## Denis Wilson, MD: Low Body Temperature as an Indicator for Poor Expression of Thyroid Hormone

Interview by Craig Gustafson

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**E. Denis Wilson, MD,** will address thyroid function and Wilson's Temperature Syndrome at the 2015 Restorative Medicine Conference in Blaine, Washington, October 1 through 4. Dr Wilson was the first practitioner to use sustained-release  $T_3$  thyroid hormone. For 20 years, he has treated more than 5000 patients with  $T_3$  and trained more than 1000 physicians on how to use  $T_3$  to improve the health of patients with low thyroid function and low body temperature who have normal blood tests. He is the author of Evidence-Based Approach to Restoring Thyroid Health.<sup>1</sup>

*Integrative Medicine: A Clinician's Journal (IMCJ)*: What originally drew your attention to issues of thyroid and metabolism?

**Dr Wilson:** A patient came to my office and she brought with her a book and she said that I should read it. It was called *Hypothyroidism: The Unsuspected Illness*, by Broda Barnes, MD.<sup>2</sup> In that book, he explains the importance of using body temperature as a guide to evaluate thyroid function. I was intrigued by that and also his suggested treatment of using desiccated thyroid as an empirical treatment to normalize the body temperature. Though I did not look at the book for a few weeks, I eventually read it and decided to try his approach in a few of my patients. To my surprise, some of those people got 100% better.

That was really illuminating to me because, according to my training in medical school, that was not supposed to happen. These people had normal thyroid blood tests and, supposedly, that meant that they could not benefit from thyroid hormone treatment. These people did not get just a little bit better; they got completely better. It did not work for all the patients I tried it with, but it worked in about 60% of cases.

I was looking at the other 40% and wondering how we could help them, too. It could be that they did not have thyroid problems, or maybe the particular treatment I was

using was not really addressing their issue. As I was trying to think of ways to increase the yield, I looked at the thyroid hormone pathways and saw that  $T_4$  is converted to  $T_3$ . It turns out that this step is really important. I thought that, perhaps, these patients have a problem with the conversion of  $T_4$  to  $T_3$ . So I started giving some of these treatment failures—these patients who had failed to respond to the pervious treatment— $T_3$  directly. A lot of those treatment failures became treatment successes. That is how it all started.

*IMCJ*: Previous to that, had you been seeing a lot of thyroid patients?

**Dr Wilson:** Not really. I was more involved in primary care practice, but when I started seeing these kinds of thyroid results, the reaction I had was, "If this isn't true, then perhaps nothing they taught me at medical school is true." The use of the thyroid hormone blood test to direct thyroid therapy is one of the most dogmatically taught principles in medical school. They acted like the blood tests are absolutely conclusive in managing thyroid health. It was really eye opening to me because this closely held dogma—I could see from my own experience—was not true.

Then I thought, "If that is not true, then maybe nothing is true." That perspective really opens up the possibilities of different things we can try to help people get better. That is when I really started diving in. When the patients do recover, there is hardly anything more dramatic than a hypothyroid patient's response to thyroid therapy. It can be very pervasive.

One thing that I have come to understand over the years is that the purpose of the thyroid hormone is to go into the nucleus of the cell, form transcriptional complexes, and dictate the speed at which DNA is transcribed. It actually dictates how fast we live. That is really what metabolism is. It is how fast we live, which is controlled by

the thyroid. When a person asks me what thyroid can affect, I respond, "Thyroid really only affects those cells that have DNA." In other words, it affects every cell. When I saw the profound ramifications a normal body temperature can have on people, I started doing that pretty much exclusively.

*IMCJ*: What are some of the more frequent symptoms that indicate to you that there may be thyroid dysfunction?

**Dr Wilson:** Certainly fatigue, chronic fatigue, and headaches—migraine headaches. A huge percentage of patients with migraine headaches have low body temperatures and I have seen so many people when they

get their temperatures corrected, their migraines sometimes disappear completely. Irritability, fluid retention, anxiety, panic attacks, PMS, hair loss, depression, decreased memorv and concentration, low sex drive, unhealthy nails, low ambition and motivation, constipation, easy weight gain for sure, irritable bowel syndrome, dry skin, dry hair, insomnia, and even some things that people wouldn't normally expect like asthma.

Even asthma and hives and allergies can sometimes respond to normalizing a low body temperature. Carpal tunnel syndrome and conditions caused by fluid retention—so there's a tremendous number of things. Some of my

favorites to treat are definitely migraines, PMS, and panic attacks. Panic attacks and anxiety symptoms are very debilitating and they are very responsive to normalizing one's temperature. That is what makes this so fun to address. There are not many good solutions out there.

*IMCJ*: In many of these cases, the tipping point marker is low body temperature?

**Dr Wilson:** I would say in every case, yes. It is not possible for a person to have symptoms of hypothyroid unless they have a low body temperature. They can have abnormal blood tests and normal temperature and they will still feel fine but the only way you can have those symptoms of hypothyroidism is by having a low temperature. *IMCJ*: Is the breadth of the symptomatology directly the result of the dysfunctional thyroid or does the low body temperature itself cause secondary symptomatology?

**Dr Wilson:** I believe that it is the temperature itself that causes the symptomatology because the correlation is so complete. My favorite theory has to do with the enzymes we talked about, the transcription of DNA in the nucleus, and that when DNA is transcribed, it makes proteins and enzymes and structural elements. Those enzymes are the key of every chemical reaction in the body and the speed at which those reactions take place and the efficacy of those reactions depend on the enzymes. The whole purpose of an

enzyme is to help a reaction take place at a reasonable temperature, like body temperature, when it would not take place at that temperature without it.

Without an enzyme, that reaction might not take place at less than 220°F or something like that. With the enzyme, the reaction can take place at a reasonable temperature or a biological temperature. Let's put it this way: It is known that those enzymes depend on their shape for their activity. The conformation, or the shape of these enzymes, is what brings reacting molecules in close enough proximity to react. If those enzymes are too hot, they are too loose. If they are too cold, they are too tight. If they are just the

right temperature, then they are just the right shape. A change in temperature can have a huge impact on the speed of these chemical reactions.

*IMCJ*: How does being "too tight" affect the shape of an enzyme?

**Dr Wilson:** An enzyme is a string of amino acids and it coils upon itself because of the electrostatic charges of the atoms forming a shape. That shape generates active sites where one active site can grab one substrate and another active site can grab another substrate and then, when those substrates are grabbed, the enzyme can change its conformation and bring the reactive species into close proximity so that they can react.



All of that depends on temperature, so what I mean by "too tight"—it is like an old-fashioned telephone cord. Sometimes they get tangled like a knot. If you pick the receiver up, the cord untangles and then when you hang it up again, the cord twists up on itself again. That is what I mean by "tight." If the cord does not tangle up on itself at all, then it is too loose and it does not really work right. If it is too twisted up on itself, then it is too tight and that does not work, either. You want the enzyme to have just the right shape and that depends on temperature.

*IMCJ*: Why are the conventional methods of treating hypothyroid inefficient for resolving these cases?

**Dr Wilson:** It is because the conventional approach is to think that thyroid function—or the adequacy of thyroid function—depends on blood tests. Ever since the thyroid-stimulating hormone, or TSH, test was discovered, or even since they discovered that  $T_4$  hormone is converted to  $T_3$ , there was an assumption made.

If you were the one who discovered that  $T_4$  is a raw hormone that is converted to  $T_3$  and that  $T_3$  is actually the active form of thyroid hormone, then at that moment you could make either of 2 conclusions. You could say, "Wow,  $T_4$ is converted to  $T_3$  and  $T_3$  is actually the active hormone. We really shouldn't focus so much on  $T_4$ . We should focus more on  $T_3$  and the effects of  $T_3$  to see if that interaction is adequate—accomplishing what we want it to accomplish." That is one conclusion. The other conclusion you could make is, " $T_3$  is the active hormone and since the body converts  $T_4$ to  $T_3$  automatically in the cells of the body, we do not need to worry about that because it happens automatically. The only thing we have to worry about is to ensure that there is adequate  $T_4$  production or supply in the blood stream."

Those are 2 reasonable conclusions with very different outcomes. For the last 50 years, the latter of the 2 conclusions has been in favor. Over the last 10 years, research supports the idea that regulation of the conversion of T<sub>4</sub> to T<sub>3</sub> happens intracellularly and it can change dramatically under different circumstances. That conversion is not measured by a TSH test. The TSH test is not a reliable indicator of thyroid status because the TSH could be normal and a person could still have hypothyroidism in the cells. There is extensive research in the last 10 years, specially, to substantiate that. The  $T_4$  to T<sub>3</sub> conversion can change under a variety of disease states. Studies of 25 different diseases show that the effects of  $T_4$ to T<sub>2</sub> conversion can be impaired or can be affected by these different disease states. In these disease states, then, TSH is not a reliable indicator of thyroid status.

That is a long answer but the short answer is this: You asked me why the conventional approach to thyroid treatment not very effective and I would just say, "Because they are measuring the wrong thing." Thyroid blood tests do not measure body temperature.

Using the blood test, if 100 people come in with hypothyroid symptoms and these people are treated based on their blood tests, 5% are going to have problems that show up on the blood test. Of those 5% of people with problems identified by the blood test, probably only 50% of those are going to get better with conventional thyroid approach. That is because only 5 people—out of 100 who have low body temperatures that could be effectively managed with treatment—are going to have abnormal blood tests. If you try to get them better by just trying to normalize their blood tests, that is probably only going to work 50% of the time.

So, you are only taking about 2.5 people out of 100 who are going to be effectively managed. On the other hand, if you take those 100 people with hypothyroid symptoms, you will find that every one of them has a low body temperature. If you were to treat their temperature, you are going to get 80% to 90% of those people's temperature to normal. For these 80% to 90%, their symptoms are going to dramatically improve, if not resolve completely.

*IMCJ*: So the crux of the matter is that because the conversion of  $T_4$  to  $T_3$  is dependent upon an enzyme, there are circumstances—including ambient temperature of the body—that will affect the ability of the enzyme to function or its functional efficiency. That is what the "conventional" approach is missing.

**Dr Wilson:** That is exactly right. If the body worked automatically and always took care of itself, there would be no disease. But there is disease. There are all kinds of diseases. All kinds of things can go wrong with the body and with every aspect of the body. In fact, I am beginning to be of the opinion that anything that happens in the body can go badly. Anything that can go wrong will go wrong in somebody, someplace, at some time, for some reason.

If you look down the chemical pathways of the human body, you will see that there is Addison's disease and there is Cushing's and there are different diseases that we label based on how things go in the chemical pathways.

One really easy way to invent a new condition or new disease is to just find some place in a biochemical pathway where dysfunction has not been named yet and just name it. There has got to be somebody that is going to have a problem in that particular part of the pathway eventually. The deiodinase enzyme depends on selenium and zinc is also important. Of course, if you have a selenium deficiency,  $T_4$  to  $T_3$  conversion goes down. If you increase selenium in those patients, the  $T_3$  levels go up. Obviously, the function of that enzyme is variable under different conditions. It is under regulation and it can be downregulated.

This is a really important point. Many of the important pathways to the body are under regulation. That is how we

maintain homeostasis and normal functioning of the body. There is something called the ubiquitin proteasome pathway. The way this system works is that key enzymes in different pathways are under regulation. When the body wants to slow down that particular pathway, it downregulates or increases the destruction of that key enzyme. If it wants that pathway to speed up, it will decrease the destruction of that key enzyme so that the pathway can speed up or increase again.

The fascinating thing about deiodinase enzymes is that researchers have looked at the things that increase the downregulation of this enzyme. What things shorten the half-life of this enzyme?  $T_4$  or thyroxin is 1 of the things and the other is reverse  $T_3$ . Of course, conventional doctors and alternative doctors, even doctors who treat low body temperature empirically in the face of normal blood testes, will often use desiccated thyroid hormone. Desiccated thyroid hormone contains  $T_4$  and, presumably, they are using thyroid hormone because they are thinking that even though the blood tests are normal, the person is not getting enough thyroid stimulation of the cell. They attempt to help the patient by giving the patient more thyroid hormone in the form of desiccated thyroid hormone. Desiccated thyroid hormone has T<sub>4</sub>, which can significantly downregulate the converting enzyme. A lot of the patients who are treated with Synthroid, or treated with desiccated thyroid, actually do not improve as much as we hoped they would. Sometimes they actually get worse.

That is because the  $T_4$  in desiccated thyroid can downregulate the enzyme. When that enzyme is downregulated, the  $T_4$  gets converted to reverse  $T_3$  and both  $T_4$  and reverse  $T_3$  downregulate that enzyme. Here we are hoping to help the person's thyroid physiology and help them benefit from more thyroid stimulation of the cell—hoping that we are going to improve their  $T_4$  to  $T_3$ conversion—and we sometimes actually inadvertently suppress their  $T_4$  to  $T_3$  conversion and suppress their thyroid hormone stimulation. Thereby, we really do not make the progress that we are looking for.

We are giving them this desiccated thyroid and we are not getting their temperatures up. I would encourage doctors, if they are using desiccated thyroid, to have the patient monitor their temperature to make sure it is going up. If temperature is not going up on desiccated thyroid, there may be a good reason for that and it may not work very well for the patient.

*IMCJ*: Will people who benefit from this therapy end up having to continue it forever? Is this something that has a definite treatment duration or is it more a situation where you have to read it by the individual?

**Dr Wilson:** It definitely does depend on the individual. Typically,  $T_3$  is not taken for life. There are different problems, which I will address in a second, but the conversion impairment problem—improving  $T_4$  to  $T_3$ 

conversion—is something that can normally be corrected in a manner of months. Often the duration is 2 to 3 months, maybe 6, maybe 8, but certainly it is reversible to the point that people do not have to keep taking the treatment for life.

I like to separate the thyroid hormone system into 3 different compartments. One is thyroid hormone supply. The second is thyroid hormone conversion and utilization. And the third is thyroid hormone expression. Historically or conventionally, our medical establishment has hoped that they could measure, predict, and manage thyroid hormone expression based exclusively on thyroid hormone supply. We figured that if we just give a person enough thyroid hormone to normalize their TSH, then the thyroid hormone expression will take care of itself and that person will be fine. My opinion is that you cannot measure thyroid hormone expression with a thyroid hormone blood test.

Thyroid hormone supply is measured with a thyroid hormone blood test. The TSH is a great measure of thyroid hormone supply but the body temperature is the best measure, as it is an exact measure of thyroid hormone expression. When I say it is an exact measure, what I mean is that the whole purpose of the thyroid system is to determine how fast our bodies live and how fast they function. That is exactly what a thermometer is. A thermometer is literally a speedometer. The higher the kinetic energy of the molecules in the air, the warmer it is outside.

As a thermometer actually measures the speed of the molecules in the air, it also measures the speed of the molecules in our bodies. When you measure temperature, you are actually measuring how fast the chemical reactions are taking place in the body. If a person has a normal TSH—they have a normal supply—and they have a low temperature, which is low expression, to me, that logically suggests that they have a problem with thyroid hormone conversion and utilization. If a person has hypothyroidism, has had a thyroidectomy, and has a thyroid hormone supply problem, then, yes, they are going to need thyroid medicine the rest of their life. Without a thyroid gland, they are going to need thyroid hormone to produce supply.

Even people who have decent supply might still have a conversion problem. They might still have a low temperature even though they have a normal TSH or even a low TSH. They could actually be hyperthyroid and still have symptoms of hypothyroidism because their temperature is too low. That is because they have a conversion problem. Anyway, to answer your question, the conversion problem is the one that is reversible.

For conversion, you can take them off their Synthroid and you could take them off their desiccated and you can give them some herbs and nutrients to support the conversion of  $T_4$  to  $T_3$  and you can give them  $T_3$  directly if they need that. So, there are some things you can do to support thyroid hormone conversion. If you are successful, in that you are going to be able to get their temperature up to normal, lots of times you can wean them off the  $T_3$  and, perhaps, put them back on the Synthroid or desiccated thyroid hormone. At that point, they may be able to maintain a normal temperature. With that medicine, they may be able to maintain a normal temperature indefinitely—or maybe in 5 years, or maybe in 10 years they have another relapse and need another tune-up.

*IMCJ*: By getting the body temperature up, a patient then is hopefully creating this enzyme in the right geometry to sustain it on their own?

Dr Wilson: Yes. It is just speculation why people tend to get better and seem to stay better. My feeling is that there are a couple of ways to address this thyroid hormone conversion problem. One is with herbs and nutrition. If you properly support the enzyme and the body, then perhaps the enzyme will start functioning better and the conversion improves and the temperature goes up. Even without weaning off Synthroid or without weaning off Armour, sometimes lifestyle, nutritional, and herbal support is enough to improve conversion to the point that they are able to have a normal temperature and to feel well. That is sometimes a good solution and, apparently, you have just supported the converting enzyme. On the other hand, some people are going to need to have their thyroid hormone pathways cleared out. Sometimes, people do not get better until you wean them off the Synthroid and wean them off the desiccated thyroid and replace them for a time with  $T_3$  by itself— $T_3$  alone. When you give somebody  $T_3$  alone, their TSH goes down and their  $T_4$  goes down and their reverse T<sub>3</sub> goes down. We have already talked about how strongly  $T_4$  and reverse  $T_3$  will downregulate the converting enzyme.

These can decrease the half-life of the converting enzyme from 40 minutes down to 20 minutes or, in other words 50%, so it can be dramatic. What if, by reducing the  $T_{4}$  and the reverse  $T_{3}$ , that that downregulation was relieved and reduced to the point that the deiodinase enzyme could upregulate? Enough to increase to the point that it could restore better T<sub>4</sub> to T<sub>3</sub> conversion? Some research suggests that there might be a genetic coding problem of the deiodinase enzyme in some people, but I think that, even with normal coding of the deiodinase enzyme, the deiodinase enzyme can get bogged down under periods of stress. And under periods of stress, the conversion of T<sub>4</sub> to T<sub>3</sub> goes down. That is well known—for decades. When that happens, the reverse T<sub>3</sub> goes up. Again, the  $T_4$  and reverse  $T_3$  can further suppress the deiodinase enzyme, so I feel that this situation can set up a persistent impairment or suppression of the deiodinase enzyme to the point that a person is going to have a hard time maintaining a normal temperature.

*IMCJ*: Then is there any direct evidence at this point that the stress hormone, cortisol, interferes with the process directly?

**Dr Wilson:** Yes. Cortisol has been shown to directly inhibit the conversion of  $T_4$  to  $T_3$  for sure. One other thing: I do want to make it clear that I do not mean reverse  $T_3$  on a blood test. I am not saying that measuring reverse  $T_3$  in a blood test is going to be useful at all, because I haven't found it to be. I have not found it to be predictive or reliable. Some people with lower reverse  $T_3$  levels still have low body temperatures. You can have a person with low TSH, which makes them look like they are hyperthyroid, and then they have a high total  $T_3$ , which makes them look like—if anything—they are making plenty or too much  $T_3$ , and they could have a low reverse  $T_3$  which makes them look like if anybody is converting  $T_4$  to  $T_3$  very well, it is this person.

They could still have low temperature regardless of anything that the blood tests say. I still think that they could have impaired conversion at the level of the cell that is invisible on the blood test. The blood tests do not measure what is happening inside the cell. They only measure what is floating around in the blood stream.

*IMCJ*: To wrap things up, when you speak at the Restorative Medicine Conference in October, what more are people going to learn at your presentation?

**Dr Wilson:** I will talk about the specifics of the nutritional and herbal support. I will talk about the specifics of  $T_3$  therapy and how to monitor and manage patients. Basically, they will learn how to normalize somebody's body temperature in a way that will help patients recover from their symptoms and hopefully remain improved even after their symptoms have been discontinued.

For more information about the 2015 Restorative Medicine Conference, please visit http://www.restorativemedicine.org/.

## References

- 1. Wilson ED. Evidence-based Approach to Restoring Thyroid Health. Lady Lake, FL: Muskeegee Medical Publishing Company; 2014.
- Barnes B. Hypothyroidism: The Unsuspected Illness. New York, NY: Harper; 1976.