

## Thyroid Function Disorders Effects Function of Thyroid Hormone

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### ABOUT THE STUDY

When it comes to female reproduction, thyroid hormone is important. The female reproductive system is replete with thyroid hormone receptors, and thyroid hormone governs important physiological aspects of reproduction, notably through the actions of follicle-stimulating hormone and Luteinizing Hormone (LH). Thyroid disease's effects are a reflection of the function of thyroid hormone. The direct effects of hypothyroidism and hyperthyroidism on subfertility are well-established modifiable risk factors for subfertility, as are indirect effects such hyperprolactinemia, changes in gonadotropin-releasing hormone sensitivity, and changes in estradiol concentrations. The justification for treatment of overt thyroid illness is well known, and recent research has demonstrated that levothyroxine therapy for euthyroid thyroid autoimmunity is not beneficial. The usefulness of levothyroxine medication for milder types of thyroid illness, namely subclinical hypothyroidism, is uncertain. Fertility experts commonly take advantage of this information gap to treat women with normal thyroid function (i.e., a TSH concentration of  $>2.5$  mU/L or below). Because of over diagnosis and overtreatment, such non-evidence-based methods generate unwarranted psychological and biologic harm. A comprehensive study and meta-analysis of thyroid function test changes under Controlled Ovarian Hyperstimulation (COH). The study of these alterations is especially important since high-quality clinical evidence is limited in this sector, and low-quality clinical data is frequently over interpreted. As a result, rather than using a non-hypothesis-based approach, future clinical research or therapies should be based on thyroid physiology. Experimental investigations and human physiology studies provide a solid foundation for the current study. Because thyroid hormone regulates 3 $\beta$ -hydroxysteroid dehydrogenase (the final step in progesterone formation), aromatase (the final step in estradiol formation), and LH/human chorionic gonadotropin receptor expression, low thyroid hormone availability is associated with suboptimal local ovarian stimulatory effects of follicle-stimulating hormone and LH. Given that COH is a condition of supraphysiologic ovarian stimulation, identifying women who have abnormal thyroid function test findings during COH may eventually enhance their outcomes. COH is also a state

of increased demand for thyroid hormone synthesis. While thyroid function test results stay consistent throughout a normal menstrual cycle, a fast rise in estradiol levels occurs. COH raises thyroxine-binding globulin levels and type-3 deiodinase gene transcription (deactivating thyroid hormone), resulting in decreased thyroid hormone availability and an increase in TSH concentration. After analyzing data from 11 studies, the studies discovered that in euthyroid women undergoing *in vitro* fertilization/intracytoplasmic sperm injection, the mean TSH concentration increased by 0.69 mU/L (95% CI, 0.30-1.08) during COH, and this effect persisted until a positive pregnancy test result, whereas the mean free thyroxine (FT4) concentration increased by 0.34 pmol/L (95% CI, 0.91 to 0.23). Sub analyses of women with thyroid autoimmunity indicated extremely varied results (effect estimates ranging from 0.1 to 2.86 mU/L), most likely due to the small sample size ( $n = 7-24$ ). The initial research used poor statistical modelling and combined thyroperoxidase antibody-positive women with thyroglobulin antibody-positive women (the latter are unlikely to present a reduced thyroid functional capacity). Women with hypothyroidism who were given a constant dose of levothyroxine were also evaluated, which is significant since thyroid function cannot be boosted due to a rise in TSH concentrations in this group, making it a more controlled experiment. The mean TSH concentration in these women increased by 1.50 mU/L (95% CI, 1.10-1.89), which lasted at least 3 months following COH. This study gives the finest quantitative evidence on the effects of COH on thyroid function, employing rigorous technique and taking into account a longitudinal element with a baseline measurement that almost eliminates reverse causation. It was not clear if practitioners may have predicated the type of COH on the outcome of the thyroid function test, making the findings of analyses after stratification for the various regimens difficult to interpret. The magnitude of thyroid function alterations, particularly in hypothyroid women treated with levothyroxine, suggests that COH is a condition characterized by an increased need for thyroid hormone synthesis. The 1.50 mU/L increase in mean TSH concentration in this group reinforces international guidelines' recommendations to aim for a TSH treatment target of 2.5 mU/L in women treated with levothyroxine before pregnancy because it reduces the risk that relevant under treatment will go undetected

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in between thyroid function assessments. Furthermore, the modest rise in TSH concentration among euthyroid women highlights that there is no reason to treat euthyroid women with levothyroxine. This study also clearly shows that the number and quality of available data are insufficient to identify women at high risk of having clinically relevant thyroid function test

abnormalities during COH that would necessitate levothyroxine medication. We agree with the scientists that more prospective trials are needed to identify subgroups with clinically significant thyroid function abnormalities. The study's design is a significant weakness. An aggregate data meta-analysis cannot appropriately account for statistical approach variations across studies.