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Jerome S Puskin
U.S. EPA

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LETTER TO THE EDITOR: REPLY TO COHEN’S RESPONSE TO EPA POSITION ON CANCER RISK FROM LOW LEVEL RADIATION

Jerome S. Puskin  □ Radiation Protection Division, U.S. EPA

The scientific basis for LNT has been repeatedly reviewed and supported by expert panels (BEIR, UNSCEAR, NCRP, ICRP, etc.). It rests on two main findings: (1) the epidemiological evidence on the Japanese atomic bomb survivors that there is an excess risk from low-LET radiation down to doses of about 0.1 Gy, with no dramatic deviation from linearity over the dose range from about 0.1 to 2 Gy; (2) a wide consensus that even a single track of ionizing radiation can produce clustered damage in DNA that may lead to a somatic mutation, which provides a plausible mechanism for a linear, no-threshold dose response for cancer induction at low doses. The panels have also concluded that, although anomalies may be found in some systems, the experimental data on carcinogenesis in animals are generally consistent with LNT so long as a DDREF adjustment is made in extrapolating from acute doses of greater than about 0.5-1 Gy to lower doses or to low dose rates.

My paper referred to several observed biological processes, which could conceivably modulate the dose response significantly at (low-LET) doses below 0.1 Gy (Puskin 2009), and Cohen (2010) cites others. However, so far, there has been little evidence that these processes act to preclude cancer induction by low dose ionizing radiation or even to reduce it significantly. Were this the case, a reduction in risk would be expected if the dose is delivered in multiple fractions or at low dose rates, but so far the epidemiological data on cohorts receiving multiple diagnostic exposures or chronic occupational or environmental exposures does not bear out this prediction (Puskin 2009).

Cohen refers to the microarray work by Yin et al. (2003), which showed a different pattern of activated genes at 0.1 Gy than at 2 Gy. But, as noted above, there is no strong indication of nonlinearity in the dose response for human carcinogenesis over this range, suggesting that the microarray results do not correlate well with proclivity for radiogenic cancer induction. Moreover, we already have evidence for risk at 0.1 Gy; the question is what happens at still lower doses, and here the microarray experiments are not at all informative. Likewise it has not been shown that the reports on immune system effects cited by Cohen have any bearing on risks from low dose chronic exposures.

Address correspondence to Dr. Jerome S. Puskin, Radiation Protection Division, ORIA (6608J), EPA, 1200 Pennsylvania Avenue, Washington, DC 20460; Phone: (202) 343-9212; Fax: (202) 343-2304; Email: puskin.jerome@epa.gov.

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My focus was on low-LET radiation because there is considerably less controversy over the linearity of the dose response for high-LET radiation—aside perhaps for the special case of bone cancer induction by bone-seeking alpha-emitters such as \(^{226}\text{Ra}\). Nevertheless, Cohen’s comments on bone cancer induced by internally deposited radium and on lung cancer due to radon exposure in homes should not stand unchallenged.

While it is true that the dose response for bone cancer induction in radium dial painters appears to be sublinear, and that no osteosarcomas have been observed among subjects receiving less than 10 Gy, this does not prove that there is a (practical) threshold, nor, in any case, does such a finding appear to be generalizable to other types of cancer. In particular, a positive association between indoor radon levels and lung cancer have been observed in case-control studies where the dose rate to presumptive target cells was about 2 orders of magnitude lower than in the bone cancer studies. The apparent increase in latency with decreasing dose rate mentioned by Cohen is likely to be a statistical artifact relating to the high probability of tumor formation at very high dose rates employed (Guess & Hoel 1977, Peto 1978). Rowland has concluded, moreover, that a “practical threshold” relating to an increased latency with decreasing dose rate was inconsistent with the dial painter data (Rowland 1994, p. 83).

Cohen continues to maintain that his observed negative correlation between lung cancer rates and average radon levels in U.S. counties implies that environmental radon poses little or no risk. A reexamination of that data has shown the negative correlation was likely due to confounding by smoking (Puskin 2003). There is also wide agreement that the residential case-control studies are a more reliable indicator than Cohen’s ecological approach and that those studies demonstrate a risk from relatively low concentrations of indoor radon (Heath et al. 2004, WHO 2009).

REFERENCES


