible, that the exposure was too low.

But 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, 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, 1951).

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.

I examine the later studies of the Radium workers in Section R

A.5 Development of Dose-Response Relations for internal emitters : the history

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.

I will comment in passing that the effects of radium on the dial painters were probably not all due to internal exposures from alpha particles. The external dose limits of the time (see Fig 3) believed to confer safety, were extremely high, as I have remarked. I own a prismatic hand bearing compass supplied to the British Army soldiers as standard issue in WW2. Soldiers wore this on their belt and held it to their eyes to obtain bearings. A calibrated Geiger Counter shows a gamma dose of 50μSv/h at 5cm from the small (2mm diameter piece) of Radium compound on the compass card. This would give an annual dose of 438mSv in a year. This is from a single dab of paint: the external doses the dial painters received would have been enormously greater since they would have had a whole paint pot of the stuff in front of them. And it is not hard to see why the child leukemia rate in WW2 suddenly increased with planes being shot down, radium paint everywhere and soldiers carrying such radioactive sources close to their testicles.

Although I have outlined the historical development of the overall dose limits in the previous section, I will here look more closely at the bodies assessing the risk from internal radiation. 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 Atomic Energy Commission. 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. External dose limits were set at 0.5R/week (260mSv/year). The reduction from the previous 1934 limit was partly based on the discovery that radiation caused genetic damage. Experiments with fruit flies by H Muller had 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 when they were reduced again to 0.3rem/week (156mSv/y).

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-- 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 E was transferred. The same *ad hoc*, and arbitrarily developed dose limit could then be applied.

These dose limits were translated into maximum permissible concentrations or body burdens (MPBB) 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 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

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 by the 1960s 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 BEIR V, BEIR VII).

A.6 *External and internal radiation: the science.*

In order to help follow the arguments about internal radiation and health I now return to review some basic principles and examine some of the assumptions at the base of radiation risk. The 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.*

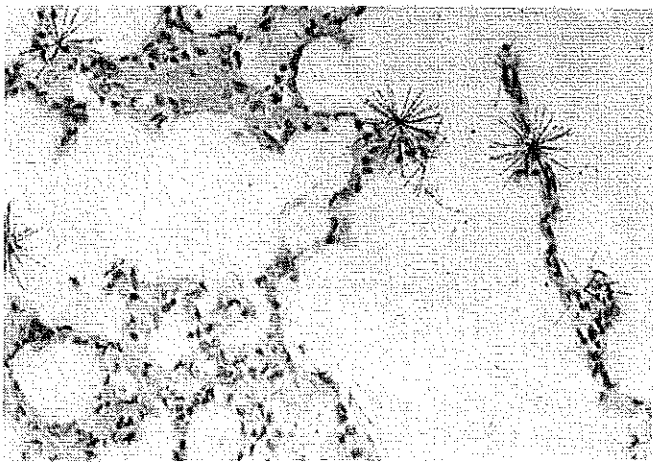
Ionising radiation acts though the damage to cellular genetic materials, the genes on the DNA, killing some cells but causing fixed genetic mutation in others, including mutations that signal to descendants a genomic instability message to increase their rate of incorporated error. These genetic and 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 or the development of a wide range of diseases. The damage to the DNA is caused either by ionisation of DNA materials themselves directly, or more usually 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 can 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. This 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. All predictions follow from this assumption, the Linear No Threshold LNT assumption.

But whatever the dose response function employed, 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 or critical parts (e.g. chromosomes, DNA) 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 modelling 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 causes 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 have shown in Fig A.3 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 ionization 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. As I have argued already, 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.*

Fig A.3 Alpha star photomicrograph showing radiation tracks emanating from hot particle in rat lung; track length has the distance of about five cells.



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 *[in the situation of] 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.* And the embarrassing paragraph above was quietly dropped from the final ICRP 2007 report (but see section 2.16).

A.7 Dose constraints and risk models after 1980

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

ICRP model and the excess leukemias was 300-fold. Note 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 (e.g. Uranium) 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 was 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 that the lymph nodes were known to be the site of leukemias in animal studies 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.

After the Sellafield discovery, childhood leukemia clusters were reported from many nuclear sites in the UK and Europe e.g. Dounreay, Aldermaston, Hinkley Point, La Hague, Kruemmel. A full discussion of the issue and how it illuminates the error in employing the external risk model is given in ECRR2003.

A.8.2 The German childhood leukemias

Most recently, in 2008, the German Childhood cancer registry (Kinderkrebsregister) published results of the largest study of childhood leukemia near nuclear power stations that has yet been carried out. By examining cases and controls by distance from all the nuclear sites in Germany between 1980 and 2005, the authors have shown that there is a statistically significant doubling of childhood leukemia risk in the age group 0-4, thus supporting the various earlier studies of childhood leukemia near nuclear sites. Scientists from the University of Mainz working for the German Childhood Cancer registry, founded in 1980, had originally investigated whether there had been similar excess risks of childhood cancer near nuclear sites by using the ecological approach employed by COMARE, that is, looking at all children within some distance of the site, in the German studies 15km. They had also, like COMARE, examined the age group 0-14, which dilutes any excess by a factor of 3 since the main age group of interest for the disease is 0-4. This may have been because, like COMARE, at that time, when the Germans were committed to nuclear power, they didn't actually want to find anything. And that is what happened: the examination of the 0-15 year group living within 15km of the sites from 1980-1995 showed no excess risk when compared with the general; national rates (RR =

modified after the discovery of the Sellafield child leukemias and the other nuclear site child leukemias. ICRP in 1985 dropped the annual dose limit 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.

As I have already pointed out, it is clear that there can be no safe dose of radiation. This has been formally conceded since the early 1990s (see e.g NRPB 1995). I repeat 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

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 even perhaps thousands 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.

A.8 The recent revolution in radiation risk perception

A.8.1 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

0.97 CI 0.87<RR<1.08). Nevertheless, examination of subsets revealed that for children living within 5km of the plant aged 0-4 there was a statistically significant 3-fold excess (RR 3.01 CI 1.25<RR<10.31). In the Kinderkrebsregister case control study published in January 2008 in the *European Journal of Cancer* (Spix et al 2008) published results from 23 years (1980-2003) of data for 6300 children. The authors reported that the best model to fit the data by distance from the nuclear plants in Germany was an inverse square root relationship and that in their model, for children aged 0-4, there was a RR of 1.61 excess risk at 5km for cancer and RR 2.19 (lower one-tailed 95% CI 1.51) for leukemia. This is further evidence of the error in employing the ICRP external radiation risk model for explaining or predicting risk from internal exposure, since these children were clearly not exposed directly to radiation from the plant, but rather inhaled or ingested radionuclides discharged from these plants. We should be clear that the doses to these children cannot explain their illnesses on the basis of the ICRP risk model by an error factor of upwards of 1000.

A.8.3 New Science

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.

A.8.4 Genomic Instability and the Bystander effect

It was discovered in the mid 1990s that a single track from an alpha particle through 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 (the clonal expansion theory). 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 potentially 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. In my opinion, this evidence came to be accepted around the end of the 1990s; that is to say, there was a revolution in the mainstream understanding of radiation risk which gathered strength from the mid 1990s and would have been largely agreed by the majority of scientists as representing a need to re-think the basic science by the year 2000.

But this discovery was followed by second very strange observation. 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 the 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. Some scientists have also argued the opposite. There is some data that suggests that low doses of radiation are protective. This process is termed 'hormesis' but it is not conceded by the official risk agencies. Risk agency models do however apply a factor to their predictions based upon a lower cancer yield for protracted doses and opposed to acute doses. In my opinion this is invalid. The application of these Dose Rate Reduction Factors to low dose radiation 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 i.e. in hormesis.

The second implication of the new scientific discoveries 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* (see ECRR2003, Burlakova 2000). 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, 2009, ECRR2010).

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.

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 Busby 1995, CERRIE 2004 and CERRIE Minority 2004)

A.8.5 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).

A.8.6 Uranium: Photoelectron amplification

I will briefly review a recent discovery which is relevant to internal radiation exposure and which is not incorporated into the current risk model. It mainly affects those who are contaminated with high atomic number elements and also subject to increased external gamma radiation. The most important elements are uranium and lead. It is an interesting and well known fact that the absorption of gamma rays of energy lower than 1000keV is proportional to the fourth power of the atomic number Z of the absorbing element. This means that high Z elements like uranium (92), gold (79) and lead (82) absorb some 100,000 times more gamma radiation than water, the main component of the body. The effective atomic number of water is 3.3 or if we take the oxygen atom as representing the highest atomic number and therefore major absorber, 8.

If the absorbing atoms or particles are bound to DNA or some critical organelle or protein, this will focus natural background gamma radiation into that tissue volume through the re-emission of the absorbed energy as photoelectrons. Thus the absorbed dose to that volume will be significantly higher than that calculated by the ICRP system. For a full discussion see Busby 2005 and Busby and Schnug, 2008. The effects will generally occur for any material with a higher atomic number than 8; indeed it was first pointed out in 1947 by Speirs that there was a 10-fold enhancement of dose to tissue near bone owing to the presence of the Calcium (Z=20) in the bone. Inhalation and concentration of uranium in the lymphatic system will increase the dose through amplification of the already enhanced background gamma radiation. Based upon these photoelectron considerations, the physical enhancement weighting factor w_j for the radiation dose coefficient for U-238 contamination has recently been agreed by the ECRR as 1000 (see below). The failure of the ICRP model to deal with the nature to the absorber is further evidence that the ICRP model is in error by a very large amount.

A.9 Chernobyl Proofs

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 was 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)*. Professor Yablokov, a member of the Russian Academy of Sciences, is also a member of the ECRR steering committee, co-editor with me of the ECRR2003 and 2010 reports and Chair of its Chernobyl sub-committee which recently published a book on the health effects of the fallout (Yablokov and Busby 2006, 2009). Yablokov also published separately together with the late Wassily Nesterenko, his son Alexey Nesterenko and the eminent US scientist Janette Sherman another book on the real health effects of the Chernobyl accident. This volume, which was published by the prestigious New York Academy of Sciences (Yablokov et al 2009) reviews the enormous body of evidence that the Chernobyl accident fallout had very serious effects on population of the contaminated territories.

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 difficult to make a connection which is unassailable in logic. In the case of the Sellafield childrens' leukemia and non-Hodgkin lymphoma (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 (Busby 2007).

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, Busby 2005, 2009). This is a simple analysis of the increase in infant leukaemia in 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 in 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.

A new epidemiological analysis of these children using the data supplied to me by the Childhood Cancer Research Group was published in 2009 together with a clear statement about the failure of the ICRP risk model (Busby 2009). Thus there is unequivocal evidence here of the failure of the ICRP model.

A.10 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).

A.11 ECRR

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. The ECRR deals with the enhancement of hazard from internal radionuclides by extending the method used by the ICRP for radiobiological effectiveness of alpha, neutron etc to situations where the chemical affinity of an internal radionuclide, or its physical decay characteristics makes it more effective at delivering ionisation to the DNA. The dose coefficients developed by the ICRP are used but with weighting multipliers w_j and w_i to represent physical and chemical enhancement mechanisms. Therefore a dose of 1mSv from an isotope that binds to DNA strongly, like Sr-90, is multiplied by a w_j of 50 and so the dose becomes 50mSv in the same way that ICRP multiply the absorbed doses from alpha emitters by 20 to obtain their equivalent dose.

A.12 ECRR in Lesvos

In May 2009 there was an International Conference of the ECRR in Lesvos, Greece, hosted by the University of the Aegean. Research papers and evidence falsifying the ICRP risk model were presented at this conference by eminent radiation scientists from Japan, Canada, UK, Germany, France, Russia, Ireland, India, Ukraine and Belarus. The proceedings are currently being prepared. However the radiation risk situation and the level of error involved was considered to be so important the conference culminated in a agreed declaration signed by all the scientists present, the Lesvos Declaration (see www.euradcom.org). This is attached with the names of the signatories. It demonstrates that a large body of eminent expert opinion exists that the ICRP model is unsafe and should no longer be used to calculate the effects of internal radiation exposure. This ICRP model is essentially the same as that of the US BEIR committee. In its stead, political and courtroom decisions should be made on the basis of the ECRR risk model. The new 2010 updated risk model was published in May 2010 and is appended as evidence in this case (ECRR2010).

A.13 IRSN

Independent support for the arguments that internal radiation effects are not properly modelled by the current ICRP risk model comes from a report commissioned by the French government and published in 2005 (IRSN 2005). A team of scientists from the official French Institute for Radiological Protection examined the 2003 report of the ECRR (above). They concluded that the criticisms made by ECRR of the current ICRP risk model were important and were valid, though the IRSN report did not agree with the way in which ECRR modified the risk model to account for the resulting errors (IRSN 2005).

A.14 Recent proofs of the failure of ICRP from Sweden and Belarus

Cancer in Northern Sweden after Chernobyl was studied by Martin Tondel who published results in 2004 (Tondel et al 2004, 2006). By comparing cancer rates before and for 10 years after the Chernobyl fallout in Sweden by Caesium-137 contamination of communities in Northern Sweden, Tondel was able to determine a risk of 11% excess

cancer incidence per 100kBq/m^2. This translates to an error factor of a minimum of 600-fold in the application of the ICRP cancer risk model to the external gamma dose associated with this contamination integrated over one year (ECRR2010). In 2004 also Okeanov reported significant increases in cancer in Belarus following the Chernobyl accident: these increases also were not predicted by the ICRP model since the mean dose to the Belarus population from Chernobyl was about 2mSv, approximately equal to the annual background radiation dose, yet the cancer incidence rates had increased by about 40% (ECRR2010).

These pieces of evidence support the assertion that the ICRP model is in error by two or three orders of magnitude when applied to internal exposures.

The ECRR2003 model accurately predicted the findings of Tondel and Okeanov and the matter is discussed in ECRR2010.

A.15 Admission of the error following the resignation of the ICRP secretary

In April 2010 I personally carried out a public interview with the ex-Scientific Secretary of the ICRP Dr Jack Valentin in Stockholm, Sweden. The interview was recorded on video and is available to the court. It is available on the internet and a transcript is available of the critical part on the website of the Low level Radiation Campaign www.llrc.org. It should be noted that Valentin was the editor and author of both of the 1990 and 2007 ICRP risk model reports (ICRP2007). In the interview, Valentin made two statements which are relevant to the present case. First, he said that since he was no longer Scientific Secretary, having resigned a few weeks earlier, that ICRP and UNSCEAR (the United Nations Scientific Committee on the Effects of Atomic Radiation), he could say that both organizations were wrong in not addressing the evidence that falsified their model evidence form Busby, from Chernobyl and from the nuclear site child leukemia clusters. Second he said that the ICRP risk model could not be employed to asses the risk of radiation in human populations because the uncertainties in internal radiation exposure modeling could be two orders of magnitude..

A.16 Non cancer effects of radiation exposure

The consequences for radiation protection of the discoveries in the area of genomic instability and bystander effects, now termed non-targeted effects and described in A.8.4, are profound. First, they show that the theoretical basis of the current risk model is unsound. But in addition, they show that since all cells are affected, all diseases are caused by or contributed to by radiation exposure. Therefore the concentration by the ICRP on cancer as an end point is wrong. The genomic instability work indicated clearly that radiation exposure will cause a kind of non specific ageing. It has been observed for many years that this is so, but because no mechanism was forthcoming it has been routinely denied (see Busby 1995, Bertell (1977) X-ray exposure and premature ageing. *J Surg Oncol.* 9 379-91). This has profound implications for radiation epidemiology, since cancer is a disease which increases in rate exponentially with age and therefore competing causes of death will affect the result of any cancer only study of radiation. You cannot die of cancer if you have died already of a heart attack or kidney failure. But it also means that a whole range of diseases and conditions are likely to be associated with prior exposure. And this is what is seen.

Because some of these conditions developed in the pipe workers I will devote

some space and to the problem. For those who develop non cancer illnesses (e.g. heart disease) the assessment of contributory causation should take into consideration the background rate of the conditions being considered but should not rule out contributory causation since rates of ageing and onset of disease processes are genetically determined: some people age at different rates from others in the absence of any stress, and indeed there may be lifestyle stresses to include in any assessment. One example is atherosclerosis and ischaemic heart disease. Atherosclerosis rates were found to be significantly higher in a number of studies of those exposed to radiation, and heart diseased is now recognised to be associated with prior radiation exposures. Here is evidence of increased rate of heart attacks in women given radiotherapy for breast cancer. I considered this matter in my 1995 book *Wings of Death* where I pointed out that the atherosclerotic plaques were monoclonal mosaics and thus benign tumours, that they were produced in animals by exposure to carcinogens; I also analysed in that book the cohort effects of the increases in heart disease in England following the weapons test exposures and concluded that the epidemic of heart disease was due to the fallout, principally Sr-90. Heart disease was found to be raised in medical radiologists. Since then results from Chernobyl and Hiroshima survivors have shown strong evidence that ischaemic heart disease and atherosclerosis are associated with exposure to radiation. Examples are to be found in ECRR2010 and in collections of reviews of Chernobyl effects (Burlakova, 1996, Busby and Yablokov 2006, 2009, Yablokov et al 2009. Since the Burlakova book is difficult to obtain I reproduce in Table 2.3 some of the data from Table 3 of Oradovskaya (Burlakova 1996) which compares certain conditions in four areas of difference contamination level in the Chernobyl affected territories.

Table A.3 Frequency of diseases in the Bryansk regions differently affected by radiation contamination from the Chernbobyl accident. (Oradovskaya I.V, Institute of Immunology, Russian Federation, in Burlakova EB 1996)

Disease	**Novozybkov N=3892**	**Vyshkov N= 1074**	**Russia control**	**Ukraine**
Mean Cs-137 Ci/sq.km	18	30	<1	<1
Ischaemic heart disease	44.7	38.3	24.8	30.7
Atherosclerosis without IHD	113.6	58.1	16.5	19.5
Arthritis, spondylitis	104.4	65.5	44.1	39.7
Chronic pharyngitis, nasopharyngitis, laryngitis	18.7	7.5	3.4	3.1
Chronic bronchitis	24.3	30.3	12.8	11.5
Chronic pyelonephritis	18.6	13.9	4.7	6.9

The same paper compares various conditions between the two contaminated regions of the Bryansk oblast. The prevalence of various clinical symptoms of immune system deficiency are compared and shown to correlate with the radioactive contamination.

Other evidence of the increased prevalence of non cancer illnesses and conditions in those exposed to radiation is presented in ECRR 2010, particularly for both Chernobyl and Hiroshima victims. These are condensed into Table A.4 and A.5. Reports of non cancer effects related to radiation and radionuclide exposure after Chernobyl are to be

found in Yablokov et al 2009. There are many examples but relevant to the current case in the report of a 3-fold increase in Type II diabetes in Belarus liquidators and evacuees (Yablokov et al 2009 p 79) and a significant excess risk of heart disease in male and female liquidators (Yablokov et al 2009, p 62) relative to the national population.

Table A.4 Non cancer illnesses per 100,000 adults of three contaminated and 5 control regions of the Brest region in Belarus in 1990 (from Malko 1990 cited in ECRR2003, 2010)

Diseases	**3 contaminated districts**	**5 control districts**	**p-value**
Altogether	62,023	48,479	<.001
Circulatory system, hypertension, IHD	12060	9300	<.001
Osteomuscular, osteoarthritis	5399	4191	<.001
Urogenital, nephritis, nephroses, kidney infections	3415	1995	<.001
Endocrine, metabolism, immune system	2340	1506	<.001

Table A.5 Comparison of morbidity rates (%) in Japanese A-Bomb survivors and the general Japanese population (Morbidity rates for 1232 victims of the A Bombing examined at the Hannan Chuo hospital, Osaka, between 1985 and 1990; Furitsu 1994 in ECRR2003, 2010)

Condition	**A-Bomb sample**	**All Japan**
Ischaemic Heart Disease	9	2
Anemia, leukopenia	12	1
Dental disease	10	<1
Diabetes	7	3
Nephritis, urethral infection	5	1
Cholethiasis, pancreatitis	4	1
Bronchitis, pneumonia	5	0.8
Lumbago	29	8

It should be clear from these tables that exposure to radiation, particularly internal exposures to fission products and uranium causes a wide range of health problems most easily seen as premature ageing. Indeed, in extreme cases like the contaminated areas of Belarus, children are found to have conditions (arrythmias, gastric conditions) which are clearly seen on biopsy to be due to cellular ageing effects (Bandashevsky references in ECRR2003,2010).
The errors introduced into radiation epidemiology from this source is seen clearly in the many studies of internal Radium and Thorotrast exposures which I look at in the next section.

Radium and Thorotrast studies

R1 Re-examining the data

The increasing pressure brought to bear on the ICRP risk model focuses intensely on the arguments about internal and external radiation exposure rehearsed in the previous section. The ICRP and the nuclear risk agencies have to concede much of the science, but fall back on the epidemiology. The problem is, very little human epidemiology has been done on internal radionuclide exposures. There are, however, two sets of studies which are said to broadly support the arguments that the current risk model is broadly correct. These are the studies of individuals medically treated with Radium and Thorotrast. The studies originally were carried out because of doubt over the use of the external based risk model to deal with internal radionuclide exposures at a time when internal exposures from alpha emitters like plutonium were increasing in proportion to the development of the A-Bombs and H-Bombs. All of these studies were of roughly the same type. A group of individuals was formalised and then records were traced, or the individuals themselves were traced to see what the number of cancers were. The end point was always cancer, since the project was to see if the ICRP cancer risk model was accurate for these internal exposures. The medical and other (e.g. laboratory) exposures to Radium had been largely before 1960; radium dial painters I have mentioned, and there were many of these who had survived from the period when they were employed. In addition there were individuals who had been exposed to Ra-224 as a treatment for various illnesses. There had been a fashion to treat syphilis, hypertension, gout, infectious polyarthritis, "muscular rheumatism", anemias, epilepsy and multiple sclerosis (Loutit 1970, Malignancy from Radium. *Brit.J.Cancer* 24(2) 17-207). Then there were many individuals who had been injected with the substance Thorotrast, an X-ray contrast medium based on the nuclide Th-232, the daughter of which is Ra-228. So these are all internal radium exposures. What was reported was that the cancer yields, mainly of liver cancer, bone cancer and leukaemia could be roughly related to the exposures and that the yield was not too far away from the yield predicted by the ICRP external type of risk model, i.e. the A-Bomb survivors. These studies are the last remaining defence that the current risk agencies can mobilise. For this reason, these studies are currently being examined by the ECRR and some of the findings of these ECRR investigations are of interest here. There are a number of fatal problems with all the radium studies:

- The study groups were assembled long after the exposures and so not all those who had been exposed were in the study group: only the survivors. Many were dead. This biased the samples
- A number of published studies give sufficient data to show that there was a high rate of death in the early period before the groups were assembled
- The doses were not isotropic; for Thorotrast, the material was stored in depots in parts of the body where cells were quite resistant to radiation

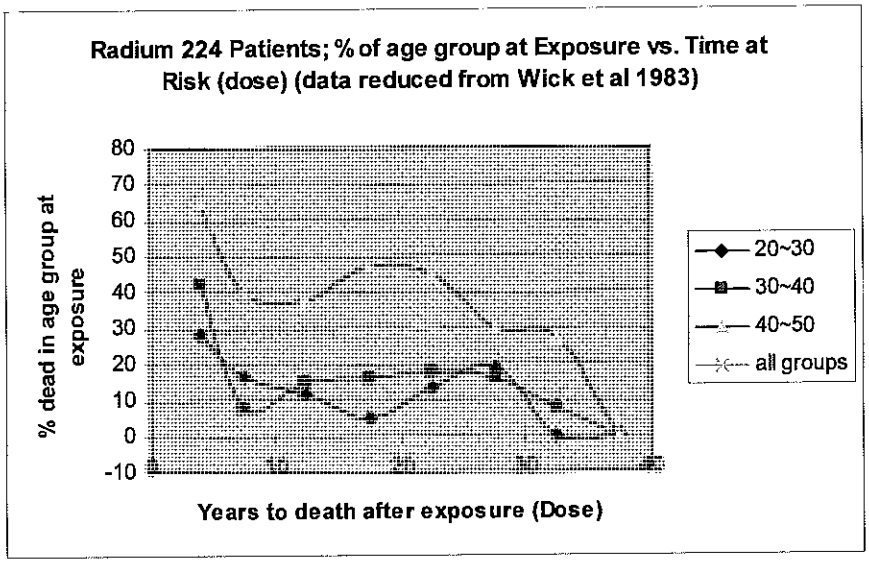
In addition, the doses were very large, so these studies were not of low dose chronic exposure but were in fact high dose internal chronic exposure.

Some of these problems were raised in 1970 in relation to the pioneering work by Robley Evans. Evans was a physicist and was concerned with the question of physical

dosimetry of small quantities of internal emitters. Writing in the *British Journal of Cancer* in 1970, JF Loutit took issue with the methodology the radium studies and pointed out that the massive bone marrow damage resulting from radium exposure (which had been reported by many authors before Evans) would result in a very large excess death rate from a range of diseases. Loutit wrote that the limiting hazard from internally retained radium acquired occupationally being bone cancer needed to be reconsidered. He pointed out that evidence already existed in the 1930s from the work of Martland (see section 2) that those with substantial body burdens of radium had considerable life shortening and that the associated pathology had not been clarified. Loutit re-examined the radium dial case reports and found that internal radium had a profound effect on the bone marrow best described as leukopenic anemia. This identifies one source of increased risk from non cancer illness and death which would have removed individuals from Radium and Thorotrast study groups. Indeed the problem with all these studies is that they exclude about half of the exposed population, who may have been lost to the researchers but are very likely to have died of cancer or a range of non cancer illnesses. In the better reported studies, where more data is made available, it is possible to see that this is indeed the case. An example is Wick et al (1983) who examined cancer in Ra-224 patients. I have reduced the data from a diagram in this paper to produce the graph in Fig R.1 which shows the percentage dead in the age group at exposure by the period between exposure and death. It is clear from the trend that for all the groups, the most deaths will have occurred in the first five years in individuals that were not in the study.

Fig R.1 Percentage of each age group at exposure plotted against years to death from exposures in the Ra-224 study of Wick et al 1983. (Wick RR, Chmelevsky D and

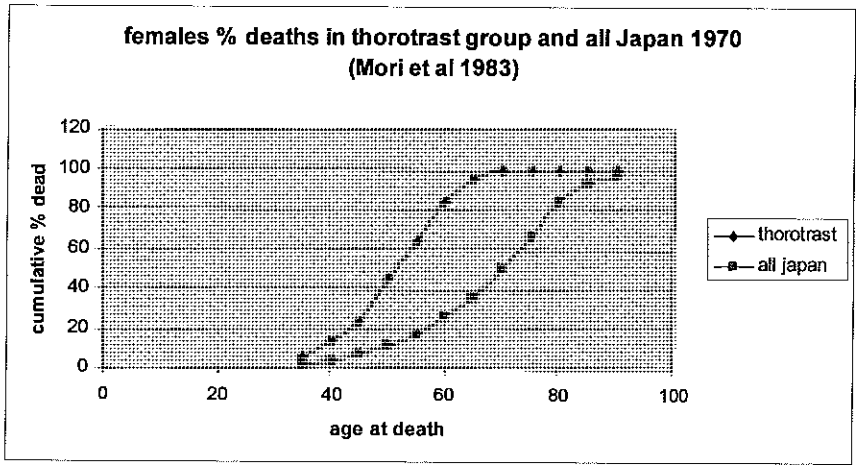
Goessner W (1985) 224Ra: Risk to Bone and haematopoietic tissue in Ankylosong spondylitis patients. in W.Goessner, GB Gerber, U Hagen and A Luz EDs *The radiobiology of radium and thorotrast* Munich: Urban and Schwarzeberg)



This Ra-224 study by Wick et al is of the exposure group of German patients who were treated between 1948-75 with Ra-224 for ankylosing spondylitis. There were 1501 total patients for which 69 were missing and 433 were dead. What did they die of? We don't hear. But 3 of them developed bone cancer, 5 developed leukemia and 6 bone marrow failure (cf Loutit above). This tiny cancer yield may approximate to the range predicted by the ICRP model (assuming that the dose could be accurately described) but what about the missing people? What about those 433 who died? If they died of conditions caused by the stress on their immune system (bone marrow failures and silent bone marrow problems) then the cancer yield is not a proper representation of the effects of the radium exposures on this group. And the cancer yield to produce an approximation to the ICRP risk predictions for leukemia is lower than in the control group. Addition of a handful of cases from the missing individuals or a handful of pre-leukemic immune-compromised individuals from the 633 dead would have a profound effect on the outcome of the study.

A similar picture is found in the thorotrast studies, where it is possible to see enough data. For example, in the paper by Mori et al 1983, 282 Japanese war wounded ex servicemen thorotrast cases are followed up. There were deaths from liver cancer, cirrhosis of the liver and also blood diseases. But in 170 deaths in the group 42% were from cancer and 37% from other causes. There was no dose response for the cancers and the cancer yield was about 20 times greater than expected from ICRP. But the most interesting aspect is that from analysis of this group, the death rate was very high and the age at death very low compared with all Japanese populations. This is missed in the report since the method employed was to choose sick pathology controls from a hospital pathology records sample. I have compared their age specific death rates with all-Japan. Plots of the survival curves in the females in this group show that 100% were dead by age 75 compared with 65% for the equivalent all-Japan population. Results are given in Fig R.2.

Fig R.2 Survival curves for female thorotrast patients studied by Mori et al 1983 compared with all Japan. Data reduced from tables in Mori et al 1983 and Japanese government publications.



(Mori T, Kumatori T, Kato Y, Hatakeyama S, Kamiyama R, Mori W, Irie H, Maruyama T and Iwata S (1983) Present status of medical study on thorotrast administered patients in Japan. 123-135 in W.Goessner, GB Gerber, U Hagen and A Luz EDs *The radiobiology of radium and thorotrast* Munich: Urban and Schwarzeberg)

Of course, about 40% of these study group women died of cancer: the effects of the thorotrast. But note that the others died from something else; they didn't live to a ripe old age nor did they live as long as the all Japan population. This is clear from the survival curves in Fig R.2 which show almost a 20 years age effect in the women. For men, the shift was about 9 years (my unpublished results, not shown).

The conclusions of this brief account of the on-going re-examinations of the radium and thorotrast studies show that they cannot be used as indicators for low dose chronic risk to internal radionuclides. Apart from the fact that the doses were (like the A-Bomb doses) very large, the main fatal flaw was and is that confounding causes of death make the cancer yield conclusions unsafe. Interestingly for ECRR, though not for the pipework cases, Loutit 1970 makes the point that the damage to the bone marrow would be likely to occur in the case of the weapons-fallout component Strotium-90, and he urges the research community to concentrate on examining risk from that nuclide, an exhortation which the research community entirely failed to take notice of. Loutit was a Medical Research Council MRC (Harwell) director; shortly after this paper was published, radiation risk was removed from the MRC (who were clearly becoming nervous about radiation effects) by government and handed to the physicists at a new outfit, the UK National Radiological Protection Board.

But what is useful for the current report on the Forsmark case in this re-examination of the radium and thorotrast studies is that first this shows that the studies cannot be employed to defend the ICRP model and also that second we should expect many other difference illnesses in those exposed to radium than merely cancer.

R.2 ECRR weightings for Radium exposure

Those familiar with the ECRR methodology will know that the committee employs weighting factors for biophysical hazard (w_J) and biochemical enhancement (w_i) due to increased ionization density at the DNA. This calculates equivalent dose in the same way as the ICRP quality factor for e.g. alpha particles (20). It seems that the ICRP itself had been aware of the problem of DNA affinity and had proposed as early as 1978 employing a weighting factor for DNA affinity nuclides (e.g. Sr-90, Ba-140, Ra-226, U-235) but somehow the idea had been shelved (personal communication Michael Jensen, SSM, Stockholm). The relevant tables in ECRR 2003 and ECRR2010 are Table 6.2 and Table 6.3. With regard to Radium-226, the committee will assign a value of 20 to Ra-226 internal exposure due to its DNA binding as Radium cation (Calcium group), and for Ra-226 as a particulate, the value will also be 20 due to local alpha field effects. However, the ECRR assignment is highly conservative, and under certain circumstance, where possible, local doses can be calculated (see skin dose and colonic epithelium doses calculations below). The ECRR factor of 20 will, of course, multiply the ICRP Q factor of 20 for alpha particles. The results of the Radium and Thorotrast studies make it clear that these weightings are probably highly conservative. They are, however, to be applied only for exposures at low and moderate dose rates, since at high dose rates (like those received by the radium/thorotrast groups, cell killing will predominate and lead to the biphasic dose response discussed in ECRR2003 and 2010). They will be applicable to the exposures considered in the Forsmark analysis since the Ra-226 represents the main component of radiation exposure in the long term..

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 of discovery continues today although the dose limits are stuck at their 1990 levels. This is because 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 (IRSN) have pointed this out: 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 were exposed at the test sites.

The modern ICRP conventional approach to dosimetry will however provide a baseline for risk assessment and I will apply it in due course. My reason for discussing the history and development of risk models is to show that all the scientific evidence is that even current statutory dose limits do not adequately safeguard human health. It has become clear that the dangers of low dose radiation should have been apparent to all who worked with radioactivity or employed those who worked with radioactivity at least from the early 1980s when the nuclear site child leukemias were widely reported and when the

dose limits were reduced to the point that they could not be reduced further without seriously affecting industry and the military.

The weight of scientific belief about the dangers from internal radiation began to change in the mid 1990s with interest on the increasing evidence from nuclear site clusters and Chernobyl effects which clearly showed that the contemporary risk models were somehow false by a very large amount. Between about 1996 and 2000, evidence began to emerge from the laboratory for genomic and bystander effects. Since the then current ICRP model was based on genetic damage and a linear relation, it was implicit by 2000 that this basis was completely incorrect. This, and various other epidemiological evidence (which had now to be re-assessed) led to the Committee Examining Radiation Risks from Internal Emitters and the 'Radiation Science Wars' of the early 2000s. The critical impact of the 2003 report of the European Committee on Radiation Risk, and the clear demonstrations in epidemiological evidence from the Chernobyl affected territories (infant leukemia, minisatellite mutations, cancer in Sweden, Belarus and Ukraine) that the ECRR predictions were close to what was seen was a turning point in a paradigm shift that continues today.

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12. Appendix B: The Lesvos Declaration

ECRR - CERI

European Committee on Radiation Risk - Comité Européenne sur le Risque de l'Irradiation

The Lesvos Declaration
6th May 2009, Molyvos, Lesvos, Greece

A. Whereas, the International Commission on Radiological Protection (ICRP) has promulgated certain risk coefficients for ionizing radiation exposure,

B. Whereas, the ICRP radiation risk coefficients are used worldwide by federal and state governmental bodies to promulgate radiation protection laws and standards for exposure to workers and the general public from waste disposal, nuclear weapons, management of contaminated land and materials, naturally occurring and technologically enhanced radioactive materials (NORM and TENORM), nuclear power plant and all stages of the nuclear fuel cycle, compensation and rehabilitation schemes, etc,

C. Whereas, the Chernobyl accident has provided the most important and indispensable opportunity to discover the yields of serious ill health following exposure to fission products and has demonstrated the inadequacy of the current ICRP risk model, especially as applied to foetal and early childhood exposures to radiation,

D. Whereas, by common consent the ICRP risk model cannot validly be applied to post-accident exposures, nor to incorporated radioactive material resulting in internal exposure,

E. Whereas, the ICRP risk model was developed before the discovery of the DNA structure and the discovery that certain radionuclides have chemical affinities for DNA, so that the concept of absorbed dose as used by ICRP cannot account for the effects of exposure to these radionuclides,

F. Whereas, the ICRP has not taken into consideration new discoveries of non-targeted effects such as genomic instability and bystander or secondary effects with regard to understanding radiation risk and particularly the spectrum of consequent illnesses,

G. Whereas, the non-cancer effects of radiation exposure may make it impossible to accurately determine the levels of cancer consequent upon exposure, because of confounding causes of death,

H. Whereas, the ICRP considers the status of its reports to be purely advisory,

I. Whereas, there is an immediate, urgent and continuing requirement for appropriate regulation of existing situations involving radioactivity, to protect the human population and the biosphere,

We the undersigned, acting in our individual capacities

1. assert that the ICRP risk coefficients are out of date and that use of these coefficients leads to radiation risks being significantly underestimated,

2. assert that employing the ICRP risk model to predict the health effects of radiation leads to errors which are at minimum 10 fold while we are aware of studies relating to certain types of exposure that suggest that the error is even greater,

3. assert that the yield of non-cancer illnesses from radiation exposure, in particular damage to the cardio-vascular, immune, central nervous and reproductive systems, is significant but as yet unquantified,

4. urge the responsible authorities, as well as all of those responsible for causing radiation exposures, to rely no longer upon the existing ICRP model in determining radiation protection standards and managing risks,

5. urge the responsible authorities and all those responsible for causing exposures, to adopt a generally precautionary approach, and in the absence of another workable and sufficiently precautionary risk model, to apply without undue delay the provisional ECRR 2003 risk model, which more accurately bounds the risks reflected by current observations,

6. demand immediate research into the health effects of incorporated radionuclides, particularly by revisiting the many historical epidemiological studies of exposed populations, including re-examination of the data from Japanese A-bomb survivors, Chernobyl and other affected territories and independent monitoring of incorporated radioactive substances in exposed populations,

7. consider it to be a human right for individuals to know the level of radiation to which they are exposed, and also to be correctly informed as to the potential consequences of that exposure,

8. are concerned by the escalating use of radiation for medical investigation and other general applications,

9. urge significant publicly funded research into medical techniques which do not involve radiation exposures to patients.

Statements contained herein reflect the opinions of the undersigned and are not meant to reflect the positions of any institution to which we are affiliated.

Professor Yuri Bandazhevski (Belarus)
Professor Carmel Mothersill (Canada)
Dr Christos Matsoukas (Greece)
Professor Chris Busby (UK)
Professor Roza Goncharova (Belarus)
Professor Alexey Yablokov (Russian Federation)
Professor Mikhail Malko (Belarus)
Professor Shoji Sawada (Japan)
Professor Daniil Gluzman (Ukraine)
Professor Angelina Nyagu (Ukraine)
Professor Hagen Scherb (Germany)
Professor Alexey Nesterenko (Belarus)
Dr Sebastian Pflugbeil (Germany)
Professor Michel Fernex (France)
Dr Alfred Koerblein (Germany)
Professor Inge Schmitz Feuerhake (Germany)

13. Curriculum Vitae (May 2012)

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Nationality: British; Resident Latvia

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:

Member: Royal Society of Chemistry
Member: Royal Society of Medicine
Member: International Society for Environmental Epidemiology
Member: Ukraine Committee: Physicians of Chernobyl

UK Government Committees

Member: (Department of Health and DEFRA) CERRIE
Committee Examining Radiation Risk from Internal Emitters 2001-2004
www.cerrie.org

Member: Ministry of Defence DUOB
Depleted Uranium Oversight Board
2002-2007
www.duob.org

Other Committees

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

Science Policy group Leader: Policy Information Network on Child Health and Environment PINCHE
www.pinche.org

1.2 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)
1997	Foundation for children with leukaemia; research on non-ionising radiation
2001	Appointed Scientific Secretary of ECRR and commissioned to prepare the report ECRR 2003- The Health effects of low doses 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
1992-2008	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; Corporate Responsibility in Science and Policy
2008	3-year research funding from The Joseph Rowntree Charitable Trust; Corporative Responsibility in Science
2008	Appointed Guest Researcher, German Federal Research Laboratories, Julius Kuhn Institute, Braunschweig, Germany
2008	Appointed Visiting Professor, School of Molecular Bioscience, Faculty of Life and Health Sciences, University of Ulster, Coleraine, Northern Ireland

2012	Appointed Visiting Scientist, Faculty of Science and Engineering, Jacobs University, Bremen, Germany

1.3 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 (annually)

1.4 ADMINISTRATIVE EXPERIENCE

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

Invited Reviewer
International Journal of Radiation Biology
Science of the Total Environment
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
The Lancet

Occupational and Environmental Medicine (BMJ)

1.5 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), MoD pension appeals tribunals for the widow of a A-Bomb test veteran and once in the Connecticut State Court for an appeal against licensing 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. He also advised on the case of Rocketdyne (Boeing) and the Santa Susana Field Laboratory childhood retinoblastoma cluster in Western Los Angeles which was settled in January 2008 and a TENORM radiation case involving Ashland Oil in Martha Kentucky, also various other TENORM cases in Louisiana. He is currently also expert witness and advisor on the UK Atomic Test veteran litigation in the Royal Courts of Justice. He has been active in several test veterans pensions appeals tribunals gaining reversal in every case of MoD refusals to pay war service pensions in respect of diseases linked to radiation exposure at the test sites. He testified in 2009 before a coroners jury in the case of the death of Stuart Dyson arguing that Dyson probably died of cancer due to his exposure to depleted uranium in the Persian Gulf. Despite opposition from the MoD the jury unanimously agreed that the uranium exposure was the probable cause of death. A full list and brief description of the court cases in which Dr Busby has been retained as an expert witness is given below.

Dr Chris Busby
Court cases as expert witness

Case and lawyer/ team	Court	Year	Details (expertise)	Result
1. R vs Hipperson et al (Charlton)	Newbury Crown Court	1998	Criminal Damage Atomic Weapons Establishment Aldermaston (radiation health effects)	Aquitted
2. R vs Helen John	Middlesex Crown Court	1999	Criminal Damage House of Commons London (uranium health effects)	Acquitted
3. Sellafield Irish Litigation (McGuill, Herr, Irish State)	Dublin High Court	1999-2001	Case against Sellafield THORP reprocessing plant (epidemiology, radiation effects, Irish Sea)	Case withdrawn in 2008

4. Millstone Reactor Public Enquiry	Connecticut State Court	2001	Opposition to relicensing of Millstone Reactor (Radiation health effects and sea dispersion)	Failed
5. Fatmir Mata (Wilson , Berry)	Immigration Appeal Court	2001-2003	Human Rights immigration appeals Kosovo (uranium health effects)	Failed
6. Lela Pelumb (Wilson & Co, Hanley)	Immigration appeal court	2001	Depleted Uranium Kosovo (uranium health effects)	Failed
7. Ladrim Spata (Clore and Co, Hirsch)	Immigration appeal court HX 06027	2001	Depleted Uranium Kosovo (uranium health effects)	Failed
8. Shaquiri, Zogu, Malo, Deda and Hidri vs. Secretary of State Home Office (Henwood)	Immigration appeal court	2002	Depleted Uranium Kosovo (uranium health effects)	Failed
9. Hadjarmata vs Sec.State Home Office (Wesley, Gryk, Amador)	Immigration appeal court	2002	Depleted Uranium Kosovo (uranium health effects)	Failed
10. Mr and Mrs Ardian Kuci vs Sec.State Home Office	Immigration appeal court	2002	Depleted Uranium Kosovo (uranium health effects)	Failed
11. Gerald Adshead vs Ministry of Defence	Pensions appeals court	2002	A-Bomb Test Veteran cancer (epidemiology, radiation health effects)	Won: appeal allowed
12. R.vs Margaret Jones and Erika Wilson (Alan Harris)	Plymouth Crown Court	2002	Criminal Damage Nuclear Submarine base Plymouth (radiation and health)	Acquitted
13. Lee Dell Craft Snr vs Intracoastal Tubular ITCO (Stuart Smith)	New Orleans	2005-	Cancer following exposure to NORM (Epidemiology, radiation and health)	Settled by defendants
14 Barbara Castell vs Intracoastal Tubular ITCO	New Orleans	2005-	Cancer following exposure to NORM (Epidemiology, radiation and health)	ongoing

CDC No 2002-12334 Dv A section 5. (Stuart Smith)				
15 Ursula Bulot et al vs Exxon Mobil Corp and others (Stuart Smith)	New Orleans	2005-	Cancer following exposure to NORM (Epidemiology, radiation effects)	ongoing
16 James Bailey vs Exxon Mobil Corp and others (Stuart Smith)	New Orleans	2005-	Cancer following exposure to NORM (Epidemiology, radiation effects)	ongoing
17 Zachary Finestone, Lowe et al vs St Lucie Power and Light (Lytal Reiter, Palm Beach Fla).	Florida USA Case 03-04040 Cohn/Lynch **BUSBY DAUBERT MOTION**	2005	Case of children with cancer near St Lucie Nuclear Power Station. (Epidemiology, radiation dispersion modelling and health effects)	Case eventually dismissed
18 R.vs Pritchard and Olditch (Bindmann and partners)	Bristol Crown Court	2005-6	Criminal Damage US bombers 2003 (uranium and health) (famous case)	Acquitted
19 R vs RV Jones and Milling (Charlton)	Bristol Crown Court	2005-6	Criminal Damage US bombers (uranium weapons) (famous case)	Acquitted after appeal to Lords
20 Brian Gay vs Ministry of Defence	Pensions Appeals Court	2007	A-Bomb Test veteran Maralinga ; was his kidney cancer caused by radiation? (Epidemiology, radiation and health)	appeal successful
21 Richard David vs Honeywell Normalair Garratt (L.I.P)	Royal Courts of Justice, Queens Bench Divn. London	2007-8	Uranium contamination and health; contaminated via aero engines filters from high altitude (Epidemiology, radiation and health)	Case collapsed; litigant in hospital
22 Cindy Mays and others vs Boeing Rocketdyne Corp (Suzelle Smith)	Los Angeles USA	2007	Did radiation releases from the Rocketdyne SSFL cause retinoblastoma in 9 Los Angeles children? Epidemiology, radiation dispersion modelling and	Settled by defendants

			health effects).	
23 Bonnie Anderson et al vs Ashland Oil (K. Mathis et al)	Lawrence Circuit Court Kentucky USA **BUSBY DAUBERT MOTION**	2008	Contamination of property by oilfield NORM (Epidemiology, radiation and health)	Case dismissed but being appealed
24. A-Bomb Test veterans vs UK Ministry of Derfence (Rosenblatts)	Royal Courts of Justice, London	2009-10	Cancer and illness in A-Bomb Test veterans (Epidemiology, radiation and health)	1st round won, but on appeal
25 Various vs Exxon Mobil Corp (Gordon)	Houston TX	2009-10	Measuring gamma and advising on NORM contamination for potential case	Ongoing
26 Derek Hatton Vs Ministry of Defence (Derek Heaps)	Pensions Appeals Court Birmingham	2009	Cancer and *polycythemia rubra vera*	See below
27 Etienne Pellegal vs Lincoln Electric Co (Garrison)	New Orleans No 2006-003684 Sec 6 Div L	2009-10	NORM radiation and laryngeal cancer	Settled by defendants
28 Stuart Dyson dec. vs MoD HMCoroner, Balmain)	Coroners Court Black Country Jury	2009	Cause of Death; did depleted Uranium cause cancer. Big case, reported everywhere	Yes
29 Colin Duncan Vs. Ministry of Defence	Pensions appeal Court	2010	Exposure to fallout in A-Bomb Tests caused cancer	Won: Appeal allowed
30 Lowell Ryman vs Regents of University of California (Howell)	Los Alamos USA	2010	Exposure to radioactivity from Los Alamos and Malignant Myeloma	Case withdrawn by attorney
31 Michael Nase vs Teco Energy (Stuart Smith)	New Orleans USA	2009	Exposure to radon and radiation and lymphoma	Ongoing
32 Dawn Pritchard vs Ministry of Defence	Pensions Appeals Court	2010	A-Bomb Test veteran widow. (Radiation and health)	Ongoing

33 L Abdale vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT 00328 2010	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
34 D Battersby vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT 00176	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
35 D Beeton vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT 00129	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
36 T VButler vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT 00078	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
37 D Hatton vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
38 NC Hughes dec vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT00065	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
39 B Lovatt vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT 00279	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
40 D Pritchard vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT 00039	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
41 L Selby vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT 00658	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
42 Denis Shaw	Pensions	2010	A-Bomb Test veteran	Ongoing

vs Ministry of Defence (Royal British Legion)	Appeals Court ENT 00054		appeal (Radiation and health)	
43 N Simons vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT 00006	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
44 H Sinfield vs Ministry of Defence (Rosenblatts)	Pensions Appeals Court ENT 00751	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
45 B Smith dec. vs Ministry of Defence (Rosenblatts)	Pensions Appeals Court ENT 00680	2010	A-Bomb Test veteran appeal	Ongoing
46 Mrs A Smith vs Ministry of Defence (Rosenblatts)	Pensions Appeals Court ENT	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
47 D Taylor vs Ministry of Defence (Chris Francis RAFA)	Pensions Appeals Court ENT 00912	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
48 Mrs W Triggs vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT 00285	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
49 Mrs M Williams vs Ministry of Defence (Royal British Legion)	Pensions Appeals Court ENT 00768	2010	A-Bomb Test veteran appeal (Radiation and health)	Ongoing
50 Kingscliffe Waste Watchers vs Augean Ltd	Public Enquiry	2010	Effects of radioactive waste on health (radiation and radioactivity dispersion, exposure and health).	ongoing

In two of the above cases, Dr Busby was deposed by defence attorneys with a view to having his status as an expert witness disallowed by the trial judges under the rules of *Daubert vs Merrel Dow Pharmaceuticals* whereby the judges have conferred on them the power to decide whether an expert witness is expert in the area of expertise being

claimed and to disallow his or her testimony if not. In both cases, in the State of Florida and in State of Kentucky the Daubert motions was unsuccessful.

In addition to the above Dr Busby has been invited or commissioned to provide expert evidence on the health effects of low doses of ionising radiation, or exposure to uranium for and to, amongst others :

The UK Royal Society Committee on Depleted Uranium
The UK Committee Examining Radiation Risk from Internal Emitters
The UK Committee on Radioactive Waste Management
The US Congressional Committee on Veterans Affairs and Security in the UK House of Lords (Depleted Uranium)
The Canadian Parliament
The Greens in the European Parliament
The UK Environment Minister Michael Meacher MP
The Federal German Agricultural Laboratory, Brauschweig
The EU Policy Information Network on Child Health and Environment, Arnhem, Netherlands
The British Nuclear Test Veterans Association
The UK House of Commons Cross Party Committee on A-Bomb Test Veterans (John Barron MP, Neil Gibson MP)
The United Nations (UNIDIR) Geneva
The World Health Organisation/ Physicians of Chernobyl (Kiev)
The Government of Belarus
The Green Party of England and Wales
SAFEGROUNDS (Nuclear Industry Organisation for waste disposal discussions)
The British Nuclear Energy Society
The British Nuclear Test Veterans Association
The Royal British Legion
The New Zealand Royal Society

1.6 APPOINTED or INVITED ADVISOR

Various national and supra-national groups have sought advice from or appointed Dr Busby as an advisor on various issues e.g.
Green Group European Parliament; Radiation and Health (Caroline Lucas MEP)
Canadian Government: Uranium and Health (appointed by Alex Atamenenko MCP, British Columbia)
UK Committee on Radioactive Waste Management (invited by Prof Gordon McKerron)
Royal Society Committee on Health Effects of Depleted Uranium Weapons (invited by Prof. Brian Spratt)
US Congressional Committee on Veterans Affairs and Security (Uranium weapons) (invited by Senator Christopher Shays)
UNIDIR Geneva (United Nations Institute for Disarmament Research) (Kirstin Vignard)

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.

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 was recognised 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. This work he updated with a chapter on Uranium and evidence of the success of the 2003 model in explaining increases in cancer near nuclear sites and also the reports of increases in cancer in Sweden after Chernobyl reported by Tondel et al. in 2010. In 2004, he (jointly with two other colleagues) published the *Minority Report of the CERRIE committee* (Sosiumi Press) which was supported and

introduced by Environment Minister the Rt Hon. Michael Meacher MP. In 2006 he produced and jointly edited with Prof. Alexey Yablokov of the Russian Academy of Sciences *ECRR2006 Chernobyl 20 Years On.* A second edition was produced in 2009.

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 (PINCHE) 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 policymakers, 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.

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

He is also currently engaged in experimental and theoretical development of a novel theory of living systems and their origin.

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 year 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 behaviour at charged interfaces and developed a Surface Raman spectroelectrochemical method as a Science Research Council Fellow at the University of Kent.

Between 1992 and 2004 Dr Busby was engaged in research 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 to inhalation of radioactive 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) 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 and Trawsfynydd, Gwynedd, Wales, which resulted in a TV documentary in 2004. 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. He has supervised a PhD student, who has subsequently graduated, 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. Since then he has measured radiation spectra in the filed in many countries and now employs a 7" detector manufactured in Kharkov to obtain rapid analysis of field gamma radiation. 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 the EU 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 conversion 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 provisional patent application for the use of photoelectrons in cancer therapy to destroy tumours.

In 2005 he was invited by various organisations in New Zealand (NZ Royal Society) 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. In 2004 he conducted a study of the health of people living in the vicinity of the Trawsfynydd Nuclear plant in Wales for HTV and later also a study of the veterans of the Porton Down human experiments in the 50s. The results of the Porton Down veterans study led to a settlement and an apology by the government to the veterans in 2008. In 2007 he began epidemiological studies of the children of A-Bomb Test veterans and also of people living near mobile

phone base stations. The A-Bomb veterans epidemiology study highlighted high rates of miscarriage and congenital illness in their children and grandchildren. The results were presented to the House of Commons committee investigating this issue in November 2007 and have led to a recent agreement by the UK government to fund further epidemiological research on this issue, research which Dr Busby will oversee on behalf of the Test Veterans. In 2005, with Saoirse Morgan he analysed data from the filters of the Atomic Weapons site in Aldermaston and employed NOAA geophysical modelling to show that uranium from Gulf War 2 weapons use had migrated to the UK. He has become interested in the use of Uranium Weapons and has recently been involved in obtaining samples from the Israeli actions in The Lebanon and Gaza and analysing vehicle filters for uranium. His discovery of enriched uranium in such samples has received significant media coverage and resulted in an invitation to write a 12-page article for the United Nations *Disarmament Forum* Journal published in Geneva in 2009. His 2010 study with Malak Hamdan and Entear Ariabi of the cancer increases and sex ratio changes in Fallujah Iraq following the US led attacks on the city has achieved considerable prominence.

He is currently an expert advisor on the Test Veterans' litigation and expert witness on their litigation against the British Government where the initial issue of limitation was recently won in a landmark case in the Royal Courts of Justice. He is official scientific advisor to the British Nuclear Test Veterans' Association and has appeared for them in many legal tribunals for Pension Appeal cases. He was appointed Visiting Professor in the School of Molecular Biosciences in the University of Ulster in 2008 where he is co-supervising research on the health effects of uranium. His uranium photoelectron theory was the top 2- page news story in the *New Scientist* of 6th September 2008 and is receiving considerable attention from the international nuclear risk agencies. Also in 2008 he was appointed Guest Researcher at the German Federal Government Julius Kuhn Institute in Braunschweig where he is co-supervising research on Uranium uptake in plants. He is also currently working on the health effects of radioactive contamination of the Baltic Sea with colleagues in Sweden, Finland and Latvia and has set up offices to organise such research in Riga Latvia and in Stockholm Sweden.

In May 2009, in his capacity as Scientific Secretary of the European Committee on Radiation Risk (ECRR) he organised an International Conference on the Greek Island of Lesvos attended by eminent radiation scientists from all over the world. The final statement from this conference The Lesvos Declaration called for the abandonment of the current (ICRP) radiation risk models which all the delegates agreed was insecure for its purpose of protecting human health from radiation exposures.

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: Health 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, Charite Hospital, 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	Hanover: Federal Agricultural Laboratories; Uranium chemistry and physics
2007	Geneva: United Nations. Health effects of Uranium weapons
2007	Geneva: United Nations. Chernobyl: WHO and the IAEA
2007	London, House of Commons Select Committee: Nuclear Test Veterans Children Epidemiology study
2007	London, Royal Society: Science Policy Advice and Scientific Dishonesty
2008	Ljubljana Slovenia: Parliament; Nuclear Energy and Human Health
2008	Malmo Sweden; Uranium and health- new discoveries
2008	Helsinki; Chernobyl effects
2008	Moscow, Russian Academy of Sciences; A new theory of living systems.
2009	Malmo Sweden, Uranium weapons and health
2009	Stockholm Sweden, ICRP, SRM, Errors in radiation risk model
2009	Lesvos Island Greece; Requirements of a Adequate Radiation Risk Model
2009	Academy of Sciences, Riga, Latvia: the ECRR and ICRP radiation risk models
2009	Arusha Tanzania: Health effects of Uranium mining
2009	Dar es Salaam, Tanzania: Health effects of Uranium Mining
2010	Geneva, Human Rights Council, Fallujah uranium effects
2010	Riga Latvia; Environment Ministry; Baltic Sea Radioactivity and Health
2010	Stockholm Sweden; Finlandhuset; Cancer and Birth Defects in Fallujah Iraq
2010	Riga Latvia; Latvian NGOs; Baltic sea radioactivity and the ECRR model
2010	Pretoria South Africa, North West University, Uranium and Health
2011	Tokyo, Fukushima health effects
2011	Fukushima, Aizu Wakamatsu, health effects
2011	Berlin, Fukushima Health effects
2011	Riga, Fukushima
2011	Chepstow UK, Fukushima
2011	Oxford Town Hall, Fukushima
2011	Vilnius University, Lithuania, Scientific Philosophy and radiation risk models
2012	Riga Latvia, Developing a new ideology for human security

2. PUBLICATIONS

PEER REVIEWED PAPERS.

Alaani Samira Tafash Muhammed, Busby Christopher, Hamdan, Malak and Blaurock-Busch Eleonore (2011) Uranium and other contaminants in hair from the parents of children with congenital anomalies in Fallujah, Iraq *Conflict Health* 5, 1-15

Busby, Chris; Hamdan, Malak; Ariabi, Entesar. (2010) Cancer, Infant Mortality and Birth Sex-Ratio in Fallujah, Iraq 2005–2009. *Int. J. Environ. Res. Public Health* 7, no. 7: 2828-2837.

Busby C.C. (2009) Very Low Dose Fetal Exposure to Chernobyl Contamination Resulted in Increases in Infant Leukemia in Europe and Raises Questions about Current Radiation Risk Models. *International Journal of Environmental Research and Public Health.*; 6(12):3105-3114. http://www.mdpi.com/1660-4601/6/12/3105

Busby Chris (2009) Depleted Uranium, Why all the fuss? *Disarmament Forum* 3 25-33 Geneva: United Nations

Busby Chris, Lengfelder Edmund, Pflugbeil Sebastian, Schmitz Feuerhake, Inge (2009) The evidence of radiation effects in embryos and fetuses exposed by Chernobyl fallout and the question of dose response. *Medicine, Conflict, Survival* 25(1) 18-39

Busby Chris (2008) Is there a sea coast effect on childhood leukaemia in Dumfries and Galloway, Scotland, 1975-2002 ? *Occupational and Environmental Medicine* 65, 4, 286-287

Busby Chris and Schnug Ewald (2008) 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.

Busby C and Howards V (2006) Fundamental errors in official epidemiological studies of environmental pollution in Wales. *J Public Health (Oxf)* 28(2) 177-8

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3. PERSONAL

Dr Busby has 7 children and 12 grandchildren and lives between the ancient Baltic city of Riga, Latvia and his 65 ton 1903 Dutch Barge *Marius* in France. His interests include music and writing and performing songs (he plays guitar, banjo, diatonic accordion, bandoneon, garmoshka, violin, viola, nykleharpa and Hardanger fiddle: see www.myspace.com/christobusby), writing poetry and sailing.