Thyroid Cancer Following Childhood Low Dose Radiation Exposure: Fallacies in a Pooled Analysis

Jerry M. Cuttler, D.Sc.
S.M. Javad Mortazavi, Ph.D.
James S. Welsh, M.D.,
Mohan Doss, Ph.D.

According to the linear no-threshold (LNT) hypothesis, there is no safe dose of ionizing radiation. Predictions of cancer in a small proportion of the persons exposed to low doses are the rationale for opposing nuclear energy and for mass evacuations in the event of a radioactive release, as at Fukushima. Such predictions also restrict the use of beneficial nuclear technology in medicine. Evidence of actual excess cancers attributed to low doses is generally restricted to thyroid cancer.

A recent article by Lubin et al.,1 which analyzes nine cohorts, illustrates common pitfalls.

The authors report that for doses <0.2 gray (Gy) and <0.1 Gy, relative risk (RR) increased with thyroid dose (P<0.01), without significant departure from linearity (P=0.77 and P=0.66, respectively). They conclude: “These analyses reinforced the existence of an excess thyroid cancer risk at doses <0.2 Gy and <0.1 Gy, and perhaps at even lower doses” and “reaffirm that the direct application of a linear relationship remains the most plausible approach for the extrapolation of radiation-associated thyroid cancer risk and adds support to the use of a linear model for ALARA [as low as reasonably achievable] assessments.” Cohorts included two of childhood cancer survivors; six of children treated for benign diseases; and one of children who survived the atomic bombings in Japan.

There is no indication that this study controlled for the myriad of confounding factors that affect cancer incidence. These include genetics, which affects susceptibility, and the significant incidence of occult thyroid cancer that depends on geographic location.

Screening for thyroid cancer has been shown to result in enormous overdiagnosis. A population-based trend study in Switzerland from 1998 to 20122 showed that the age-standardized annual incidence of thyroid cancer increased from 5.9 to 11.7 cases/100,000 among women (annual mean absolute increase: +0.43/100,000/year) and from 2.7 to 3.9 cases/100,000 among men (+0.11/100,000/year). The increase was limited to the papillary subtype, the most indolent form of thyroid cancer. There was no comitant rise in mortality, and the screening may have resulted in unnecessary thyroidectomies. South Korea’s thyroid cancer “epidemic”3,4 was the result of screening and overdiagnosis. Hoang and Nguyen5 concluded that indiscriminate workup of incidental thyroid nodules is not cost-effective and is potentially harmful.

While Lubin et al. model radiation-induced cancer using a linear relationship, the 1956 National Academy of Sciences (NAS) recommendation to use the LNT model to assess the risk of radiation-induced mutations (cancer) has been progressively discredited for the past 8 years.6

It is well known that DNA mutations overwhelmingly result from attack by reactive oxygen species, which are produced abundantly and constantly by aerobic metabolism. All organisms have powerful protection systems, which prevent, repair, and remove damaged cells. The rate of mutation induction by low-level radiation is negligible when compared with the rate of endogenously-induced mutations.7 Low dose radiation stimulates the protection systems, resulting in a reduction in mutations.8 The immune system destroys cancer cells, and therefore cancer generally appears when the immune system has been weakened or damaged.9 Low-dose radiation stimulates immunity,10 so the idea that increased thyroid cancer follows an exposure to low-dose radiation simply contradicts biology.

In 1957 the UK had its most serious nuclear accident when there was a fire at the Windscale reactor No. 1 and plutonium production plant in Northwestern England. Emergency measures “started with the knowledge that cancer of the thyroid in children had been known to occur following X-ray doses greater than 200 rad (2 Gy). No cases were known to have occurred following exposures to smaller doses.”11 A recent study of the leukemia incidence of 97,000 Hiroshima survivors identified a threshold at about 500 mSv.12 Since the blood-forming cells are more sensitive to radiation than the thyroid gland, it is reasonable to expect the threshold for radiation-induced thyroid cancer to be higher than 500 mSv.

Radioiodine has been employed to treat hyperthyroidism for more than 70 years. The Franklyn et al.13 study of many cancer rates following this treatment showed there was a significant increase in rare thyroid cancer mortality; however, “the decrease in overall cancer incidence and mortality…is reassuring.” Continuing concerns about the risk of cancer have led to many other studies. The review by Cuttler and Pollycove in 2009 did not identify a conclusive link between low doses of radiation and thyroid cancer.14

The natural history of thyroid cancer strongly suggests the existence of self-limiting cancers, which are truly malignant but do not progress to lethal cancers, a first-time observation in the history of medicine. Early detection of self-limiting cancers results in overdiagnosis. Ultrasonographic screening of the thyroid in the young should be avoided. Lethal thyroid cancers, whose origin is still unknown, appear suddenly after middle age, writes Dr. Toru Takano.15

In conclusion, the Lubin et al. study on thyroid

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cancer following low-dose exposure lacks credibility. The radiation level needed to induce thyroid cancer is far above environmental levels, even after a hypothetical severe accident. Applying ALARA is not necessary for protection; it is very detrimental. The ALARA standard sustains the unwarranted cancer scare and impairs important applications of low-dose radiation in the diagnosis and treatment of serious illnesses, as well as in industry.

Mass screening for thyroid cancer after low-dose radiation exposure has led to unnecessary treatment with its inherent risks and has not been shown to save lives.

Jerry M. Cuttler, D.Sc., is a nuclear scientist. Contact: Cuttler & Associates Inc, 1104 – 11 Townsgate Drive, Vaughan, Ontario, L4J 8G4, Canada. Email: jerrycuttler@rogers.com. S.M. Javid Mortazavi, Ph.D., is a visiting scientist, Department of Diagnostic Imaging, Fox Chase Cancer Center, 333 Cottman Ave., Philadelphia, PA 19111. James S. Welsh, M.S., M.D., F.A.C.R.O., is a radiation oncologist, Department of Radiation Oncology, Stritch School of Medicine, Loyola University-Chicago, 2160 S. 1st Ave., Maywood, IL 60153. Mohan Doss, Ph.D., M.C.C.P.M., is a medical physicist, Department of Diagnostic Imaging, Fox Chase Cancer Center, 333 Cottman Ave, Philadelphia, PA 19111.

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