

COMMENTARY:
NEANDERTALS AND THE THYROID GLAND:
THE SELENIUM CONNECTION

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*I*n an intellectually stimulating *Geographical Review* article on “The Iodine Factor in Health and Evolution,” Jerome Dobson argues that “distinctive Neandertal skeletal traits are identical to those of modern humans who suffer from cretinism.” He further proposes that “this new evidence, coupled with recent mitochondrial DNA findings, suggests that a single genetic alteration, which improved the ability of the thyroid gland to extract and utilize iodine, may account for differences between Neandertals and modern humans” (1998, 1). Clearly, if Dobson is correct, the key to understanding the Neandertal enigma is the operation of the Neandertal thyroid gland (Shreeve 1995).

THE THYROID GLAND

In adult humans the body content of iodine is between 20 and 50 milligrams. Most of this trace element is concentrated at the anterior base of the neck, in the thyroid gland. All vertebrates require iodine and possess a similar gland or tissue somewhere in their bodies (Mervyn 1985). Dietary iodine is rapidly absorbed by the intestinal tract, chiefly as iodide, and is then transported by the blood to the thyroid gland. There iodide is oxidized to iodine and combined with the amino acid tyrosine to produce mono- and di-iodotyrosines. Further conversion, which results in the formation of the hormones thyroxine (T^4) and triiodothyronine (T^3), takes place in the epithelial cells of this gland. Both hormones are essential for human health and are involved in brain development, growth, and metabolism. Mentality, speech, and the condition of the teeth, skin, hair, and nails are all dependent on a thyroid gland that functions well (Nutrition Search 1984; Mervyn 1985).

Not only is iodine essential for the production of triiodothyronine, selenium is, as well. Researchers from the Hahn-Meitner Institute in Berlin first discovered that selenium plays a key role in deiodinase, an enzyme required to catalyze the conversion of thyroxine to triiodothyronine. When a selenium deficiency is present, deiodinase levels are depressed, and so too is the production of triiodothyronine (Contempré, Vanderpas, and Dumont 1991; Toro 1991). Depressed serum thyroxine levels, however, do not necessarily imply low serum triiodothyronine. When severe iodine deficiency is present, serum triiodothyronine usually remains at normal levels, or it may even rise as thyroxine levels fall (Pharoah, Buttfield, and Hetzel 1971). This is because triiodothyronine contains less iodine, weight for weight, than does thyroxine yet is more active metabolically. It is thus produced in quantity by the

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