

Moscow, 22 April 2006 - Georges MOUTON MD

THYROID GLAND, POLLUTION AND AGEING

The incidence of thyroid disease in elderly patients was previously unrecognized [1]. Since, several publications have demonstrated the high prevalence of subclinical hypothyroidism in elderly subjects and the increased prevalence of *auto-immune thyroiditis* among these patients [2]. Though the prevalence of thyroid auto-antibodies steadily increases through the decades, being tightly age-dependant, this phenomenon is not seen after the ninth decade of life. Such observations lead to the concept of "**healthy centenarians**" [3].

Radioactive iodine has been produced by atomic bomb testing (such as the 1951 Buster-Jangler test series in United States of America) and by nuclear accidents (such as the 1986 Chernobyl accident in Europe). But in fact, exposure to ionizing radiation represents the only established environmental risk factor for thyroid carcinoma [4]. Besides, exactly as therapeutic use of radioactive iodine does in case of hyperthyroidism (due to Basedow's disease), it also can progressively induce hypothyroidism.

Thyroid hormonogenesis may also be impaired by other environmental chemicals. Wildlife observations in polluted areas clearly exhibit a significant increase in the incidence of goiters and thyroid imbalances [5]. Among most harmful xenobiotics for the gland are **polycyclic aromatic hydrocarbons (PAHs)** [6], **dioxins** [7, 8], **polychlorinated biphenyls (PCBs)** [7-10], **organochlorinated pesticides** (among which **DDT**) [8, 9] and **brominated flame retardants (BFRs)** [11]. These groups of chemicals have a striking similarity with the thyroid hormones and interfere with thyroid hormone homeostasis [8, 11].

Exposed populations show a significantly higher frequency of thyroid auto-antibodies (especially anti-thyroperoxidase), an increased thyroid volume (also expanding with age), more thyroid nodules and more frequent hypoechogenicity shown by ultrasound [9, 10].

Thyroxine (T4) can be considered as a prohormone, which can be transformed into the much more active triiodothyronine (T3) from a deiodination performed by **5'-deiodinase**. This reaction doesn't occur in the thyroid gland, but in the periphery, especially in the liver and kidneys. Oppositely, the inactive reverse T3 (RT3) is produced from T4 by **5-deiodinase**. Higher serum RT3 levels among the elderly seem to result from a decreased metabolism of thyroid hormones due to the aging process itself [12].

All forms of stress - physiological, pathological and emotional - block the activity of **5'-deiodinase** in favor of **5-deiodinase**, leading to a lack of active T3 and to an excess of inactive reverse T3. Several **heavy metals** [13], such as **mercury** [14], **lead** [15] and **cadmium** [16], impair the activity of **5'-deiodinase** and may induce clinical hypothyroidism.

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