## **Chronic Fatigue Syndrome Might Have a Crucial Hormonal Link**

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For years, a line has divided millions of patients from their doctors, separating those who experience the debilitating effects of <u>chronic fatigue syndrome</u> from a medical establishment that has traditionally refused to acknowledge or agree upon the condition. Now, finally, that barrier is beginning to crumble.

In recent times, a series of studies has <u>identified evidence</u> of <u>biological mechanisms</u> that could <u>contribute to the disorder</u> – and now new research from the Netherlands is being hailed as an important advance in our understanding of the illness.

Researchers at the University Medical Centre Groningen have discovered a link between <u>chronic fatigue syndrome</u> (CFS) – aka <u>myalgic encephalomyelitis</u> (ME) – and lower thyroid hormone levels.

If the findings can be confirmed by additional research, it could be a first step toward finding a treatment for this maddening, mysterious disease.

Part of the problem with exploring what's behind CFS is recognising it in the first place. Often, it's diagnosed by ruling out any other underlying medical conditions, using a process of deduction to eliminate viral, bacterial, and other medical explanations that we have established tests for.

The condition, which has no definitively known cause, is marked by long-term fatigue, post-exertional malaise, sleep problems, difficulty in thinking clearly, and a host of other varied physical symptoms, characterised by overall discomfort, aches, and pains (sometimes extreme).

The severity and prevalence of these symptoms – estimated to affect over <u>1 million</u>

<u>Americans</u> and 2.6 percent of the global population – has made CFS one of the world's

most controversial medical disorders, with patients and researchers <u>lamenting the</u> <u>inadequacy</u> of our existing understanding and <u>'treatments'</u> of the <u>disease</u>.

The new study, led by biochemist Begoña Ruiz-Núñez, compared thyroid function and markers of inflammation between 98 CFS patients with 99 healthy control participants. What they found was that CFS patients had lower serum levels of two key thyroid hormones – called triiodothyronine (T3) and thyroxine (T4) – but normal levels of a thyroid-stimulating hormone that's usually present at higher levels in <a href="https://hypothyroidism">hypothyroidism</a> – the better understood condition that also displays low thyroid hormone production.

To the extent they can characterize it so far, the researchers hypothesize that CFS is caused by low activity of thyroid hormones in the absence of thyroidal disease, since the patients in the study had regular amounts of the thyroid-stimulating hormone, called thyrotropin.

In addition, the CFS patients demonstrated low-grade inflammation generally, plus higher levels of another thyroid hormone called "reverse T3" (rT3), which is thought to contribute to the overall reduction in T3 hormones.

"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," <u>says Ruiz-Núñez</u>.

"This strengthens our test results considerably."

While we don't yet understand how these altered hormone levels are related to the myriad symptoms of CFS, isolating this imbalance in the thyroid could be a major step forward in learning more about what's triggering this strange illness – bringing us hopefully closer to more targeted trials, and one day, treatment.

"This new research into thyroid gland hormones in ME/CFS represents an important advance in our understanding of hormonal abnormalities in this illness," <u>explains</u> physician Charles Shepherd, a medical adviser to the UK's ME Association, who wasn't involved in the study.

"If these findings can be replicated by other independent research groups, it suggests that the cautious use of thyroid hormone treatment needs to be assessed in a <u>clinical trial</u> – as it could be an effective form of treatment for at least a subgroup of people with ME/CFS." The findings are reported in <u>Frontiers in Endocrinology</u>.