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Letter to Editor

Familial Goiter and Related Disorders

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Synthesis, storage, secretion, delivery, and utilization of thyroid hormones are dependent upon specific enzymatic activity; inherited disorders may cause thyroid disease. Studies of a cretinous patient from consanguine marriage have indicated a limited response to thyrotrophic (TSH), both in synthesis of the specific thyroid protein thyroglobulin and in cell growth and division. Other similar response has been observed in a strain of mice. Familial goiter and hypothyroidism may occur when there is failure of the thyroid cell to transport iodide. Familial goiter may also result from failure to convert inorganic iodide into iodine in the thyroid gland. Several families of patients have been studied who have a defect in the coupling of iodotyrosines into iodothyronines. Family groups are known to be unable to deiodinate iodotyrosines. Familial goiter may occur in humans and sheep because of impaired synthesis of thyroglobulin. A syndrome of famitial goiter, nerve deafness, and stippled epiphyses has been described. Familial hypothyroidism may arise from several different disorders of hypothalamic or pituitary control of the thyroid. In most of the above conditions, errors are inherited as autosomal recessive traits.

In our perspective, common “primary” hypothyroidism could be a subtle epigenetic embryo-foetal disorder with anatomo-clinic stages of histologic damage of the thyroid gland and even the peripheral metabolism of thyroid hormones on tissues effectors.

Obesity could be a molecular tissues effectors peripheral thyroid hormones metabolism disorders.