

## Research Shows Beneficial Effects of Low Doses of Radiation

By Jerry Cuttler

### Summary of the Work

Ron Mitchel, Senior Scientist in the Radiation Biology and Health Physics Branch at Chalk River Laboratories and three co-workers have just published an outstanding paper in the journal *Radiation Research*.<sup>\*</sup> It presents evidence that a single radiation dose of 10 or 100 milliGray\* (mGy) (1 or 10 rad) cobalt-60 gamma rays to cancer-prone, radiation-sensitive young (~50 day old) mice delayed cancer death substantially. The dose of 10 or 100 mGy is approximately 4 to 40 times the average natural radiation dose per year, but it was delivered at 0.5 mGy/min.

The research employed genetically modified Trp53+/- mice whose cells lack the cancer-fighting gene Trp53 in one of their two chromosomes. Such mice get cancer in middle age instead of old age, as would happen in normal mice (Trp53+/+ ones). Not only are Trp53+/- mice cancer prone, they are also radiation sensitive. Such mice do model cancer-prone and radiation-sensitive people including those who would be part of a workforce.

Normal mice have a mean life span of  $578 \pm 138$  days ( $1.6 \pm 0.4$  y). Exposure of these normal mice to the large dose of 4 Gy (400 rad) at a high dose rate results in a life-span loss of 125 days or 22% of their normal life. In comparison, Trp53+/- mice with a life span of  $375 \pm 103$  days (1.0 y), when exposed to 4 Gy, have their life reduced by 148 days or 40%. Trp53+/- mice are therefore more sensitive than normal mice to a high dose.

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### The Importance of This Work.

*Hormesis* is an adaptive response of living organisms to low levels of chemical, biological or radiological stress or damage – a modest overcompensation to a disruption – resulting in improved fitness. Observations of this reproducible phenomenon (low-dose stimulation and high-dose inhibition) have been widely reported in the scientific biomedical literature since the 1880s, and form the basis for all immunology treatment. Despite the growing body of evidence for adaptive response to low-level radiation, however, epidemiological (population) studies are generally inconclusive. The reason given is that the dose-response is lost against the noise of background cancer incidence. Public and occupational health policies, which rely on such studies, therefore revert to the Linear-No-Threshold (LNT) hypothesis. Unfortunately, misinterpretation of the LNT hypothesis, that it implies proof of a negative health effect at any level of radiation exposure, has contributed to societal radiophobia and anti-nuclear propaganda. This Chalk River paper reports on quality research carried out on radiation-sensitive animals, and demonstrates that a low dose of radiation, while neither increasing the average life span nor reducing the frequency of tumor initiation in the sample population, did provide protection against spontaneous cancer by delaying death due to cancer. This, and other related research, suggest the need to reconsider the conventional “ever lower is better” criterion for low-dose radiation limits. Such a policy shift would also enable the possible use of low-dose radiation therapy in many important medical applications.

*Jerry Cuttler*



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Radiation in low doses does not significantly affect the normal life span of these radiation-sensitive, cancer-prone mice; however, the effect on cancer latency (the time between spontaneous initiation and progress to malignant tumors which cause death due to cancer) is significant. Trp53+/- mice spontaneously develop a variety of cancer types. Two types that provide a good measure of latency are bone cancer in the spine (indicated by the onset of paralysis) and lymphoma (leading rapidly to death). The research demonstrated that the radiation exposures had little effect on tumor frequency, indicating no effect on tumor initiation. The 10 mGy exposure reduced the risk of both cancer types by increasing latency (by up to ~120 days for spine cancer); i.e. it reduced the rate at which spontaneously initiated cells progressed to malignancy. The effect of this adaptive response persisted for the entire life span of all the animals that developed these tumors. Exposure to 100 mGy delays lymphoma latency longer than the 10-mGy dose, however, the 100 mGy dose increased spinal cancer risk by decreasing latency compared to unexposed control mice. The higher dose is in the transition zone between reduced and increased risk; the dose at which the transition occurs varies with tumor type.

Stated more concisely, a small dose (10 mGy) did increase the latency period for lymphoma and for bone cancer; i.e., it did reduce the rate at which spontaneously initiated cells progressed to malignancy and the effect of this adaptive response persisted for the entire life span of all the animals that developed these tumors.

° Mitchel RE, Jackson JS, Morrison DP, Carlisle SM. Low Doses of Radiation Increase the Latency of Spontaneous Lymphomas and Spinal Osteosarcomas in Cancer-Prone, Radiation-Sensitive Trp53 Heterozygous Mice, *Radiat Res* 159 (3):320-7, 2003 March

\* A measure of radiation dose per unit animal weight (1 Gray is 1 Joule radiation energy per kg)

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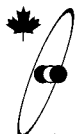
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