Health Risks following Exposure to Aerosols produced by the use of Depleted Uranium Weapons

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Presentation to *Res Publica* conference on DU
Prague, Czech Republic, 24 and 25th Nov 2001

Occasional Paper 2001/12
Aberystwyth: Green Audit
November 2001

1. Background

Veterans of the recent war in Iraq have been suffering from a mysterious ailment called 'Gulf War Syndrome' which has all the indications of a consequence of chemical or radiation poisoning. The spectrum of ill health effects in this group of people suggesting exposure to a mutagen, or carcinogen. In addition, the population of Iraq in the areas where DU weapons were used seems to be suffering a similar fate, but in these territories also there have been reported increases in childhood cancer and malformation rates, again indication of a mutagen source.

It is now generally conceded that about 350 tonnes of Depleted Uranium was used in the war and that sub-micron diameter oxide particles became dispersed in the areas that were bombed or strafed. Uranium is known to be a radiation hazard, and radiation exposure is a known cause of all the effects observed in the veterans and the children of Iraq. These facts taken together suggest that the exposure to DU may be the main cause or one of the causes of the observed effects

More recently, there have been reports of increases in ill health, particularly leukaemia, in soldiers involved in peacekeeping duties in Kosovo, where about 10 tons of DU ammunition was used by the NATO forces. Investigations by UNEP and others confirmed the widespread existence of DU in soil and water in Kosovo and recent measurements made on the population confirm the existence of DU in urine specimens. There are also reports of sharp and anomalous increases in cancer rates in Bosnia, where DU was also used in the mid-1990s.

The argument marshalled against the suggestion that radioactive exposure to DU is the cause of all these health problems is that Depleted Uranium is not considered to be a serious radiological hazard since it is so weakly radioactive. This is owing to its long half-life and the fact that its alpha emissions have a very short range. It is argued that on the basis of the 'known relationship between radiation dose and subsequent cancer' the exposure to DU suffered by the Gulf War veterans, the Iraqi children, or the Kosovo peacekeepers cannot have been sufficiently high to be a cause of cancer or mutagenic illness. This is familiar territory to any scientist who has looked into the area of the health risks of exposure to man-made radioactivity. Similar arguments are routinely advanced to exonerate radiation as a cause of childhood cancer and leukaemia clusters near nuclear reprocessing sites like Sellafield, Dounreay and La Hague, increases in infant mortality and cancer in populations exposed to weapons fallout and fallout from nuclear accidents, cancer increases in nuclear workers and their children, and whole ranges of observations and experience which to most people seem clear evidence of causation. For these people, therefore, the questions about Depleted Uranium, are the latest in a long series of questions about the health effects of low level radiation [Busby, 1995].

In this presentation, I will begin showing that recent incontrovertible evidence defines the existence of a very large error in the presently accepted model for the health consequences of exposure to low-level man-made radioactivity, and that therefore this same model which underpins the presently accepted assessment of harm from Depleted Uranium, is likely to be similarly flawed. In addressing this issue, I will ask if Scientific Method has been properly used in the historical assessment of risk from exposure to low-level radiation and suggest that it has not.

In order to explain how DU may be having a serious impact on health at what is conventionally seen as low dose, the cellular and molecular basis of radiation action must be examined. I will outline two possible mechanism which address the particular type of radiation exposure involved with Depleted Uranium and argue that these types of exposure cannot be modelled by the system of assessment of risk used by ICRP and based on the external irradiation which occurred at Hiroshima. I will briefly review some of the evidence from the studies of my group which support this new model and show how it applies to the Depleted Uranium controversy. Finally I will review recent evidence on the dispersion of battlefield DU and its consequences.

2. Depleted Uranium: Properties

Depleted Uranium is a by product of the nuclear industry where the fissile isotope U-235 in natural Uranium ore is concentrated to produce reactor fuel consisting of 'enriched Uranium'. The isotope discarded by this process is Uranium 238 which is generally classed by the risk agencies as a low radiation hazard material owing to its long half life (4.47 x 10^9 years) and its weak gamma emission of 48keV. However, it is an alpha emitter and thus poses an ingestion risk owing to the high ionisation density of alpha tracks and their high biological effectiveness in inducing mutation. In addition, there is a major risk from the beta-emitting daughter isotopes Thorium234 (beta, 0.26MeV, half life 24 days) and Protoactinium-234 (beta; 0.23MeV, half life 6.75 hours) which decay through one another to Uranium-234, also an alpha emitter with a half life of 2.47 x 10^5 years. The overall activity of Uranium 238 therefore increases as soon as it is produced due to ingrowth of the beta daughters and by 30 weeks these are in total secular equilibrium. The activities per kilogram are given in Table 1 below.

Weeks	U-238 (α,γ)	Th-234 (β)	Pa-234 (β)	U-234 (α,γ)
0	12.43	0	0	0
5	12.43	7.89	7.84	0.001
10	12.43	10.77	10.75	0.004
20	12.43	12.21	12.21	0.01
30	12.43	12.4	12.4	0.017

Table 1. Increasing specific activity (MBq/kg) of DU due to ingrowth of daughters.

Over centuries, the specific activity of U-234 should be the same as that of the parent U-238, and thus the environmental concentrations of these isotopes is generally the same if the source is natural. The specific total activity is thus about 37MBq/Kg. It should be pointed out that DU material recently found in battlefields in Europe contains small quantities of isotopes of Plutonium, Neptunium and other fission products: thus the source of this DU is refinement of nuclear reactor waste. However, the quantities are very small and are not considered by the authorities to be of serious radiological significance.

Owing to the high density of Uranium, (19g.cm^{-3} metal and 10.96 g.cm^{-3} for the dioxide) and the fact that the metal is pyrophoric (burns in air) the substance is used in the manufacture of armour piercing shells, missile nose cones and penetrators and certain ballast materials in some aircraft (e.g. helicopter rotors, commercial aircraft counterweights). A single Abrams 120mm tank shell contains about 3kg of DU (111MBq of radioactivity) and there is 275g in a 30mm GAU3A A-10 Thunderbolt Gatling Gun round.

The military penetrators explode on impact with hard targets with about 80% conversion to micron diameter Uranium Oxide particles of a 'ceramic' nature. These particles are highly mobile and extremely long lived in the environment, owing to the very high degree of insolubility of Uranium Oxides UO_2 and U_3O_8. They can be inhaled and the sub-micron diameter particles are translocated from the lung to the lymphatic system, building up in the tracheobronchial lymph nodes and potentially able to circulate everywhere in the body. Alpha and beta disintegrations from these particles cause very high and repetitive doses to cells local to the range of the disintegration i.e. about 30microns for the alpha and 450 microns for the beta tracks.

3. Errors in the ICRP low level radiation risk model

The model used by the risk agencies and the military to predict the health consequences of such exposure is that of the ICRP and is based on the cancer yield of the Hiroshima bomb. The group of survivors of this single large acute irradiation exposure (in which many people were killed) have been collected into the Life Span Study or LSS and their cancer rates have been compared with controls from the same town who were shielded or outside the town at the time of the bomb. The cancer yield in this LSS cohort has been used as a basis for predicting cancer risk and other health detriments for all types of radiation exposure. It has been assumed that the relationship between dose and cancer yield is linear and so low levels of exposure have been assumed to carry no significant risk on this basis.

This approach has been criticised extensively, and considerable evidence has become available in the last twenty years to suggest that the increases in cancer and leukaemia near nuclear sites are examples of a failure of the model to adequately address risk from internal radiation. However these arguments have always been countered by the risk agencies on the basis that other possible causes for the observed phenomena exist. However, very recently, two unequivocal pieces of evidence have defined errors of between 100 and 2000-fold in the ICRP risk models as applied to internal radiation risk. This evidence has forced the UK government to set up a new committee to examine the situation and assess the failures of the ICRP risk model applied to internal radiation exposure [CERRIE, 2001].

In addition, the European Parliament has called for a similar process to be undertaken by the European Commission [EU,2001], and the recent WHO conference on Chernobyl in Kiev in 2001 came to a similar conclusion [WHO 2001].

3.1 The Chernobyl Infants

Following the Chernobyl accident in 1986, in five different countries, the cohort of children who were exposed in their mother's womb to radioisotopes from the releases suffered an excess risk of developing leukaemia in their first year of life. This 'infant leukaemia' cohort effect was first reported in Scotland [Gibson et al, 1988], and then in Greece [Petridou et al, 1996], in the United States [Mangano, 1997] and in Germany [Michaelis, et al. 1997]. We first reported increases in childhood leukaemia in Wales and Scotland following the Chernobyl accident in 1996 [Bramhall, 1996] but more recently examined the specific infant leukaemia cohort in Wales and Scotland [Busby and Scott Cato 2000].

Unlike the earlier researchers, who merely showed the existence of a significant rise in infant leukaemia, we decided to examine the relationship between the observed numbers of cases and those predicted by the present radiation risk model. This was an invaluable opportunity since the specificity of the cohort enabled us to argue that the effect could only be a consequence of the exposure to the Chernobyl fallout. There could be no alternative explanation, like the 'population mixing hypothesis' advanced to explain away the Sellafield childhood leukaemia cluster. However implausible such theories may be, they have acquired popularity, and their proponents status, as a consequence of their utility to the nuclear lobby. However, population mixing may not occur at Sellafield but it cannot occur in the womb.

Because the National Radiological Protection Board had measured and assessed the doses to the populations of Wales and Scotland and because they themselves had also published risk factors for radiogenic leukaemia based on ICRP models it was a simple matter to compare their predictions with the observations and test the contemporary risk model. The method simply assumed that infants born in the periods 1980-85 and 1990-92 were unexposed, and defined the Poisson expectation of numbers of infant leukaemia cases in the children who were *in utero* over the 18 month period following the Chernobyl fallout. This 18 month period was chosen because it was shown that the *in utero* dose was due to radioactive isotopes which were ingested or inhaled by the mothers and that whole-body monitoring had shown that this material remained in the bodies of the mothers until Spring 1987 because silage cut in the Summer of 1986 had been stored and fed to the cattle in the following winter. The result was startling. First, there was a statistically significant 3.8-fold excess of infant leukaemia in the combined Wales and Scotland cohort (p = 0.0002). Second, the leukaemia yield in the exposed 'in utero' cohort was about 100 times the yield predicted by the model. Table 2 compares the effect in the three main studies. In passing it should be noted that this number, 100, is very close to the error required to explain the Sellafield childhood leukaemia cluster.

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

And since the World Health Organisation has given approximate exposure levels in Greece, Germany and the United States, it was also possible to examine the leukaemia yield in the infant 'exposed cohort' reported by the several other studies and establish a dose response relationship. This is shown in Fig 1. It is a curious shape and goes up, down and up again, and this shape should be noted. I will return to it below.

3.2 Minisatellite DNA in Chernobyl children

Since the discovery of the DNA minisatellite characterisation method, 'DNA testing' it has been increasingly applied to those who were exposed to the fallout from the Chernobyl accident. In a series of papers, Dubrova et al. showed an association between exposure of children in Belarus [Dubrova et al.1997] found a doubling in the mutation rate in children from the high exposure territories of Belarus compared with controls from low exposure territories. This discovery was astonishing to those who adhered to the ICRP risk model for genetic mutation since this was based on the belief that the Hiroshima exposures, which were hundreds of times higher than the average dose in Belarus, had produced no genetic effect on any offspring of those exposed. A doubling of the mutation rate thus pointed to an error of some 1×10^5 . Others pointed out that even if the minisatellite DNA was mutated, this was not an effect which had any significance since there were no phenotypical changes associated with minisatellite DNA. Shortly after this, it was reported that barn swallows which migrated to Belarus had similar changes in their minisatellite DNA and these were associated with plumage pattern alterations which destroyed their camouflage and thus might be harmful. [Ellegren et al. 1997]

The question of proper controls and the reality of the effect was answered very recently in an elegant study by Weinberg et al.[2001]. They examined minisatellite DNA changes at various loci in the offspring of the Chernobyl 'liquidators' who were born after the accident and compared their DNA to their siblings born before the accident. Results showed that there was a significant difference of up to seven-fold. The dose response relationship appeared to be biphasic. Based on the natural mutation rate in the minisatellite DNA, the finding showed an error in the ICRP risk factor for mutation of between and 700-2000 fold. This series of studies thus demonstrates finally and unequivocally that the ICRP risk model for internal exposure is wildly inaccurate.

I must ask how it is that some fifty years after the atom bomb, and following a huge amount of research into the subject, we can have discovered such a huge error in the science of radiation risk. To understand the answer, we must look at the scientific method a little more closely.

4. Radiation Risk and Scientific Method

The classical exposition of the scientific, or inductive method (originally due to William of Occam) is what is now called Mill's Canons, the two most important of which are:

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

In addition, the method relies upon the *Principle of Accumulation* which states that scientific knowledge grows additively by the discovery of independent laws, and the *Principle of Instance Confirmation*, that the degree of belief in the truth of a law is proportional to the number of favourable instances of the law.

Finally to the methods of inductive reasoning we should add considerations of *plausibility of mechanism.* These are the basic methods of science [Mill, 1879; Harre, 1985; Papineau, 1996]

Let us first define our question. It is this. What are the health consequences of exposure to novel internal radioisotopes at whole organ dose levels below 2mSv? Because we are looking at battlefield DU, we should add that in this case, although the element is 'natural', the exposure is novel, and due to internal sub-micron Uranium Oxide particles embedded in tissue.

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 exposure to low-level radiation. There is a nuclear establishment one, which is that which is presently used to set legislation on exposures and argue that DU is safe, and a radical one, which is espoused by the anti-nuclear movement and its associated scientists. I show these two models schematically in Fig 2.

The two models arise from two different scientific methods. The conventional model is a physics-based one because it was developed by physicists prior to the discovery of DNA. Like all such models it is mathematical, reductionist and simplistic, but because of this is of great descriptive utility. Its quantities, dose, are average energy per unit volume or dE/dV and in its application, the volumes used are greater than 1kg. Thus it would not distinguish between the average energy transferred to a person warming themselves in front of a fire and a person eating a red hot coal. In its application to the problem at hand, the internal, low-level, isotopic or particulate exposure, it has been used entirely deductively. The basis of this application is that the cancer and leukaemia yield has been determined following the external acute high-dose irradiation by gamma rays of a large number of Japanese inhabitants of the town of Hiroshima. Following this, arguments based on averaging have been used (quite spuriously) to maintain that there is a simple linear relationship (in the low-dose region) between dose and cancer yield. This Linear No Threshold (LNT) assumption enables easy calculations to be made of the cancer yield of any given external irradiation.

By comparison, the radical model shown in Fig.2 arises from an inductive process. There have been many observations of anomalously 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. In addition, 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) or because of work in the nuclear industry or military. A review of these findings is available [Busby, 1995] and a more recent literature review of studies showing these effects if published by the Low Level Radiation Campaign [LLRC, 2000]. In addition, the radical model is based on biological considerations and considers each type of exposure according to its cellular radiation track structure in space and in time. It is not, therefore, possible to employ this 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.

These models are mutually exclusive: which one is correct? What considerations can we use to choose?

The answer is that the conventional LNT model must be rejected because it is not scientific. Its conclusions are based on deductive reasoning. It falsely uses data from one set of conditions, high-level, acute, external exposure to model low-level, chronic, internal exposure. It 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 radical model 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 the downwinders, and to the fallout-exposed populations. This satisfies the *Canon of Agreement*. The contingency analysis tables with control populations for such studies show that the *Canon of Difference* is also satisfied: people living in more remote regions than the downwinders show lower levels of illness. We must by now also have some faith in a *Principle of Instance Confirmation*, since so many studies have shown that increases in cancer and leukaemia follow these exposure regimes at low dose. Indeed, the Gulf War Syndrome, might be considered as such an instance confirmation. We are left only with 'Plausibility of Mechanism', which will be addressed briefly below.

5. Mechanistic Considerations

Averaging Dose

I want to look more closely at the averaging model and its predictions at low dose. It is essentially what used to be called a colligative model: the dE/dM formulation of dose requires that energy transferred from absorption of the consequence of a radioactive disintegration is averaged over the target site, usually the whole body or organ. Whatever lip service is made to considerations of what is now called 'microdosimetry', close examination of calculations done to establish risk near nuclear sites shows this to be the case. The documents NRPB R-276, *Risk of Leukaemia and other Cancers in Seascale from All Sources of Radiation* published in 1995 is a good example. In this document, doses to the lymphatic system were calculated by modelling it as 'liver, lung, kidney, spleen, pancreas, uterus and intestines'. A physiologist would not recognise this list as the 'lymphatic system', so why was it used? The answer is that breathing introduces the particles of plutonium that exist in the air near Sellafield into the lungs of the children who live there. From the lungs, these particles are scavenged to the two small tracheobronchial lymph nodes which have a combined mass of perhaps one gram. If NRPB had divided dE by 1 gram, the resultant dose to this part of the lymphatic system would have been extremely high. Given that this organ has been identified as a source of lymphoma and leukaemia in animals, this sounds very like the cause of the Sellafield leukaemia cluster. But dilution of the plutonium decay energies into the whole mass of guts used for dM reduces the 'dose' to an acceptable small level. This process, incidentally, is very relevant to the DU exposures.

Figure 3 shows a phantom used by ICRP to calculate doses from external radiation fields. This is the model that is presently used to calculate internal doses. Of course, in the low dose region, cells are either hit or not hit, so the cell dose is very different from the tissue dose. Nevertheless, the model is valid as a means of establishing a quantity, 'dose' which can be correlated with some health consequence like cancer, so long as each cell in the body, or target region, has an equivalent probability of being hit (or more properly intercepted by a track). Dudley Goodhead has written of the low-dose region [Goodhead, 1988]:

Most situations of practical interest are characterised by cells receiving occasional single tracks well separated in time from any other tracks which may impinge on the same cell. From Natural Background, there is, on average, about one track per year through each cell nucleus. Therefore it is highly unlikely that there will be multiple tracks in short times (< 1 day) over which repair of radiation induced damage within cells is usually observed to take place.

It is these (essentially external irradiation) considerations that enable the model to assume the linear dose response relationship that is the basis for radiation risk. But there are two situations of practical interest that Goodhead's arguments do not address. The first is that a cell's response to radiation damage is not constant over its lifespan: cells are very sensitive to radiation when they are in their repair and replication cycle. The second is that for internal radionuclide decays, either from sequential emitters or from 'hot particles' the microscopic local radiation flux, or energy density, may be very high, even though the average dose may be low. For internal exposure, these are common situations. Here the concept of 'dose' no longer applies and the conventional model breaks down. I will address these in turn.

Cellular responses to radiation: the Burlakova dose response

It has been known from almost the beginning of the radiation age that rapidly replicating cells are more sensitive to radiation damage [Bergonie and Tribondeau, 1906]. Indeed, this is the basis of radiotherapy for cancer where it is the rapidly proliferating cancer cells that are preferentially destroyed. Most cells in a living organism are in a non-replication mode, sometimes labelled G0. These cells are contributing to the organism as part of the normal living process and do not need to replicate unless there is some signal requiring this, perhaps because of tissue growth, damage or senescence. Throughout the growth and lifespan of individual organisms, there is a constant need for cellular replication, and therefore there are always some small proportion of cells which will be replicating: the magnitude will naturally depend upon the type of cell. When cells receive the signal to move out of stasis or G0, they undertake a fixed sequence of DNA repair and replication, labelled G0-G1-S-G2-M, with various identifiable check points through the sequence which ends in replication M or Mitosis. The period of the repair replication sequence is about 10 to 15 hours and the sensitivity of replicating cells to damage including fixed mutation is extremely high at some points during this sequence. This has been known for some time: Fig 4 shows the results of early experiments on Chinese hamster cells indicating up to 600-fold variation in the cell radiation sensitivity over the whole cycle. [Morton and Sinclair, 1966] If we display this response variation on a scale that shows the normal cell lifespan in the organism, rather than just over the cell cycle *in vitro,* the window of opportunity for cell mutation at high sensitivity becomes apparent Fig 5.

So the picture of isotropic dose to equivalent cells, the 'bag of water' phantom model outlined by Goodhead has to be reviewed. Perhaps 1 percent of these cells are actively dividing and are in repair replication sequences that we will assume, for argument, are 600 times more sensitive to being 'hit' by a track. What would we expect the dose-response to look like? Well as the dose was increased from zero, the sensitive cells would begin to be damaged and a proportion of these hits would results in fixing a mutation and increasing the possibility of cancer. As the dose increased further, eventually this rise in response would peak as these sensitive cells were killed. The mutation yield would then begin to fall. However, at some point, the insensitive G0 cells would begin to be damaged and the whole process would begin again, with a rise in cancer. Ultimately there would be a second fall, but this level of exposure would probably result in the death of the organism (although such considerations have been used to explain an observed fall-off in effect from alpha emitters at high dose). So the dose response would look like that in Fig 6. This type of response was shown to occur in several experiments by Burlakova, although she gave a different explanation for it, involving a combination of increasing damage and induced repair curves.

She showed that such an effect can be seen by plotting the results of a large number of separate radiation and leukaemia studies, a graph reproduced in Fig 7.

The results of animal studies on beagle dogs and mice also show these biphasic effects in the low-dose region [Busby, 1995] . Note that this type of curve is seen in the Chernobyl infant studies collected together in Fig 1.

The Second Event Theory

There is large variation in sensitivity over the cell lifespan. Although naturally dividing cells may accidentally receive a 'hit', this process can be modelled by averaging over large masses of tissue, even if the dose response curve is not linear, as thought. However, unplanned cell division, preceded by DNA repair can be forced by a sub-lethal damaging radiation track: this is one of the signals which push the cell out of G0 into the repair replication sequence. It follows that two hits, separated by about eight hours, can generate a high sensitivity cell and then hit this same cell a second time in its sensitive phase. This idea, the 'Second Event Theory' is described and supporting evidence advanced in Busby 1995 and its mathematical description has been approached slightly differently in Busby 2000. It has been the subject of some dispute by NRPB (Cox and Edwards, 2000, Busby, 2000a)

Very recently, developments in micro techniques have enabled some new evidence that supports the two hit idea to emerge. Miller et al., [1999] in a consideration of Radon exposure risks, have been able to show that the measured oncogenicity from exactly one alpha particle hit per cell is significantly lower than for a Poisson distributed mean of one alpha particle hit per cell. The authors argue that this implies that cells traversed by two alpha particles or more contribute most of the risk of mutation, i.e. single hits are not the cause of cancer.

There are two types of internal exposure for which there would be expected to be an enhancement of risk from this Second Event source. The first, due to sequentially decaying radioisotopes like Strontium-90 has been discussed in Busby 1995, Cox and Edwards, 2000 and Busby, 2000. Following an initial decay from an Sr-90 atom bound to a chromosome, the second decay from the daughter, Yttrium-90, whose half-life is 64hrs can hit the same cell in the induced replication sequence with a probability that is simple to calculate. The same dose from external radiation has a vanishingly small chance of effecting the same process. The second type of Second Event exposure, referred to in Busby 2000a, is from micron or sub-micron sized 'hot particles'. If lodged in tissue, these will decay again and again increasing the probability of multiple hits to the same cell inside the 10 hour repair replication period. It is this process that is relevant to the Depleted Uranium problem.

Second Events from DU particles.

The US Defence Department commissioned research into the levels of Uranium Oxide particulates produced by the impact of Abrams M1A1 Tank ammunition at the Nevada test site in 1986 [USBRL 1986]. The impact on armour of Depleted uranium penetrators results in about 80% conversion to Uranium Oxides UO_2 and U_3O_8 in the form of ceramic particles of diameters in the micron region. These aerosol particles are very mobile and can clearly be inhaled. In this regard the hazard is of a similar nature to that from the Plutonium oxide particles resuspended from Sellafield discharges to the Irish Sea which were considered as a possible cause of the Sellafield leukaemia cluster by COMARE and NRPB and referred to earlier where it was recorded that the ICRP66 models used to estimate doses did so by diluting the particles energy into large masses of tissue.

For particles below 1 micron diameter, self absorption of the alpha particle decays may be considered second order and the dose to tissue in the range of these alpha decays calculated. Table 3 shows the calculated doses in spheres of tissue within the 30micron range of the alpha decays. Also tabulated is the number of hits per day to this sphere of tissue. The table shows that for particles as small as 0.2 microns diameter, average annual alpha dose to the (lymphatic) tissue surrounding the particles is about the same as the total annual average background dose of 2mSv. For larger particles the dose rapidly increases. Between 0.5 and 5 microns, Second Event processes are stochastically likely. This is shown by Fig 8 where the number of hits per day is plotted against the particle diameter.

These 'hot particle' processes have been known about for a long time: Fig 9 shows a radiographic photomicrograph of a plutonium oxide 'hot particle' in lung tissue. Overlapping tracks can be seen.

Energy density and risk

The consequence of aggregating decays into a small sphere around a 'hot particle' is, of course, that the number of different cells capable of being hit elsewhere is necessarily reduced: we have converted a number of tracks well separated to the same number of tracks close together. If all tracks carry the same risk of mutation in cells in the track, i.e. all hits are equivalent, then there should be no hazard enhancement. The hazard enhancement proposed arises not from some 'hot coal' type of energy concentration process but from the fact that cells may be triggered into a sensitive repair replication sequence which carries a very high sensitivity weighting. It may, of course be true that there would be other reasons why concentrated irradiation of a small cluster of cells could produce unstable cell replication or cell communication fields such as those recently proposed by Sonnenschein and Sato [1999] and this itself may lead to a tumour promotion advantage but this is another matter.

Beta emissions from DU

Before collecting together these considerations there is one further matter which may have been overlooked in the case of DU. It was pointed out that Uranium-238 is an alpha emitter but depleted Uranium is also a beta emitter: indeed in the solid form the two beta-emitting daughter isotopes, Thorium-234 (beta; 0.26MeV, 24 days) and Protoactinium-234 (beta 0.23MeV, 6,75 hrs) are in equilibrium with the parent after 20 weeks (Table 1). These beta emissions are the main radiological hazard in handling the bulk material. In Iraq, I recently measured 24,000 counts per second at the surface of a stray A-10 30mm penetrator which was just lying on the ground. This represented a dose of about 1mSv/hour to the hands of anyone holding the penetrator. However, most of the beta (and alpha) decays were absorbed inside the bulk material, and only surface disintegrations were emerging to be absorbed in the scintillation counter head.

The equilibrium beta activity of DU is about 37MBq/kg. But most of this energy is absorbed in the bulk material: oxidation of the material on impact to produce some 10^{14} 1 micron diameter Uranium Oxide spheres per kilogram would enable all of the decay energy to be potentially available for human exposure. The enhancement of efficiency in release of beta radiation is thus greater than 1000-fold.

Environmental Mobility of the DU particles

In order to be define the population at risk, it is necessary to know the fate of the Uranium particles subsequent to impact. At the Nevada test site, the atmospheric concentration at 100m from impact exceeded the UK NRPB Generalised Derived Limit for Uranium in Air by a factor of about 5 [Busby 1999]. Dietz has reviewed data which establishes that DU particles are able to travel at least 100km from their impact source [Dietz, 1997]. I recently made measurements of alpha radiation levels in Iraq in three areas, the southern battleground near tanks destroyed by DU fire, the same area remote from the tanks, the town of Al Basrah and the city of Baghdad. Results showed that the alpha activity in the battleground area was more than five times higher than in Basrah and ten times higher that in Baghdad. In addition, and remarkably, levels on the surface of the ground near the damaged tanks did not generally show high levels of alpha or beta signal from Uranium and its daughters except in the case of one tank where a yellow contaminant, probably UO_3, showed high levels of beta activity. In addition, the insides of tank turrets which had radioactive holes in them from A10 hits, did not show high levels of beta or alpha activity. The generally higher alpha levels in the whole area, coupled with these observations suggest that the Uranium particles has been efficiently dispersed by some mechanism. I believe that this mechanism is the repulsion of charged particles by themselves and by the earth's permanent electric field of 150V/m. I have argued elsewhere that this effect operates in the Kennet Valley near the Atomic Weapons plant at Aldermaston and results in the preferential concentration of charged radioactive particles near electrostatic discontinuities between strata with different conductivity [Busby, 1997] . A similar effect near high voltage power lines was recently found by Henshaw et al. [1999].

Conclusions on Mechanism

Thus we can conclude that the external bag-of-water model is not an accurate representation of the kind of processes that occur at the cellular level and that the physics-based descriptions do not apply to internal irradiation. The Uranium Oxide particles are capable of travelling very large distances [Deitz, 1997]. They may then be inhaled and will become trapped in the lymphatic system where they may be transported to any part of the body. Here they may cause sequential moderate dose irradiation of local tissue volumes where the risk of mutation is far higher than is suggested

The enhancement of mutation efficiency that follows from exposure to inhaled Uranium oxide hot particles is capable of explaining the 'anomalous responses to low dose exposure' found near Sellafield and other nuclear sites and also 'Gulf War syndrome' etc. We are not, however, reduced to looking only at the Gulf War Syndrome and the Iraqi children for supporting evidence though I shall return to these later. There are other indicators, and our springboard for these is the 1983 observation of a childhood leukaemia cluster at Sellafield. In the last four years Green Audit been funded by the government of the Republic of Ireland to study cancer incidence close to the Irish sea. The study has used both Wales Cancer Registry and Irish Cancer Registry data to examine and explain variations in cancer risk with distance from the sea. The results of this work will be published elsewhere but since they cast considerable light on the DU problem, some of the findings will be briefly reviewed here.

6. Sea coast cancer risks and resuspended hot particles.

In three separate investigations between 1997 and 2000, Green Audit discovered profound and statistically significant evidence of excess risk of cancer incidence and mortality in coastal populations in Wales, Ireland and Somerset. The excess risk has been found for most of the cancer types and sites and in the following data:

- Incidence data for small areas in Wales from Wales Cancer Registry from 1974-89
- Incidence data for small areas of Ireland from the Irish National Cancer Registry for 1994-1996.
- Mortality data for census wards in Somerset from the Office for National Statistics for 1995-1998

In each area the trend with distance from the sea shows a sharp rise in the group of people living within 800m of the sea coast. It is driven by proximity to areas of intertidal sediment known to be contaminated with radioisotopes from Sellafield discharges. In the case of the Somerset study, which was investigated as a hypothesis test the drying, offshore, mud bank, known as the Steart Flats, was contaminated by historic releases from the adjacent Nuclear Power site at Hinkley Point.

As one example of the type of result found, the all malignancy relative risk for populations of all ages in Wales from 1974-89 is shown plotted against distance from the Irish Sea in Fig 10. Note the sharp rise in risk near the coast. Sufficient evidence has now accumulated from these studies to support the hypothesis that this cancer risk is a consequence of an exposure route involving inhalation of resuspended radioisotopes, particularly Plutonium Oxide particles. The trend in concentration of Plutonium with distance from the sea in Cumbria has been established and is shown in Fig 11. The radioactivity is brought inland by seaspray scavenging mechanisms which are quite well understood: indeed, the ocean is the source of about 30% of all PM10 particles in the UK. It is therefore not surprising that NRPB workers found Plutonium in the tracheobronchial lymph nodes of autopsy specimens from all over the UK in proportion to their distance from the west coast, particularly Cumbria [Popplewell, 1986]. Table 4 shows some results of these studies, which were, incidentally, omitted from the considerations in the COMARE report on the Sellafield leukaemia cluster. Nor is it surprising that Plutonium is found in children's teeth in the UK at levels which reflect a similar trend with distance from the Irish Sea [Priest et al, 1996]

7. Recent evidence on DU exposure risks and response by UK government

DU, leukaemia, cancer and birth defects in Iraq

There have been reports from within Iraq of serious health problems emerging after the Gulf War. These problems are apparent in the soldiers, in civilian adults living in the south near the war zone and also in children. They take the form of a range of conditions similar to those categorised as 'Gulf War Syndrome' in the US and UK veterans and also in large and significant increases in cancer and leukaemia in adults and children and also birth defects including novel types of birth defect. I visited the country in September 2000 with Al-Jazeera TV and toured the hospitals in Baghdad and Basrah, speaking to senior doctors and health service researchers. Cancer registry data reflect the increases in cancer and show that the main increases are also in the parts of the country, south and north of Baghdad, where DU ammunition was mainly used. Significant pieces of evidence are the first, the geographical pattern of cancer and second the cohort effect in childhood leukaemia which shows the main excess in the cohort aged 5-9 in 1998. This is an unusual finding for childhood leukaemia which normally peaks in the 0-4 age group and indicates that it was the war birth cohort that showed the greatest leukaemia effect. The geographical pattern of cancer also broadly correlates with the measurements I made of alpha activity in air in the country, which again reflects the distribution of DU based on the areas where the material was mainly used.

DU in Kosovo

No cancer data is available in Kosovo owing to the large changes which have been taking place there after the war. I was able to visit western Kosovo in January 2001 with Nippon TV and we used UN maps supplied by the Italian Army to locate areas where DU had been used. Using a survey scintillation counter I found areas where high beta counts indicated the presence of significant amounts of DU and took samples for analysis by alpha and gamma spectroscopy and also thermal ionisation mass spectrometry. Two main conclusions could be drawn from the results, which are shown in Table 5.

First, some 18 months after its use, significant quantities of DU either were resuspended in or remained suspended in the atmosphere to be precipitated with snow and to pool under the snow when it melted. The ratio of daughter isotopes to parent U-238 was remarkable. Instead of there being a 1:1:1 equilibrium ratio, the activity of U-238 in the sample was much smaller than the activity of the daughter isotopes. Since Uranium is largely insoluble (or would not have been there if it were soluble) this result shows that the Uranium particles had become resuspended between the time the snow melted and the time I measured the activity (about 2 weeks)

UNEP report on Kosovo

Following concerns about the possible health effects of radioactive contamination from Depleted Uranium weapons used by NATO in the actions in Kosovo in 1999, a number of scientists and experts were assembled under the auspices of the United Nations Environment Programme to visit Kosovo between 5-19th November 2000 to investigate levels of contamination and report on possible health hazards. Details of the expedition and its protocols and findings are to be found in the report [UNEP, 2001]. I have analysed their findings in a presentation to the European Parliament in Strasbourg in 2001 [Busby 2001, www.llrc.org] but will briefly outline UNEP's findings and their conclusions.

UNEP made three main claims relating to their findings.

a. There was no widespread dispersion of DU in areas of Kosovo where the shells were fired. DU measurements showed only local contamination, i.e. there was no evidence of DU further than 10-50 metres from a direct hit site.
b. There was no contamination of water sources.
c. There was no health hazard to humans anywhere with the possible exception of some slight danger from handling shell fragments for a long period.

	Sample A5 Gjakove Surface road dust	Sample A6* Gjakove Surface road dust	Sample A5A Cermjan Soil
Field Beta cps at 5cms	14	27	4.5
NATO Grid Reference	DM545937	DM545937	DN534026
Number of A10 rounds	225	225	655
Date of attack (NATO)	7th June 1999	7th June 1999	7th June 1999
U238	353 (6.5)**	5443*	19.6*
U235	6.8 (1.20**	69.6*	0.86*
U234	26.1 (2.3)**	91.08 (18)*	NA
Th234	1721 (52)	4988 (98)	NA
Pa234m	1836 (98)	5352 (433)	NA
Pb214	1.7 (.2)	1.1 (.3)	NA

Bi214	1.5 (.3)	1.3 (.3)	NA
Mass Ratio U238/U235	353	504	146 or 138.4[a]
Natural Uranium Ratio	137.8	137.8	137.8
DU present	Yes	yes	Yes

*Note 1: * Uranium by Thermal Ionisation Mass Spectrometry; **Uranium by Alpha Spectrometry; all others by Gamma Spectrometry.*

Note 2: Electra with DP2 Dual Phosphor 4-inch Scintillation Counter (NE Beenham Reading) gave average background beta counts per second in the field of 3-3.4cps. This is slightly greater than average levels in the UK of about 2.7cps.

[a] 146 was the value using ammonium carbonate extraction of Uranium, 138.4 was using nitric acid extraction which dissolves all the Uranium, not just the adsorbed Uranium.

Table 5 DU dust does not harmlessly disperse in the environment. Results of tests on samples from Kosovo collected 19th / 20th Jan 2001, more than 18 months after the attack

Examination of the tables of results shows that all three of these conclusions are incorrect and that the results showed the presence of widespread contamination by DU both by aerosol dispersion of particles greater than 0.2 micron diameter and decay products of U-238.
I conclude that the analysis of the results given in the tables was either biased or badly interpreted. Significantly, the tables of results were not attached to the report when it was sent to the Press. Consequently it was only the conclusions which were addressed at the Press conference [Parsons, 2001, Fleming, 2001]

Nic Priest's study for the BBC
Shortly after my visit, which followed UNEP's visit, in Spring of 2001, BBC Scotland commissioned Nic Priest, of Middlesex University to visit Kosovo and Bosnia and measure DU in urine samples taken from members of the population living in Eastern Kosovo in areas where bombing had occurred. I advised them to visit Djakove, where I had found DU and so had UNEP, and they took samples from inhabitants of this town, among others. Astonishingly, the samples showed that all the people tested had significant amounts of DU in their Urine samples. This included the BBC cameraman who had only been there for the week of the visit. Nic Priest's report is to be published and so I cannot give them here, but have made some averages of his results which were reported by the BBC and which I have given in Table 6 below.

Location	Number of adults	Mean 24hr DU excretion (ng)
Djakove, Kosovo	5	8.3
Klina, Kosovo	6	24
Bratunac, nr Sarajevo, Bosnia	3	22
Cameraman, Scotland	1	6.9

Table 6 Mean DU in urine samples from the areas in Kosovo and Bosnia visited by BBC Scotland in Spring 2001.

Epidemiology of Balkan peacekeepers
There have been many misleading statements from government ministers regarding the significance of leukaemia deaths among Balkan peacekeepers. Recently a UK government minister suggested that 42 leukaemia deaths per 100,000 peacekeepers was a reasonable sum and that therefore the handful of deaths observed should be seen as a normal situation.

Table 7 shows the numbers of deaths from leukaemia by age in males in England and Wales in 1998 and calculates the overall rate.

ages	deaths	population (m)
20-24	27	1984394
25-29	24	2168819
30-34	24	1967765
35-39	41	1711844
40-44	27	1760461
45-49	49	1700017
50-54	86	1360926
55-59	106	1281777
60-64	138	1228076
65-69	217	1129274
70-74	316	919901
	1055	17213254

Rate = 0.612 per 10,000

Table 7 Leukaemia deaths in men in England and wales in 1998 by age group

The value, 0.612 is for all ages 20-75 combined and is not correct for soldiers who are younger. Leukaemia rates increase markedly in people above 50 as you can see from the table and this would suggest a higher expected number of deaths if this large age group were used as a basis for any comparison. It is unlikely that there would have been many soldiers older than 40. Assuming an age range of 20-40 (which is conservative) there should be 0.15 deaths per 10,000 exposed per year (i.e. the death rate in the men aged 20-40 is about 116/7832822 = 1.48 E-5 which is 0.148 per 10,000 per year. So in the year since the bombing we should expect approximately 0.15 per 10,000 or 1.5 deaths in 100,000).

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

Cancer increases in Sarajevo

There has been an extraordinary increase in cancer and leukaemia in Sarajevo since the bombing. Sarajevo is close to the town where Nic Priest took urine samples and found DU contamination in people at least 6 years after the bombing. I append the latest figures from the Sarajevo Registry in Table 7.

Tumour Site	1995	1996	1997	1998	1999	2000	
Mouth and Throat	1 (1.1)	-	—	2 (2.1)	4 (4.3)	4 (4.3)	
Digestive	15 (16.0)	50 (53.2)	36 (38.3)	55 (58.5)	68 (72.4)	82 (87.3)	
Respiratory	12 (12.8)	15 (16.0)	20 (21.3)	34 (36.2)	44 (46.8)	51 (54.30	
Skin and ligaments	-	2 (2.1)	1 (1.1)	10 (10.6)	8 (8.5)	9 (9.6)	
Breast	3 (3.2)	11 (11.7)	14 (15.0)	29 (30.9)	34 (36.2)	37 (39.4)	
Urogenic	8 (8.5)	8 (8.5)	11 (11.7)	18 (19.2)	27 (28.7)	28 (29.6)	
Eyes	3 (3.2)	-	1 (1.1)	2 (2.1)	1 (1.1)	4 (4.3)	
Lymphatic and Blood		1 (1.1)	6 (6.4)	1 (1.1)	7 (7.4)	19 (20.2)	2 6 (27.7)
Divers	-	1 (1.1)	11 (11.7)	18 (19.2)	11 (11.7)	7 (7.4)	

All above	43 (45.3)	93 (99.0)	95 (101.0)	175 (186.)	216 (230)	248 (264)

Table 7 Cancer incidence in Sarajevo 1996-2000. Cases (crude rates per 100,000).
(*Source: Sarajevo Tumour Registry)*

Time lag considerations.

It is incorrect to discount such increases on the basis that the time lag is too short. The time lag between initiation and expression is given by the theoretical equations of Armitage and Doll, developed in the 1950s. The outcome of an exposure is biphasic [Busby 1995] since cancer development may follow immediately in cells which have a pre existing genetic lesion or later in cells for which the exposure causes a first lesion which is then developed following geometrical expansion of the cell line.

Chromosome testing UK vets

UK Gulf War veterans have recently had blood samples tested for chromosome aberrations in Germany. Results show a significant excess number of aberrations relative to German controls and are compared with Chernobyl levels reported by Shevchenko and Snigiryova [Burlakova 1995] in Table 8.

Group	**Number of chromosome aberrations DiC + CR per 1000 metaphases scored**	**Mean Dose in excess of natural background**	**Number of metaphases scored**
Gulf Veterans	7	0 + DU?	1001
German controls	0.5	0	
Chernobyl NPP staff	5.8	300-470mGy	6015
Chernobyl liquidators	4.4	220-350mGy	4937
Chernobyl controls	0	2mSv	34791 3605

Table 8 Chromosome aberrations in Dicentric Ring and Centric Ring rearrangements in Gulf War veterans compared with measurements made on groups exposed to the Chernobyl accident. (Schott 2001, Burlakova, 1996)

There is a fourteen-fold increase in the frequency of the unstable rearrangements leading to centric rings and dicentric rings relative to German controls. Data from the Chernobyl exposures published by Shevchenko and Snigyryova in Burlakova 1996 suggests that the DU exposure of the veterans is equivalent to more than 500mGy externally delivered, supporting the belief that the ICRP calculations of dose are in error by an amount of the order of 2000-fold or more if we assume that the average dose of the veterans tested was 0.25mSv. Similar unexpected chromosome aberrations following exposure to Uranium dust have been recently reported for Uranium miners by Zaire et al [1997]

8. Overall Conclusions

The Gulf War Syndrome and the increases in cancer and congenital effects in veterans of the Gulf War, the Balkans and in Iraqi populations are merely more and recent evidence of the serious error in the way in which the health consequences of ionising radiation exposures are presently modelled.

References

Bergonie, J. and Tribondeau, L. (1906), ‘De quelques resultats de la radiotherapie et essai de fixation d’une technique rationelle’, *Comptes Rendu des Seances de l’Academie des Sciences*, 143: 983.

Burlakova, E.B, A. N. Goloshchapov, N. V. Gorbunova, G. P. Zhizhina, A. I. Kozachenko, D. B. Korman, A. A. Konradov, E. M. Molochkina, L. G. Nagler, I. B. Ozewra, L. M. Rozhdestvenskii, V. A. Shevchenko, S. I. Skalatskaya, M. A. Smotryaeva, O. M. Tarasenko, Yu. A. Treshchenkova, ‘Mechanisms of Biological Action of Low Dose Irradiation in E. B. Burlakova (ed.), Consequences of the Chernobyl Catastrophe for Human Health (Moscow: Centre for Russian Environmental Policy, 1996).

Busby, C. C. (1995), *Wings of Death: Nuclear Pollution and Human Health* (Aberystwyth: Green Audit)

Bramhall R (1996), Busby C in Bramhall, R. (ed.), *The Health Effects of Low Level Radiation: Proceedings of a Symposium held at the House of Commons, 24 April 1996* (Aberystwyth: Green Audit).

Busby, C. C. (1998), ‘Childhood leukemia and radioactive pollution from the Atomic Weapons facilities at Aldermaston and Burghfield in West Berkshire: causation and mechanisms’, *Occasional Paper* 98/1 (Aberystwyth: Green Audit).

Busby C, (1999) Deposition and testimony in the Case of Regina vs Helen John, Middlesex Crown Court, Dec 1999

Busby C.,(2000), ‘Reponse to Commentary on the second event theory by Busby’ *International Journal of Radiation Biology* 76 (1) 123-125

CERRIE (2001) Consultative Exercise in Radiation Risk from Internal Emitters. A new committee set up by the UK government to investigate the ICRP risk model fror internal irradiation.

EU (2001) Motion passed unanimously in March 2001 by the European Parliament calls for reassessment of the ICRP risk models as applied to inrternal radiation effects.

MoD (2001) UK Ministry of Defence set up a committee to measure DU in the urine of veterans and correlate with ill health.

Deitz L (1997) in *Metal of Dishonour p134* New York City: International Action Center

Dubrova,YE Nesterov VN, Jeffreys AJ *et al.* (1997), ‘Further evidence for elevated human minisatellite mutation rate in Belarus eight years after the Chernobyl accident.’ *Mutation Research* 381 267-278

Eakins, J.D and Lally, A.E., (1984), ‘The transfer to land of actinide bearing sediments from the Irish Sea by spray.’ *Science of the Total Environment* 35 23-32

Edwards, AA and Cox R (2000), ‘Commentary on the second event theory of Busby’ *International Journal of Radiation Biology* 76 (1) 119-122

Ellegren, H, G.Lindgren, C.R.Primmer, and A P Moeller, (1997), ‘Fitness loss and Germline mutations in Barn Swallows breeding in Chernobyl,’ *Nature* 389/9, 583-4

Gibson, B. E. S., Eden, O. B., Barrett, A. *et al.* (1998), ‘Leukemia in young children in Scotland’, *Lancet,* 630.

Harre R (1985) *The Philosophies of Science* Oxford: University Press

Henshaw, D.L, Fews, A, Keitch, P, Close JJ, Wilding, RJ (1999) ‘Increased Exposure to Pollutant Aerosols under High Voltage Power Cables’ *International Journal of Radiation Biology* 75/ 12:1505-21

LLRC, (2000) see the website www.llrc.org/compendium/html

Mangano, J. (1997), ‘Childhood leukemia in the US may have risen due to fallout from Chernobyl’, *British Medical Journal,* 314: 1200

Michaelis J, Kaletsch U, Burkart W and Grosche B, (1997) ‘Infant leukemia after the Chernobyl Accident’ *Nature* 387, 246

Mill J.S (1879) *A system of Logic* (London: Longmans Green)
Miller R.C, Randers-Pehrson, G Geard, C.R, Hall, E.J and Brenner, D.J (1999) 'The oncogenic transforming potential of the passage of single alpha particles through mammalian cell nuclei.' *Proc. Natl. Acad. Sci. USA* 96: 19-22
Papineau D (ed) (1995) *The Philosophy of Science* Oxford: University Press
Petridou, E., D.Trichopoulos, N.Dessypris, V.Flytzani, S.Haidas, M.Kalmanti, D.Koliouskas, H.Kosmidis, F.Piperolou, and F.Tzortzatou, (1996) 'Infant Leukemia after in utero exposure to radiation from Chernobyl' *Nature*, 382:25, 352

Popplewell, D.S (1986) 'Plutonium in Autopsy Tissues in Great Britain' *Radiological Protection Bulletin No 74* Chilton: NRPB
Schott A (2001), 'Chromosome aberration analysis: Pilot study of nine depleted Uranium Contaminated British Veterans. Decoding Paper No 3' WODUC: Berlin Dahlem

Sinclair, W. K. and Morton, R. A. (1966), 'X-ray Sensitivity during the Cell Generation Cycle of Culture Chinese Hamster Cells', *Radiation Research*, 29: 450-74.
Sonnenschein C and Sato A (1999) *The Society of Cells* (Harvard :University Press)
Weinberg H. Sh, Korol A.B, Kiezhner V.M, Avavivi A, Fahima T, Eviatar Nevo, Shapiro S, Rennert G, Piatak O, Stepanova E.I, and Skarskaja E (2001), 'Very high mutation rate in offspring of Chernobyl accident liquidators.' *Proc Roy. Soc. London D, 266: 1001-1005*

Zaire R, Notter M, Riedel W, Thiel E (1997) 'Unexpected rates of chromosome instabilities and alterations of hormone levels in Namibian Uranium miners' *Radiation Research* 147 579-584