

Diagnosis of Thyroid-Related Problems

[Q&As are placed in reverse chronological order. In other words, the latest Q&As come first. Earlier ones are further down the page.]

Date: September 7, 2000

Question: Several alternative doctors on the Internet are now saying that the free T3 is the ultimate thyroid test to use in adjusting our dose of thyroid hormone. Do we finally have a blood test that matters?

(For purposes of courtesy, we've deleted the names of the alternative doctors the writer mentioned.)

Dr. Lowe: No, we don't finally have a blood test that matters—not unless a doctor's goal is to treat another lab value rather than his patients. I recently communicated with one of the doctors you mentioned. He had copied to me an e-mail he'd written to another physician. In his e-mail, he boldly advocated adjusting thyroid hormone dosage according to the free T3. He also stated that he wasn't going to attend the upcoming Broda Barnes Foundation meeting. His reason was, "Although I retroactively recognize his genius for his times, the latest FREE-levels of the T4 and T3 hormones—if one uses them properly and regularly—make his methods obsolete." I've included below my reply to the doctor. The content of my reply explains why I disagree with him that the free T3 is the ultimate method of adjusting thyroid hormone dosage. I wrote to him:

"I'm confident you're getting clinical results superior to those other physicians get by using the TSH level and Synthroid. But I'm confident that you're *not* getting results superior to those we achieve by adjusting patients' thyroid hormone dosages by the responses of their tissues particular hormone dosages.

" My understanding is that those who work under the banner of "doing Broda Barnes work" have expanded the measures by which they assess therapeutic change and to adjust patients' dosages. These doctors now use other measures along with the basal body temperature. I don't know, however, what their clinical tract record. I don't know because hardly any one in "the movement" (away from conventional clinical thyroidology), except for my research group conducts clinical trails to assess the effectiveness their methods of adjusting dosage. Without experimental assessments of the different methods, we really can't accurately judge which is truly most effective.

"Nonetheless, I'm confident that your results are usually good from using free T3 and T4 levels. And reaching an priori conclusion that your method is most effective is certainly a first step in scientific thinking. Bold hypothesizing is essential advanced sciences. But it is of little use scientifically unless we following hypothesizing with rigorous attempts to disprove our own hypotheses. My research group has taken this scientific approach. This is why we *know* objectively that our method is highly effective. But without studies that compare our method of dosage adjustment with those such as yours, we can't dogmatically pronounce that ours is superior to them—nor can the advocates of these other methods, such as yours. I suspect, however, that you and I will agree that your method and ours is superior to that of using the TSH and Synthroid.

"My concern is that you won't follow your bold hypothesizing by the next essential step in good science—stringent efforts to logically or experimentally disprove your own hypothesis. If not, you'll be making the same mistake as conventional thyroidology—dogmatically believing that your good *tentative* hypothesis is a well-established scientific fact. In this way, thyroidologists have built a superstructure of false beliefs upon their fundamentally wrong hypotheses. In the process, they've ruined the health of incalculable numbers of people. Hopefully, those of us involved in this new movement will learn from that gargantuan mistake in medical history and not repeat it.

"I feel good that you're on our side of the fence regarding health problems resulting from too little thyroid hormone regulation of tissues. Those of us on this side of the fence agree that the conventional approach to diagnosing and treating thyroid-related problems is seriously flawed and doesn't serve most patients well. Yet we disagree about which approach serves patients best. Our beliefs about how best to diagnosis and treat patients with too little thyroid regulation differs from yours. Such differences in beliefs are healthy for the movement. Our group must remain open to differing view points, just in case we've missed something that could make our treatment regimen even more effective than it is. We maintain this attitude although our beliefs and treatment regimen are based on tough logical analyses and scientific studies. (See our studies reprinted in an appendix in my book, [The Metabolic Treatment of Fibromyalgia](#)."

This doctor had written in the e-mail he copied to me: "There is an absolute explosion of physicians now "winging it on their own" re the Rx of hypothyroidism." He then wrote: " I believe my approach is the best in the world: Tell me why it's not and why yours is better!" I replied:

I agree that most clinicians are "winging it on their own." They don't have established criteria by which they make dosage thyroid hormone adjustments. I think that the movement we're all involved in will be strengthened if we can influence these clinicians to come to use assessment measures that increase their therapeutic effectiveness.

However, I personally don't believe that measuring the free T4, free T3, or any other circulating hormone level, is the most effective clinical approach. My belief is based partly on the the studies of Escobar-Morreale and colleagues in Spain.[1][2] Their study results make one thing clear: Circulating free T3 and T4 levels don't allow us to accurately predict the T3 concentration in the cells of most tissues. The evidence suggests that there is simply too much variability between different tissues in the same patient. Moreover, there's too much variability between the tissues of different patients. Even more difficult is accurately predicting the physiological and clinical effects of different circulating free T3 and T4 levels. Again, there's simply too much variability to allow accurate predictions.

Barnes was right when, long ago, he wrote that circulating levels of hormones don't measure what's most important—how the patient's tissues are responding to a dosage of thyroid hormone. Our regimen involves multiple measures of how tissues are responding to a particular dosage, repeated at short intervals in a highly systematic way. Our model of assessment is taken from behavior modification, in which I was trained in the early 1970s. We know from hundreds of trial runs that we can precisely control the metabolic status of most patients only by using these multiple measures of tissue response. We adjust each patient's dosage until these measures tell use we've achieved normal tissue metabolic status—regardless of what the patient's circulating hormone levels are. I concede that you can do some fairly good tweaking by using free T3 and T4 levels. But still, if the patient's tissue responses aren't carefully assessed, the clinician isn't focusing on what's most important—the patient's physiological and clinical responses to treatment.

I wrote to this reply to the doctor's e-mail months ago. I've never received a response from him.

Mary Shomon publishes the valuable online newsletter titled [Sticking Out Our Necks: The Thyroid Disease News Report](#). At the top of each issue, she writes, "We're patients, NOT Lab Values!!" The alternative doctors you refer to should heed Mary's emphatic assertion. As usual, she's right, and the doctors are wrong.

References

1. Escobar-Morreale, H.F., Obregón, M.J., Escobar del Rey, F., and Morreale de Escobar, G.: Replacement therapy for hypothyroidism with thyroxine alone does not ensure euthyroidism in all tissues, as studied in thyroidectomized rats. *J. Clin. Invest.*, 96:2828-2838, 1995.

2. Escobar-Morreale, H.F., del Rey, F.E., Obregón, M.J., and de Escobar, G.M.: Only the combined treatment with thyroxine and triiodothyronine ensures euthyroidism in all tissues of the thyroidectomized rat. *Endocrinology*, 137(6): 2490-2502, 1996.

Date: November 1, 1998

Question: I'm a 29-year old woman with fibromyalgia. My blood tests showed a normal T4 level and low T3 level. My doctor said that this lab result does not mean I have a thyroid-related problem. Do you agree?

Dr. Lowe: The answer depends on what we are referring to as a "thyroid-related problem." Most conventional thyroidologists use the word "thyroid" as a synonym for "thyroid gland." For the moment, let's accept this qualification for the sake of illustration and rephrase your question: Is there a thyroid gland problem that could result in a normal T4 level and a low T3 level? Theoretically, the thyroid gland may dysfunction in such a way that it secretes normal amounts of T4 but less-than-normal amounts of T3. This could result in a normal circulating T4 level and a low T3 level. T3 is the most metabolically active thyroid hormone, but a low circulating T3 level may not result in slowed metabolism and related symptoms. The reason is that most T3 inside cells, where the hormone drives metabolism, is derived from the conversion of T4 to T3. As long as enough T4 reaches the cells and the cells convert enough T4 to T3, metabolism may be normal despite the low circulating T3 level.

Using the term "thyroid" to refer to the thyroid gland, however, is an unfortunate convention. The absence of the qualifier "gland" can leave one confused as to what mechanisms are included in the term "thyroid-related problem." Some writers use this term to refer to problems related to the cellular processing or cellular action of thyroid hormones. If we use this meaning of the term "thyroid-related problem," there are several mechanisms that may cause a normal circulating level of T4 and a low level of T3. These mechanisms may or may not result in slowed metabolism and related symptoms.

For example, various man-made chemical contaminants may induce liver cells to selectively clear T3 from the circulation at a faster rate than they clear T4. Again, if enough T4 enters cells and is converted to T3, metabolism may remain normal despite the rapid clearance of T3 through the liver. Also, for various reasons, the enzymes that convert T4 to T3 (5'-deiodinases) inside cells may not be catalyzing the conversion at a normal rate. This may cause enough slowing of metabolism to result in symptoms, although there is some debate about this in the thyroidology literature.

These examples certainly don't exhaust the possible mechanisms that can result in this pattern of hormone blood levels. What is important to appreciate is that problems of either the thyroid gland or the cells of various tissues can produce normal T4 but low T3 levels. If you have symptoms of hypometabolism, and your lab result (a normal T4 and low T3) is consistent with repeated testing, further investigation is warranted to determine exactly what the mechanism might be. Since you have fibromyalgia, which means you have symptoms of hypometabolism, then further testing and investigation is certainly warranted in your case.

So, to simply answer your question: "My doctor said that this lab result does not mean I have a thyroid-related problem. Do you agree?" If the lab result is consistent with repeated testing, then I disagree. Your test result does not rule out a thyroid gland problem, and it does not rule out a possible problem in the cellular processing or action of thyroid hormones. Your symptoms are as important as your test results. Since your fibromyalgia symptoms indicate that you are hypometabolic, then some "thyroid-related problem" is a distinct possibility. Certainly, further testing is justified.

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