High Risks at Low Doses

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Since a wise man may be wrong, or a hundred men, or several nations and since even human nature, as we think, goes wrong for several centuries on this matter or that, how can we be certain that it occasionally stops going wrong and that in this century it is not mistaken
Montaigne (1533-92): The Essays.

1. Introduction

Thank you for inviting me to this conference. Some of you may recall the last BNES conference that I attended in Stratford-on Avon, I was accompanied by Death himself, who chained himself to the stage in order to remind delegates that what we are discussing as scientists has serious and fatal consequences for ordinary people. Although Death and I were ejected by the police, what we said then is still true today, 'Death is present at all your conferences'.

If you did not already know that low-dose radiation was safe, you would certainly assume, from the available evidence, that it was really dangerous. But how do you know that it is safe? Through the following assumptions:

1. You can express the stress to the organism through a quantity 'dose' which is energy absorbed per unit mass (Joules per kilogram) integrated over kilogram quantities of tissue.
2. At low doses, the target is the cell nucleus which is intercepted by a single ionization track.
3. The biological consequence to the organism is fatal cancer or heritable genetic damage measured as morphological change (cleft palate, Down's syndrome etc.)
4. Cancer or leukemia diagnosed within 5 years of the exposure cannot be a consequence of the exposure.
5. Response is linearly proportional to dose with no threshold.
6. Irradiation from internal isotopes may be assessed in the same way as external irradiation by integrating the decay energy over the mass of the organism or organ. In practice, the organ masses are 'adjusted' to enable averaging of doses over large masses e.g lymphatic system modelled by NRPB and COMARE in the UK as all organs in the abdomen [NRPB R-276, COMARE IV]
7. A study of the survivors of the Hiroshima A-Bomb (who were exposed to a very large single acute dose of external radiation) have expressed a yield of cancer and leukemia over the period of their follow-up from 5 years after the exposure to the present day which is taken to represent the pro-rata cancer yield for all exposures down to the very lowest.

These assumptions have been built into what I will term the 'ICRP model' due to the International Commission on Radiological Protection and now universally adopted as a basis for radiation risk assessment. In this paper I will argue that the consequence of this has been the avoidable deaths of millions of people exposed to low doses from weapons fallout, licensed releases from nuclear sites and other sources and power station accidents.

First I will remind you of the background to this conference. The purpose of our discussion is essentially to establish whether the nuclear industry and the military have been inadvertently killing people. When radioactive materials are released to the environment they end up in the food, the air and water and ultimately living creatures. We know this because we have measured weapons fallout isotopes in the bodies of the most isolated tribes on earth, and we have found plutonium from Sellafield in the teeth of children from across the whole of the UK (Priest et al. 1997). Most scientists now take the view that there is no safe dose: this is a consequence of the discrete nature of ionizing track events. Cells are either hit or not hit, even at the smallest dose. And if they are hit, then there is a finite probability of a mutation in the genetic data on the chromosomes, and a finite chance of this eventually leading to serious or fatal illness.

The nuclear project was originally countenanced politically at a time when scientists were less knowledgeable (the DNA structure was not even known in 1952). It was developed through a period of Cold War, when atomic bombs were thought to be necessary, and dissenting views about the health effects were not permitted

The assumptions of the ICRP model are wrong assumptions. What has emerged in the last thirty years is a body of evidence that the science which is presently used to define the health effects of exposure to internal, novel, man-made radionuclides is in error by a very large amount. The data showing this are now so compelling that the European Parliament passed a resolution in Spring of 2001 asking for a reassessment of the risk model. On July 31st, the UK government set up a new group, the Committee Examining Radiation Risk from Internal Emitters (CERRIE) in order to examine the evidence

and report on the security of the ICRP model. Thus the immediate background to this conference is a large question mark over the belief of the ICRP that low-dose radiation is safe.

2. The ICRP model and Radiation Risk

Scientific knowledge becomes either refined or altered as a consequence of the ability of its models to explain and predict real world data. There have been many cases of scientific models being discarded and replaced following failures to explain observation. Early crude theories in chemistry and physics, like the ICRP model, were based on mathematical averaging of large amounts of material: litres and kilograms whatever could be conveniently experimented upon. Such theories had to be revised.

In the ICRP model, doses are defined as energy per unit mass or $\Delta E/\Delta M$. The quantity employed is that of an organ or larger. One Gray is the absorption of 1 Joule by 1 kilogram of tissue. Very early on, ICRP had to recognize that this model was inadequate since experiments showed that it was the ionization density that was the important factor in cell killing, and so they added a weighting for this to the Gray to give the Sievert. For alpha decays, 1 Gray becomes 20 Sieverts. For internal irradiation there are a number biokinetic and dosimetric concerns. Biokinetic problems include:

- Anisotropy of dose at the sub cell level due to chemical affinity for chromosomes (Sr-90, Pu-239).
- The movement and distribution of radioactive particles smaller than 1μ are not addressed nor are their doses or effects considered.

Dosimetric problems include:

- Organs modelled as 'bags of water' into which energy is uniformly distributed as radiation tracks.
- Isotopes only distinguished in their affinity for organs; microdoses to organelles are not calculated or estimated.
- No consideration given to the state of the cell, its responses to radiation stress, its repair systems or their induction or destruction.
- No consideration given to exposures where individual cells receive high doses due to proximity to internal sequential emitters or internal warm (e.g Uranium) or hot (e.g Plutonium) particles: every dose is averaged in space and time.
- No consideration given to transmutation effects (e.g. Tritium to Helium)

But the main failure of the system used to calculate dose is that the result is an average, in space and in time. The external dose calculation has just been applied to internal dose by averaging all energy of the decays which occur in a bag of water the same size and shape of the organ over its mass. Why is this wrong? Because it is individual cell doses which decide the magnitude of the biological effect, and for internal emitters which are point sources, some cells will receive very high doses whilst other cells receive none. Some groups of cells will receive high doses whilst others will receive none. This is not the case with external irradiation where the source is effectively planar and thus all cells receive the same dose. There are other problems also which must be addressed. And because the theoretical dosimetric model is wrong, it is not possible to use epidemiology of external irradiation to inform us of risks from internal exposure. Table 1 addresses the way in which doses have been correlated with cancer and genetic damage. Here it is clear that scientific method has not been applied properly. Cancer yield in the Hiroshima survivors who suffered almost lethal acute external doses has been linearly extrapolated to internal chronic doses at levels close to background radiation levels. Massive evidence of harm at low dose exposure from internal isotopes has been routinely dismissed by ICRP and its satellites on the basis of the deductive application of this external Hiroshima model.

3. Philosophical arguments

The way in which science has been misused to support the nuclear military project has been addressed at some length elsewhere (Busby 1995, 2000) but I will briefly summarise the arguments here. The scientific method is empirical and inductive (Mill, 1879; Harre, 1985). Induction looks for what is common between the antecedent conditions of a phenomenon and assumes this is the cause or related to the cause. Also, what is different between the conditions under which an effect occurs and those in which it does not defines the cause or is related to the cause. The method also relies on instance confirmation and additive evidence through the accumulation of knowledge, but essentially, if we are to 'do science', we gather together all the cases where a phenomenon occurs and look to some common possible plausible cause. What we cannot do is employ deductive logic and this is just what the ICRP model has done and the risk agencies and advisors to government after them.

The questions of interest are:

(1) What are the health consequences of exposure to external radiation doses at levels below 2mSv, the approximate dose received from natural background?
(2) What are the health consequences of exposure to novel internal radioisotopes at whole organ dose levels below 2mSv?
(3) Is the concept of dose applicable to internal radiation exposures?

Although risks from exposure to high levels of ionising radiation are generally accepted, since they are fairly immediate and graphic, the situation with regard to low-level exposure is curious. There are now two mutually exclusive models describing the health consequences of such exposure. There is

the ICRP one which starts from the Hiroshima survivors' cancer yield and there is the anti-nuclear movement model which begins with the observations of excess cancer and leukemia in populations exposed to low level internal radiation. The latter model arises from an inductive process and considers high levels of cancer and leukaemia in populations living near nuclear sites, especially those where the measurements show that there is contamination from man-made radioisotopes, e.g. reprocessing plants. It includes populations who have been exposed to man-made radioisotopes from global weapons tests, downwinders living near nuclear weapon test sites and those exposed to these materials because of accidents (like the Chernobyl infant leukaemia cohort, which I return to) or because of work in the nuclear industry or military.

Stage	Problem
Assume linear response	1. The basis is that below 1mGy uniform irradiation each cell takes one hit per year. In the low dose range, the number of cells hit linearly increases with dose. This is not true for internal irradiation. 2. A large body of empirical evidence now suggests non linear or biphasic low-dose response.
Use external acute high-dose to model internal chronic low dose.	1. Theoretically invalid since the internal exposures are qualitatively different 2. Empirical evidence points to much higher cancer and leukemia yield at low internal doses. 3. Populations have different radiosensitivity 4. Wartime Japanese survivors are a more healthy sub-group.
Check against internal studies	1. The few internal studies considered are of natural isotopes and high dose exposures. 2. Animal studies are of short lived species and because of cost have usually used high doses.
Ignore non-cancer effects	A whole range of non-cancer effects has been ignored.

Table 1. Problems of ICRP model relating to health consequences of exposure.

Mechanistically, in contrast to the averaging approach of the conventional model, a more accurate biological model considers each type of exposure according to its cellular radiation track structure in space and in time. It is clearly not possible to employ such a model to predict risks from 'radiation dose' to 'populations' but only from microscopically described doses from specific isotopes or particles whose decay fractionations are considered to interact with cells which themselves respond biologically to the insults and may be in various stages of their biological development. The dose-response relationship following from this kind of analysis might be expected to be quite complex.

In deciding between such models we should employ the rules of scientific method. We might argue that the ICRP model is scientifically sound in its application to acute, high dose, external irradiation and its extension to acute, external, low level radiation may be justified on the basis of theory, since the plausibility of the model rests on the idea of uniform density of radiation track events in microscopic tissue volumes.

It cannot be applied to internal exposures. The basis for this conclusion is that such a process involves deductive reasoning. ICRP uses data from one set of conditions, high-level, acute, external exposure to model low-level, chronic, internal exposure. The procedure is scientifically bankrupt, and were it not for political considerations, would have been rejected long ago. On the other hand, it should be clear that the model which begins with the epidemiological observations (which suggest high risk) conforms to all the requirements of the scientific method listed above. Man-made radioisotopes, often in the form of 'hot particles' are common contaminants to the areas near nuclear sites where there are cancer and leukaemia clusters, and to nuclear site and test site downwinders, and to fallout-exposed populations. We must start with the observations, and work back to the causes, not use our prior beliefs to dismiss the observations.

4. Mechanistic arguments

The target for radiation action is probably the nuclear chromosomes and supporting apparatus of the living cell. Thus it is the impact on the cell and its supporting environment that should be the measure of radiation stress. The ICRP model uses units of dose which remove the calculation of cell dose from the cell nucleus to the organ or the whole body. Radiation stress should properly be assessed in terms of tracks or hits to each cell. For low LET radiation, such as X-rays or gamma rays, there are approximately 70 ionizations across a 8μ diameter cell nucleus, equivalent to about 1mGy dose, although individual tracks may exceed this value because of track stochastics. However, within the cell nucleus, low LET radiations can produce dense regions of ionization, and these may be important in the case of isotopes, particularly Auger emitters bound to DNA. For high LET alpha particle tracks of 4MeV, there are about 23000 ionizations and .an absorbed dose of about 400mGy. In one year, natural background external radiation levels, measured as 1mGy to the

whole body, ensure that each cell in the body receives one track or 'hit'.

The health consequence of this to the whole organism is purely a function of the overall probability that the cell will acquire a harmful fixed mutation. If all cells were equal in their sensitivity to radiation this would be merely a function of the number of cells hit and it is this argument that is the basis of the linear dose response relationship assumed by ICRP. But living cells and their cooperative support and communication systems do not conform to the kind of stress/strain relationships exhibited by steel wires.

The damage from a single track of ionizing radiation is either lethal or else causes the cell to enter a DNA repair and cell replication sequence which lasts about 10 hours. This sequence, once begun, is irreversible, and the daughter cells may be exact copies of the parent or, as a result of inefficient DNA repair, may have acquired a fixed mutation. This fixed mutation may be harmless, lethal to the cell, or harmful, in that it may be a critical part of the process that leads to unchecked replication or cancer. Therefore the probability of acquiring either the initial or some later critical step in the path to mutation or final clinical expression of cancer following a radiation dose is seen as a binomial probability calculation involving a sequence of very low probabilities. Assessing the mechanistic basis of such a risk comes down to examining each stage in this process. Clearly any factor which alters each of the probabilities in the chain of events, particularly those affecting the sensitivity of the cell to radiation, will increase the level of risk from the exposure being considered.

For internal point source exposures, there are a number of important cases where the averaging approach breaks down.

4.1 The double hit in space

For external whole body irradiation from gamma rays and X-rays, both human epidemiology at high dose and animal studies support a linear quadratic dose response for cell transformation and certain cancers e.g.leukemia in the intermediate dose range i.e.50-5000mGy. Outcome can be written:

$$\text{Response} = A(\text{dose}) + B(\text{Dose})^2$$

Where A and B are constants.

Kinetic theory suggests, and most now agree, that this result is best interpreted as showing that at low doses, single events dominate the risk but that at higher doses, the quadratic term demonstrates that two correlated events can occur (resulting in enhancement of effect). Because of the 2-stranded complementary nature of DNA, and for other reasons, it is suggested that the two correlated events are breakages of separate DNA strands opposite each other, called a 'double strand break'. Such a process leads inevitably to high risk of a fixed mutation since, unlike a single strand break, there is no complementary copy available to use for a repair. The two correlated hits must occur on the same section of the chromosome, and must occur either together or within the 10-hour repair replication period. The quadratic portion of the dose response curve occurs at high external dose, when the radiation fluence or consequent ionization density is sufficiently high to confer a high probability of two correlated hits occurring in the same cell and same section of chromosome.

At natural background radiation levels of 2mSv, a double hit to a cell within the 10 hour repair replication period is very unlikely. It can easily be calculated from Poisson cumulative probability and based on 500keV events is about 4×10^{-4}, some 40 times higher than the natural heritable genetic damage frequency in human populations.

So we can say that, for external irradiation, correlated double hits do not take place in the low dose range and on this basis a linear dose response is mechanistically justified. However, this is not true for internal irradiation from an immobilised point source where there are sequential decays. Micron and sub-micron diameter 'hot particles' represent such a risk. Tables 2 below shows calculations of hits and dose to local tissues of two common alpha emitting particles that have been introduced into the environment, plutonium oxide (Sellafield, weapons fallout) and ceramic depleted uranium (military use in the Persian Gulf, Bosnia and Kosovo). In both these cases, inhalation and translocation to lymphatics results in high and repetitive local doses to cells. These exposures are modelled by ICRP, after dilution of the decay energy into large masses of tissue, as vanishingly small doses. The response to these small doses is then modelled as a linear response. But strictly, if there are two hits inside the repair replication period, the response will be proportional to the square of the dose. Miller et al (1999) have recently demonstrated that in the case of alpha particles, it is a two-hit process that causes cell transformation rather than a single hit.

Dia. (μ)	PuO2 Hits/d	PuO2 Sv/d	U3O8 Hits/d	U3O8m mSv/d
0.2	8.3	1.2	3.3e-5	4e-3
1	1002	146	7.6e-3	0.9
2	8294	1220	0.03	3.7

Table 2. Hits per day and dose per day to sphere volume defined by the 30μ range of alpha decays from various common environmental particles of Plutonium and Uranium oxides (Busby 2000)

4.2 The double hit in time

The response of cells to exposure in the low dose range has been referred to. Measurements of variation of transformation sensitivity to radiation in different phases of the cell cycle demonstrate that cells in repair replication are hundreds of times more sensitive to radiation than cells in quiescent phase.

(e.g. Sinclair and Morton, 1966). This has been known for most of the radiation age, and is the basis of radiotherapy for cancer, where the rapidly dividing cells are preferentially killed. The first consequence of this is that this variation of sensitivity of cells between the two phases should lead to dose-response relations which reflect two populations. It was pointed out by Elkind [1991] that in the living animal, there are always cells engaged in repair-replication which are therefore highly sensitive to mutation and killing by radiation. It follows that living systems should theoretically exhibit a biphasic dose response resulting from the sequence: high sensitivity mutation-death followed by low sensitivity mutation. Such a dose response is commonly seen in experimental and epidemiological systems e.g. Burlakova's meta-analysis of leukemia studies [Burlakova 1996] the Chernobyl infants [Gibson et al 1988, Petridou et al 1996 , Michaelis et al 1997, Mangano 1997, Busby and Scott Cato 2000, 2001, 2001a) ,Weinberg's minisatellite studies of Chernobyl liquidator children (Weinberg et al 2001) the nuclear industry workers and their children (Draper et al 1997, Roman et al 1999). This has implications for epidemiology since, if this is so, then if a dose-response which is not continuously increasing is found in a radiation health study, this is not evidence that the radiation is not causing the health problem.

Clearly if it is possible to move quiescent (G0) cells into repair-replication by a sub lethal hit and then subsequently hit the cell in repair-replication this two hit pattern represents an enhancement of hazard over the same dose given at once. That this type of exposure represented an enhanced hazard for mutagenesis was first suggested by Busby and Busby in 1987 in what was termed the 'Second Event Theory'. There are two main types of such hazard. The first is immobilized sequential decay isotopes like Sr90/Y90 or Tl-132/I-132 [Busby 1995, 1998] Cox and Edwards of NRPB recently attempted to show that for the case of Sr90, there is almost but not quite the equivalent probability of effecting a double hit from external radiation at background doses. Despite some erroneous assumptions in their paper, they have nevertheless implicitly shown that for more effective Second Event isotopes like Tl-132, or other more efficient second event sequences than Sr-90/Y-90 the probability is very much higher[Cox and Edwards 2000, Busby 2000c].

The second type of Second Event system of interest is the hot particle e.g the immobilized plutonium oxide or uranium oxide (DU) particle [Busby 2000d, 2001b] Table 2 indicates the sizes of particles most likely to effect second event enhancements. It is of interest that the most common size of plutonium oxide particle found in the environment, of diameter 0.2 to 1 μ, is also the size that can most efficiently deliver two hits to a cell in its immediate environment in ten hours and also can pass through the lung into the lymphatic system.

Early formulations of the second event theory assumed that a specific cell, or even cell nucleus had to be sequentially hit. However, recent research has revealed the existence of a cell communication field, whereby a hit to one cell causes predisposition to genetic mutation in nearby cells, the 'bystander effect'. This has implications for the Second Event theory since the target for the second hit now may be any cell in the field and this target is thus is made very much larger. Interestingly, Sonnenschein and Sato [1999] have drawn attention to the fact that cancer cells do not develop when transplanted into a normal cell matrix whereas normal cells are transformed into cancer cells when transferred into cancer tissue. They argue that this suggests a field effect necessary for cancer promotion. Such a field would be more easily disrupted by high local dose from internal immobilised particles and this would mean that such exposure would be more hazardous than the averaging model predicts.[Zhou et al, 2001]

4.3 *Threshold and hormesis*

The idea of a threshold below which radiation damage is mostly repaired seems plausible and there is evidence for the existence of mechanisms whereby repair systems may be induced by 'priming doses' of radiation. Since repair mechanisms are now well accepted, clearly there must be a point where they become overwhelmed, and above this point, in dose terms, the effect will begin to increase at a greater rate than below it. The dose response effects of the complex interplay between damage and repair have been discussed at length by Burlakova (2000)

Furthermore, if, as in all other physiological systems, the repair efficiencies may be increased by acclimatization or induction, it could be argued that low doses of radiation might effect such an increase in repair efficiency, and there is evidence from animal studies that there are such processes.

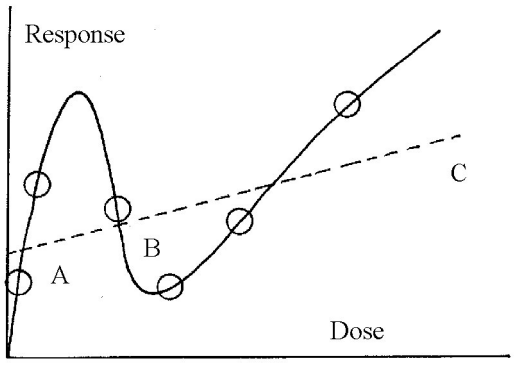

Fig 1 Biphasic dose response due to high and low sensitivity cell populations. Cell killing of high sensitivity population gives rise to apparent 'hormesis'

at B. Dotted line is linear no-threshold assumption drawn through data points with intercept at apparent 'background' incidence .

The question therefore is, why do these systems not remain on the highest level of efficiency all the time? The probable answer is that if the cell were on high alert and repair efficiency was always high, there would be a much higher rate of repair replication all the time, and this on its own would result in greater erosion rates and ultimately an overall loss of efficiency in the organism. The result would be a shorter overall life span of the DNA, with more periods of high sensitivity available for DNA damage from other endogenous or oxidative sources. There is no such thing as a free lunch, and in this case, continuous rates of high repair efficiency (hormesis) would put the organism itself at risk of accelerated ageing. It is of interest that the two-population biphasic dose response appears to show hormesis, so long as the origin point of 'no dose- no effect' is ignored (Fig 1).

Population studies, claiming to show hormesis are usually based on ecological comparisons of people living in high Radon areas and are unpersuasive since they do not take account of social class confounding and other exposures.

Finally, if thresholds exist, then the question becomes, what types of exposure are likely to bypass the systems which give rise to the threshold?

5. Evidence

5.1 Early concerns

Cancer and leukemia have been associated with ionizing radiation since the 1920s. Childhood leukemia incidence has increased steadily since the 1920s. Bramhall has pointed out that this correlates well with the world production of Radium, used extensively for luminous dials up until the 1960s. Fig 2 displays the trend in Radium production (Encyclopaedia Brittanica) against child leukemia mortality.

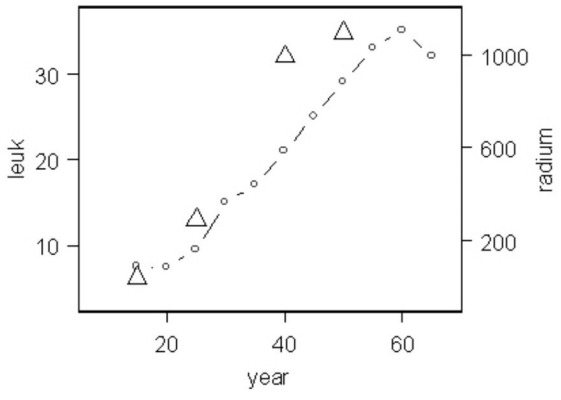

Fig 2 Trends in childhood leukemia mortality (line) rate per 100,000 and world Radium production (grammes).

Doses from such dials (e.g. wristwatches, army compasses) are significantly high. However, other exposure sources also rapidly multiplied over the same period, Uranium production, medical X-rays, including mobile chest X-ray units, etc.

There was a rise in childhood leukemia and cancer following the period of development of the atom bomb and in investigating the cause of this, Alice Stewart (Stewart et al, 1956) showed that a 10mSv dose to the foetus was associated with a 40% increased risk of cancer in childhood. However, she was not able to account for much of the increased rate of childhood cancer.

In 1945, the A-bombs dropped on Hiroshima and Nagasaki caused almost complete destruction of those cities and thousands of deaths due to deterministic effects; the studies of the survivors (which are still on-going) began collecting data in the early 1950s, more than five years after the exposure. Deaths from cancer in this period are thus excluded from the study. First reports published in the 1960s were confusing, with US funded scientists reporting a clear excess of leukemia only (Beebe et al 1963) , although Japanese scientists saw increases in solid tumours also (Harada and Ishida 1963). These dissonances have not since been resolved. In addition, it was discovered that the control group in Hiroshima showed increases in leukemia relative to all-Japan (Busby 1995) suggesting that residual exposure to fallout might be a significant factor. The heritable genetic effects of the A-bomb exposures, assessed as morphological effects in offspring, were reported to be below the background spontaneous rate of about 10^{-5} and although sex ratio effects were clear, these were discounted by the researchers. The bias and inaccuracy of these studies have been pointed out by Padmanabhan (2001).

The period 1955-63 saw increasing contamination of the environment by global weapons tests. The concerns that contamination of milk by the bone and chromosome seeking isotope Strontium-90 might be causing ill health was dismissed by Doll and others on the basis of A-bomb and ankylosing spondylitis data on external irradiation (MRC 1957). The next significant development was the discovery by Ernest Sternglass of increases in infant mortality in line with exposures from global weapons fallout peaking in 1963 (Sternglass 1971). Sternglass showed that the deviation from the temporal trend for infant, perinatal and neonatal mortality in the US and many other countries correlated with fallout contamination doses in the period 1955-70. More recently, Whyte (1992) has followed the trend lines to the mid 1980s and demonstrated that the effect is real. In the UK, over the period 1959-63 which represented the peak fallout period for Sr-90, there was a 14% increase in infant mortality in England and Wales for a parental cumulative dose of about 0.5mSv. The effect was highest in areas of high rainfall like Wales, where the doses were about twice the average England dose

(Busby 1995) and the infant mortality was also higher. Over England and Wales, this represented about 12,000 infant deaths in the five-year period 1959-63. Although nothing was said in public, the government were clearly concerned and ordered a confidential enquiry. The results, not published until 1970 confirmed the effect (DHSS 1970) but could not find a cause (radiation was not considered). Sternglass's findings were savagely attacked by the Atomic Energy Commission but his report came to the attention of President Kennedy who moved rapidly to negotiate and sign the 1963 Atmospheric Test Ban Treaty. These infant mortality effects have never been considered by the ICRP or any of the risk agencies and no reference to infant mortality may be found in any of the reports on the health effects of low-level radiation.

Also ignored by the ICRP was the report in 1963, by Luning et al who demonstrated some astonishing genetic effects in the offspring of male mice injected with Sr-90 (Luning et al. 1963). Foetal deaths occurred in offspring of Sr-90 exposed males but not after Cs-137 or control exposure. The effect continued into the second (F2) generation: this was particularly worrying since the levels of Sr-90 in milk had been rising inexorably with the weapons fallout. Later Smirnova and Lyaginskaya (1969) were to show that the foetal deaths were caused by heart and circulatory system defects. Based on this, I examined infant mortality records in England and Wales and was able to show a high degree of correlation between Sr-90 deposition and infant mortality from congenital heart defects in the period of peak testing (Busby 1995).

Recent correspondence with Alexey Yablokov in Moscow has revealed some interesting data which is relevant to this matter of infant mortality and childhood cancer, and supports the belief that the infant mortality yield following internal exposure to fission spectrum isotopes is between 20-40% per mSv. Two nuclear cities in the Soviet Union. part of the Mayak nuclear reprocessing and fabrication site in the South Urals are Snezhinsk and Ozersk, 100km apart and enjoying exactly the same population types, weather patterns and natural background radiation but different doses. Infant mortality was reported by Petrushinka et al (1999) for the period 1974-1995. Table 3 shows the rates.

Mortality/ 1000	**Ozersk (n =20983)**	**Snezhinsk (n = 11994)**
Average (mSv) effective dose	1.60 (0.05-3.36)	0.98 (0.04-2.04)
Infant mortality /1000 live birth	14.9	11.7
Stillbirths	7.0	5.8

Table 3 Infant mortality and stillbirths at the two Mayak cities Snezhinsk and Ozersk (1974-1995). N is number of children.

Comparison of these two cities gives an excess of 27% infant mortality for a dose differential of 0.6mSv, about twice the rate found from the weapons testing fallout in England and Wales.

5.2 The contemporary cancer epidemic

The infant deaths were caused by mutations due to radiation damage from internal fallout isotopes. The question is, what were the consequences of sub-lethal doses in the children who survived and the adults who were also exposed?

The West is experiencing a cancer epidemic which is not due to an ageing population. Cancer is a genetic disease expressed at the cellular level and is widely believed to be caused by exposure to environmental mutagens. Recent studies of twins show that inherited genetic factors account for less that 20% of most cancers (Lichtenstein et al., 2000). What is also known is that although pre-cancerous conditions are promoted by radiation, the final clinical expression of the main solid cancers following mutation may be twenty years (e.g. lung cancer and smoking). The present age adjusted cancer incidence rate (all malignancies) began to rise in England in the early 1980s and in Wales (where fallout doses were twice as high) in the mid 1970s (ONS). I have examined the relationship between cancer incidence trends in Wales and cumulative Sr-90 exposure 20 years earlier. There is a good correlation between the two, even exhibiting changes in Sr-90 exposure brought about by the partial test ban treaty in 1959 (Busby 1995). The two are compared in Fig.4.

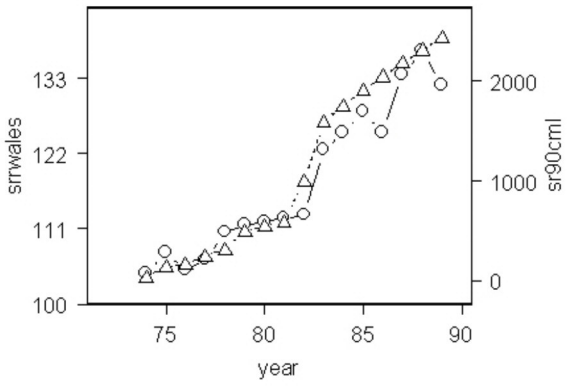

Fig 4. Standardised Incidence Ratio (SIR) all malignancies in Wales 1974-1989 (Wales Cancer Registry), circles and cumulative dose (µSv) from Sr-90 1954-79 (ARC Letcombe Research Laboratory, Annual Reports) displaced by 20 years. (Busby 1995)
Regression equation is:
[SIR] = 0.012 [Sr-90] + 104.5
R-square = 0.96; $p<0.00000$

This is a 12% increase per mSv. The pattern of cancer increase is similar in other countries of northern Europe exposed to weapons fallout.

I have been able to show also that there is evidence that the children born to those who were themselves born over the peak years of global weapons fallout have a higher risk of developing leukemia. The numbers used for this unpublished study were small and obtained from a leukemia charity but the results suggest that a more comprehensive study of this cohort would be instructive. Attempts to obtain the data from ONS have failed.

5.3 Nuclear sites

There are statistically significant childhood leukemia excesses at Sellafield, Dounreay and La Hague. Childhood cancer is high near Sellafield [Beral et al 1994, Viel and Poubel, 1997]. There are many other nuclear site childhood leukemia clusters which you all know. These collectively suggest an error in the Hiroshima risk model of about 300-1000 times based on an aggregate of external and internal dose as calculated by NRPB and COMARE. The most studied of these sites is Sellafield, which has come to be a representative case for the discussion.

Here, there is a large question mark over internal doses from inhalation of radioactive material in the air. The calculations of internal doses made by NRPB and used by COMARE to exonerate the radiation from the leukemia in COMARE IV have not been published nor peer-reviewed [NRPB 1995, COMARE IV1996]

The main nuclear industry childhood leukemia clusters are lieted in Table 4. What these discoveries have in common, besides the correlation between child leukemia and proximity to a radioactive discharge source is that the measured doses have been too small to predict or explain the leukemia by a factor of between 300 and 1000-times if the ICRP model is employed. Rather than looking for errors in the model, this fact has been employed to argue that the leukemia is not caused by the radiation, and other unpersuasive theories have had to be devised. The main contender is population mixing (Kinlen 2000) which was advanced to explain how immune system stresses resulting from exposure to novel infections might lead in rare cases to leukemia . The idea is now being extended to solid tumours (Dickinson et al, 2002) without any plausible biological argument.

One valid question is why not all nuclear sites have significant leukemia or cancer clusters. There are many factors affecting the probability of internal contamination of local people. They include the quantities of materials discharged, the type of discharge e.g. sea, air etc and the local population density. The three European reprocessing sites discharge very large amounts to the sea and this material is transferred back to shore by wind and wave action, with effects on local and more distant coastal populations as I shall outline.

Nuclear Site	Year	ICRP error if radiation
Sellafield	1983	300
Dounreay	1986	300-1000
La Hague	1993	300-1000
Aldermaston	1987	300-2000
Hinkley Point	1988	300-2000
Kruemmel,Ge.	1992	100-1000
Julich, Ge.	1996	300-2000
Barsebaeck, Sw,	1998	300-2000

Table 4. Range of required ICRP model error to account for the results of studies which have established excess leukemia and cancer risk in children living near nuclear sites.

5.4 The Irish Sea

A three year study of cancer in Irish Sea coastal populations in Wales and Ireland by Green Audit supported by the Irish government revealed significant excess risk associated with living close to the sea in areas where there was measured radioactive contamination from Sellafield. Using data from Wales Cancer Registry Cancer for 1974-89, Standardised Incidence Ratios (SIR) were calculated for 100 small areas in Wales and related to covariates which included proximity to the coast, rainfall, radon, plutonium in soil, plutonium in grass, plutonium in air (modelled on Harwell measurements), Carstairs deprivation, Welsh Index of Deprivation and altitude. Temporal and spatial development of variation in risk followed releases from Sellafield and proximity to coastal areas where they concentrated. For all the different cancers studied (with one or two exceptions) there was a sharp and significant excess risk very close to the coast, i.e.within a kilometre or so, particularly in children and particularly in towns in north Wales close to known offshore sediment banks contaminated with radioisotopes. Figs 5 shows the trend for all cancers, breast, lung, leukemia and colon cancer [Busby 2000] For children, brain tumour and leukemia risks in some small coastal areas were extremely high. The trend with distance from the coast was the same as that for sea to land transfer of plutonium particles and also the penetration of salt spray particles inland [Busby 2000]

In a study of Irish National Cancer Registry 1994-1996, small area data was supplied to Green Audit. Standardised Incidence Ratios were calculated for these small areas and aggregates of areas were made into bands by distance from the sea with transects from east to west, west to east and south to north. Results showed significant sea-coast effect on cancer in women of all ages for the east coast but not the south or west coasts. For men, the effect existed in young men but not older men. The trend with distance from the sea on the east coast was similar to that found in Wales. In order to investigate the effect at high spatial resolution, Green Audit and the Irish group STAD undertook a questionnaire study of

Carlingford, Co Louth, cancer cases were mapped in an area where Sellafield pollution was concentrated in offshore sediment. Results showed that the trend in cancer was similar to the trend in sea to land transfer of plutonium. The effect was extremely local, and showed itself in a 100metre band near the contaminated intertidal sediment.

The authors concluded that sea to land transfer of plutonium or other material produced airborne particulate radioisotopic pollution which was inhaled or ingested by those living near coasts where this material was present. Sellafield discharges of plutonium to the Irish Sea become associated with fine particles of silt and sediment and it is the factors which affect the distribution of these particles which decide where the material becomes deposited. The main factor is tidal energy rather than distance from Sellafield and so certain parts of the Irish and Welsh coasts have become contaminated due to the existence there of low tidal energy conditions. The area of coast in the north of Ireland around Dundalk has extremely low tidal energy as do areas in north Wales near the mouth of the Menai Strait near the Town of Bangor. Wave action and wind cause contaminated particles trapped in and measurable in the offshore drying mud flats (like the Lavan Sands in Wales) to be resuspended and driven ashore in rough weather. The trend in plutonium concentration with distance from the sea correlates with the trend in cancer in all the above studies [Busby 2000].

5.5 Coastal nuclear sites

Radiation measurements in estuaries or of coastal offshore and intertidal mud banks confirms that these areas have become depots of radionuclides from reprocessing discharges, nuclear power stations and also weapons fallout, which washes to the sea via rivers. If we believe that internal low dose exposure causes cancer and other diseases, then we would expect to find a sea coast effect in populations living close to such sources, since they will be at greater risk of inhaling the material. Cancer incidence data for small areas exists but is kept secret. However, from 1995, mortality data for wards has become available.

Green Audit has studied cancer mortality 1995-99 near three coastal nuclear sites in the UK, Hinkley Point, Oldbury and Bradwell [Busby Bramhall and Dorfman 2001,Busby , Dorfman and Rowe 2000a, 2000b, 2000c, Busby, Dorfman et al 2001] The purpose was to see if proximity to contaminated mud banks and estuaries was a risk factor for cancer mortality. Results confirmed a statistically significant trend in prostate, lung and all cancers with distancefrom the contaminated mud bank near Hinkley Point. There was a breast cancer mortality cluster of about twice the national expected value in the town closest to the contaminated sediment, Burnham on Sea.

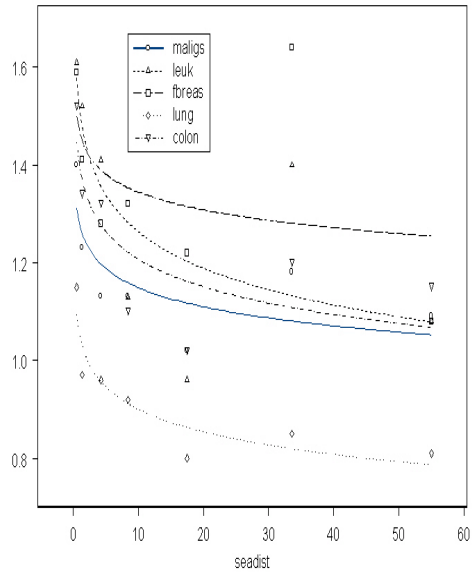

Fig 5 Trend in standardised cancer incidence by distance (km) from the sea of population centroids of six groups of 'Areas of Residence' health authority units in Wales (excluding S.Wales) between 1974-89. Base population England and Wales 1979. All malignancy, leukemia, lung, colon and breast. Exponential fit to data points.

Preliminary work on cancer mortality near Bradwell nuclear power station in Essex identified a significant breast cancer mortality excess for the period 1995-2000. Comparison of Breast cancer in wards along the Blackwater estuary (Bradwell power station) with those along the nearby Crouch Estuary (no nuclear power station) shows a significant difference and highlights the epidemiological problem of using concentric rings to establish the health effects of a point source. Table 5 shows some results for groups of wards. Use of concentric rings shows no effect because this is not the way the radioactivity is distributed [Busby et al 2001a].

There is independent evidence that bears upon the effects of contaminated coasts and estuaries. Alexander et al. [1990] studied childhood leukemia in populations living close to estuaries and the sea. The study hypothesis, which was supported by the results, was that radioactive contamination might be the cause of childhood leukemia and that those living near the sea and estuaries suffered excess risk. Child leukemia excess associated with living near the Hinkley Point site was identified in the late 1980s by workers at Somerset Health Authority [Ewings and Bowie 1988]. Viel's case control study of childhood leukemia near La Hague identified playing on the beach as a risk factor [Viel 1997] Green Audit recently discovered a significant child leukemia

excess on the Welsh coast near Chepstow, on the estuary of the river Wye where there are contaminated mud banks [Busby, Bramhall and Dorfman 2001]

Group	Obs/Expected	SMR (p-value)
Radial < 4km	6/4.84	1.23
Radial 4-17km	198/169	1.17 (0.05)
Blackwater	62/44.9	1.44 (0.003)
Non-Blackwater	133/127	1.05
North Crouch	11/15.4	0.71

Table 5 Breast cancer mortality in five groups near Bradwell power station 1995-1999. The bottom three relate to coastal ward dichotomy. The Crouch has no nuclear power station

5.6 Hot Particles and their local dose effects as explanations: plutonium in humans

Following the discovery in the 1980s of sea to land transfer there have been some studies which indicate that plutonium from this source is contaminating people. These include the studies of sheep droppings which show the trend in plutonium from the west to east coasts across the country, the local plutonium in air coastal studies, the report of plutonium in children's teeth by distance from Sellafield and the autopsy studies which show that the tracheobronchial lymph nodes are where the material concentrates [Popplewell 1986, Priest at al 1997, Eakins and Lally 1984, Eakins et al 1984]

Plutonium and Uranium Oxides exist in the environment as long-lived, virtually insoluble micron sized particles, capable of suspension in air and of travelling long distances. There was early evidence of plutonium dispersion from Sellafield in the various Cawse and Horrill grassland and soil surveys [Cawse et al 1988, Cawse and Horrill 1986] Further, there has been evidence from the AWE reports of high activity particles (up to 60,000Bq/kg of beta, 2000Bq/kg alpha) being trapped in passive airshade filters in Berkshire near Reading and Aldermaston [e.g AWE 1992].

These indications, taken with the cancer epidemiology from the coastal populations suggest that the distribution and dispersion of radioactive particles in the environment may result in local areas of concentration and excess cancer risk.

5.7 Unequivocal evidence

(1) There is unequivocal evidence for the existence of an error of 100 to 500-fold in the ICRP model, capable of supporting the contention that the nuclear site clusters and other evidence of serious harm from low level internal radiation exposure are real causal effects. This is the set of studies showing increases in infant leukemia after Chernobyl in five countries: Greece, Germany, US, Scotland and Wales[Gibson et al 1988, Petridou et al 1996, Mangano 1997, Michaelis et al 1997, Busby and Scott Cato , 2000, 2001].

We compared the predictions of the NRPB/ ICRP leukemia model with the observed cases based on estimates of the exposure published by NRPB and WHO [Busby and Scott Cato 200]. Results showed a 100-fold error based on the first year of life, which would extend to more than 500-fold if the risk continued in the cohort. Because the effect occurred with statistical significance in five countries, the overall significance was extremely high ($p<0.0000000001$) Thus it was not a chance finding. Because the cohort was so tightly specified, those who were *in utero* over the period of the internal contamination, the effect could only be due to the radiation exposure.

The results across the different exposure groups and countries showed a biphasic dose response relation. The observations demonstrate unequivocally that the ICRP risk model for this exposure are invalid. This observation was communicated with COMARE in 1999 but they rejected it as unlikely. Following publication of the report in *Energy and Environment* and its invited presentation at the WHO conference on Chernobyl in Kiev, NRPB wrote a criticism of the finding and its significance. This criticism, written by Colin Muirhead and defended by Roger Clark in letters to Green Audit, is based on the defence that 'it is not possible to say that the children who developed leukemia had not received a much larger dose' than that provided by NRPB which the calculation of expected numbers was based upon. This is a critical judgement since it undermines the whole area of radiation risk assessment. For how is it possible to maintain the present linear risk model for cancer causation by radiation but also say, if it fails to predict the correct number of cancers, that this anomalous result is not a failure of the risk models but due to the cases being caused because they received a higher than average dose?

(2) Minisatellite mutations following exposures

The application of the minisatellite DNA test to exposed populations has revealed that there is unequivocal evidence of significant mutation on the minisatellite DNA in offspring of people exposed to low level internal radiation from the Chernobyl accident. The first study by Dubrova et al demonstrated a doubling of mutation in children living in contaminated territories with average background exposure of 2mSv, but it was criticized for poor controls. The latest study by Weinberg et al uses a sibling control and shows a 600-fold error in the perception of germline mutation risk from ICRP models [Weinberg et al 2001]. Such mutation in swallows have been shown to be associated with phenotypic changes and so have to be taken seriously as a measure of the damage caused by low level radiation [Ellegren and Lundgren 1997]. They are

certainly disturbing the equilibrium of those scientists who have consistently supported the ICRP risk model. Bryn Bridges, Chair of COMARE (who regularly find that the Sellafield and other leukemia clusters are not caused by low-dose radiation) has asked if it is time for a paradigm shift. (Bridges, 2002).

In general, technological advances in the last ten years have enabled analysis of objective biological indicators of harm from low dose radiation. The minisatellite DNA mutation tests are beginning to reveal evidence of both significant harm from low dose radiation and most recently transgenerational effects (Dubrova et al. 2002). A range of other techniques are becoming available. For example, chromosome aberration tests are showing significant radiation related problems in those living in Chernobyl affected territories of the ex-Soviet Union (Burlakova et al., 1995, 2000). Chromosome tests by Albrecht Schott of Berlin (unpublished) have recently revealed the existence of a seven-fold increase in aberrations in a group of Gulf War veterans exposed to internal depleted uranium dust. Levels compare with aberrations in Chernobyl workers with external recorded doses of 500mSv (some results reported in Busby 2001).

5.8 Arguments about Chernobyl effects

Nowhere does the argument about low dose radiation effects seem more bizarre or cruel than in the analysis of Chernobyl effects in the worst affected territories. Here, people are forced to live in dangerously contaminated regions and watch their children fall ill or die whilst being told that the radiation levels are safe on the basis of unscientific armchair deductions and advice form the International Atomic Energy Agency (IAEA). This may be because, in the area of the health effects of radiation, World Health Organization, is subordinate to the IAEA through a 1959 agreement. At the 2001 Kiev conference of Chernobyl and health there was fierce debate with contributions showing major health problems and others showing no health problems. However, when the time came to produce the final conference conclusion, the organizing committee provided a statement which formally agreed that the conference showed that the exposures had had no effect and only the highest dose groups should now be studied. There was an uproar, and this statement was altered following further acrimonious debate from the floor to one where the risk model was to be questioned. However, the final conference resolution, now published, has been restored to an emasculated version of the final agreed form of words (which I provided). Fortunately, the whole shoddy affair was recorded by a Swiss TV cameraman and has been now produced on film by Swiss TV [Atomic Lies, 2002].

Close examination of health statistics in many countries where contamination from Chernobyl occurred shows clear effects. This is true for child leukemia in Wales and Scotland. It is true for infant mortality, stillbirths, congenital malformation and cancer in adults in many countries where we can get raw data. But by the time the data is filtered through the official level, the effects all miraculously disappear. In affected countries like Belarus, there are horrifying increases in general morbidity, blamed by the WHO on poor nutrition and radiophobia. Yablokov has reported that in the worst affected territories, only one in five children is born healthy. The governments are colluding with this airbrushing. Recently, Bandashevsky showed a significant correlation between cardiac damage in children and measured Cs-137 concentration [Bandashevsky 2000]. He was arrested on a bogus charge and sent to gaol. The results of the Chernobyl exposures can be seen in all health indicators and in objective biological measurements. Examine the results, for example, in the collections by Burlakova [Burlakova, 1996, 2000] and Imanaka [Imanaka 1998]. Goncharova, the eminent geneticist has found serious genetic damage in fish and in small mammals. Voles develop genetic defects over twenty generations. (Goncharova 2000). Wherever we look closely there are effects: in Poland, for example, there were increases in tumours, infant mortality, congenital defects, miscarriages and Downs syndrome [1992].

6. Conclusion

With the 17th Century mystic Comenius who perhaps foresaw the releases of man-made radioactivity to the environment I conclude:

They have given wings to Death, so that it could, in an instant, travel everywhere, both near and far.

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