An Interview with John Dommisse, MD
Unique Theories About Hypothyroidism Treatment

by Mary Shomon

Mary Shomon: Can you introduce yourself, tell us a bit about your background, and how you came to be interested in thyroid disease, and how people can find out more about you if they're interested?

John DOMmisse, MD, FRCP(C): I'll start by making 1 or 2 disclaimers. I am not an endocrinologist, or even an internist; my MD is from the U of CapeTown and my (Canadian) board-certification was in psychiatry (accepted in Arizona and some other states). In 1987, I ordered a TSH on myself and it was 6.0 mIU, 1.0-2.0 points above the 'normal range'. I saw an endocrinologist, who repeated the tests and then started treating me with Synthroid (T4). He was an open-minded person who also happened to be fascinated by the stories of psychiatrists and others 'bringing people back to life' with T3 (Cytomel). So, when I never felt that the T4 'did the whole job', I started experimenting with adding Cytomel to - or substituting Armour Thyroid for - the Synthroid. I have never looked back. After all the studying I did on the subject, I started treating patients for hypothyroidism as well, and now regard myself as an expert on the subject. My correction of nutritional and metabolic deficiencies in the blood has led me to treat also isolated aspects of other specialties; it would not be feasible to specialize and take specialty training in each one of these diverse areas of medicine, so I just call myself a nutritional-metabolic physician (for which there is no board-certification) and treat the conditions I am able to. Psychiatry is now only my 3rd specialty-area, after nutritional and metabolic medicine. My publications are listed - and some printed out - on my website, where there are about 160 pages of free info, plus my semi-annual Natural Medicine NewsLetter, containing case-reports from my practice, are for sale at $3-5.00 each, depending on the number ordered: www.JohnDommisseMD.com.

MS: What are your thoughts about the use of the TSH test and its "normal range" as a primary means of diagnosis of hypothyroidism?

JD: Not much. 2 reasons: (1) The TSH is an indirect measure of the height of thyroid hormone function and is influenced by many factors in addition to the height of the free-T4 and free-T3 serum levels. Endocrinologists are confusing the great sensitivity of the test (for measuring the height of the thyroid stimulating hormone serum level) with the idea that it is therefore the most accurate and appropriate measure of T4 and T3 thyroid hormone function, which it is not - the free-T4 and free-T3 serum levels are the best arbiters of that! They are still 'stuck' in the time when the Total-T4 and Total-T3 (and other even-less-accurate measures) were the only measures available. They have not adapted to the paradigm-shift in accuracy of the free-levels. Besides, an elevated TSH can only suggest primary hypothyroidism (in which the defect is in the thyroid gland itself), not secondary, tertiary or nonthyroidal-illness hypothyroidism (in which the defect is in the pituitary, hypothalamus, and peripheral enzymatic conversion of T4-to-T3 with 5'deiodinase, respectively, and TSH is not elevated in these types of hypothyroidism, may even be below its 'normal range'). If the TSH has any usefulness at all, it only has it in a trio of tests including the FT4 and FT3 as well. There are many circumstances in which one can dispense with the TSH test, but never, in my opinion, with the FT4 and FT3 levels.

(2) The so-called 'normal range' is way too high. Even in conventional circles it has become acceptable to make a diagnosis of grade-3 hypothyroidism (the mildest grade) when the TSH level is "in the upper half of its 'normal range'" (above 2.0, rather than 4.0 or 5.0 mIU), although very few conventional physicians will do this, or treat this 'mild, sub-subclinical' degree of hypothyroidism. But isn't that strange? Doesn't that immediately render the TSH

normal range cut in half, to a new normal range of 0.4-2.0? For several years I adhered to the grade-3 primary hypothyroidism range, with 2.0 as my cut-off point. But, eventually, I ran into too many patients who had classic hypothyroid symptoms, which cleared completely on appropriate thyroid treatment, and whose TSH was below 2.0 (but above 1.5) and with FT4 and FT3 levels in the low ends of their 'normal ranges'. So I lowered my range to 0.2-1.5. For several months I was happy to be helping more people (those whose TSH fell between 1.5 and 2.0). Finally, I found some patients with several symptoms and signs of hypothyroidism whose TSH was between 1.0-1.5; so I lowered my range, for the last time, to 0.1-1.0; I now treat primary hypothyroidism with a TSH of >1.0 (if the FT4 and FT3 are low-normal, not above the middle of their 'normal ranges'). Some physicians are still waiting for the TSH to go above 6.0 or even 10.0 mIU before they'll agree that the patient's hypothyroidism needs treatment!

MS: How do you typically diagnose hypothyroidism in your patients?

JD: I diagnose primary hypothyroidism in the way indicated in my first answer, above. Pituitary, hypothalamic and nonthyroidal-illness hypothyroidism require a FT3 level, at least, if not the FT4 as well, to be below its 'normal range'. That raises the question of what the normal range for FT3 really is. When I first started treating hypothyroidism, in 1988/9, the national chain-lab that I used most used a normal range for free-T3 of 280-540 (or 2.8-5.4, depending on the size of the units). It later lowered that range to comply with the ranges used at most other large chain-labs: 2.3-4.2 (or 230-420). I have often wondered whether that lab should have stuck to its guns and stayed with the higher range, and I have occasionally returned to that higher range in treating certain patients who particularly seemed to cry out (not literally or emotionally but in their symptoms and signs) for that higher range.

MS: What is your advice to patients who have physicians who insist that having a normal range TSH value means that hypothyroidism cannot be diagnosed?

JD: This claim is only reasonable for primary hypothyroidism, not for any other type. Even with regard to primary hypothyroidism, I would disagree, and I would repeat what I have just said, above, in answer to your previous questions.

MS: What about the TRH Stimulation Test?

JD: Thyroid stimulating hormone is also known as thyrotropin. It is secreted from the pituitary gland, to stimulate the thyroid gland to secrete enough thyroid hormones to carry out all the metabolic functions required of these crucial hormones, T4 and T3. The pituitary, in turn, is dependent on a certain amount of stimulation by the hypothalamus, a small part of the lower-anterior brain. The hypothalamus does this by secreting the thyrotropin-releasing hormone (TRH). Researchers like Evered, Ormston, Wentzel, Gold, Pottash and Extein discovered that there are a certain number of patients with normal TSH levels whose TSH level will jump up by more than 20 points 30 minutes after an injection of a certain amount of TRH. These were the first patients to be called grade-3-primary-hypothyroid (grade-1 being those whose FT4 and FT3 levels are below their normal ranges despite the best efforts of the TSH; and grade-2 being those whose TSH is elevated above its normal range but this TSH elevation is managing to keep the FT4 and FT3 levels in the low parts of their normal ranges).

With the development of increasingly-sensitive TSH tests, it was realized that a basal sensitive-TSH is sufficient to diagnose grade-3 if one reads all those who are in the upper half of the TSH's 'normal range' as being grade-3. When I first read Gold, Pottash and Extein's papers about the TRH stimulation test uncovering mild or 'subclinical' degrees of hypothyroidism, whose treatment then rescued them from refractory depression and other conditions resulting from this low-level hypothyroidism, I called them up and asked whether

the patients whose TSH shot up >20 points after the TRH injection were not also the ones whose TSH was hi-normal in the first place, and Dr Extein admitted that this was the case. At that time, the TRH stimulation test cost $600.00, so I bypassed that expense for my patients and started to diagnose all cases with TSH > 2.0 (and FT4 and FT3 low-normal) as grade-3 hypothyroidism, and started treating them. All of them improved dramatically.

So when physicians talk about 'subclinical' hypothyroidism (meaning 'no symptoms'), I am shocked because I realize they have simply not elicited the history or signs of classic hypothyroidism that seem, to me, to invariably accompany grade-3 hypothyroidism. As I've said, I now treat people whose TSH is > 1.0 (with FT4 and FT3 lo-normal) and, so far, have always obtained an excellent improvement in the patient's symptoms, cholesterol level, or whatever they have presented with. For some reason that I don't understand, there is more fear of treating these minor degrees of hypothyroidism than of treating the more-severe degrees, the implication being that, since they are only slightly low, they are that much closer to being overtreated. The reason I don't understand this is because one obviously uses smaller doses in the milder cases than in the more-severe ones, and one monitors their FT4 and FT3 levels at the point when they reach their maximum effect for each dosage-level anyway, so I fail to see what the danger is. I don't believe I have ever seen a patient's FT4 and/or Ft3 level go too high after the initial dose of thyroid hormone(s); one starts with a lowish dose, re-tests, increases the dose, etc., until both levels are optimized, whether the patient started out as a severe case or a mild case.

MS: What are your thoughts about the need for T3 in addition to T4 as a treatment for hypothyroidism?

JD: In my opinion, it is essential in most cases, in order to obtain the optimal response to treatment. There is a minority of hypothyroid cases that is able to convert T4 sufficiently to T3 in the peripheral tissues to produce an optimal free-T3 level, as well as an optimal free-T4 level, without prescribing any T3-containing preparation. I don't have any fixed combination of T4 and T3 that I use. The most important thing to do when prescribing any T3-containing preparation is to write it as a twice-daily, after brkfst and supper, prescription. This is because, unlike the T4-hormone, T3 is rather short-acting, with a half-life of 8-12 hours (meaning that half of it is already metabolized away in that time).

This is the fact that has scared many conventional physicians off from prescribing any T3-containing preparations. But this fear and objection is entirely taken-care-of by this prescribing regimen. So easy! This is not to imply that there are not many dangers involved in prescribing any thyroid preparation - and, even with this regimen, T3 is still more volatile than T4. e.g., If a patient misses a daily dose of T4, it will not significantly affect the blood-level drawn on that day; but, if a patient misses any dose of a T3-containing preparation in the 48 hrs leading up to the blood-draw, esp. the morning dose on that day, the serum free-T3 level is going to look lower than it has actually been running when the patient took every dose regularly. And temporary surges of free-T3 are all that are needed to cause cardiac arrhythmias and other significant problems in susceptible individuals. So perhaps it is not for the faint-of-heart, esp. if the prescriber endeavors to maintain the free-T3 level close to the maximum tolerable level for each particular patient. Frail, elderly, cardiac-arrhythmic and seriously-ill persons' free-T3 levels should not be run at hi-normal levels but only in the mid-range. But, with careful dosing, it is completely 'do-able' and well worth the extra attention needed.

If I start out with a T4/T3 combination like Armour Thyroid (because the free-T3 level in that patient is significantly lower, relatively-speaking, than the free-T4 level), this becomes the only treatment, again, in a minority of cases. In most cases, the free-T3 level will be elevated higher than the free-T4 level by Armour Thyroid. In that case, I add small to moderate daily doses of T4 - whatever is required to optimize both the free-T3 and free-T4 levels. If I start out with T4-only (Levoxyl or Levothroid*), I will add T3-only (Cytomel) twice-daily if the FT4

level is totally optimized but T3 is lagging; and I will add Armour Thyroid twice-daily if the FT4 level is still suboptimal and the FT3 level is even lower. There is also a synthetic T4/T3 combo, called Thyrolar, but it is much more expensive and w/o any real advantages except in cases of sensitivity to the pork thyroid gland in Armour (very rare). Sensitivities to cornstarch and other fillers are much more common; in such cases, I do compounded prescriptions, to be filled by a compounding pharmacist who puts all the ingredients together himself, from scratch. There are several of these kinds of pharmacies dotted around the country, and they have gotten quite heavily into mailing supplies to patients in remote locales. *I avoid Synthroid unless the patient's insurance mandates this brand for payment purposes, because the then manufacturers of Synthroid, the Boots Co., did some very unethical stuff in relation to the non-publication of an article in the early 90's that proved that Levoxyl ('Levoxine' at the time) and Levothroid were equal to, or very slightly better than, Synthroid.

MS: Many patients have told me that they've brought the New England Journal study and Dr. Ridha Arem's book to their doctors, but the doctors have said that the study wasn't enough to prove anything, dismissing the results, and decrying the study as flawed or inadequate. Why do you think this is happening, and there's such resistance to the use of T3?

JD: I've explained some of the resistance to T3 above. The rest of the resistance has to do with the conservatism that many doctors are influenced or coerced to adopt because of the huge survival- and career-jeopardizing pressures that physicians who 'step out of line' are subjected to, by medical boards, plaintiff lawyers, other physicians, medical schools, etc., etc.. As you probably know, the very issue of the New England J of Med (that published the Bunevicius et al article about T4+T3 being better than T4-only for many hypothyroid patients) contained a sort of disclaimer-editorial, written by Prof. Anthony Toft of Edinburgh, in which the significance of the article was placed under the qualification that 'further studies are required to verify these findings'. Prof. Toft said his impression is that most hypothyroid patients treated with T4-only are quite satisfied with their treatment!

Let me also say this about the Bunevicius et al paper in the NEJM on Feb. 11. These 4 workers merely substituted a fixed amount (12.5 mcg) of T3 for 50 mcg of the T4 that 33 patients were taking for their hypothyroidism; they did not, by any means, optimize the FT4 and Ft3 serum levels. And STILL, many of the patients improved when the T3 was substituted. My contention is that they would have improved even much more if their FT4 and FT3 levels had both been optimized.

MS: Some doctors claim that T3 is dangerous to use because it increases the risks of osteporosis or heart palpitations, among other claims. Are there any proven dangers in use of T3?

JD: There definitely are dangers inherent in prescribing T3 if one doesn't know how to do it. The reverse-side of that is that, if one knows how to prescribe it, the dangers can be minimized and kept to a level that is no higher than the dangers inherent in prescribing T4-only. The dangers are increased if one has the approach that both thyroid hormone levels should be kept optimal (for that individual) at all times. There is not much danger of over-treatment if all you are trying to do is "put the TSH and, perhaps the T4 level, in its normal range" because you are sailing 'far from the edge' in that case. But your patient is going to suffer the overall disadvantages of continuing to run a low-normal, sub-optimal level of both hormones. In my view, these disadvantages (which I'm sure most people reading this interview are very familiar with) are far, far greater than the potential dangers of over-treatment, if one knows how to prescribe T3 and monitors the FT4 and FT3 levels regularly.

The commonest cause of palpitations, in my experience, is intracellular hypokalemia (a low potassium level in the red blood cells). This test is hardly ever done; the serum level is the

one usually done but it won't pick up most cases of low potassium where it counts, which is INSIDE the cells, not outside them, in the serum. The red cell level reflects the level inside heart cells, muscle cells, etc.. It is tragic how many sportspeople are forced out of competitions because their physicians didn't measure the INTRACELLULAR potassium level and pick up their deficiencies that cause muscle cramps, heart problems, etc.. But a high T3 level can add to the risk of cardiac arrhythmias, so patients who have this tendency should have their free-T3 levels kept in the mid-range and not near the top of the normal range, which is where I like it in young, middle-aged otherwise-healthy people.

The danger of osteoporosis is way-overblown and is the reason why many hypothyroid cases are not diagnosed as such (because then the physician has to subject himself to this supposed high risk of being sued for having caused or aggravated the osteoporosis). It is strange that the free-T3 level should be picked on in this fearful strategy because hardly any of the studies that have been done on thyroid treatment causing osteoporosis have even measured the free-T3 levels of those patients! In fact, the free-T3 level is seldom obtained, period. And this is the level that does 90% of the thyroid function! The main reason why I know the fear of osteoporosis is overblown is that all my patients who are both hypothyroid and osteoporotic see their bone mineral density x-ray scans not only not deteriorate every year, but actually improve - by as much as 30 percentage-points in one year! If osteoporosis can be caused or aggravated by aggressive, hi-normal thyroid treatment, then surely my patients would be prime suspects to show this phenomenon. But they don't. Admittedly, I do also correct many mineral, vitamin, amino-acid and other hormone deficiencies, many of which cause or aggravate osteoporosis, but at least I have proven that, in a fairly-unique practice that does attend to such deficiencies, there is not only no danger of osteoporosis, there is reversal of osteoporosis. And this is without the use of Fosamax, calcitonin or any other drugs that are usually prescribed for osteoporosis.

MS: What do you think of Prof. Ridha Arem's new book, 'The Thyroid Solution', published in June by Ballantine Books?

JD: I read it in a few days, fascinated, because here, virtually for the first time, was a conventional endocrinologist who (1) actually listened to his patients, including the female ones, and responded to their needs rather than, in the usual cold academic way, brushing them off with "The problem is not in your thyroid gland, it is normal. Next case, please."; (2) admitted that the standard approach is not the answer in many cases, that some patients 'with normal blood values' do seem to be hypothyroid and to respond to treatment with thyroid hormone (T4); (3) added small doses of T3 2-3 times per day in some cases who did not respond favorably to T4-only. and (4) beautifully and seamlessly integrates physical and psychiatric symptomatology and response to treatment.

So I called him up, to congratulate him and to ask if he would be interested to see my approach of the last 11 years, which has been to include all 3 the accurate tests in all screening and most monitoring, and to re-define the normal range in the TSH, and possibly also in the free-T3 level. Rather than accepting that 'the tests are normal but the patient needs treatment'. As I expected, judging from his open-mindedness in his book, he was very interested and we are exploring ways in which we can collaborate in some writing in the future.

MS: What is your opinion about 'Wilson's Syndrome' and Drs Wilson and John C Lowe's treatment with T3-only?

JD: First of all, now that we have the free-T4, free-T3 and ultra-sensitive (3rd-generation) TSH tests, there is no need to rely on the non-specific 'low basal body temperature' method of diagnosis of hypothyroidism popularized by Broda Barnes decades ago, when these new tests were not available. If one does all 3 these blood-tests, and reads them sensitively-enough, one can diagnose the mildest cases of hypothyroidism accurately. The low basal

body temp. is not specific for hypothyroidism, although common in hypothyroidism. However, when the only test that is normally done to screen for hypothyroidism is the TSH, I can see where patients will become desperate enough to use any method they can to try to establish a diagnosis of hypothyroidism when this TSH test is "in the normal range".

Secondly, treating with T3-only is almost as bad as treating with T4-only in most cases and worse than T4-only in some cases. I say 'almost as bad' because, since 90% of thyroid function is carried out by T3, correcting the T3 level is a good thing. However, the brain needs T4 to be present in the blood in a good amount because T3 doesn't cross the 'blood-brain barrier' and get into the brain directly. T4 has to get into the brain first and then convert to T3 in the brain tissues. So the cognitive effects of a low T4 level would continue because T3-only treatment raises the T3 level a lot, often way above normal (with all the dangers inherent in that situation), and, by lowering the TSH level, this also lowers the T4 level to way-below normal. I cannot understand why anyone would want to treat with T3-only and not use both thyroid hormones, as needed to optimize BOTH free-levels. This is not to deny that many people treated with T3-only will improve in many ways; after all, T3 is a very important hormone; but they would improve much better and with less ill-effects if both their FT4 and FT3 levels are optimized and neither one is overtreated or undertreated.

Thirdly, my understanding is that these people who treat with T3-only do rather poor monitoring of thyroid blood levels and, if they measure the T3 level at all, it is usually the much-less-accurate total-T3 level and not the free-T3 level, which is the only 100%-active fraction of the T3 in the blood. I discussed all these issues with Dr Dennis Wilson by phone from my then-office in Portsmouth VA in the early 90's; I am disappointed that he didn't see the obvious merit in my arguments and has not modified his approach at all. My understanding is that John C Lowe, DC, doesn't necessarily think that his fibromyalgia patients who respond to T3 are necessarily hypothyroid but that boosting the T3 level to high-normal or even higher is somehow beneficial for such patients. I have seen some fibromyalgia patients respond quite dramatically to thyroid hormone treatment, but this is not the answer in all or even most cases. I am also pursuing relaxin hormone and cetyl myrist-oleate treatment in these cases, with benefit in most of them.

(Note from Mary Shomon: For information regarding Dr. John Lowe's perspectives, see my article Fibromyalgia Aches and Pains as a "Symptom" of Hypothyroidism: A Look at the Theories of Dr. John Lowe, and please visit Dr. Lowe's site, at www.drlowe.com. Dr. Lowe's theories and practice are also featured in Mary Shomon's book, Living Well With Hypothyroidism.)

MS: What other deficiencies have you found often associated with hypothyroidism that respond well to supplementation?

JD: The commonest conditions I see resulting from hypothyroidism are: Fatigue; depression; abnormal weight-gain; dry skin and eyes; excessive hair-loss; memory and concentration problems; apathy; constipation; high 'bad-cholesterol' and low 'good-cholesterol'; decline in academic and physical/ sports performance; etc., that are all virtually impossible to reverse without correcting the hypothyroidism first. With optimal correction of the hypothyroidism, these conditions will virtually all clear up, although of course some of them may require additional measures as well, such as depression, obesity, and dry skin. When the necessary other measures are taken, success is usually achieved in eliminating these conditions.

MS: Have you found any kind of treatment program that helps thyroid patients effectively lose weight?

JD: In addition to optimizing both the FT4 and FT3 serum levels, I find that correcting blood-deficiencies of chromium and manganese optimally; correcting a high free-insulin level (Syndrome-X) with a hi-protein, lo-carbohydrate diet; correcting many nutritional deficiencies

optimally; and encouraging plenty of exercise, even walking, will usually achieve total or partial excess weight loss that can be sustained indefinitely.

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Finally, I'd like to salute all the hypothyroid patients, like yourself, who have refused to accept the half-life that results, in most cases, from the standard treatment-approach. 'Good on you', guys!! I hope - and believe - that you will all be richly rewarded.

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