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Editorial

Uranium Epidemiology

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If the illumination of epidemiology is there to identify threats and save lives it is high time its searchlight beam was seriously directed at the element Uranium and its health effects.

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 the bombs and the 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 tests was dispersed as 1 micron (um) oxide particles [1]. 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 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 [2, 3] it was discovered that Uranium had a very high affinity for chromosomal DNA. And 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 which cause ionization (and therefore genetic damage) in local tissue [4]. 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 [5- 9].

There are two routes of exposure to Uranium: by ingestion and by inhalation. Studies which underpin the current radiation risk model for Uranium exposures already give quite different dose conversion factors for the two routes. This reflects the exclusion of Uranium by the gut. If Uranium is inhaled, the dose conversion coefficient is between 64 and 178 times greater [10]. 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 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 [11].

So discussions of health outcomes of exposures to bombs like the Marshall Islands study [12] 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-235. 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 [13]. 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 [14] and the bones of those living near the Semipalatinsk test site. [15].

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 [16]. 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 [17], Uranium nuclear energy workers [18] , New Zealand nuclear test veterans [19] and Gulf war veterans [20]. 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 [21, 22], Gulf War veterans [23-25] and civilians in areas where DU was deployed [26, 27] all show such effects.

Nuclear Uranium worker studies show excess rates of cancer [28, 29]. The series of studies of Uranium workers by Guseva Canu et al. 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 [28]. 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 [30]. The other place where this condition has a high rate is in offspring of workers at the Sellafield reprocessing plant in the UK [31].

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) [32].

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 [33]. This is because the "absorbed dose" necessary to cause cancer would require grams of U-238 to be inhaled. 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 [34]. 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 me) at the inaugural MELODI conference in Paris in 2011, but nothing seemed to develop. I said 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 [34]:

. . . 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.

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