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

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Short Version

The problem of the health effects of exposure to material from Depleted Uranium weapons is part of a much larger argument. This is the question of the health effects on exposure to internal man-made radioactive materials. These substances, like Strontium-90, Caesium-137, Plutonium-239 etc. have been released routinely to the planetary environment since the first use of the atomic bomb in 1945. They have become incorporated into the food chain and exist in the air and water and now contaminate all living systems. Research into the effects of this has been discouraged by nuclear nations during and after the Cold War and evidence of their serious harmful effects, in the form of cancers and genetic problems has been routinely suppressed.

It is now fairly clear, from our research and that of others that the present cancer epidemic is largely a result of the exposure to weapons fallout which peaked in the period 1959-63 when atmospheric tests were banned by Presidents Kennedy and Krushchev. After the weapons fallout stopped, there were new sources of radioactive pollution, particularly the atomic fuel reprocessing plants at La Hague in France and Sellafield in the UK. Sellafield has contaminated the Irish Sea with Plutonium, Uranium and other fission isotopes, mostly the same as the weapons fallout isotopes. The results of our 3-year study of cancer in Wales and Ireland shows that people exposed to radioactive material from Sellafield washed ashore on the coast of the Irish Sea suffer significant excess risk of cancer. The trend with distance from the sea of airborne plutonium oxide particles is identical with the trend in cancer and this draws attention to errors in the present modelling of risk from these particles using the averaging model developed by the International Commission on Radiological Protection (ICRP). This model was recently questioned by a new independent committee, the European Committee on Radiation Risk (ECRR) whose new rational

model distinguished between external and internal exposure. The report ECRR2003 calculated that over 60million people worldwide have died as a result of the releases of man-made radioactivity to the environment (ECRR2003).

The ECRR report draws attention to the wide environmental dispersion and local dose effects of hot and warm particles like Plutonium and Uranium Oxides. This has implications for DU exposure. Despite the fact that Uranium is much less radioactive than Plutonium, the fact that very large quantities of DU have been used in wars means that in terms of activity density their effects may be comparable. For example the 350 tons of DU used in the first Gulf War represents an area density of 130,000 Bequerels (Bq) per square kilometre, assuming that the localisation was into 100km² compared with releases of 20,000Bq/km² releases of Plutonium into the Irish Sea from Sellafield or 37,000-111,000Bq/km² in the 30km Chernobyl exclusion zone as defined by the IAEA. Indeed the IAEA define a contaminated area as >37000Bq/km².

The Low Level Radiation Campaign (www.llrc.org) and Green Audit (www.greenaudit.org) have been researching this problem since 1992 and looking at DU and its internal effects from 1995 when we suggested DU as a cause of Gulf War syndrome and the Iraq illnesses in children. We have now succeeded in having the ICRP risk model for internal radiation re-examined at a high level through the new CERRIE committee of the UK government (www.cerrie.org) and have put pressure, through the Royal Society on the UK Ministry of Defence to examine the DU problem more closely. The CERRIE process, which is an official process of the UK government will end with a report in February 2004 and will conclude that there are large question marks over the internal risk model of the ICRP. These follow from theoretical and epidemiological evidence. Theoretically, the work of the committee focuses on the fact that internal radioactive materials can give high doses to some cells close to their decays and cannot therefore be modelled by averaging techniques which are the basis of the ICRP method. Thus cells can receive more than one hit over the period the cell is trying to repair damage. In the area of epidemiology, there are many studies showing high levels of cancer and leukemia in children and adults exposed to internal radiation from the weapons fallout to people living near nuclear sites. Most critical to the arguments are the observations of increases in infant leukaemia in those who were in the womb at the time of the Chernobyl fallout. These increases have been seen in several countries by different teams of workers and together they show an error of 100-500-fold in the predictions of the ICRP model for exposure. In addition, recent measurements by Dubrova et al. and separately by Weinberg et al demonstrate the existence of subtle genetic damage effects in children born after the Chernobyl accident, drawing attention to an error of about 2000-fold in the ICRP model of genetic damage after radiation exposure. One of the areas which will be reported on by CERRIE is DU. Our work naturally divides into a number of headings:

- Theoretical considerations of mechanism
- Epidemiological considerations and evidence
- Environmental dispersion measurements
- Human contamination and biokinetics

Our group has made studies and comments of other work in all these areas.

Theoretically, DU particles are incorrectly modelled by the ICRP averaging model because they deliver relatively high doses to local tissue. We calculate for a 2micron diameter particle that the equivalent dose to a 30micron radius sphere of cells within the range of the particle decay is 1500mSv per year and will involve 11 alpha tracks

from the U-238 and 22 beta electron tracks from the two daughter isotopes Pa-234m and Th-234. The ICRP calculation give a dose of 2.1×10^{-7} mSv to the 800g lymphatic system, thus highlighting the enormous difference in the dose calculated by the two methods and giving some level of insight into the kinds of error factors needed to explain differences between the levels of illness suggested by ICRP models and those observed in the real world. In addition to this, there is another source of radiation damage to local cells. This is from the borrowing and re-scattering of radiation by the Uranium particle from natural background gamma rays. Because gamma photon absorption occurs as the fourth power of the atomic number and also by density, Uranium absorbs between 400 and 1200 times more radiation from Natural Background than the equivalent volume of tissue (depending on the photon energy). The energy borrowed is re-emitted as short range photoelectrons which have high ionising power. This effect is most significant for low energy gamma photons below 100keV which make up almost half the gamma background. The effect does not occur for particles larger than 10microns since the photoelectrons produced by primary absorption are reabsorbed by the uranium within the mass of the larger particles and pieces of shrapnel. Uranium is very dense and the particles have an enormous combined surface area. For 0.2 micron diameter particles a 5mg dose represents 10^{11} particles with a combined surface area of 250cm^2 . The combined effect of this and the short range alpha particles from the U-238 decay result in very large doses to local cells and multiple tracks to local cells, both effects do not occur at natural background dose levels.

On the matter of environmental dispersion of the particles, results show that the mean particle size is around 1 micron diameter and that the particles can be carried by wind several hundred miles. Our contribution has been the investigation of DU in Iraq in 2000 and in Kosovo in 2001. We were able to show, from measurements of daughter isotope ratios in material I collected in Gjakove, Kosovo that the DU was being re-suspended in sunny weather and then washed out by rainfall. This means that there are significant amounts in the atmosphere in dry weather for inhalation and translocation to the lymphatic system. Later measurements by Nic Priest of Urine samples in Kosovo confirmed contamination of local people a year after the bombing. In addition UNEP's later studies in Bosnia and Montenegro found airborne DU in filters.

Humans who inhale the DU dust are therefore contaminated, and measurements in animals suggest that a proportion of the DU is immobilised in the lymphatic system and has a half life of about 13 years. On this basis we would expect a daily excretion of about 20ng DU some 10 years after an inhalation of 5mg in which 10% was translocated and 50% was insoluble form. This seems to be what is measured in Gulf veterans. Under these conditions, if 20% of the total insoluble material was trapped in the tracheobronchial lymph nodes this represents 2×10^9 particles of 0.2 micron diameter or about one particle per cell.

There is some evidence that these loadings have caused observable effects. Chromosome damage to peripheral lymphocytes, found recently (2003) by Schroeder et al can be analysed on the basis of relationships deduced by Schmitz Feuerhake and Hoffmann in 1999. These suggest a dose of 50mV or more in the year prior to the measurement. This dose is massively greater than any dose obtained by calculation using the ICRP averaging approach. If I assume a 5mg lung loading the ICRP calculation gives a dose of 1.4×10^{-3} microGray in two years. This underscores the huge difference in the doses calculated by ICRP and those measured by the chromosome tests. However, the chromosome test results can be explained on the

basis of the local dose calculations I have made, suggesting that it is local dose which is the relevant parameter.

If it is dose to the lymphatic system that is important, then health effects associated with lymphatic system damage should be the first to emerge, and this is what is found. In Iraq, leukemias in children have been highest in the cohort who were born around the time of the bombing. I compare the 0-4 and 5-9 and 10-14 year old leukemias and lymphomas in Iraq with controls from England and Wales. Results show that for leukaemia and Hodgkins lymphoma excess risks of more than 3-fold exist in the Iraqi war cohorts who were aged 5-9 in 1995-99. This is most unusual since childhood leukaemia peaks in all normal populations in the 0-4 age group showing that the Iraqi 5-9year olds have been affected by some leukemogenic agent.

There is also evidence of lymphatic system involvement from the Italian study of peacekeepers in Kosovo and Bosnia which I re-analysed for the UK Depleted Uranium Oversight Board. These results of 39,491 peacekeepers showed a significant excess of lymphoma. Allowance for the healthy worker effect increased the significant excess risk found by the Italian epidemiologists of 3-fold to about 7-fold. Very recently, the UK parliament was told that the risk of lymphoma in UK veterans of the Gulf War is significantly higher than that in a matched control

The military authorities and scientific committees have continually denied or minimised evidence of the health effects of DU. However, their methods have been generally pathetic and demonstrably inaccurate. In the US Department of Defense report on the Balkans DU, almost all of the referenced contained in the report were to the NATO website and none of the references were to any peer reviewed report. My conclusion is that there is enough evidence to show that DU is a serious health hazard and that its effects are being covered up systematically by the politicians, the military and scientists employed by them. A major reason for this is that any acceptance that DU posed a risk would lead to questions about the effects of low dose radiation from internal isotopes which are routinely emitted by nuclear power stations and the nuclear fuel cycle, e.g. Sellafield, Chernobyl, weapons tests. The resulting cost, in loss of energy and litigation is too great for any government to gamble on and it is this that is the source of the cover-up.

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