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New Zealand nuclear test veterans' study

Thank you for sending me a copy of the report by Rowland, Podd and Wahab.

I have some comments, primarily with regard to the radiation exposure pathways postulated in the report, which may be of interest in placing the value and conclusions of the report in context.

I have noted the review of the report by Stephen Robertson and in particular his understanding that the small differences in observed SCE frequencies between the study group and controls have no transgenerational implications, and his concerns about the potential for confounding by smoking.

It is clear from the report that the observed frequencies of SCEs in individuals vary widely in both the study (1 to 34) and control (2 to 28) groups, with similar wide standard deviations about the means for both groups. Further, the mean SCEs observed are not greatly different but generally lower than those observed in normal populations elsewhere (p.20 of the report). While the small difference in the mean SCE scores is of statistical significance the potential for confounding factors in comparing the two groups needs to be recognised. While the authors collected information on a number of potential confounders these are not explored in any detail. The very different smoking habits of the test veterans compared with controls is noted but discounted on the basis that the impact of smoking on SCE frequencies is not long lived. However, no information is given on the current smoking habits of the groups and how these may differ. Furthermore no correlations are attempted between current individual smoking habits and SCE scores, which might have provided some indication of smoking as a factor giving rise to SCEs.

The authors also provide no information on whether there is any difference in medications being taken by the two groups, nor do they explore the possibility of drug-induced effects on SCE scores. If the study group, for whatever reason, has a different socio-economic level with different welfare or sickness benefit dependency, there could be an associated difference in medication use.

The authors also seem to imply that SCEs caused by smoking are of short-lived duration, while those caused by putative radiation exposure are longer lasting (e.g. on p.7). No explanation is offered as to why this might be the case. However, on p. 26 of the report they state that if the difference in the mean SCE scores of the study group versus the controls were to be attributed to radiation exposure "this would imply, *a priori*, that radionuclides were still present in their system". They conclude (on p.28)

that the small difference they observed in mean SCE scores between the study group and controls is attributable to "residues of radiation particles that may still be present in the bodies of the veterans", and that small body burdens of alpha emitters (e.g. uranium, plutonium) "could be the source responsible" (first paragraph on p.27).

This postulate is totally implausible.

Firstly it should be recognised that the human body contains quantities of naturally occurring alpha emitters as a result of ingestion and inhalation of the naturally occurring long-lived radionuclides in the uranium 238 and thorium 232 chains. Annual intakes are typically some tens to a hundred becquerels (Bq) of alpha emitters but vary widely from region to region and with diets. Concentrations of alpha emitters in bone are typically a few Bq/kg (UNSCEAR 2000).

Secondly, a plausible pathway of exposure to alpha emitters has to be established. If the test veterans had some small exposure to airborne fission products from detonations, the intake by *inhalation* of long-lived alpha emitting radionuclides at these times can be explored. From data in the UNSCEAR 2000 report the airborne release of plutonium isotopes as a fraction of the combined activity of all fission products can be estimated. This indicates that the airborne activity of all plutonium isotopes combined (Pu-239, Pu-240 and Pu-241) at a time of 4 hours post detonation would have a concentration less than 6×10^{-7} (i.e. less than 1 Bq in a million) in relation to the activity concentration of fission products produced in the detonation. The beta/gamma emissions of the fission products would give rise to very high external radiation exposure rates before any significant concentration in air of plutoniums was reached. The report of the Advisory Committee on the Health of Veteran's Children noted that the ships "were stationed upwind and well away from areas likely to be contaminated if a surface burst occurred accidentally". Further, "The ships could if necessary be sealed and they contained wash-down equipment. Radiation detection systems were in place which included personal film badges, and integrating monitoring instrumentation and ratemeters by which doses at the time were estimated. These recorded no significant radiation". The fact that the ships were never in an area of moderate external exposure rates indicates inhalation of alpha emitting plutoniums was not possible.

The possibility that the test veterans may have *ingested* alpha emitters in fallout can be dismissed. (The ships were not themselves exposed to any significant fallout, nor was there any on Christmas Island (McEwan, A.C. et al, 1981. NRL Report 1981/9). Further, the transfer factors for Pu from environmental media into fish and plants is low and the gut transfer factor for ingested Pu is also low.)

I conclude that the New Zealand Test Veterans Study provides no evidence whatever for radiation exposure of the test veterans, and that the postulated pathway of continuing exposure from incorporated long-lived alpha emitters has no scientific foundation.

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