Expert Witness Statement for Tribunal Case
Don Battersby and Anna Smith
vs.
Secretary of State for Defence

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1. I am Malcolm Hooper the Emeritus Professor of Medicinal Chemistry at Sunderland University where I pursued an academic career from 1959-1992. My qualifications are B.Pharm, PhD from the Faculty of Medicine University of London and C Chem, MRIC (by election). I have been involved with veterans of the first Gulf War, 1990-1, GW-1, since 1997 following my retirement from the University and serve as their Scientific Advisor since that date. I was appointed President of the National Gulf War Veterans and Families Association, NGVFA, in 2000. I have been involved with advising the sick veterans and writing documents in support of Tribunals and Appeals. I wrote a major document for the Select Defence Committee, published in 2000 and have given evidence orally and in writing to Coroners Courts and the major Inquiry that accepted Gulf War Syndrome as a legitimate term to describe the multi-system illness that is Gulf War Syndrome. In connection with this work I served on 2 Government Committees as the veterans representative, the Possible Interactions between Vaccines and Pyridostigmine Bromide Assessment Panel, 2000-2006. I was a member of the Royal British Legion Gulf War Group for many years until its final demise. I have advised and written reports for the late Lord Morris of Manchester and the Countess of Mar in support of their Parliamentary activities on behalf of the veterans.

2. I have been asked by Dr Chris Busby, representing two nuclear test veterans the late Don Battersby and Anna Smith (for the late Barry Smith) to provide expert evidence about the developments in the understanding of radiation and health in relation to internal exposures to Uranium. I have read The Upper Tribunal Appeal from the First–Tier Tribunal regarding Donald Battersby and Anna Smith and also the Statement of Case and associated documents in the new remitted Tribunal hearing.

3. I was a member with Dr Busby of the Depleted Uranium Oversight Board (DUOB) which was set up in 2002 to develop and supervise the measurements of DU in the urine of British servicemen potentially exposed to DU particles in Iraq and the Balkans. Part of the remit of the DUOB was to review and discuss evidence for the safety of the current radiation risk model for predicting or explaining the genotoxicity and associated health effects of exposure to Uranium.
I much appreciated working with Dr Busby in this Board and in putting together our minority report that was submitted because of the failure of the Board to come to a clear conclusion on the science presented. Sick Gulf War veterans from the 1990-1 conflict were ill-served and are now largely forgotten or ignored despite our efforts. The ICRP dosage assessments, which use only external dosage based calculations as a measure of exposure applied some 10 years after the event, were the sticking point. Dr Busby is the leading national and international expert on radioactivity and its effects on people and the environment. His publications alone make that clear and I would strongly recommend him as an expert in any cases involving consideration of

evidence about radionuclide exposures and their biological effects. His qualifications and experience are second to none.

4. Since the fundamental basis of all nuclear weapons is Uranium I have been asked to review the evidence that the element has anomalous genotoxicity especially when inhaled as a nanoparticle. This route of exposure has become much studied since the deployment of DU weapons and significant research has been carried out and published showing that the radiobiological effects of internal Uranium exposure are much greater than expected on the basis of the average radiation absorbed dose to bulk tissue.

5 It is true that there are many fission and fusion weapons which principally involve the fission of Plutonium-239. However, all weapons have neutron reflectors and/ or stages which consist of large amounts of Uranium, the bulk of which is massive amounts of U-238. The high temperatures of the nuclear fireball ensure that any Uranium will be vapourised and will condense as nanoparticles and larger micron sized particles [Glasstone and Dolan 1979] and that these particles will be available for inhalation when they come to earth either as fallout or in tropical or maritime regions as rainout caused by the sucking up of moist air by the rising fireball.

6. A review of the evidence for the health effects of Uranium is the 2010 Uranium report of the European Committee on Radiation Risk (ECRR 2010). The ECRR is an independent body of scientists and experts in radiation effects which was founded in 1998. Its position is that certain types of internal radiation exposures are not safely assessed by the current radiation risk model and that theoretical errors in employing the current ICRP averaging model are likely to be large. This especially true for elements like Uranium which have high chemical affinity for DNA, the target for radiation genotoxicity. I was one of those who argued this in the DUOB in 2002-2006 and scientific developments since then have strengthened this position.

7. **The anomalous health effects of Uranium**

Uranium is a common element in the Earth's crust, and has been locked up as insoluble ores in most rocks and soils throughout evolutionary history. But after the discovery of radioactivity, the initial rush to extract Radium, and then later develop bombs and nuclear energy, a Uranium economy developed. After the 1950s, because of nuclear weapons tests and nuclear energy the quantities of the material released into the biosphere increased enormously.

Uranium in the atmospheric bomb test was dispersed as 1 μm oxide particles [Glasstone and Dolan 1979]. The fuel that drives the nuclear energy cycle is routinely released from the stacks and remains in the final waste. From the 1990s Uranium was dispersed into the air as weaponised Depleted Uranium (DU) particles. The element is present in nearly all phosphate fertilisers and contaminates the food grown with them.

Because of its enormously long half-life of 4.7 billion years, the radioactivity of Uranium is considered to be low, and radiation dose coefficients tabulated in the current radiation risk models, the basis of legislation, are also therefore low. But as early as 1963 [Huxley and Zubay 1961, Nielsen et al 1992] discovered that Uranium had a very high affinity for chromosomal DNA. It is widely distributed in body tissues, and has a long retention time

[Miller 2010]. The other important fact about Uranium is that it has the highest atomic number (92) of all naturally occurring elements, and this makes it the most radiation opaque substance on earth. That is to say, it blocks and absorbs natural background gamma radiation, X-rays and even ultraviolet radiation extremely effectively. It releases the energy mostly as short range photoelectrons, ionizing radiation equivalent to β^{-}radiation capable of causing genetic damage in local tissue [Busby and Schnug 2008]. The interest in the genotoxic effects of Depleted Uranium following the use as a weapon in Iraq and Kosovo in the 1990s and later Iraq in 2003 led to a significant number of studies in cell cultures and animals that identified anomalous genotoxicity [Miller et al 2002, 2002a, 2010; Coryell and Stearns 2006, Craft et al 2004, ECRR 2010b]. Reports from the first Gulf War 1990-1, GW-1, recorded many animal and human birth defects often resulting in still born babies [Guenther 1999] with extreme numbers of anophthalmos, babies born without eyes [Salman and De Sutter 2001]**.**

Two routes of exposure to Uranium need to be considered with regard to the Atomic Veterans of A-bomb tests, ingestion and inhalation. (Embedded fragments of DU are important for veterans who have been involved in military action where DU shells were used, e.g. first Gulf War veterans. This is an allied area that has been much studied, [Miller 2010.])

The current radiation risk model for Uranium exposures already give quite different dose conversion factors for the two routes, ingestion and inhalation. This reflects the exclusion of Uranium by the gut. However, I would expect damage to the gut microbiome and cells lining the gut. If Uranium is inhaled, the dose conversion coefficient is between 64 and 178 times greater [ICRP 1996]. And there are many situations where Uranium or Uranium compounds are present in the air to be inhaled. Nuclear atmospheric bomb tests, for example, were of bombs that were made mostly of Uranium, the central devices and the so-called Tampers, approximately one ton of Uranium per Megaton of yield.

The ‘pure’ substance Depleted Uranium (DU) was deployed in recent conflicts and is still part of the arsenal of the USA, UK and (we assume) other countries. Impacts from DU weapons generate aerosols of nanoparticles of long-lived Uranium Oxides. Nuclear atmospheric tests will have done the same, yet no measurements of Uranium in fallout and rainout were published. The standard accepted document on which predicted health effects from fallout is based, the Lawrence Livermore Hicks printout, does not even list Uranium 238 [Hicks 1981]. So discussions of health outcomes of exposures to bombs like the Marshall Islands study [Simon et al 2010] entirely miss out the Uranium exposures. These are dominated in dose terms by the isotope U-234 which is present in the U-235 enriched Uranium. This isotope is more radioactive than U238 and U-234. U-234 is extracted into the enriched Uranium that is separated from natural Uranium because it is lighter even than the U-235. The high levels of U-234 in enriched Uranium have recently been confirmed by the release of a previously restricted document from the Oak Ridge separation plant in the USA [Kazanjian 1976]. Like the other Uranium isotopes it is a pure alpha emitter and invisible to Geiger counters. U-234 was found in soil in the black rain areas of Hiroshima [Takada et al 1983] and the bones of those living near the Semipalatinsk test site. [Yamamoto et al 2006].

But in mass terms, most of the fallout is Uranium-238. And this was the colouring agent for the “black rain” which followed the air-burst 1945 Hiroshima bomb. The residual Uranium particulates contaminated all of the areas where the rain fell and will have been resuspended

and available for inhalation, particularly in the early months when reconstruction was being carried out. Thus the first control group chosen in the Life Span Studies, the 5000 or so Hiroshima Not-in-City-Early-Entrants (NIC-EE) should be the group to watch for any effects. But in the ongoing ABCC studies these people were later mixed with a much larger group (20,000) of later entrants (NIC-LE) and any effects diluted. Only one of the Technical reports was ever published in which these two groups were distinguished. That was Report 7 in 1973 [Moriyama and Kato 1973]. It showed that the NIC-EE groups had a quite anomalous health response, identifying a biased selection of a more healthy survivor group. Note that these groups were all chosen 5-7 years after the bomb, and after a time when many of the original cohort might have died from early effects of exposures, resulting in a selection bias. In the NIC combined group there were also epidemiological oddities with high levels of cancer in the young, but overall less cancer and ill health in the old. The confusion resulting from this led to the ABCC organisation abandoning the NIC controls altogether and using the low dose 0-9 rad group as the control for its findings. This early assumption of no fallout exposure was unfortunate, and colours the credibility of the main epidemiological study underpinning the entire edifice of radiation risk.

If Uranium, an alpha emitter and photoelectron amplification agent also binds to DNA in chromosomes we might expect chromosome damage in those exposed. There were chromosome damage effects reported in Namibian Uranium miners [Zaire et al 1997], Uranium nuclear energy workers [Martin et al 1991], New Zealand nuclear test veterans [Wahab et al 2008] and Gulf war veterans [Schroeder et al 2003]. All these findings are in those who have received very small doses on the basis of the current risk model, doses far less than annual natural background.

Chromosome damage in germ cells will have effects on offspring. Epidemiological studies of the offspring of Test Veterans [Busby and de Messieres 2014, Rabbitt Roff 1999], Gulf War veterans [Doyle et al 2004, Araneta et al 1993, Kang et al 2001] and civilians in areas where DU was deployed [Alaani et al 2011, 2012] all show such effects.

Nuclear Uranium worker studies show excess rates of cancer [Guseva Canu et al 2011, McGeohegan and Binks 2000]. The series of studies of Uranium workers by Guseva Canu et al. 2011 in France show significant excess risks of leukemia at doses which can be estimated to represent at least 1 thousandth of the dose which the current risk model would require to explain. The paper was careful to avoid stating this obvious conclusion.

Then there is a well-accepted but rare genetic-damage based cancer Retinoblastoma, which is diagnosed in children and for which the Rb gene mutation is known. The highest rates for this cancer are in the Navajo tribes who inhabit areas where there are Uranium mine tailings [Bercow 1983]. The other place where this condition has a high rate is in offspring of workers at the Sellafield reprocessing plant in the UK [Morris et al 1993].

Some groundwater studies in areas of the USA differential Uranium levels indicate that cancer rates follow the Uranium levels (and of course the decay chain nuclides like Radium and Radon) [Wagner et al 2011].

There are thus important and major indicators that for Uranium exposure and health, radiation risk science, so called Health Physics, has hitherto got it very wrong. The conventional dosimetry error seems to be upwards of 1000-fold. In its report on the health effects of Depleted Uranium in 2001, the UK Royal Society (and all the global radiation risk agencies)

concluded that there could be no cancer effects until "choking" quantities of Uranium have been inhaled [Royal Society 2001]. This is because the "absorbed dose" necessary to cause cancer would require grams of U-238 to be inhaled. This is clearly absurd since genotoxicity associated with a precancerous state and excess cancers are found in various groups, see above. It is high time to revisit this issue and carry out some proper epidemiological research of the effects on humans of the element Uranium, and also to incorporate what is known even now into changes in legislation and changes in the Health Physics understanding of Uranium.
There does at last seem to be some measure of concern evolving in this area [DOREMI 2015]. The European Union radiation research organisation MELODI has finally moved into action, led by the French radiation protection agency IRSN for whom the epidemiologist heroine of all this, Irina Guseva Canu worked. The matter was raised (by Dr Busby) at the inaugural MELODI conference in Paris in 2010, but nothing seemed to develop. He pointed out that there are likely to be dose estimation problems associated with internal exposure to nuclides which bind to DNA, and particularly Uranium, the essence of the failed CERRIE committee deliberations in 2001-2004. Now at last a project has been proposed: CURE: Concerted Uranium Research Europe. In the report launching this development in March 2015 the authors write:
. . . a large scale integrated collaborative project will be proposed to improve the characterization of the biological and health effects associated with uranium internal contamination in Europe. In the future, it might be envisaged to extend collaborations with other countries outside the European Union, to apply the proposed approach to other internal emitters and other exposure situations of internal contamination, and to open the reflections to other disciplines interested in the effects of internal contaminations by radionuclides.

8 Relevance to the veterans' case of the changing view of the radiotoxicity of Uranium

The changing view of the genotoxicity of Uranium is clear from the development in Europe of a large scale and expensive project (CURE) to re-examine the issue in the light of the recent scientific evidence which emerged since the DUOB discussions. It is unarguable that the weapons tests created large quantities of Uranium particulates and that by mass these would have represented more than 99% of all the contamination from bombs which were exploded as air bursts (not incorporating ground level material). Whatever the specific mechanism of enhanced Uranium genotoxicity, the evidence is such that it cannot be denied. One way of looking at the issue has been to classify Uranium as partly chemical and partly radiological agent, but this means that the ICRP and other current radiation risk models deployed by inter alia the Royal Society in the case of DU and the MoD Defence in the case of the veterans cannot be seen as safe.
Uranium exerts its genotoxic and other physiological effects by a variety of mechanisms which include;-

1. Chemical –
    a. strong binding to the DNA molecule and allied proteins – other non-radioactive 'heavy metals' also do this.
    b. its large atomic number- the photoelectron effect, see above, has not been considered.

c. the strong oxidising capacity of uranium oxides depletes the protective molecules of cells, e.g. glutathione, thereby generating destructive reactive oxygen species associated with inflammatory diseases (Wu et al 1999; Iyer and Lehnert 2000; Lorimore et al 2003).

2. Physiological –Neurotoxic changes have been reported in animals (Miller 2010)

3. Radiological/physiological responses.

a. The effects of internal doses of uranium in animals show that the dose effects are cumulative i.e. chronic multiple small doses are more damaging than an acute single dose for the same quantity [Miller 2010]. This is particularly worrying in the light of the ubiquitous presence of uranium in the environment which could lead to ever accumulating toxic effects.

b. The bystander effects in which non-irradiated cells respond to low dose α–radiation in remote cells by the generation of messenger molecules leading to genotoxic changes and oncogene production [Iyer and Lehnert 2000].

It is now known that irradiation of the cell cytoplasm, and not just the nucleus where the DNA is contained, also leads to mutations and increased sister chromatid exchanges [Iyer and Lehnert 2000; Wu et al 1999]. "Radiation-induced …… bystander effects may reflect inter-related aspects of inflammatory-type responses……and contribute to the variety of pathological consequences of radiation exposure" [Lorimore et al 2003]

3. Direct radiological effects which lead to breakages in DNA molecules with loss of genetic integrity. Mechanisms for the repair of DNA damage are disrupted leading to genomic instability [Yellowhair 2011]. Uranium in animals induces damage to germ cell DNA. Preconceptual paternal exposure induces genomic damage in unexposed offspring. Epigenetic effects are reported in animal studies [Miller2010].

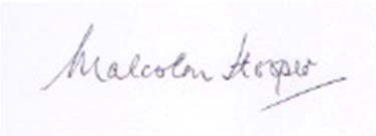
It is disingenuous to try to maintain that measurements of external based average radiochemical dose can accurately indicate the impact of internally absorbed radionuclides of uranium or to ignore or exclude the basic chemistry and physiologically properties of uranium and uranium compounds in contributing to the genomic and other affects of this element. The ICRP approach is grossly inadequate and misleading and ill-serves the demand for justice by the atomic veterans and their families.

It is my understanding that the law, as related to the Pensions appeals is that the appellant only has to show that there is some reasonable doubt based on evidence which is not fanciful that their condition could have been caused by their exposures. It seems to me that if the EU can have enough doubt about the radiogenicity of Uranium to give several million Euros to a major scientific project to investigate the issue, that must be good enough for the Tribunal to find that there is sufficient doubt about the MoDs assertion that the cancers developed by the test veterans (and indeed also the Gulf War and other veterans exposed to DU) were not caused by their exposures.

9 I have omitted discussing the levels of Uranium in the weapons or in fallout and rainout, and have merely assumed that there must be Uranium exposures to those deployed at the test sites. I reserve the right to add to this report in the light of new information that may become available, in particular measurements of Uranium isotopes on the ground at the test sites, in the weapons before detonation, and in other relevant samples. I understand that Disclosure requests have been made to the Ministry of Defence representatives.

10 **Witness Statement**

I understand my duty to the court and the information in this report is to the best of my knowledge truthful and accurate. I do not know any of the clients in this case personally and have had no dealings with them prior to the referral of this case from Dr Busby



Malcolm Hooper

Date 01/10/2015

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