Thyroid Cancer after Chornobyl
Increased Risk Persists Two Decades after Radioiodine Exposure

During the 1986 Chornobyl nuclear accident, the then-Soviet republic of Ukraine was hit hard with iodine-131 fallout. Since then, numerous studies have demonstrated a relationship between I-131 exposure from Chornobyl and thyroid cancer risk. Much of the published research, however, has relied on grouped radiation dose estimates rather than individual estimates of radiation exposure. Now a new study using measurement-based individual dose estimates has shown the risk of developing thyroid cancer after I-131 exposure persists two decades later [EHP 119(7):933–939; Brenner et al.].

The U.S. and Ukrainian authors studied 12,514 individuals who in 1986 were under 18 years of age and living in one of three Ukrainian oblasts (states) contaminated with I-131 fallout from Chornobyl. The individuals’ I-131 exposure from the Chornobyl accident had been estimated using individual radioactivity measurements taken within two months of the accident, interview data, and ecological models of the fallout pattern. The study participants also were screened for thyroid cancer and other thyroid diseases a total of four times between 1998 and 2007. The current study did not include people who had been diagnosed with thyroid cancer during the first screening examination (1998–2000).

Sixty-five people in the cohort were diagnosed with histopathologically confirmed thyroid cancer after the first screening examination. Statistical analysis indicated the dose response was linear and that the estimated risk of thyroid cancer almost doubled for every Gray (a unit of absorbed radiation) of exposure. There was no reduction in the estimated radiation risk over time. Estimated risk varied significantly by oblast of residence. It also rose with younger age at exposure, and females were at a slightly increased risk over males, as were people with benign thyroid conditions such as diffuse goiter. However, none of these differences were statistically significant.

An earlier analysis of pooled data on external irradiation and thyroid cancer in other populations suggested the increased risk associated with radiation peaked 15–19 years after exposure but was still apparent 40 years later. It is unknown whether long-term effects of I-131 on thyroid cancer risk follow a similar pattern. The study’s comprehensive and detailed analyses strengthen existing evidence relating I-131 exposure and thyroid cancer risk; additional follow-up of this cohort is necessary to more accurately describe excess risk by time since exposure.

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Plastics and Food Sources
Dietary Intervention to Reduce BPA and DEHP

Purchasing fresh, unpackaged foods and avoiding the use of plastic food processing and storage equipment may help consumers minimize their exposure to bisphenol A (BPA) and bis(2-ethylhexyl) phthalate (DEHP), according to a small but systematic study of associations between diet and people’s levels of these chemicals [EHP 119(7):914–920; Rudel et al.].

At the time of this January 2010 study, BPA and DEHP were used widely in food packaging, including cans, plastic wraps, and food storage containers.1 Evidence from in vitro and animal studies demonstrates BPA’s potential to disrupt endocrine function in a number of organ systems. Animal studies have linked exposure to DEHP to inhibition of testosterone synthesis and adverse effects on the developing male reproductive system. Some epidemiologic evidence also links urinary levels of phthalate metabolites to effects on boys’ reproductive development, male hormone levels, semen quality, and neurobehavioral end points.

Although some scientists believe diet is likely to be a major source of exposure to BPA and DEHP, few empirical data exist to verify this belief. One of the stated goals of the current study was to assess the relative importance of food packaging as a source of exposure to these chemicals. BPA and DEHP are relatively easy to study because they have short biological half-lives and exposure biomarkers that can be measured in urine.

The study involved five 4-person families, including adults and children, who were chosen in part because they reported frequently eating canned foods. Urine samples were collected over 8 days. On days 3–5 family members consumed only food prepared by a caterer who avoided using plastic (including plastic utensils and storage containers) in preparing and packaging the meals and snacks made from fresh ingredients. Families were given stainless steel water bottles and lunch boxes and advised to use only the containers provided. Coffee drinkers were instructed to use a French press or ceramic drip in place of a plastic coffee maker.

When consuming their customary diets, family members’ urine levels of biomarkers for both BPA and DEHP were in the range of the general U.S. population’s, as estimated by the Centers for Disease Control and Prevention’s National Health and Nutrition Examination Survey program. During the time the families were on the fresh-food diet, the geometric mean concentration of BPA dropped by 66%, and mean concentrations of DEHP metabolites decreased by 53–56%.

The researchers note they cannot determine exactly which changes in food sourcing and handling caused the observed exposure changes. In addition, their intervention did not completely eliminate all sources of BPA and DEHP. They state food contamination may occur during premarket processing of whole foods or from the presence of phthalates and BPA in the environment where the food originates.

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NOTE
1. After this study was published online, a representative of the American Chemistry Council sent a letter to EHP’s editors stating that “to the best of the industry’s knowledge, [DEHP] is no longer used in the food packaging products that the authors removed from the subjects’ dietary routine.” The letter, which will appear in a future issue of EHP, does not state when these uses of DEHP were discontinued.