Chronic fatigue syndrome possibly explained by lower levels of key thyroid hormones

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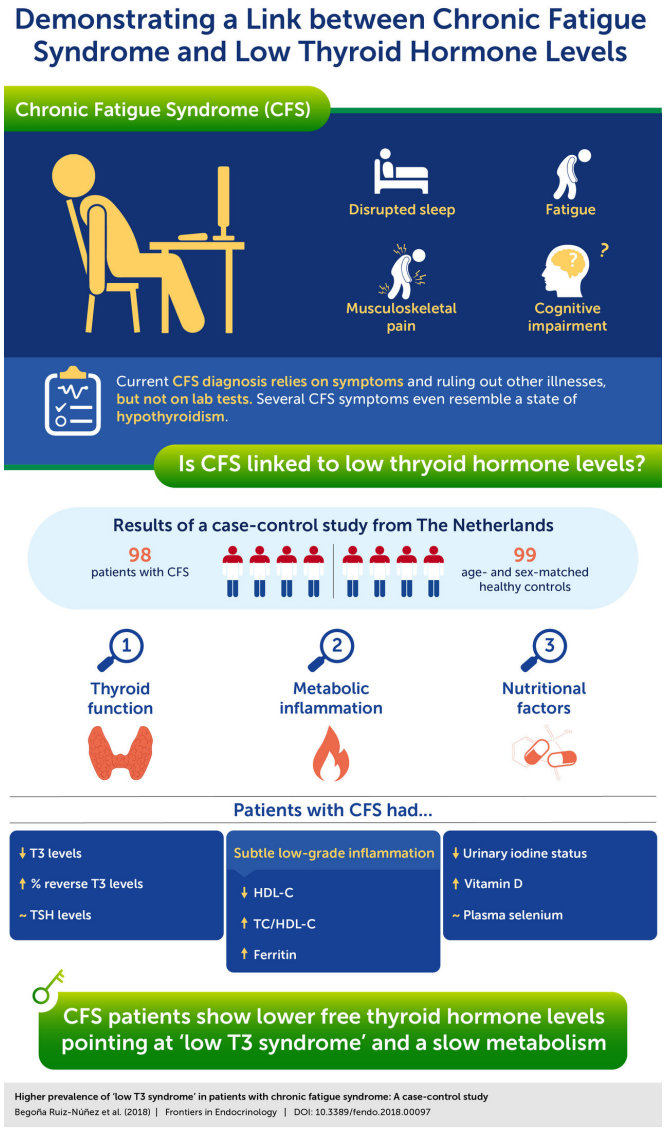

Overview of the study methods and results, which demonstrate a link between chronic fatigue syndrome symptoms and lower thyroid hormone levels Credit: © 2018 Ruiz-Núñez, Tarasse, Vogelaar, Dijck-Brouwer and Muskiet. *Front. Endocrinol*. doi: 10.3389/fendo.2018.00097

New research demonstrates a link between chronic fatigue syndrome (CFS) symptoms and lower thyroid hormone levels. Published in *Frontiers in Endocrinology*, the study indicates that CFS, a condition with unknown causes, can be explained by lower thyroid hormones—but may be distinct from thyroidal disease. This finding can be seen as a first step to finding treatment for a debilitating illness for which there is no recognized treatment.

Chronic fatigue syndrome is a common disease marked by lengthy spells of weakness, fatigue and depression. Its diagnosis is predominantly based on symptoms and on ruling out any underlying medical condition, rather than on laboratory tests and physical examination.

Interestingly, several symptoms resemble those of hypothyroidism—a condition where the thyroid gland does not produce enough thyroid hormone. In hypothyroidism, the body tries to encourage thyroid hormone activity by releasing more thyroid-stimulating hormone—however, this does not happen in patients with chronic fatigue syndrome.

This contrast in thyroid-stimulating activity led the study's authors to hypothesize that chronic fatigue syndrome is caused by low activity of thyroid hormones in the absence of thyroidal disease.

Led by Dr. Begoña Ruiz-Núñez at the University Medical Center Groningen, The Netherlands, the researchers compared thyroid function and markers of inflammation between 98 CFS patients and 99 healthy controls. Remarkably, the CFS patients had lower serum levels of certain key thyroid hormones such as triiodothyronine (T3) and thyroxine (T4), but normal levels of thyroid-stimulating hormone.

Additional analyses indicated that CFS patients had a lower urinary iodine status and low-grade

inflammation, which possibly mirrored the symptoms of patients with hypothyroidism. These CFS patients, however, had relatively higher levels of another thyroid hormone called "reverse T3" or rT3. This appeared to be due to a shift in hormone production, where the body preferred to convert T4 to rT3 instead of producing T3. The low T3 levels found in CFS patients coupled with this switchover to rT3 could mean that T3 levels are severely reduced in tissue.

"One of the key elements of our study is that our observations persisted in the face of two sensitivity analyses to check the strength of the association between CFS and thyroid parameters and low-grade inflammation," says Dr. Ruiz-Núñez. "This strengthens our test results considerably."

The researchers believe inclusion of patient information, such as duration of illness, would enable a correlation with their biochemical profiles. Further, even though the study demonstrates a link between chronic fatigue syndrome symptoms and low levels of key thyroid hormones, a definitive cause for CFS remains unknown.

If the study findings are confirmed by additional research, it may pave the way for a treatment for chronic fatigue syndrome.

More information: *Frontiers in Endocrinology*, DOI: 10.3389/fendo.2018.00097

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