

Chemistry

Janet Raloff reports from New York City at the spring national meeting of the American Chemical Society

Geologically induced goiters

Worldwide, an estimated 400 million people suffer from goiter, or enlarged thyroid glands — a condition that can, if left untreated, lead to reduced thyroid function. While the majority of goiters can still be traced to insufficient dietary iodine, roughly 25 percent cannot. Many of the 100 million persons who fall into this latter category may be victims of goiter-producing geological factors, according to Eduardo Gaitan, chief of endocrinology at the University of Mississippi School of Medicine in Jackson. Studies that he and his collaborators from a number of institutions have conducted in Colombia and the United States now indicate that natural environmental pollutants in drinking water appear capable of inducing goiter and interfering with thyroid function.

For example, in some Appalachian coal-mining regions of Kentucky, despite adequate iodine, “as many as 37 percent of the children have enlarged thyroid glands,” Gaitan says. (Children are more susceptible to goiter, he notes, and if untreated can suffer physical and mental development problems.) Their studies in these regions over the past four years now show that resorcinol, a known thyroid inhibitor, is in much of the well water serving the area. “We also found other compounds in the water — phthalates — which under the action of bacteria can be transformed into dihydroxy benzoic acids,” Gaitan says. Research by his team has shown that these can also interfere with thyroid function. Recently, they found both compounds in the drinking water serving high-goiter areas in Colombia.

Resorcinol and dihydroxy benzoic acids are among coal-processing wastes, their work shows. Gaitan says it thus appears “the geological composition” of the Kentucky and Colombian areas — especially limestone rich in organic compounds such as coal — may be contributing these compounds.

While many goiter symptoms affecting the Colombian and Kentucky populations they study are similar, all of their contributing sources may not be. In two high-goiter counties of Kentucky, Gaitan’s group found that a third of affected children “make antibodies against their own thyroid glands” — a condition known as autoimmune thyroiditis. No similar antibodies were identified in any of the Colombians studied. Moreover, Gaitan points out, two chemicals isolated from the Kentucky water — methoxy anthracene and bromoform — are not found in the Colombian water. Studies conducted by other researchers have shown that buffalo rats fed these two compounds will develop autoimmune thyroiditis, Gaitan says.

Goiter? Do you eat millet?

Over the past five years or so, another natural source of goiter has been identified: flavonoids — plant pigments providing most of the red, yellow and blue coloring in flowers and fruits. New data from Robert Cooksey and his colleagues at the Veterans Administration Medical Center in Jackson, Miss., the University of Alabama at Birmingham and the National Research Council of Canada in Saskatoon, show that flavonoids in the hulls of millet, when broken down by bacteria in the gut, can be transformed into resorcinol; a functionally similar variant known as “substituted” resorcinol; dihydroxy benzoic acids; and yet another potent thyroid-function inhibitor, ferulic acid. This may explain, at least in part, the previously puzzling correlation between millet consumption and goiter incidence in many developing countries, Cooksey notes.

But flavonoids may not be the whole story. The researchers have found another antithyroid compound in millet, thiocyanate, which when consumed with the flavonoid has an additional inhibitory effect on thyroid function. Most perplexing, they found that when millet is cooked and then stored for a week — a practice common in many cultures — its antithyroid activity increases six-fold.

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Environment

Long-UV light may cause cancer . . .

In recent years, suntanning salons have sprung up in the United States and Europe claiming to use “safe” wavelengths of ultraviolet (UV) light. Their tanning lamps emit wavelengths primarily in what is known as the longer, or “A,” portion of the UV spectrum. But a new Food and Drug Administration (FDA) study indicates these lamps may not be safe after all.

The study found that UV-A — the most prevalent form of UV in sunlight penetrating to earth’s surface — can cause mutations in cultured mouse lymphoma cells, a standard screening test for potential human carcinogens. According to C. David Lytle, acting director of the biophysics division at FDA’s Center for Devices and Radiological Health, in Rockville, Md., “We interpret these findings as indicating the potential of the radiation to cause skin cancer.” Unlike shorter-wavelength UV-B — which is also present in sunlight and is believed to play an active role in skin cancer formation — UV-A has generally been considered virtually benign.

The tests, conducted at the FDA laboratory in Rockville by Victoria Hitchins, exposed the cells to pure UV-A, using only wavelengths in excess of 340 nanometers. Mutation rate increased with dose, based on single exposures of up to 60 joules per square centimeter. In fact, Hitchins told SCIENCE NEWS, at the highest dose — one comparable to what manufacturers of their lamp suggest for humans seeking a tan — 90 percent of the cells died. However, Lytle adds, the cells used in this test were growing fast and were unprotected by an outer skin layer, the stratum corneum. As a result, he says, one would expect the test cells “to be much more sensitive than the [vulnerable] cells in somebody’s skin.”

Recent studies at Argonne (Ill). National Laboratory have shown that UV-A can damage DNA in both bacteria and human cells via mechanisms quite different from those of shorter-wavelength UV (SN: 11/30/85, p. 342). In fact, Lytle says, Hitchins’s new data “are very consistent with that [the Argonne findings], and are basically a continuation of that type of work.”

Lytle says it’s not yet clear whether such data will have much effect on UV-A tanning salons, since their lamps already must carry timers and labels warning that the light may contribute to aging and cancer. But it’s possible, he says, that these new data may provide ammunition “for the Federal Trade Commission to keep people from advertising these devices as ‘safe.’”

. . . and destroy natural anticarcinogens

UV-A exposures also reduce the blood levels of carotenoids — plant substances suspected of offering some natural protection against cancer — according to research by Daphne Roe, a Cornell University nutritionist, reported recently in St. Louis at the annual meeting of the Federation of American Societies for Experimental Biology. Carotenoids, of which beta-carotene is the best known example, are the yellow pigments in plants, obtained by eating such foods as squash and tomatoes.

In separate studies, 12 women and 12 men were exposed to 11 UV-A exposures over a two-week period, receiving a skin dose from each less-than-3-minute exposure of on the order of 20 to 25 joules per square centimeter. That’s “not a casual exposure,” Roe says, but instead what might be encountered in very intense sunlight. As a result of these exposures, total-carotenoid levels in blood dropped on the order of 30 percent, something Roe terms “very significant indeed.”

She says these data “strongly suggest that sunlight can break down beta-carotene in white and oriental people who are repeatedly exposed to average summer sunshine,” thereby limiting much of the carotenoid’s potential “protective benefits against many kinds of cancer.” She hopes to conduct follow-up studies in East Africa to see if intense sun exposures there have similar effects on dark-skinned people.

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