

The Common Thread: What the New Research Shows

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Keywords: hypothyroidism • TSH • clinically hypometabolic • indirect calorimeter

Thyroid Science began a little over a year ago, and we were immediately accepted by the research community as a respected, open-access online journal. My job as Executive Editor, in addition to helping to prepare manuscripts, is to review and assess how we've done in our first year. As I began to reflect upon the studies, letters, and editorials we've published, I realized that they all seemed to point in the same direction. The common thread, so to speak, is startling considering the fact that we publish relevant submitted research without prejudice or subject selection criteria. In other words, we did not pre-decide what types of papers we would publish.

Because *Thyroid Science* was created simply to foster truth in science, we've been fortunate to be able to publish papers from around the world that clearly seem to contradict the conclusions of the papers you'll find in more "conventional" journals. What jumped out at me—as I reviewed the new research papers as a body of evidence—is that the current standard of practice in endocrinology with regard to hypothyroidism is gravely deficient. More especially, using the TSH as a universal and solitary gauge for both diagnosis and treatment should now be seriously questioned.

Each of the studies we've published in this first year (save Frank Thompson's on depression that didn't deal with actual treatment technique) used a better and more direct assessment of patients' thyroid status than the TSH test. And interestingly, the assessment method was different in each study!

Dr. Bjørn Øverbye of Norway used the Van Vincent Bio-Electrical-Terrain-Analysis, a system that evaluates the metabolic status of structurally-supportive and essentially-producing tissues. In his clinically hypometabolic patients, he found evidence of acidosis in connective tissues and lymphatic and blood vessels. "The acidosis can be explained by a lack of thyroid hormone effect at the cellular level."^[1] For almost all of his patients, the TSH was "in the normal range," whether they had been treated (actually under-treated) or not.

In our most recently published paper, Dr. John Dommis of Arizona, USA describes what he believes to be a better assessment of patients' metabolic status than the TSH.^[2] He measures his clinically hypometabolic patients' free T₄ and free T₃ levels by the tracer-dialysis method both for diagnosis and treatment. He prescribes whatever type of thyroid hormone the patients need, including T₃, to "optimize" the free T₄ and free T₃ levels. Most importantly, he doesn't mind suppressing the TSH in the process.

The group of studies from Sweden by Dr. Bo Wiklund and Dr. P.O. Sanberg may be the most influential in persuading practitioners that the sole use of the TSH and other blood tests is inadequate for diagnosing autoimmune hypothyroidism.^{[3][4][5]} Many of Dr. Wiklund's clinically hypometabolic patients had normal range blood tests—TSH, thyroid hormone levels, and antibody levels. But when Dr. Wiklund performed fine-needle aspiration of the thyroid gland on these patients, cytologist Dr. P.O. Sandberg found many samples to show lymphocytic infiltration. In other words, these patients had evidence of autoimmune thyroid disease in the tissues of the thyroid gland that did not show up in any blood test. Dr. Wiklund believes that not only should a normal range TSH *not* be the goal for these patients, but that the TSH should be suppressed because "TSH signals trigger and maintain autoimmune activity."^[3]

I want to briefly mention here that Frank Thompson's recommendation of T₃ for depression implies that he, too, is willing to ignore the "standard" approach to thyroid treatment.^[6] (Thompson is from California, USA.) Though he doesn't say how he assesses depressed patients' thyroid status and whether or not he uses the TSH in any way, the fact that "conventional" thyroid treatment allows only T₄ and Thompson recommends T₃ tells us that he has indeed found a better way to work with his depressed patients than the one dictated by the endocrinology specialty.

Next, we come to the critiques of the flawed studies^{[10][11][12][13]} done on adding T₃ to T₄ in patient treatment. Dr. John Dommissé's letter^[7] about these studies and Dr. John Lowe's extensive examination of how they were flawed^[8] both tell us that the conventional endocrinologists who performed the studies were unwilling to give up their use of the TSH as the "gold standard." And by using the TSH to decide how much thyroid hormone to give patients, the results were pre-determined. It didn't really matter whether they used T₄ and T₃ in combination, if the dosages merely normalized patients' TSH levels; the treatment was doomed to fail because these symptomatic (clinically hypometabolic) patients already had normal range TSH levels.

Finally, what we consider to be the most important study published in the last 10 years for fibromyalgia patients is our paper "Lower Resting Metabolic Rate and Basal Body Temperature of Fibromyalgia Patients Compared to Matched Healthy Controls."^[9] Dr. Lowe used the indirect calorimeter to measure resting metabolic rates in fibromyalgia patients. After controlling for almost all the factors that could influence resting metabolic rate—including lack of exercise—our results showed that indeed, fibromyalgia patients have lower resting metabolic rates and basal temperatures than controls. By definition, then, fibromyalgia patients can be said to be clinically hypometabolic. In addition, the patients' TSH levels failed to correlate with their resting metabolic rates. In other studies, the TSH has not been shown to reliably predict resting metabolic rates, so we cannot rule out inadequate thyroid hormone regulation as the mechanism of the fibromyalgia patients' low metabolic rates. Once again, then, we have evidence that the TSH is not the proper gauge for judging whether or not a patient is hypometabolic.

The publications in *Thyroid Science*, as a body of evidence, all relate to clinically hypometabolic patients who were failed by the use of the TSH to diagnose and treat them. And, most of the studies offer better ways to assess and treat patients than the TSH test. Remember that we did not set up criteria for publication in *Thyroid Science* that would lead to the above "common thread." But when a group of unrelated and unselected papers all show the same thing, honest scientists and clinicians must consider that the conclusions of these publications come

much closer to the truth than what was previously believed. The reason why we have the compilation of international studies in *Thyroid Science* that we now have, and the reason they come to similar conclusions, is simply that research done openly, without prejudice or politics playing a role, will always come closer to the truth.

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