Comments on the Report of Professor A Rowlands on SCE studies of Nuclear Test Veterans

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The report is well written and is an excellent review of the field. The study reported has been well performed and the data carefully analysed. The problem is clearly described; measurement of biological effects some 50 years after putative exposure to ionizing radiation is not an easy task. The author recognizes that detection of such late effects will most likely require employment of other techniques such as mFISH (multiple Fluorescence In Situ Hybridization) that can detect chromosomal translocations arising from original damage to bone-marrow stem cells, and such studies are in progress. However, the SCE method is a very sensitive assay and able to monitor ongoing damage to cells and can give a measure of genotoxic damage to blood cells. It is unlikely that the SCE assay will pick up actual damage inflicted 50 yrs ago. However, it may record ongoing genomic instability resulting from such damage. Genomic instability can be caused by past radiation exposure, as shown by numerous studies, including those on bone-marrow cells (e.g. Kadhim, M.A., Lorimore, S.A. Hepburn, M.D., Goodhead, D.T., Buckle, V.J. & Wright, E.G. Alpha-particle-induced chromosomal instability in human bone marrow cells. Lancet, 344, 987-988, (1994). The question arises as to whether the findings reported here are indicative of such ongoing instability (resulting from past exposure), or whether they arise from current exposure to genotoxins of some kind. A key point in the report is the exposure of the subjects to tobacco smoke (and possibly also alcohol). Although the average length of time of smoking is cited as some 27 years for the Veterans and 17 years for controls, it is really the current tar intake which would be the crucial factor since as the report mentions it has been shown that the frequency of SCE decreases to background on cessation of smoking. Thus past smoking record would be unlikely to be a determinant of current SCE frequency. The author notes that '....an adjustment is made for smoking'. (p17, parag. 3). It is not made clear how this can be (or has been) done.

In summary, the data reported clearly shows a definite trend to higher frequencies of Veterans than controls showing heavily damaged cells. Thus, the question arises whether this trend is due to former radiation exposure and consequent genomic instability, or whether it is due to ongoing damage due to heavy consumption of tobacco and/or alcohol. The heavy consumption could presumably be ascribed to psychological stress arising from anxiety over possible long-term effects of the past exposure to radiation. While the effects of minute

(undetectable) amounts of radionuclides causing the elevated SCE levels in Veterans cannot be completely ruled out, smoking would seem to be the most likely cause of the effect seen. As a starting point for further studies the work is of value and should be published. However, it would be advisable and helpful if further data could be included concerning the smoking habits of the subjects.

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