

**A hypothesis: radiation carcinogenesis may result from tissue injuries and subsequent recovery processes which can act as tumor promoters and lead to an earlier onset of cancer**

Cancer risks in humans following an exposure to radiation are observed as increased mortalities when compared with mortality rates in unexposed people. The difference between the two mortality rates typically has been expressed as “fold increase.” Exposure of one gray of radiation increases cancer risks about 1.5-fold (or, 50%). This means that about one-third of the individuals exposed to 1 Gy of radiation and who died of cancer were affected by the exposure and about two-thirds were unaffected.

On the contrary, the present study reviewed past mouse-experiment data from a different perspective and observed that irradiation resulted in all mice dying prematurely. The data resulting from this new interpretation could not be explained by the idea that some mice were affected and the rest were not (that is, the idea that irradiation “increases” cancer mortality). How can this gap in understanding of radiation’s effect on mortality be explained in a reasonable way?

We realized if the idea that irradiation hastens the onset age of cancer, the increased mortality with exposure in humans was not inconsistent with the experimental results in mice. This is because cancer mortality even in unexposed people increases rapidly with increasing age, starting at around 50–60 years.

The idea that all cancers were affected by radiation exposure might at a glance seem unconventional, but radiation has long been known to cause inflammation in irradiated tissues. Our bodies work to heal this inflammation, and during that process, a variety of factors are secreted to repair the damage. As these factors are also beneficial for the growth of potential cancer cells, the hypothesis of earlier onset of cancer following irradiation makes good biological sense.

Based on this idea, cancer risks from radiation can now be expressed in a new way: namely, as a loss of life span. Moreover, if radiation exposure increases cancer risk through inflammation, it may become possible to reduce radiation-related cancer risks through pharmacological intervention to restrain the inflammation.

As the above hypothesis is untested, further study is necessary.

*doi: 10.1259/bjr.20190843*

RERF’s objective with this brief outline is to succinctly explain our research for the lay public. Much of the technical content of the original paper has been omitted. For further details about the study, please refer to the full paper published by the journal.