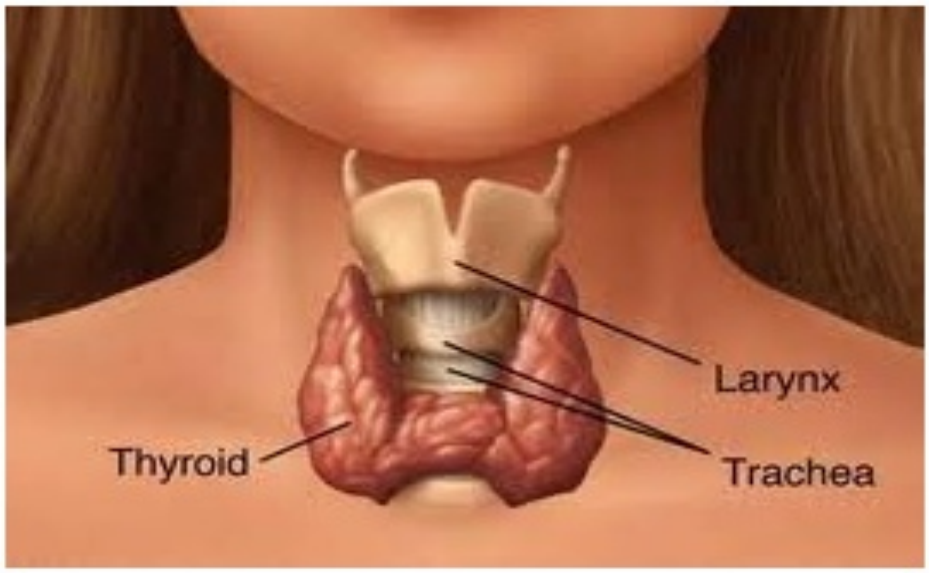
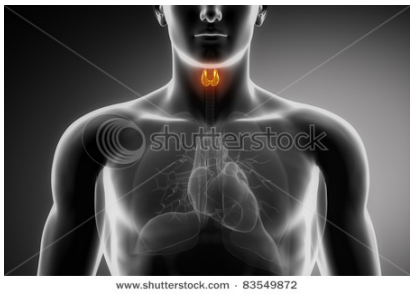
Mastering the Thyroid



Mastering the Thyroid, Presented by Apex Energetics, April 27-29

A Seminar Geared to Helping Clinicians Master the Concepts of Thyroid Physiology, Assessment & Clinical Applications



As many of you know, I recently attended a seminar on "Mastering the Thyroid" by Apex Energetics.

So many of you expressed an interest and made requests to "tell me everything" that I thought it would be fitting to address some of the details about thyroid physiology and biochemistry in a newsletter.

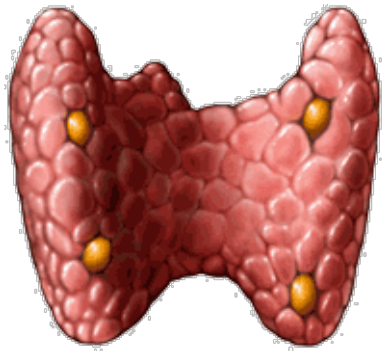
As can be expected of anything related to Apex Energetics, the seminar was excellent; and covered many aspects of thyroid health at a level that simply cannot be distilled into a brief non-technical summary! I will do my best to give you a rendition in layman's terms.

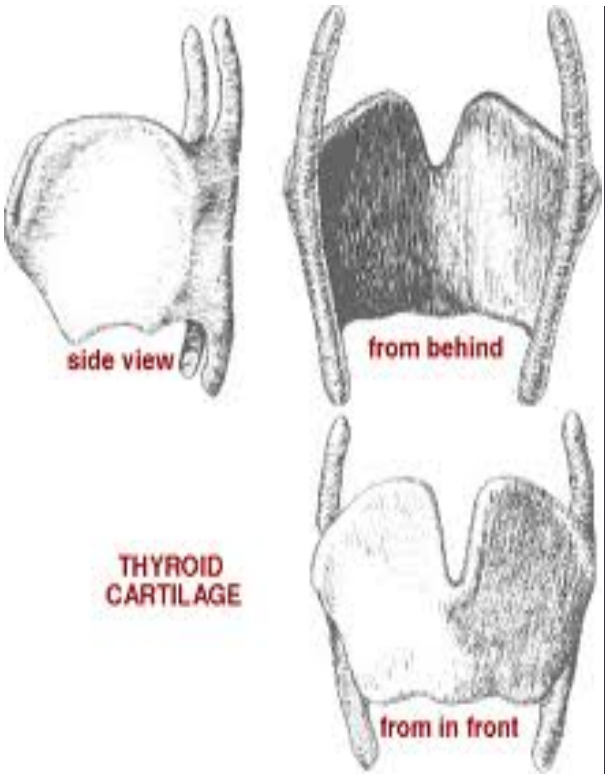
The program was geared to help clinicians master the concepts of thyroid physiology, assessment and clinical applications.

It was a great group of participants and I really enjoyed working with people in other fields of practice. I was the only NTP in attendance; the other practitioners were MDs, DOs, NDs and DCs.

The presenter was engaging. He knew his material and presented it with flair. So we had a really fun time while recommitting different biochemical processes to memory.

The next Apex seminar I will be taking is focused on Neurotransmitters and Brain Function. I will keep you posted.





Every Cell in the body has receptor sites for thyroid hormone.

"Thyroid hormones are responsible for the most basic and fundamental aspect of physiology, the basal metabolic rate."

Basal metabolic rate is the amount of daily energy expended by humans and other animals at rest.

Dr. Datis Kharrazian

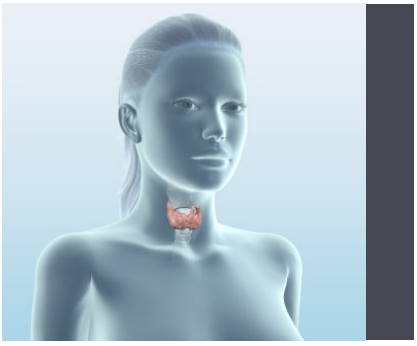
Why should you be interested in the health of your thyroid? Because the tiny butterfly shaped gland nestled under your Adam's Apple influences EVERY CELL IN THE BODY; *every cell* in the body has receptor sites for thyroid hormone. So the balanced and proper functioning of the thyroid is critical for balanced and proper functioning of *every cell* in the body.

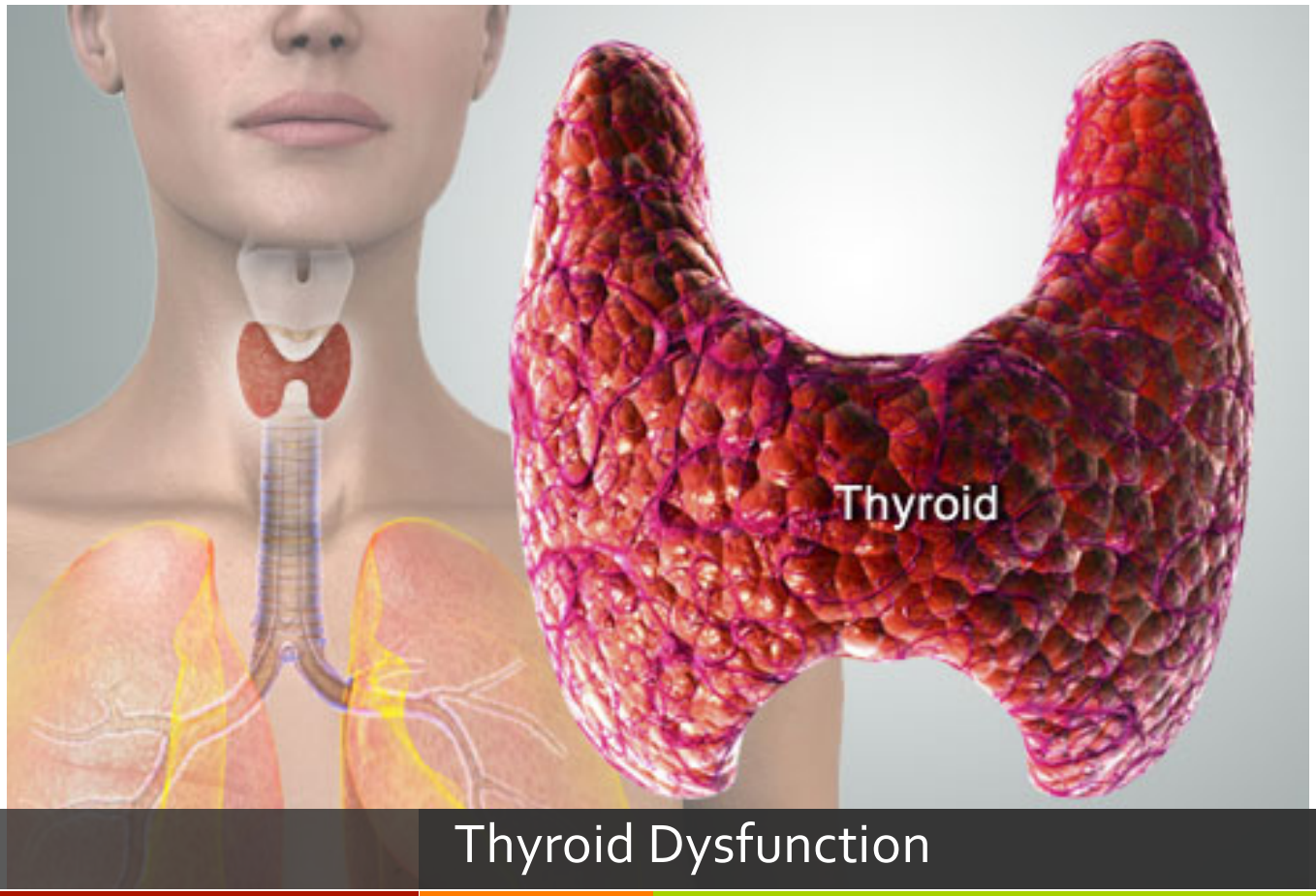
Essentially there are 10 Metabolic Steps for Proper Thyroid Hormone Synthesis and Activity:

- Proper serotonergic and dopaminergic activity in the hypothalamus
- Proper pituitary responses to hypothalamic signals
- Proper thyroid-peroxidase enzyme activity
- Proper iodine uptake to the thyroid gland
- Proper amino acid synthesis of tyrosine
- Proper attachment and detachment of thyroid binding proteins
- Proper 5' deiodinase enzyme activity
- Proper intestinal sulfatase and acetylation enzyme activity
- Proper nuclear receptor site sensitivity responses
- Proper thyroid proteomic responses

The thyroid axis begins with the hypothalamus where there is a group of nuclei called the paraventricular nucleus (PVN). The PVN is modulated by the signaling pathways of neurotransmitters: serotonin and dopamine. Once the PVN has been stimulated by serotonin and dopamine the hypothalamus releases TRH (thyroid releasing hormone) to stimulate the pituitary.

TRH stimulates the pituitary to release TSH (thyroid stimulating hormone). TSH stimulates metabolic activity of the cells and stimulates the TPO enzyme (thyroid peroxidase) to produce inactive T4 thyroid hormone and active T3 thyroid hormone. T3 hops a ride to the cells on TBG thyroid binding globulin. And T4 hops a ride to the liver on TBG. In the liver: 5' deiodinase converts 60% of the T4 to T3, 5 deiodinase converts 20% of T4 to rT3 (irreversibly inactive) and the remaining 20% of the T4 gets taken to the gut where intestinal sulfatase converts it to T3. The T3 hops another ride on TBG and goes to an open active receptor site on a cell.





Thyroid Dysfunction

As Dr. Kharrazian summarized: *thyroid hormone physiology starts with brain synaptic activity, goes to the pituitary, then to the thyroid gland, then to peripheral tissues such as the liver and gastrointestinal tract before it has a chance to become activated and bind to receptor sites on every cell. Any defect along this chain can decrease the potential for thyroid hormone synthesis and affect its effect on bodily tissues.*

Several mechanisms are important for ideal thyroid hormone synthesis and activation. So it is not surprising that there are 24 patterns of thyroid dysfunction that develop out of a breakdown in four main categories of thyroid physiology and biochemistry.

The first main category of dysfunction focuses on the hypothalamus-pituitary axis. Stress, excess cortisol, low stomach acid, liver congestion and cytokines can disrupt the serotonergic and dopaminergic activity in the hypothalamus and the pituitary responses to TRH. (Cytokines are cell-signaling immunomodulating protein molecules. They are how the cells of the body communicate with one another during an immune response).

The second area of dysfunction occurs in the thyroid itself. Thyroid peroxidase enzyme (TPO) activity can be compromised by autoantibodies. This can lead to a defect in iodine uptake in the thyroid or a defect in the amino acid synthesis of tyrosine; both of which inhibit the thyroid's ability to make T4 and T3.

The third broad category of dysfunction occurs in the liver and in thyroid hormone circulation. Thyroid binding proteins carry thyroid hormone to different areas of the body. When the system is irritated by an antigen, like gluten, the body can make autoantibodies against the thyroid binding protein TBG. When this occurs there is a breakdown in the method by which thyroid hormone travels through the body, the hormones simply can't get to the cells or to the liver for conversion.

Speaking of the liver, if the liver is congested, then 5' deiodinase and 5 deiodinase enzyme activity can become dysregulated. In an optimally functioning system, 5' deiodinase converts 60% of inactive T4 to active T3 in the liver, and 5 deiodinase converts about 20% of inactive T4 into irreversibly inactive rT3. If there is a deficiency in the way 5' deiodinase works you will end up with low levels of T3. Likewise, if 5 deiodinase is over active, you will end up with too high levels of rT3 and diminished T3. When there are low levels of T3 the metabolism of every cell in the

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body is effected.

The next area of dysfunction occurs in the gut. Systemic gastrointestinal inflammation diminishes intestinal sulfatase and acetylation enzyme activity (Acetylation is a biochemical reaction that conjugates toxins for removal from the body).

As you recall, 60% of the inactive T4 that is sent to the liver gets acted upon by 5' deiodinase and turned into T3, 20% gets acted upon by 5 deiodinase and turned into rT3 … the remaining 20% is sent to the gut to be turned into active T3 by intestinal sulfatase. If there is insufficient intestinal sulfatase due to an imbalance in gut flora, then the body will not be able to make active T3.

The final broad category of dysfunction lies in the thyroid hormone receptors in each cell. Receptor site sensitivity responses can be disrupted by stress, cortisol, cytokines and vitamin A deficiency. Thyroid receptors are found on the nucleus of the cell, in contrast to steroid receptors that are found on the plasma membrane of a cell. In the absence of thyroid hormones thyroid receptors bind to DNA. This leads to transcriptional repression … an essential mechanism in the precise control of gene expression. When in the cell, thyroid hormone is responsible for creating proteins needed for different biochemical processes. When receptor sites are not working properly, then the metabolic pathways in which proteins function are disrupted.

So you can see there are many ways in which the thyroid can be pushed out of optimal function. There are a total of 24 different patterns of thyroid dysfunction, and each one needs to be addressed differently. And, only ONE pattern, primary hypothyroidism, can actually be properly addressed by supplemental thyroid hormones (Synthroid, Levothyroxine, Cytomel, Armour, etc).

Primary hypothyroidism is a true dysfunction of the thyroid gland. It is the ONLY pattern of hypothyroidism that can be effectively managed by thyroid replacement hormones. In standard health care, doctors prescribe thyroid hormone if TSH is elevated.

Even though 90% of hypothyroidism in America is caused by Hashimoto's autoimmune reaction, doctors typically do not screen for Hashimoto's in people with an elevated TSH because it does not change their treatment strategy. This causes a problem because thyroid replacement hormones will not stop hypothyroid symptoms caused by an autoimmune reaction. The replacement hormone will only affect the TSH levels.

When a person with autoimmune hypothyroidism is treated with thyroid hormones the body adapts to the dosage of replacement hormone. When the body adapts, the TSH will elevate again because the main cause of the thyroid dysfunction (the autoimmune condition) has not been addressed and the disease process is still underway.

In the standard treatment model, the patient will require an even higher dosage of replacement hormone. This can be an ongoing cycle that repeats itself until the thyroid receptor sites on the cells are pushed into dysfunction by the flood of excess hormones. By this time the flood of excess hormones will also contribute to liver congestion. Over time the patient will exhibit several patterns of thyroid dysfunction simultaneously.

As Dr. Kharrazian points out: *thyroid replacement hormones are a first line of defense for many doctors, prescribed with the promise of wiping out a number of symptoms in one fell swoop. But taking that approach is turning a blind eye to what caused the thyroid to become depressed in the first place. Gut infections, adrenal problems and hormonal imbalances can all significantly depress thyroid function.*

So, if you have functional or pathological hypothyroidism you can see the importance of being able to identify exactly what is causing the dysfunction so that your thyroid can be treated appropriately.

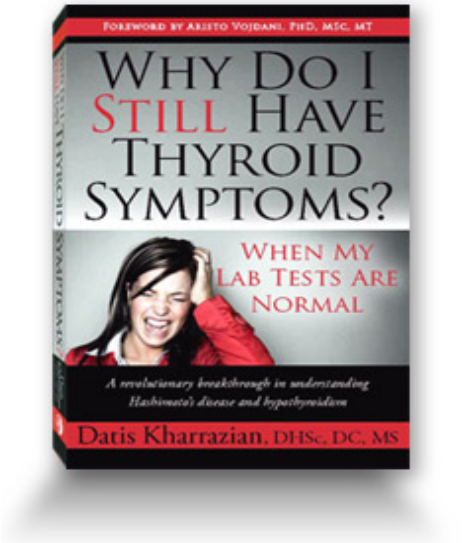
The 24 patterns of thyroid dysfunction are caused by thyroid illness and non-thyroid illness.

Thyroid illness includes primary hypothyroidism, primary hyperthyroidism, autoimmune Hashimoto's and autoimmune Grave's.

Non-thyroid illness, or euthryoid, consists of defects in thyroid metabolism that are not related to actual pathology in thyroid tissue, but rather to defects in thyroid physiology. Euthyroid disorders cause the clinical symptoms of hypothyroidism, but their laboratory test patterns are typically overlooked or dismissed in the standard healthcare system.

Both thyroid illness patterns and euthryoid can cause defects in thyroid physiology that lead to the 24 patterns of thyroid dysfunction.

To learn more about why taking thyroid hormones and even some popular thyroid supplements may not help and can even make your symptoms worse I recommend Dr. Kharrazian's Thyroid Book.



What your doctor hasn't told you about hypothyroidism, and what you need to know:

For 90% of Americans, hypothyroidism is caused by Hashimoto's, an autoimmune thyroid disease.

Thyroid replacements (Synthriod, Armour, Cytomel, etc) may normalize TSH, but they do not manage the autoimmune disease symptoms.

If you have a thyroid disorder, you MUST avoid gluten – strictly. Studies link gluten intolerance with Hashimoto's and Grave's disease.

Pituitary function plays a role in underactive thyroid symptoms.

Adrenal function plays a role in underactive thyroid symptoms.

Thyroid hormone resistance, under- and over-conversion to T3 and other metabolic factors drive hypothyroidism symptoms.

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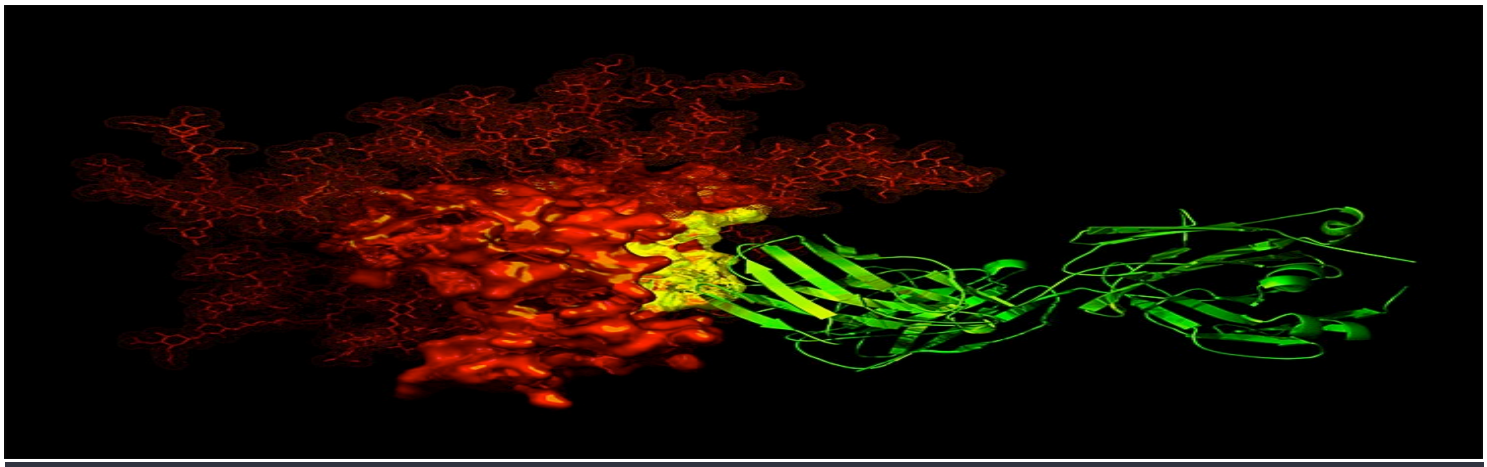
The 24 Thyroid Patterns

1. Hypothalamus paraventricular defect related to central nervous system deficiency of serotonin leading to low TSH
2. Hypothalamus paraventricular defect related to central nervous system deficiency of dopamine leading to low TSH
3. Hypothalamus paraventricular defect promoted by cytokines leading to low TSH
4. Hypothalamus paraventricular imbalance promoted by elevated prolactin
5. Pituitary suppression from cortisol leading to low TSH
6. Thyroid tissue imbalance related to thyroid peroxidase autoimmune response
7. Thyroid tissue imbalance related to thyroglobulin autoimmune response
8. Thyroid tissue imbalance related to thyroid stimulating hormone autoimmune response
9. Autoimmune response to (T4) and (T3)
10. Down-regulated thyroid peroxidase activity related to progesterone deficiency
11. Down-regulated thyroid peroxidase activity from deficiency of cofactors
12. Down-regulated 5'-deiodinase activity from deficiency of cofactors
13. Down-regulated 5'-deiodinase activity from gastrointestinal dysbiosis and lipopolysaccharides
14. Down-regulated 5'-deiodinase activity from elevated cytokines
15. Down-regulated 5'-deiodinase activity from elevated cortisol
16. Down-regulated 5'-deiodinase activity from deficiencies of serotonin
17. Down-regulated 5'-deiodinase activity from peripheral deficiencies of dopamine
18. Up-regulated 5'-deiodinase activity from elevated testosterone
19. Elevations of thyroid-binding globulin leading to decreased percentage of free fraction thyroid hormone
20. Depression of thyroid-binding globulin leading to increased percentage of free fraction thyroid hormone
21. Thyroid resistance promoted by an elevation of cytokines
22. Thyroid resistance promoted by an elevation of cortisol
23. Thyroid resistance promoted by deficiencies of vitamin A
24. Thyroid resistance promoted by elevations of homocysteine



What's the Big Deal about TPO antibodies?

TPO AB is the antibody that the body makes against thyroid peroxidase enzyme … an enzyme responsible for the production of thyroid hormone. If you test positive for TPO AB it means that your body has started the autoimmune process … it is not a question of *if* you have an autoimmune reaction it is a question of WHEN you will develop the serious signs of disease. TPO AB is produced when you have a 'leaky gut' reaction to gluten. If you test positive for TPO AB it is imperative that you follow a strict gluten free diet. The half-life of TPO AB is 4 months; one bite of gluten can affect you for nearly a year!

Don't Waste Another Day feeling lousy because you're trapped in outdated standards of health care. Read Why Do I Still Have Thyroid Symptoms by Dr. Kharrazian and make an appointment to have your Functional Blood Chemistry Analysis and Nutritional Coaching Session.

Remède Physique

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Hello Good People!

Thank you for your interest and support.

Email questions, comments or requests for future newsletter topics to Vanessa Hendley

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