Thyrotropin-Releasing Hormone

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Thyrotropin-releasing hormone (TRH), also called thyrotropin-releasing factor (TRF), thyroliberin or protirelin, is a tropic, tripeptidal hormone that stimulates the release of thyroid-stimulating hormone and prolactin by the anterior pituitary.

Excess levels inhibit dopamine, which stimulates the release of prolactin, which in turn decreases GnRH.

A TRH test may be indicated if secondary hypothyroidism is suspected. Some patients may have low levels of circulating thyroid hormones and secondary hypothyroidism as a result of damage to the hypothalamic or pituitary control mechanisms that regulate thyroid function. The hypothalamus secretes the thyrotropin-releasing hormone that directs the synthesis and secretion of TSH from the pituitary gland. If these normal regulatory mechanisms are interrupted, the pituitary may not be able to produce appropriate levels of TSH and levels of thyroid hormones may decline, although the TSH remains appropriately normal.

Growth Hormone

An increase of serum GH concentration followed TRH injection in patients with primary hypothyroidism. (Hamada, Uoi et al. 1976)

Cancer-Related Fatigue

A randomized controlled study found that thyrotropin-releasing hormone may be useful as a treatment for cancer-related fatigue. TRH administration was associated with significant improvement in fatigue and was also associated with a positive impact on quality of life. TRH administration was associated with transient increases in blood pressure and heart rate. (Kamath, Feinn et al. 2011)

Melatonin

Walter Pierpaoli published a report on the experimental evidence that, while melatonin alone exerts a low-level age-postponing activity, its age-delaying effects are greatly enhanced and accelerated when given in combination with a pineal peptide, thyrotropin-releasing hormone (TRH). (Pierpaoli, Bulian et al. 1999)

Obesity

A recent study examined the effects of long-term intraperitoneal injection of thyrotropin-releasing hormone (TRH) on aging- and obesity-related changes in body weight, lipid metabolism, and thyroid functions on anterior hypothalamus-lesioned obese mice and genetically obese mice. The treatment provoked a mobilization of triglycerides in the peripheral blood, a decrease of leptin and a loss of body weight. The weight loss did not depend on TSH-mediated stimulation of thyroid hormone secretion with consequent metabolic hyperthyroidism. The levels of blood cholesterol were not affected or even suppressed. Even at a very high dosage TRH did not affect the obesity of genetically obese mice. (Pierpaoli and Lesnikov 2011)

References

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