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Radiation Limits Revisited

NMBU scientists are co-authors of a study by the Norwegian Institute of Public Health (Folkehelseinstituttet, FHI) and published in Nature Communications (1). Together with other studies, the results reported in this paper suggest that current risk estimates for radiation damage may need to be revised.

All radiation can cause breaks in DNA with higher doses causing more damage than low doses. The challenge for regulatory authorities, therefore, has been to define radiation doses that constitute a significant risk for the health of humans and other organisms. The European Commission's latest recommendation from 2013 sets a dose limit for occupational exposure at 20 mGy (milligrays) per year. By comparison, average annual background exposures to radiation are about 3 mGy and a single X-ray exposure at the dentist's office corresponds to about 1 mGy.

In the experiments conducted by FHI, with FHI's Anne Graupner as senior author, mice were exposed to gamma irradiation at a dose rate of 1.4 mGy/h during 45 days. Even though a 1.4 mGy/h rate of exposure corresponded to approximately 4000-fold compared to background

exposures, the authors considered it to be a 'human relevant low dose rate'. Interestingly, the levels of DNA damage measured with the comet

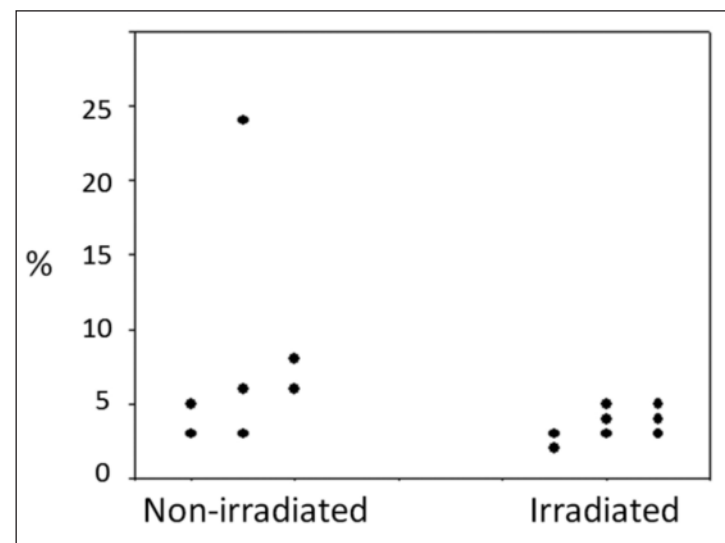


Fig. 1. Lower levels of DNA damage in irradiated versus non-irradiated mice. Irradiated mice were exposed to 1.4 mGy/h gamma radiation for 45 days before blood cells were examined for DNA damage (% tail DNA) with the comet assay. Each point shows % tail DNA results for an individual mouse. Figure is redrawn from results in reference 1.



Anne Graupner.

assay in blood cells were higher in non-irradiated mice compared to irradiated mice (Fig. 1). This result is similar to positive radiation effects reported in popular articles in both Aftenposten (2) and NBS Nytt (3). In the Aftenposten article, X-ray physician Tor Ole Kjellelland reviewed results on low dose

radiation which suggest that other factors may be playing a role at low dose rates. For example, several studies of human exposure to low dose radiation resulted in lower cancer incidences than would be expected by extrapolating from cancer incidences caused by acute exposures. A second consideration is

the extensive evidence that low dose exposures may protect against damage caused by subsequent acute exposures, a phenomenon known as hormesis.

Another relevant article on low dose radiation used plants as experimental models, showing that the repair of double-strand breaks after X-ray exposures is delayed because the expression of key enzymes (e.g. BRCA1 and RAD51) has to be induced (4). The induction of repair enzymes by low dose radiation may explain hormesis.

Kjellelland says, "There is now enough evidence to say that low dose radiation is not dangerous. Rather, it can be beneficial."

References

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4. Einset, J. and Collins, A.R. DNA repair after X-irradiation: lessons from plants. Mutagenesis 30 (2015) 45-50.

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