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# EVIDENCE SUPPORTING NONLINEAR EFFECTIVE THRESHOLD DOSE-RESPONSE RELATIONSHIPS FOR RADIATION CARCINOGENESIS

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Nonlinear effective threshold (NET) relationships describing the occurrence of radiation-induced cancer are the natural result of compensatory biological responses to cellular DNA. In addition, they are demonstrated by the application of nonlinear mathematical relationships in the analysis of epidemiological data. In this context, an effective threshold is a dose below which the cancer risk decreases to negligible (but not necessarily zero) and undetectable levels. Nonlinear relationships that display effective threshold doses are predicted when the normal background levels of biochemically-induced cellular DNA alterations are considered. Protracted radiation exposures from internally deposited radionuclides in beagles (including ✂ emitters $^{226}$Ra, $^{239}$Pu, $^{238}$Pu, and $^{241}$Am and 🖋 emitters $^{90}$Sr, $^{91}$Y and $^{144}$Ce) yield non-linear cancer induction as a function of time. At lower dose rates the long latency time required for development of radiation-induced cancer exceeds natural life span, yielding a life-span effective threshold for death associated with radiation-induced cancer at lifetime cumulative absorbed doses to the target tissues from about 0.9 to 1.4 Gy for ✂ irradiation or from about 28 to 70 Gy for 🖋 irradiation. The beagle results were scaled to predict human bone cancer risks for internally-deposited $^{226}$Ra yielding a life-span effective threshold of about 2 Gy to the skeleton; which agrees well with the U.S. human $^{226}$Ra data. Likewise, the predicted occurrence of human lung cancer from inhaled $^{239}PuO_2$ yielded a life-span effective threshold of about 1 Gy to the lung, which agrees well with recent human data from the former Soviet Union. Other studies have shown a significant reduction in carcinogenic potential associated with protracted radiation exposures in contrast to acute exposures.