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

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1. The DU story is part of a wider concern

For there is nothing hid that shall not be manifested;
neither was anything kept secret, but that it should come abroad
Mark 4,22

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

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

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

Let us try to fit the dispersion of Depleted Uranium into this perspective. In terms of disintegrating atoms, radioactivity is measured in Bequerels. One Bequerel represents one disintegration per second. This is a reasonable way of quantifying amounts of radioactivity. The average Natural Uranium content of soil is about 10-20 Bequerels per kilogram, including all the Uranium isotopes. Most people excrete as much as 0.1mBq (0.0001Bq) per litre of Urine as a result of absorption of natural Uranium in food they eat. Pure Depleted Uranium contains about 12,400,000Bq of U-238 per kilogram and in Kosovo, some soil samples analysed by the United Nations Environment Program (UNEP) contained 250,000Bq/kg (UNEP 2001, Annex). The 350 tonnes of DU used in the first Gulf War represents 4.3 TBq (4.3×10^{12} Bq) of Uranium alpha activity (13.0×10^{12} if the radioactive beta emitting daughter isotopes are included-more of these below). If Dai Williams (2003) is correct and about 1700 tonnes were used in the latest war, then that represents 63 TBq of activity dispersed

mainly into a populated area of perhaps 100km^2 . This gives a mean density of deposition of radioactivity of 630,000Bq/m^2. These sums are instructive and are collected together in Table 1.

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

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

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

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

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

Like DU, these Plutonium Oxide particles are also long lived and mobile. Plutonium from Sellafield has been measured in autopsy specimens across the UK, in sheep droppings on the east coast of England 100 km from Sellafield at the same latitude and even in the teeth of children up to 200 km from the site in south east England. Both Uranium-238 and Plutonium-239 are alpha emitters, although

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

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

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

I have compared Plutonium and weapons fallout with DU to demonstrate that we are dealing with the same problem, the health effects of low level exposure to radioactive substances that irradiate our bodies from the inside. The weapons fallout, and other pollution from nuclear sites like Sellafield has been responsible for the present cancer epidemic, the one that everyone has experienced. It has been a major project of the nuclear military complex, and for governments who have been involved in releases of radioactivity, to cover up the link between these exposures and cancer or other ill health. This is why all these committees are controlled and steered by the same people. Recognition that DU caused cancer, leukaemia or lymphoma at the doses experienced by those who were contaminated after its use would lead to inevitable recognition that the weapons fallout substances, the Strontiums and Plutoniums and Caesiums also caused cancer, leukaemia and lymphoma. The reverse is also true. Recognition of the cause of the Sellafield lukemia/lymphoma cluster would lead to reassessing the risk models to the point where it would be clear that DU would have serious health effects. This is the origin of a massive cover up which extends to the cancer registries and the cancer research organisations.

2. Green Activists

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

Gandhi

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

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

As a result of the Green Activist approach, the Low Level Radiation Campaign has persuaded the UK government to set up a new committee to examine these effects. We pointed out, following the 'Mad Cow Disease' committee failings that the only way to get to the truth in science advice was to fund both sides and have them argue the case out in committee. The first committee of this kind is this new Committee Examining Radiation Risk form Internal Emitters (CERRIE, www.cerrie.org). Here, there are scientists from both sides of the debate on low-level internal radiation arguing out the various pieces of evidence that the ICRP risk model is in error and that internal radiation exposure, like that from fallout, from Plutonium, or DU represents a serious health hazard. CERRIE reports finally in 2004, but its preliminary report was considered at an international workshop in Oxford in July 2003. The report drew attention, for the first time, to the existence of major scientific uncertainties in the area of risk from internal radioisotopes.

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

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

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

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

3.1 Theoretical considerations

External radiation produces ionization tracks in tissue that are uniformly distributed. Thus each cell receives on average one track per year and the linear dose response used by the ICRP to predict cancer from the Hiroshima survivors breaks down if there is more than one track intercepting a cell in the time it takes for the cell to repair damage, about ten hours. For internal sequentially decaying isotopes and for internal long lived, hot (or warm) particles the probability of a cell local to the internal decay receiving two or more hits is very much higher than the equivalent probability for the same dose delivered externally. There are two consequences. The first is that the cell response is in the 'dose squared' region of the accepted ICRP model and the dose response in no longer linear. This is because the probability of a DNA double strand break occurring increases sharply for two or more hits to the cell. Such a lesion carries a high degree of certainty that a fixed mutation will follow. The second possible consequence is that the first hit to the cell will either induce a repair replication cycle in the hit cell, or if the cell is killed, in local cells which will begin to replicate to supply a replacement. Whilst replicating and repairing the initial lesion, a second hit at the critical point in the replication process will cause a fixed unrepairable mutation. This is the second Event Theory. There are further problems with internal isotopes which relate to their chemical affinity for DNA. Both Strontium (e.g. Sr-90, Sr-89) (Sr^{++}), Barium (Ba-140, an Auger emitter) and Uranyl UO_2^{++} ions bind strongly to DNA (Wu et al, 1996) and so their decays will be extremely hazardous since they are localised near the target of interest. Work with the covalently bound Auger emitter Iodine-129, and also manmade Auger emitters like Cr-59 with bind to DNA show that these localisation effects carry very high risks which are not modelled by their apparent average doses. U-238 itself is an Auger emitter (31 % decay 10keV) and the high concentration gradient of of UO_2^{++} ions near the surface of a UO_2 particle would result in a high level of DNA localisation near the particle. Particles are, of course, highly likely to cause second event and multiple hit effects to nearby cells and the local doses from DU particles are considered below.

In the last ten years, evidence has emerged that low doses of radiation cause genomic instability in cells that are hot, but also in cells that are near the cells that are

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

3.2 Epidemiological considerations

If we cannot extrapolate from external radiation and Hiroshima, and we cannot use linear no threshold dose responses to mathematically model health effects where does that leave risk assessment? The scientific answer is that we have to look at the effects themselves and use them to define risk. This is done by epidemiology of populations exposed to the radiation sources we are interested in. It is not good enough to say that the model does not predict the cancers, as the risk agencies said about the Sellafield leukaemia cluster. If the models is theoretically unsound, we must re-examine the issue and consider whether the cancers were caused by the radiation. When we do this for the famous Sellafield child leukaemia cluster, we find that the error in the ICRP risk model needed to account for the cancers is about 300-fold. Looking at the other leukaemia clusters the error needed to explain the cancers is between 300-fold and 2000-fold. This may seem like an enormous error, but if it consistently turns up, we should as scientists begin to look at how it can occur. Tamplin in 1972 examined hot particles of plutonium and concluded on the basis of theoretical assumptions that they, were more hazardous that the ICRP model suggested by a factor of 115,000, so these large numbers are not as silly as they may seem. They essentially represent the difference between local dose to tissue and averaged dose to body from a hot particle. And since it is the tissue that develops the cancer over a long period by amplification through cell division of various DNA lesions, it is not surprising that it is the tissue dose that is important, and not the whole body or whole organ dose.

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

There were two pieces of evidence that forced the UK government into a reappraisal of the issue of internal radiation. The first was the Chernobyl infants and the second was the minisatelite DNA mutations.

3.3 The Chernobyl infants

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

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

Because the National Radiological Protection Board had measured and assessed the doses to the populations of Wales and Scotland and because they themselves had also published risk factors for radiogenic leukaemia based on ICRP models it was a simple matter to compare their predictions with the observations and test the contemporary risk model. The method simply assumed that infants born in the periods 1980-85 and 1990-92 were unexposed and defined the Poisson expectation of numbers of infant leukaemia cases in the children who were *in utero* over the 18 month period following the Chernobyl fallout. This 18 month period was chosen because it was shown that the *in utero* dose was due to radioactive isotopes which were ingested or inhaled by the mothers. Whole-body monitoring had shown that this material remained in the bodies of the mothers until Spring 1987 because silage cut in the Summer of 1986 had been fed to cattle in the following winter. The result showed a statistically significant 3.8-fold excess of infant leukaemia in the combined Wales and Scotland cohort ($p = 0.0002$). The leukaemia yield in the exposed *in utero* cohort was about 100 times the yield predicted by the ICRP model. Table 3 compares the effect in the three main studies. In this table, the B cohort were those children exposed to the internal exposure from Chernobyl *in utero* in the 18 month period following the event and born between June 1987 and January 1988. These exposure periods were defined by the whole body monitoring results. The control periods A and C were the ten years before (1975-85) and the four years after 1988 for which data was available.

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

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

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

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

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

3.3 Minisatellite mutation rates in Chernobyl children

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

Although subtle genetic effects on sex ratio were apparent in the LSS offspring, the RERF researchers excluded them from the study because they did not accord with their notions of the expected direction of such an effect [Padmanabhan, 1997]. Neels's exclusion of the sex ratio effects resulted in the belief that the genetic effects of 10mSv in the first generation would be unmeasurable. Thus BEIR V gives the incidence of total genetic effects including chromosomal effects (unbalanced translocations and trisomies) at 6 per million offspring compared with the natural rate of 4,200. It predicts a 10mSv excess risk of 10 cases of congenital malformation in a natural rate of 25,000 per million offspring and similar vanishingly small increases are given for autosomal dominant, X-linked and recessive disorders. Using a combination of mouse studies and the epidemiology of the LSS, the doubling dose for spontaneous genetic burden has been estimated to be 1 Sievert. [e.g.BEIR V, 1990 p 70]

However, the development of molecular techniques has enabled objective measurements of the consequences of irradiation to be investigated in human populations. There have been several studies of minisatellite DNA mutation in children living in parts of the ex-Soviet Union and exposed to radiation from Chernobyl. Using the technological development of 'DNA testing' in which minisatellite DNA is separated into bands which are characteristic of its genetic identity, it has been possible to show that children living in Belarus and exposed to radiation from fission-product isotopes and particle fission fragments which contaminated their environment suffered a doubling in genetic mutation. [Dubrova,

1996, 1997]. Similar work with barn swallows exposed in Belarus showed that these genetic changes were also present in these birds and were associated with phenotypic changes in their plumage patterns as well as reduced survival, therefore underlining the potential importance of such mutations. [Ellegren *et al.* 1997].

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

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

I will now turn to the effects of DU.

4. The health effects of Depleted Uranium

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

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

3.1 Particle doses and hot coals

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

how it is done for a 2 micron diameter particle of DU trapped in the lymphatic system of a person who inhaled it.

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

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

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

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

3.2 Borrowing radiation energy from background: second order scattering
There may be a second source of error here although it is difficult to quantify. Uranium is very dense and the particles have an enormous combined surface area. It is possible to calculate that for the smaller particles of 0.2μ diameter a 5mg inhalation loading represents some 10^{11} particles with a combined surface area of about 250cm^2.

Now small particles smaller than the wavelength of incident scatter incident radiation so that the particles act as secondary scatterers for the gamma rays from natural background radiation or medical X-rays or other internal emitters including other local particles. In addition, the lower energy component of this radiation, below 100keV photon energy will quantitatively be converted into photoelectrons from the particle surfaces. These are short-range highly ionising electrons which will increase the ionisation density in the immediate vicinity of the particles. This effect is increased because Uranium happens to have a very low photoelectron work function and even releases electrons when irradiated by UV and visible light so that Uranium salts are light sensitive and can be used for photography. In addition the release of photoelectrons from the particle surface will cause it to acquire an electric charge and attract negative ions which will perturb the biochemistry taking place close to the particle with unknown consequences. None of these considerations are included in the ICRP model.

3.3 Particle environmental dispersion

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

I also found widespread DU in southern Iraq when I visited there in September 2000, or rather, I found areas of high beta counts on the ground in the area of the 'Mother of All Battles' and saw a few A10 penetrators lying on the ground also. In

Iraq, I found significantly higher alpha activity in the air in this area. Unfortunately the Iraqi authorities would not let me remove any samples.

3.4 Human contamination and biokinetics

Shortly after my visit to Kosovo in January 2001, Prof Nic Priest visited the same region with BBC Scotland and took urine samples from some 20 people including his BBC cameraman. Priest has access to sophisticated mass spectrometry equipment and can measure Uranium isotope ratios in urine. He found that all the urine samples were contaminated, including the cameraman Donald Macleod who had only been there for five days. These results have now been published (Schroeder et al, 2003) and they show conclusively that the people in the area are contaminated with DU. We also have the results of measurements on the urine of Gulf War veterans by at least three teams. All show the presence of DU in the urine some ten years after the exposure.

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

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

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

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

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

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

3.5 Chromosome aberrations in Gulf Vets

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

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

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

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

3.6 Epidemiology

3.6.1 Iraq

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

Cancer Registry, Baghdad 1999]. I show some data in Table 4 for male children (I should really say little boys) where I compare the numbers of cancer cases with those expected on the basis of the England and Wales rates for the same cancers and in Table 5 show the relative risk in the war birth cohort, those aged 5-9 in 1995-99. This calculation uses the rates in England and Wales to calculate the expected numbers of cases in each age group is the Iraq children had the same rates as the England and Wales children.

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

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

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

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

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

3.6.2 The Italian Kosovo Study

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

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

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

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

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

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

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

3.6.3 Cancer in the UK Gulf Veterans

The UK government have been very poor at examining the health effects of DU. But various questions have been asked in Parliament by individual MPs and the Gulf Vets themselves and non-Governmental Organisations like the Low Level Radiation Campaign have put pressure of the Ministers to investigate risk. The MoD set up a Gulf Veterans Illness Unit and these people produced a report in November 2002 which compared deaths in all Gulf Veterans compared with deaths in a matched control group who were not deployed in the Gulf. Results show that there were 19 deaths from leukemia and lymphoma combine compared with 11 in the control group.

This is a statistically significant finding (p = 0.018) but nothing was said about the finding, and my attempts to obtain a breakdown by type of cancer have so far failed.

4.The US Department of Defense.

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

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

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

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

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

My conclusions are that the position taken by the establishment is not based on science, but on wishful thinking. The NATO website and other NATO documents are reports of NATO meetings where everyone agreed that there was no problem. These positions were informed by a few meetings where military investigations agreed there were no problems. Other reports of the results of environmental surveys found no DU. This was probably because they were deploying Geiger Counters which only detect gamma rays. Later on, when there were some discoveries of DU made by the second UNEP survey, the statement 'no widespread dispersion of DU' was changed to, 'no widespread dispersion of DU at levels that would constitute a health risk'. And of course, these levels are those predicted by the ICRP risk models.

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

These organisations all agree with each other that there is no health consequence of exposure to DU. They have all produced reports stating this. All these reports are 'armchair' reports based on the health model of the ICRP. None of them have used scientific induction to look at the health of people who are exposed and work backwards to the exposures. Instead they look at the cancer yield in the Hiroshima survivors and say that at the doses imparted by the DU there can be no ill health. This is not science, as I argued in my first paper for the Royal Society (Busby 2000). Scientific method is based on induction. The deductive conclusion about DU and health is similar to the deductive conclusion that the Sellafield leukaemia cluster is not caused by radiation from Sellafield. Both arguments are scientifically bankrupt.

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

In 2000, Molly Scott Cato, Richard Bramhall and I published a small book, *I Don't know much about Science* (Scott Cato et al 2000). In it we analysed the results of questionnaires sent to UK Members of Parliament to see what qualifications in Science they had. We also addressed the question of scientific advice to government in the immediate post Mad Cow disease period and asked how such a situation could have come about. We concluded that science advice committees were biased in the direction of Industry. Molly, who has studied politics and philosophy at Oxford suggested that the only way to allow for such bias was to have oppositional science committees. In these structures, there are scientists from both sides of the argument, funded by government, who debate the issue within the committee and finally publish a report which draws attention to the consensus but also to the disagreements with suggestions for research that might resolve these. Shortly after this book two committees came into being where this approach was adopted (but without the funding). These were CERRIE, which I have mentioned above and the Depleted Uranium Oversight Board. Because the DUOB has members from all sides of the argument, from the Veterans and the Defence establishment, it is possible to ensure to a large extent that there are honest investigations of DU in the urine of the veterans. We have tried to ensure, by elaborate mechanisms of coding and questionnaires which are photocopied and redistributed to several organisations, that there can be no James Bond exercise in which the MoD dilute the samples or alter the questionnaires. And so at the end of this process, I believe that we can get a real understanding of the levels of DU, some ten years after the Gulf War I. However, the Chair, David Coggon, has been able to force the question of asking the veterans who are being tested whether they have been diagnosed with cancer off the questionnaire. It was clear from the discussions that he was terrified that this question, properly answered, would enable us to analyse the samples to show that there was a significant effect. This process has to be left to 'expert epidemiologists' which many feel means 'tame scientists' who will find nothing. This serious for us and for the veterans because we do not trust the epidemiological studies that are supposed to be happening, if indeed any happen at all.

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

6. Summary: Depleted Science

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

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

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