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"TSH is Not the Answer," report Dr. Carol Rowsemitt and Dr. Thomas Najarian: Their explanation and verification

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Recently, we had the privilege of publishing two papers by Carol Rowsemitt, PhD, RN, FNP and Thomas Najarian, MD. These superb papers support views shared by Dr. John Dommisse, Dr. Kenneth Blanchard, and many other advanced, outside-the-box thinkers in modern thyroidology. However, to their credit, Dr. Rowsemitt and Dr. Najarian persuasively elaborate and document that thinking.

Rowsemitt and Najarian Papers. One of Rowsemitt and Najarian's papers is an exemplary example of heavily-substantiated hypothesizing—hypothesizing that has profound practical implications for the health and well-being of hypothyroid patients. The other paper is a laudable description of their clinical protocol based on the hypothesis. I am confident that many of our readers will print, circulate, talk about, and share the papers with other clinicians and patients.

Many mainstream clinicians, of course, are at least temporarily still anachronistically stuck in "TSHism." By this term I mean in part the simplistic belief that a low TSH is synonymous with hyperthyroidism or thyrotoxicosis. And a corollary belief accompanies the first: that is, due to the low TSH, the patient should not use thyroid hormone at all or should reduce his or her dose. Rowsemitt and Najarian convincingly refute both of these beliefs.

I doubt that self-asserting promotors of TSHism will respond to Rowsemitt and Najarian's papers. These authors' arguments are simply too logical and too grounded in science, as the propositions and citations in their theoretical paper shows. Hopefully, however, those promotors will open-mindedly consider Rowsemitt and Najarians' way of thinking about weight-burdened thyroid patients and hypothyroid patients in general.

Not Anti-TSHism. In doing so, those advocates should not mistake the authors' stance as anti-TSH. Instead, their work is respectful of science and rationality and motivated by humane concern for patients' well-fare, and their paper clearly puts TSH in proper perspective. Consider a concluding statement of theirs:

> "We submit these ideas hoping that others will join us in re-evaluating thyroid treatment when maladaptive hypothyroidism occurs during weight loss attempts. Clinicians must use clinical skills and patient-centered concerns in the optimum evaluation and treatment of their patients and not succumb to blindly following an arbitrary system of defined normal lab values in making therapeutic decisions that greatly affect the well-being of their patients."

Lowered Pituitary Set Point. The authors' papers, I believe, also show a noteworthy drive to be didactically helpful. In that vein, they have cast light on a realistic and fortunate feature of the thyroid system: that when a patient is in a low caloric state, it adapts with sophisticated and complexity to the chronically low intake of calories by slowing metabolism. But, as the authors write, "The patient is often told to get more exercise and that s/he must be eating more calories than realized." And they show that reducing the patient's thyroid hormone dose is likely to slow metabolism even further. This misguided treatment approach hinders the adaptive mechanism, reducing calorie expenditure, and perpetuating the patients' retention of excess weight.

Rowsemitt and Najarian write that upon finding a lower in-range TSH, “The provider is likely to conclude that there is nothing wrong with the patient’s thyroid function despite the symptoms.” As they explain, though, the provider is correct in one respect—there is nothing wrong with the patient’s thyroid function. The patient’s lowered TSH is caused by the pituitary’s lowered set point. That lower set point is a life-preserving evolutionary adaptation to low calorie intake.

Early humans and even earlier hominids underwent feast/famine cycles that were beyond their control. Modern humans in developed countries have largely eliminated famines. But in times when feast/famine cycles were fairly common, those whose metabolism slowed down during famines had an evolutionary advantage—that is, it enabled them to survive. The survivors, through generations, spawned offspring progressively better able to survive protracted times when food was scarce.

They note that humans still have this adaptive advantage despite no shortage of calories for most people today. The adaptation that benefited our ancient ancestors now leads to many hypothyroid patients continuing to suffer from hypothyroid symptoms and excess weight. The reason is that clinicians in general lack an understanding of the adaptive mechanism of a lowered pituitary set point. The resulting lower TSH prompts them to reduce or stop patients thyroid hormone doses, worsening their symptoms and weight gain.

Our main problem for many hypothyroid patients today is the abundance of calories that would nullify our need for the adaptive mechanism—had we enough time for evolutionary deletion of the set point phenomenon.

I have touched on some of Rowsemitt and Najarian’s points from my enthusiasm for their brilliant and insightful exposition. I am convinced that their papers will do much needed good for hypothyroid patients, especially the weight-laden ones. I strongly encourage patients and clinicians alike to read, print, and disseminate Dr. Rowsemitt and Dr. Najarian’s extraordinary papers.