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Iodine intake as a determinant of thyroid disorders in populations

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Depending on the availability of iodine, the thyroid gland is able to enhance or limit the use of iodine for thyroid hormone production. When compensation fails, as in severely iodine-deficient populations, hypothyroidism and developmental brain damage will be the dominating disorders. This is, out of all comparison, the most serious association between disease and the level of iodine intake in a population.

In less severe iodine deficiency, the normal thyroid gland is able to adapt and keep thyroid hormone production within the normal range. However, the prolonged thyroid hyperactivity associated with such adaptation leads to thyroid growth, and during follicular cell proliferation there is a tendency to mutations leading to multifocal autonomous growth and function.

In populations with mild and moderate iodine deficiency, such multifocal autonomous thyroid function is a common cause of hyperthyroidism in elderly people, and the prevalence of thyroid enlargement and nodularity is high. The average serum TSH tends

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to decrease with age in such populations caused by the high frequency of autonomous thyroid hormone production.
On the other hand, epidemiological studies have shown that hypothyroidism is more prevalent in populations with a high iodine intake. Probably, this is also a complication to thyroid adaptation to iodine intake. Many thyroid processes are inhibited when iodine intake becomes high, and the frequency of apoptosis of follicular cells becomes higher. Abnormal inhibition of thyroid function by high levels of iodine is especially common in people affected by thyroid autoimmunity (Hashimoto's thyroiditis).
In populations with high iodine intake, the average serum thyroid-stimulating hormone (TSH) tends to increase with age. This phenomenon is especially pronounced in Caucasian populations with a genetically determined high tendency to thyroid autoimmunity. A small tendency to higher serum TSH may be observed already when iodine intake is brought from mildly deficient to adequate, but there is at present no evidence that slightly elevated serum TSH in elderly people leads to an increase in morbidity and mortality.
Conclusion: Even minor differences in iodine intake between populations are associated with differences in the occurrence of thyroid disorders. Both iodine intake levels below and above the recommended interval are associated with an increase in the risk of disease in the population. Optimally, iodine intake of a population should be kept within a relatively narrow interval where iodine deficiency disorders are prevented, but not higher. Monitoring and adjusting of iodine intake in a population is an important part of preventive medicine.

Iodine is relatively abundant in the aquatic environment of the earth, the sea, but scarce in most parts of the terrestrial environment. To compensate for the low amount of iodine available for thyroid hormone production, a series of mechanisms have developed in human and other mammals.

When the supply of iodine is below a certain limit, autoregulatory intrathyroidal mechanisms enhance the activity of the many processes involved in the use of iodine for production of thyroid hormones.[1] If this adaptation is not sufficient to keep thyroid hormone production normal, a further enhancement of thyroid activity will take place through hypothalamic/pituitary feedback, leading to an increase in the synthesis and secretion of thyroid-stimulating hormone (TSH). Moreover, there are other mechanisms both inside the thyroid and in the peripheral tissues that tend to stabilise the production of active thyroid hormone.[2] Assisted by such mechanisms, even small supplies of iodine will be sufficient to keep the thyroid function at a level allowing reproduction and other activities necessary for the survival of the species.

However, the reproduction and survival may be impaired not only by insufficient thyroid hormone production caused by iodine deficiency, but also by excessive thyroid hormone production, leading to thyrotoxicosis. If all thyroid processes involved in use of iodide are up-regulated to compensate for iodine deficiency and the individual then suddenly is exposed to iodine excess, for example, from eating seaweed, then the individual is prone to develop hyperthyroidism. Fortunately, the thyroid also harbours mechanisms that almost immediately detect iodine excess and subsequently down-regulate the processes involved in thyroid hormone synthesis and secretion[3] to protect against hyperthyroidism.

Thus, the thyroid gland contains an advanced set of processes that may enhance or block the use of iodine for thyroid hormone production. A likely cause for many thyroid disorders is some degree of complication to this complexity of processes. Because the processes activated during low and high iodine intakes are different, the type of disease that dominates thyroid pathology in populations may be different depending on the level of iodine intake.