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The following are a few brief comments on this report by M. A. Bender and A. B. Brill dated October 12, 1979:

1. In general, this is an excellent report.

2. The report accepts the dose measurements of Robinson et al. (1979) without providing the reader with any of the pertinent information needed so that he can judge its adequacy. For example, there is no breakdown of the dose between that which is external and that which is internal. There is no indication whether internal dose values include a contribution from the actinide alpha-emitters, yet one would expect that some of the islands have appreciable quantities of ^{239}Pu. It is not stated, but I assume their dose values are almost entirely from ^{90}Sr + ^{90}Y and ^{137}Cs plus ^{239}Pu. I would expect the contribution from other radionuclides to be negligible.

3. It seems odd that values are given only for total body dose. Since, as stated above, the dose is mostly from ^{90}Sr + ^{90}Y, ^{137}Cs and ^{239}Pu, one would expect the external dose to be primarily beta-dose because ^{90}Sr and ^{90}Y are pure beta-emitters and ^{137}Cs is a strong beta and x-ray emitter. One wonders if the beta bremstrahlung dose was included with the total body dose.

4. What would their estimate be on the skin cancer induction from this skin dose. UNSCEAR gives a wide variation of skin cancer coefficients of 2×10^{-7} to 1.8×10^{-5} skin cancers per person rem. I doubt these values apply here, however, because some of the beta-radiation in this case has high energy and can penetrate 1 cm into

tissue (i.e., far beyond the 0.007 cm penetration depth assumed by Standards setting bodies in estimating skin dose. Also, one should determine whether or not there are co-relations or synergistic relation between beta-radiation and UV as there are between UV-A and UV-B in the induction of skin cancer. One might suspect that skin cancer is the predominate malignancy on the sun baked islands.

5. Since a large fraction of the radioactive contamination on the islands should be $^{90}Sr + {}^{90}Y$, and since 99 percent of Sr is deposited in the skeleton, why did the authors not discuss bone dose and radiation induced bone sarcoma and carcinoma as well as leukemia from active bone marrow irradiation in the trabecular bone matrix?

Published values of bone cancer coefficients range from 2×10^{-6} to 2.2×10^{-4} cancers per person rem depending on age, radionuclide, type radiation, etc.

6. Some of the comparisons of population exposure given do not add to the quality of the report. If natural background radiation in the U.S. causes 6×10^{-4} (c/pr) 80 mrem/y $\times$ 220 $\times 10^{6}$ persons $\times 10^{-3} \approx$ 10,000 lethal cancers/y in the U.S., the objective should be to reduce this background radiation - especially that due to phosphate rock, etc. - and not use this as an excuse to permit more malignancies. One bad thing does not justify another! The comparison with exposures to radiation workers in the U.S. weakens the report.

7. It seems odd that these writers were able to use data from BEIR III report. I have been trying unsuccessfully to get a copy of this unpublished report for over a year. I guess the fact that this report is paid for by tax monies does not entitle university professors to a copy?

8. In estimating the genetic risk, it is not stated whether or not the risk was reduced by a factor of 10 (as is often the practice) because the exposures are at low dose and low dose rate, i.e.:

3 (dose rate effect for spermatogonia) x 2 (2 sexes)
x 2 (dose effect) $\approx$ 10.

Data of Lyon et al. (Nature New Biol. 101, July 1972) suggest use of this factor of 10 may not be warranted at very low dose rates.

9. When the authors suggested small doses of radiation might even be beneficial genetically, they might have added also that influenza

given as 6 x 10 $^{-}$ to 1.1 x 10 $^{-}$ genetic mutation/gentically signifi-cant rem. This upper value is greater than the upper value of cancer risk so the reader should be given the final estimates of genetic risk.

11. The report is in error in stating there are no human exposure data at low dose ranges, e.g. studies of in utero exposure and data on Hanford radiation workers are low dose studies.

12. The report uses only the linear and linear quadratic models, yet much of the data on human population exposure conforms best with a super linear model (e.g. effect = c $\sqrt{\text{dose}}$). In other words, the cancer coefficients are a power of dose less than unity in a number of cases or the cancers induced per rem are greater at low doses than at high doses because of overkill at high doses, damage to the reticuloendythelial system, etc.

13. It may not be a good assumption that the cancer risk on these islands is the same as that in the U.S. because the natural background radiation here is between 1/3 and 1/2 that in the U.S. and the Hanford radiation worker data suggest that about half the cancer per year in the U.S. are the result of natural background radiation.

14. I question that leukemia is one of the best understood cancers. The lack of leukemia induction by radiation in Olmstead County of Minnesota (Linos et al. - New Eng. J. Med. 1111, May 15, 1980) and in the Hanford worker data (Mancuso, Stewart, and Kneale) suggest that low chronic exposure to normal population (those not subjected to fire, blast, disease such an ankylosing spondylitis, etc.) die preferentially of forms of cancer other than leukemia.

15. There is a peculiar statement on page 28 to the effect that the BEIR III relative risk model gives a cancer risk 2 to 4 times the risk estimates of UNSCEAR 1977 and so it seems reasonable to accept the linear risk model instead.

16. Why was the life span of these islands chosen as 50 years? The U.S. life span is 70 years.

REPOSITORY PNNL

COLLECTION Marshall Islands

BOX No. 5682

FOLDER

DOCUMENT DOES NOT CONTAIN UCI

Reviewed by _____ Date _____