Response to *Draft Report of the High Level Expert Group on European Radiation Risk Research of 8th Sept 2008*

On behalf of

The Green Party of England and Wales

Green Audit

The Low Level Radiation Campaign

The European Committee on Radiation Risk

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1. Introduction

The *Draft Report of the High Level Expert Group on European Radiation Risk Research of 8th Sept 2008* outlines the reasons for scoping and implementing a major research effort into re-examining the current risk models for assessing and interpreting the effects of low doses of ionising radiation. Conceding that there is considerable uncertainty about the health effects of ionising radiation at low doses, it goes on to list the areas of concern where research effort is to be concentrated and proposes a research strategy.

I should point out at the outset that the currently most consulted radiation risk agencies, which I list in Table 1, do not presently concede that their risk models, which are largely the same, have any significant uncertainty at low doses. The publication of this HLEG report, and its contents, therefore already represents a significant question mark over the adequacy of the radiation risk model which currently underpins legal constraints on exposures in the European Community and which are laid out in the Euratom Safety Standards Directive 96/29.

Table 1. The most influential International Risk Agencies or Committees that either underpin current exposure standards or disagree with current standards

Agency	Relevant publication	Note
(1) ICRP	ICRP2007 (2007)	Based in UK; extrapolates from external doses from Japanese A Bombs; linear no threshold dose response
(2) UNSCEAR	UNSCEAR (2000/ 2008)	Geneva/ USA; Extrapolates from external acute doses; as above
(3) BEIR	BEIRVII (2006)	USA; Uses external acute doses; as above
(4) ECRR	ECRR2003 (2003)	Based in Europe, Brussels; addresses internal exposures and dose response relationship

(1) The International Commission on Radiological Protection
(2) The United Nations Scientific Committee on the Effects of Atomic Radiation
(3) The Biological Effects of Ionising Radiation sub-committee of the US National Academy of Sciences
(4) The European Committee on Radiation Risk/ Comite Europeen sur le Risque de l'Irradiation

National risk agencies in each country, and each member state of the EU, take their model from that of the ICRP, essentially the same model as that with is presented by 1-3 of the above. On the other hand, the model of the ECRR (ECRR2003) pragmatically addresses the various problems which are the subject of the HREP report and is increasingly employed in radiation health litigation and in advice to governments on specific issues. Indeed, it is an open secret that the report of the ECRR and the two reports of the UK CERRIE committee (CERRIE 2004a, 2004b) has so destabilised public and government belief in the adequacy of the current risk framework that moves have been made to investigate the issues which have been raised (e.g. IRSN2005). The report I am addressing here and its proposals are clearly a response to the concerns initially raised in the ECRR report and its forerunners.

The agreement between the models of the ICRP, UNSCEAR, BEIR and the national committees is unsurprising. It results from two things. First these committees base their risk model on sophisticated and complex analysis of the Japanese A-Bomb lifespan studies (with some supporting evidence from other acute external radiation

epidemiology). They ignore internal radiation from fission products and uranium. They also ignore the massive evidence that their models are incorrect which has emerged from the post Chernobyl accident landscape where internal radiation is the main factor. Second, they share many individual members between themselves and also the national radiation risk committees, particularly in the UK where ICRP is based.

The decision by the EU to invest energy and money into the investigation of these important issues is a valuable step and is to be welcomed. In general, the process is too long and the questions asked could already be answered with a considerable degree of accuracy. Children die of leukaemia near nuclear plants. People exposed to Chernobyl radiation are dying of cancer and other illnesses. Infant leukemia in 5 European countries defines errors in the risk models of 500-fold or more. Uranium kills and deforms children in war zones.

Given however that the process is underway, the investigation must not be allowed to become diverted or biased by those who have an interest in showing that the concerns being examined are without foundation. The Policy Information Network on Child Health and Environment, PINCHE drew attention in its final reports to the ways in which interpretation of scientific data, or choice of research area, could influence the conclusions of any scientific investigation. The case of the classification by the EU of the carcinogenicity of trichloroethylene was used as an example of how industry culture was brought to bear on decisions which were made at the highest level (Ruden 2003). PINCHE concluded (van den Hazel et al 2006) that committees which examined scientific issues involving public health must be oppositional and transparent if correct answers were to be found and if the public were to have faith in the results. The PINCHE report on radiation risks to children drew conclusions about research efforts some (though not all) of which I see have been included in the scope of the HLEG report (Busby and Fucic 2006). HLEG is not currently transparent. The HLEG scientists have not been identified although their organisations have. This is unacceptable. Many of the members of these organisations are also members of the international agencies or committees whose risk models are being investigated. There is therefore the likelihood of cultural scientific bias; the HLEG is not seen to be transparent or independent and its findings will therefore be weakened or discounted by the public and their representatives.

What is also unacceptable, within the current and widely published political policy of the EU to take information and evidence from all available expert sources, is the failure to include in the HLEG, representation from experts from the ECRR. It was the ECRR arguments, raised at the 1997 EC STOA meeting on radiation risk models, outlined in the ECRR2003 report and presented to the UK CERRIE committee, and the further ECRR evidence on Chernobyl effects (ECRR2006) which have clearly been a major (though unacknowledged) driving force behind the HLEG process. Radiation risk is in disarray; its models will only become accepted by the public and the stakeholders if the processes that underpin them are created transparently and through unbiased examination of all of the evidence. It is the bias that has been historically built in to the current radiation risk models though secrecy and public unaccountability and through historic connections with the military that has led to an alarming situation where the whole edifice is thought to be so massively inaccurate that it urgently needs to be re-examined.

This is a serious matter. Those who are responsible for this re-examination should be aware that if they do not carry it out properly, it will eventually be carried out anyway though litigation in the courts, and the result will be personally

embarrassing and possibly painful for those involved. Since people have died or will die in future as a result of incorrect modelling of radiation exposure risk, this may ultimately result in civil or criminal actions against individuals involved in advice on decision making in this area. I now turn to the draft itself.

2. Policy questions and important issues

The report claims to identify the key policy issues, the state of the science and the research challenges. It proposes a way forward. I present the questions asked by the HLEG and the issues they believe are involved below in Table 2.1. There I also briefly comment on the issues identified and add other important issues which have been identified by independent scientists but which are not addressed by HLEG.

Table 2.1. Issues identified in the HLEG report, other issues (*in italics*) not addressed by HLEG and some comments

Issues and questions	**Comments and answers**
1. How robust is the current system of radiation protection?	Seriously in error for internal chronic exposures to specific radionuclides and Uranium
2. How can it be improved?	By setting up a proper, transparent and unbiased discussion on the issues and taking forward specific research proposals from all sides of the analysis.
3. (What is) the shape of the dose response relation for cancer?	This depends upon the dose range, the type of exposure (external/internal) the tissue, the history of the tissue, and the cancer. It is generally biphasic or supralinear.
4. (Are there) tissue sensitivities for cancer induction?	Yes. This is generally mostly known and further research is unnecessary
5. (Is there) individual variability in cancer risk?	Clearly. This is already known but is not factored into radiation risk models.
6. (What are the) effects of radiation quality (type)?	This also is known from many types of radiation and is (except for Auger) factored into risk models.
7. (What are the) risks from internal radiation exposure?	For some isotopes and exposure regimes the current model is in error by up to three orders of magnitude. The risks from Uranium (and other high Z elements) photoelectron enhancement of gamma background are not considered. These statements are backed by epidemiology and theoretical considerations.
8 (What are the) risks of and dose response relationships from non cancer diseases?	The risks are finite and serious: all the evidence shows this.
9 (What are the) risks and dose response for internal exposures to the foetus?	*Significantly higher than currently modelled.*
10. (What are the risks) from weapons Uranium particles due to local doses and photoelectron enhancement?	*Theoretically and epidemiologically significant.*

11. What are the affinities of radioisotopes for DNA?	*It is extraordinary that this question has not been answered: the research is simple.*

The conclusion of the report is that there should be a *new trans-national organisation capable of ensuring an appropriate governance of research*. This organisation is to be called MELODI.

If such an organisation is formed, it will be made up of members of the HLEG and/or the groups which are part of this organisation. If this occurs, the proposed organisation will be composed of individuals with affiliations to organisation whose current risk models they are effectively investigating. There will be conflict of interest. Since it is conceded that no individuals are without cultural bias, it will be necessary to ensure that the final organisation includes independent scientists and individuals members of groups who are critical of the current risk model. The most relevant organisation here is the European Committee on Radiation Risk, founded in 1997.

3. Comments on the text of the report

I have concentrated on those parts of the report I believe to require comments. I will add my own account of the history of radiation protection and the current radiation regulations at the end of this paper.

3.1 Shape of the low dose response

The text correctly identifies the shape of the low dose response as an important factor. However it is only important in a system where the raw data employed to determine low dose risk is obtained from high dose acute exposure. In science, it is necessary to compare like with like. This is the basis of scientific induction, the scientific method. To compare high dose acute exposure (called 'dose') with low dose internal chronic exposure (also called 'dose') is not induction: it is deduction, and is not therefore science unless it can be shown that the mechanisms are identical. These two 'doses' are only approximately the same in an ionization chamber. Only a theoretical physicist with no knowledge whatever of biological systems would think they could be applied to living systems. For internal exposures, the situation is bizarre, since ionisation density caused by one single internal isotope decay at the target, DNA, can be many orders of magnitude higher for internal exposures than for the whole body external exposures delivered by the A-Bombs. In general whatever the shape of the dose response curve, over a small range it will be effectively linear, and so the way to establish risk factors is to compare cancer rates in those exposed to a particular radiation type or source over a narrow range to those unexposed. When this is done (and it has been done in many cases) the risk from that exposure can be calculated and applied to other similar situations (e.g childhood cancer and nuclear installations). In addition, the clear theoretical and epidemiological evidence for biphasic dose response make the use of regression and correlation methods insecure. This is an important point. The easiest analogy is the irradiation of the foetus where above a certain dose, foetal death/ miscarriage results in a reduction of the level of e.g. leukemia in the child. The resultant dose response is positive, then negative as dose increases. The negative region could be seen (by the stupid) as a hormesis where radiation reduces the incidence of leukaemia! I point out that Fig 2 in HLEG does not include a biphasic response though evidence for such responses is in the literature and

indeed, also in the A-Bomb data. Again, there is no reference to ECRR2003 where the effect is discussed.

In general, given the complexity of biological systems and their responses and ranges of effect it is very unlikely that a simple dose response relation could be decided upon and applied across the board for radiation protection purposes. All types of dose response have been reported by different groups and probably exist under different conditions. The question which must be asked is this: what is the most dangerous dose response curve? It is this one that should be applied to radiation protection: for, if in some situations there is a threshold, this cannot make people sick and die. But if in other situations there is a supralinear or biphasic relation, those situations become the limiting factor on an ethical basis and protection of the public must be made on that basis.

3.2 Internal exposure risks

The HLEG devote one page of bland comment on this issue, which is the most critical one in radiation protection. A number of simple studies could be undertaken, some of which were begun in the CERRIE process but cancelled by the Chair.
Epidemiologically there are a number of natural experiments: Weapons fallout, Chernobyl, Reprocessing plant and nuclear site downwinders. I suggest:

- The investigation of weapons fallout as a determinator of child leukaemia risk though comparing children with leukaemia by cohort year of birth of mother and father with controls.
- The measurement of Sr-90 and U-238 in the teeth/ bone of parents with children with leukaemia vs. controls
- The measurements of Sr-90 and U-238 in bone or teeth of adults with cancer vs. controls.

There are other studies which are important:

- The measurement of the affinity constant of DNA for Strontium, Barium and Uranium in cell cultures and in animals. Concentration partition coefficients for blood Uranium/ Strontium and cellular/ germ cell DNA levels.
- The residual ionisation of an atom after it has decayed e.g. when Sr-90 or U-235 decays where does the recoil energy go? Is it released into the location of the decaying atom as an Auger shower?
- To what extent is photoelectron conversion of natural background gamma or X-rays a cause of local ionisation damage from high Z elements bound to critical organelles like DNA?
- What is the dispersion of radioactive contaminants (e.g.Uranium) in the greater environment?
- What are the local effects of uranium nanoparticles; do these particles bind to DNA or to chromosomes?

3.3 MELODI: Holistic approach

This proposed organisation is a machine to target relevant research funding and to put together all the different disciplines. This could be a good or a bad thing. The obvious political question is what is the point, when national bodies are already funding low dose radiation research? The answer given is that MELODI would ‘better integrate these programmes’ and thus make ‘better use of limited resources’. Cynics might point out that such a structure would be necessary to prevent any single organisation,

one perhaps in a non-nuclear state, from discovering or reporting something awkward. A similar arrangement in the late 1950s (when Strontium was raining from heaven with the weapons fallout, the cause of the current cancer epidemic according to some) resulted in all the radiation research being transferred from the World Health organisation to the International Atomic Energy Agency. It is an extraordinary fact that no research has ever been published showing the affinity of Strontium-90 for DNA even though this was a major political concern in the period and ultimately resulted in the 1963 Test Ban. Why?

On the other hand, taking this development at face value, and plugging in the necessary constraints, bringing in independent scientists and making the arrangement transparent, allows for a resolution of the important issue. A litmus test of probity for me, and for the independent scientists and organisations that I represent, will be the response by the HLEG to this paper and their acceptance of some or all of the suggestions for research topics that I have suggested: a preparedness to discuss these issues and include independent scientists and examine their findings. For if there is a rejection or ignoring of these suggestions, without proper consideration, the MELODI will be seen by us, and by members of the public, as another EU gravy train whose hidden agenda, (operated by the invisible, though funded by the public) is to ensure the continued operation (or perhaps even the relaxed operation) of a system of radiation protection which has resulted in the deaths of countless innocent people. And the enrichment both of uranium and certain individuals and organisations.

4. The history of radiation protection

Ionising radiation and health

4.1 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?

4.2 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 waterthrow unit could encompass either a cupful of boiling water in the face or a bucket of water at room temperature: more information is needed before the health consequence can be assessed. 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 in the test veterans.

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 4.1 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 4.1 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). $1/100^{th}$ 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 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 reality 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. Maybe this was because they were medical practitioners, campaigners for workers' rights and newspapers eager for the human interest angle on any story. Maybe their investigation enjoyed some liberty because the dial painting industry was owned privately, rather than by any government, and because at that time the fate of the free world did not seem to hang on the outcome.

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 must have been emerging everywhere of the effects of atmospheric dispersion of fission products, infant mortality and leukaemia, 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 is forced to leave radiation and health investigations to the latter, 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.

The 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). 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 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.

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. 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 2.2 and the plots (Figs 2.1 and 2.2) 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 4.2 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 4.1 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)

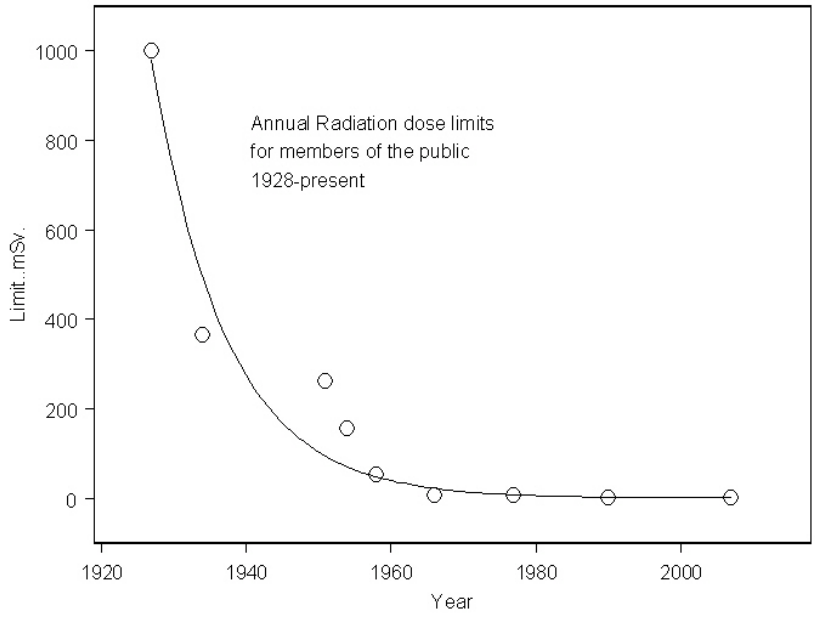
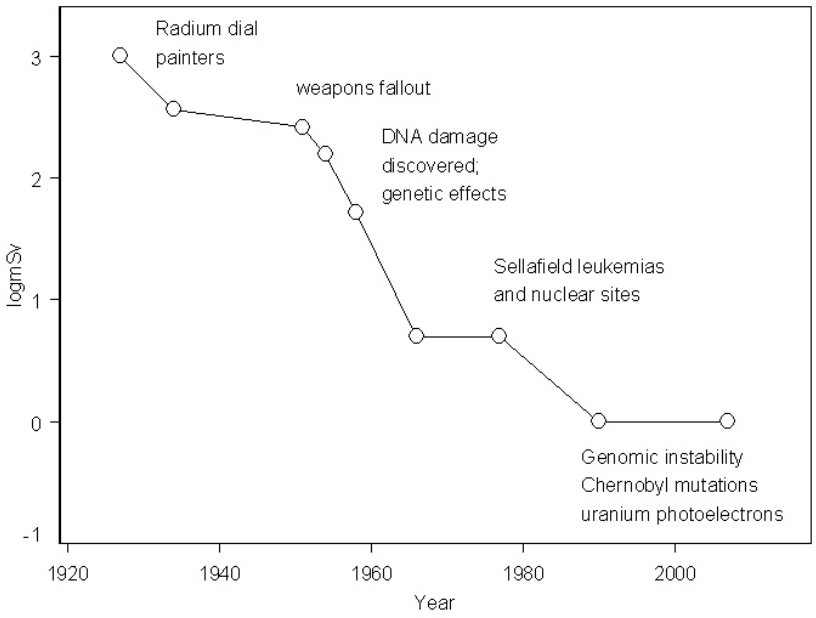


Fig 4.2. Log plot of statutory annual radiation dose limits to members of the public over the radiation age 1920-present (log(mSv))with some radiation exposure events influencing reduction of dose limit. Note that new discoveries in radiobiology and Chernobyl effects since 1985 cannot reduce the limits further as the industry cannot take this.



4.3 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 the same disease. 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.

4.4 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).

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.

4.5 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 of ten' for people who might be exposed for thirty years or more. This represented official unease about the differences between acute external and chronic internal exposure: the conflict between the understanding of physics and that of biology.

There was much argument about the adoption of recommendations from Morgan's group, and the final report did not include the proposals for people likely to receive prolonged exposure

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).

4.6 *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. These issues are key to an understanding of the Test Veterans exposures. 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

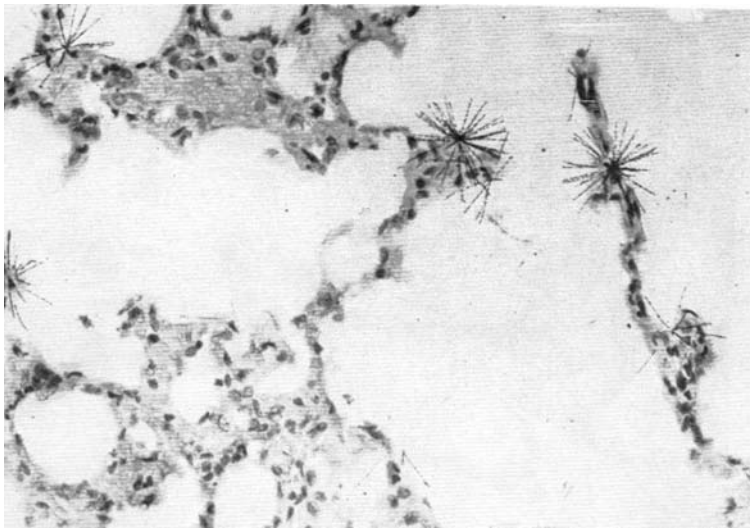
increase their rate of incorporated error. These genetic and genomic mutations are now known to be the main initiation point in the development of cancer and leukemia and also the origin of heritable damage and increases in many illnesses that were not originally thought to be radiation related. It is 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 2.3 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 4.3 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.*

4.7 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 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.

4.8 The recent revolution in radiation risk perception

4.8.1 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 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 <u>that the lymph nodes were known to be the site of leukemias in animal studies</u> and yet accepted calculations of the doses to the lymphatic system from enhanced levels of Uranium from Plutonium that used the ICRP dilution model, in this case diluting the energy into an assumed body organ mass of 11kg. Since dose is Energy divided by Mass this dilution reduced the dose by 1000-fold.

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.

4.8.2 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 = 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.

4.8.3 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.

4.8.4 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 of the Test veterans. 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).

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)

4.8.5 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).

4.8.6 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. It is an interesting and well known fact that the absorption of gamma rays of energy lower than 500keV 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 to 500,000 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 of the A-Bomb veterans will increase the doses to their lymphatic system 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). Since contamination by Uranium was significant on the test sites this makes a big difference to the equivalent doses received from inhaled and ingested dust.

4.9 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)*.

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

on ICRP considerations) had been well described and measured. The only known cause of child leukaemia is ionising radiation. The differences in the levels of leukaemia rates in the exposed cohort and the rate predicted by the ICRP model is greater than 100-fold but varies inversely with the dose.

The CERRIE Majority Report conceded this p88 Table 4A6 where it gives the central estimate of error in the ICRP model for Great Britain as 200X, for Greece as 160x and 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.

4.10 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).

4.11 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.

4.12 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).

Summary of Part 4

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.

It is now clear, with hindsight, that the risk models in operation at the time of the tests were wrong. They failed to recognise the dangerous nature of internal exposure and did not even measure it. They concentrated on external exposure, and as I will show, even then applied limits which were too high and which they just accepted, limits which were clearly cobbled together in order to permit the development of nuclear weapons. When these limits were questioned by many independent scientists, by the media and in several books, those responsible for the safety of the personnel dismissed or ignored the claims.

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 fro 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 leukema, 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. Indeed it is this that has led to the HLEG process and the suggestion to form the MELODI initiative.

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UNSCEAR 2000 United Nations Scientific Committee on the Effects of Atomic Radiation, 2000 report to the General Assembly. Volume II Effects (New York: United Nations).

CURRICULUM VITAE (July 2008)

PERSONAL DETAILS

Name: Dr Christopher Busby

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Date/Place of Birth: 01/09/45, Paignton, Devon UK
Nationality: British

FURTHER/HIGHER EDUCATION

Education: 1966-69 Chemistry, University of London

TRAINING AND QUALIFICATIONS

BSc, PhD, C.Chem, MRSC

Qualifications: in

1969 University of London First Class Honours Special Degree 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: 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

Policy Information Network on Child Health and Environment
PINCHE

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.' **[£24,000]**
1997-2000	Directed research at Green Audit Funded by Irish State to research health effects of Sellafield **[£97,000**]
1997	Appointed UK Representative of European Committee on Radiation Risk (ECRR)
1997	Foundation for children with leukaemia; research on non-ionising radiation **[£15,000].**
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**) [£6000]**
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' **[£24,000**]
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* **[£68,000]**
2004	Leader of Science Policy for(EU) Policy Information Network for Child Health and Environment ***PINCHE*** based in Arnhem, The Netherlands **[€5000]**
2005	3 year research funding by Joseph Rowntree Charitable Trust; Corporate Responsibility in Science and Policy **[£68,000]**
2008	3-year research funding from The Joseph Rowntree Charitable Trust; Corporative Responsibility in Science **[£76,000]**
2008	Appointed Guest Researcher, German Federal Research Laboratories, Julius Kuhn Institute, Braunschweig, Germany
2008	Appointed Visiting Professor, School of Biomedical Sciences, Faculty of Life and Health Sciences, University of Ulster, Coleraine, Northern Ireland

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

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)

Editorial boards (Current):
European Journal of Biology and Bioelectromagnetics

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

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), one MoD pension appeals tribunal 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. The third, a landmark case involving childhood cancer near a nuclear plant in Florida is currently being appealed in the US Supreme Court. 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 also a TENORM radiation case involving Ashland Oil in Martha Kentucky. He is currentluy also expert witness and advisor on the UK Atomic Test veteran litigation in the Royal Courts of Justice.

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. His analysis of the increases in childhood leukaemia in Wales and Scotland following Chernobyl was recently published in the journals Energy and Environment and the International Journal of Radiation Medicine.

From 1997-2000 he was funded by the Irish Government to carry out research into cancer incidence and proximity to the coast. In June 2000 he was invited to present evidence to the Royal Society committee on Depleted Uranium and health, and shortly after this was invited to Iraq to measure DU in the country and relate exposure to health effects which followed the Gulf War. In 2001 he was asked to visit Kosovo to investigate the dispersion of DU using field monitoring equipment. He discovered DU in many areas from analytical measurements made on samples he collected (paid for by the BBC) he showed that there was atmospheric resuspension of DU particles. His work and expertise in the field of environmental health and radioactivity 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. In 2004, he (jointly with two other colleagues) published the *Minority Report of the CERRIE committee* (Sosiumi Press). In 2006 he produced and jointly edited with Prof. Alexey Yablokov of the Russian Academy of Sciences *ECRR2006 Chernobyl 20 Years On*.

Between 2004 and 2006 he was leader of the Science and Policy Interface Group of the EU funded Policy Information Network for Child Health and Environment and organised the discussions and collation of information leading to their final report on the issue which he wrote large parts of. The culmination of this project which involved over 40 scientists and physicians from all major EU countries was the recommendation that as a result of bias in scientific advice to 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.

From 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.
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. 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. Most recently he has conducted a study of the health of people living in the vicinity of the Trawsfynydd Nuclear plant in Wales for HTV and also a study of the veterans of the Porton Down human experiments in the 50s. 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. He is currently an expert advisor on the Test Veterans' litigation and official scientific advisor to the British Nuclear Test Veterans' Association.

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	Vilnius Lithuania; Chernobyl effects
2008	Moscow, Russian Academy of Sciences; A new theory of living systems.

2. PUBLICATIONS AND SUBMITTED PAPERS

PEER REVIEWED PAPERS.

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

Busby C C and Howard CV (2006) **'**Fundamental errors in official epidemiological studies of environmental pollution in Wales' *Journal of Public Health* March 22nd 2006

Busby C and Fucic A (2006) Ionizing Radiation and children's health: PINCHE conclusions *Acta Paediatrica* S 453 81-86

Van den Hazel P, Zuurbier M, Bistrup M L, Busby C, Fucic A, Koppe JG et al (2006) Policy and science in children's health and environment: Recommendations from the PINCHE project. *Acta Paediatrica* S 453 114-119

Koppe JG, Bartonova A, Bolte G, Bistrup ML, Busby C, Butter M et al (2006) Exposure to multiple environmental agents and their effects. *Acta Paediatrica* S 453 106-114

Van den Hazel P, Zuurbier M, Babisch W, Bartonova A, Bistrup M-L, Bolte G, Busby C et al, (2006) 'Today's epidemics in children: possible relations to environmental pollution' *Acta Paediatrica* S 453 18-26

Busby CC (2005) Does uranium contamination amplify natural background radiation dose to the DNA? *European J. Biology and Bioelectromagnetics.* 1 (2) 120-131

Busby CC (2005) Depleted Uranium Weapons, metal particles and radiation dose. *European J. Biology and Bioelectromagnetics.* 1(1) 82-93

Busby CC and Coghill R (2005) Are there enhanced radioactivity levels near high voltage powerlines? *European J. Biology and Bioelectromagnetics.* 1(2) Ch 7.

Busby Chris and Bramhall Richard (2005) Is there an excess of childhood cancer in North Wales on the Menai Strait, Gwynedd? Concerns about the accuracy of analyses carried out by the Wales Cancer Intelligence Unit and those using its data. *European J. Biology and Bioelectromagnetics.* 1(3) 504-526

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