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Exaggerated Risk Perception of Low-Dose Radiation: Motives and Mechanisms

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Radiation is a known carcinogen because of the world-wide health scare that was created in 1960.[1] However, there is no evidence that radiation is a carcinogen below some threshold.[1,2] Prof. Edward Calabrese pursued the emergence of the linear no-threshold theory (LNT) and questioned whether it ever had a scientific basis.[2,3]

After the Chernobyl accident many publications appeared with LNT-based prognostications, for example, of millions of victims from nuclear accidents, reviewed previously.[4] Apparently, certain writers' exaggeration of medical and ecological consequences of the moderate anthropogenic increase in the background radiation contributed to a strangulation of the atomic energy, which was in the interests of fossil fuel producers.

Some dose-effect correlations may be attributed to a dose-dependent selection, self-selection and recall bias noticed in exposed cohorts.[4] It can be reasonably assumed that individuals, knowing that they had higher doses would be more motivated to undergo medical examinations being at the same time given more attention. Therefore, diagnosis of diseases would be on the average more likely in people with higher doses. For example, the dose-dependent increase in incidence of cardio- and cerebrovascular diseases among Mayak Production Association (MPA) workers was not accompanied by a corresponding elevation of mortality,[5,6] which can be attributed to a more frequent recording of mild cases in people with higher doses. The excess relative risk per unit dose for leukemia among MPA workers, using incidence data, has been considerably higher than that using mortality data.[7] A more efficient detection of latent leukemia with occasional registration of unverified cases is a probable explanation.

Elevated risks of non-malignant diseases have been found in Chernobyl, MPA, and Techa River populations.[4] For example, the excess relative risk of cerebrovascular diseases, per unit dose, among MPA workers was reportedly higher than in the atomic bomb survivors, where the exposure was acute and thus would be expectedly higher.[5,8] Remarkably, the dose-dependent incidence increase in cerebrovascular and ischemic heart disease among MPA workers was not accompanied by any increase in mortality,[5,6] which can be explained by a dose-dependent diagnostic efficiency with recording of mild and borderline cases in people with higher dose estimates. According to the same researchers, the incidence of cerebrovascular diseases was significantly increased among MPA workers with cumulative external doses ≥ 0.1 Gy.[8]

For comparison, UNSCEAR could not make any conclusions about immediate causal relationships between doses ≤ 1–2 Gy and excess incidence of cardiovascular or generally of non-malignant diseases.[9] The value 1–2 Gy may be an undervaluation due to bias in epidemiological studies. Doubtful correlations between low-dose exposures and non-malignant conditions call into question the cause–effect character of such correlations for malignancies reported by the same researchers.

A promising approach for research of dose-response relationships is lifelong animal experiments. Life span duration is known to be a sensitive endpoint, attributable to radiation exposures, which can reflect the net harm or potential benefit within a certain range, according to the concept of hormesis. The experimental evidence in favor of radiation hormesis is considerable. Admittedly, not all experiments supported hormesis, for example, showing no life lengthening of exposed mice; other studies did report life lengthening of rodents and other species; details and references are in reviews.[4,10]

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Finally, but not of least importance, unfounded suppositions about enhanced aggressiveness of malignancies, in areas contaminated with radioactive materials, may be conductive to overtreatment.[11]

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