

Help, My body is Killing Me!

Solving the connections of autoimmune disease to thyroid problems, fibromyalgia, infertility, anxiety, depression, ADD/ADHD and more

By Dr. Kevin Connors

Section 5

Let's remember the RULES:

RULE 1 – Is the patient autoimmune? There are symptoms that mimic autoimmune disorders that may not yet be an actual immune attack on one's tissues. If the patient has already been diagnosed with an autoimmune disease, this step is complete. Move to RULE 2.

RULE 2 – What is the Antigen and How are we going to get RID of the antigen? You will learn that, by definition, an autoimmune disease is a disorder where your immune system is creating antibodies of self-tissue. Whatever you have antibodies for, be it a bacteria or your thyroid tissue, your killer side of your immune system will destroy it. I don't think ANY autoimmune patient that I have ever consulted over the decades has had this addressed by their doctor! It is crucial to know what the antigen is, and as you'll see through this book, a person cannot have an autoimmune condition without initially having an antigen that the immune system originally was trying to kill.

When this question is posed to the doctor by the patient – “Doctor, if I have an autoimmune disease, what is the antigen?” – The blank stare and awkward pause that follows should be a sign that the discussion probably won't end well. Immunology 101 states that the immune system does many things but the single-minded focus of the cell-mediated, TH1 response (more on this later) is to KILL the foreign invader – AKA the ANTIGEN.

In autoimmune disease, the immune system is killing self-cells but it didn't start this way. Initially, it was attempting to kill a pathogen and

LATER started creating antibodies to self-tissue – usually because the antigen was able to HIDE inside the patients cells! We'll discuss this as we go.

As long as the antigen remains in the patient's body, there is NO way to correct the situation. The immune system may be likened to a viper that will never let go until it has killed its prey. You NEED to find out WHAT the antigen IS and then RID it from your body!



RULE 3 – Is the patient Th1 or Th2 dominant? When a patient is autoimmune, there is usually one 'side' of their immune response that is in hyper-response mode. You'll learn about these two important parts of the immune response and their role in self-destruction. Knowing if you are Th1 or Th2 dominant is CRUCIAL for several reasons but most importantly, it dictates proper care.

RULE 4 – How are we going to balance the rest of the organ systems? When a person has any long-standing illness, there will be other organs that have paid the price. The barrier systems of the gut and brain are most assuredly compromised; the hormones, adrenals,

pituitary, and thyroid are strained; and other systems like liver detox pathways and kidneys need support.

We MUST investigate all these systems with SPECIFIC functional testing and then develop appropriate protocols to support and re-charge the cellular batteries.

Adrenal Stress

Adrenal insufficiency and adrenal stress is measured through the Adrenal Stress Index. The adrenal glands are two glands that are embryologically neurological tissue that sit right above each kidney. They are very important in function of hormonal activity, energy and stress management. The adrenal glands are made up of an exterior covering or cortex of the gland which produces steroid hormones cortisol, aldosterone, progesterone, and DHEA. And then, the inner part of the adrenal gland is called the medulla that produces the catecholamines such as epinephrine and norepinephrine which have to do with your sympathetic nervous system function.

Our nervous system is made up of two basic components, a voluntary nervous system and an involuntary nervous system. The involuntary nervous system (the autonomic nervous system or one might say an automatic nervous system) is made up of two separate components that work in harmony and balance. They are the sympathetic part of the autonomic nervous system and the parasympathetic part. The sympathetic part may be best termed as the fight, flight, or freeze mechanism; the parasympathetic part has to do our metabolism, digestion, and the calming aspect of the autonomic nervous system. Both need to be in balance. If a person is hyper-sympathetic or is functioning in a very high stress situation for extended periods of time their adrenal glands are in a state of hyperfunction and can end up

burning out. We weren't meant to live in a society where we're constantly running away from a grisly bear.

Burning the candle at both ends tends to cause a high degree of hyper-sympathetic nervous system function, which relates to greater adrenal output and adrenal stress. Prolonged stress leads to adrenal fatigue and exhaustion, not to mention exhaustion to the other parts of the brain that are the stimulators of the adrenal and the pituitary output. A lifestyle issue may have been an initiator for this, but adrenal fatigue is a negative cycle that will drive the autoimmune patient into the ground if not concurrently addressed.

Lisa's Story

“Bill and I have been married for fourteen years since I was 22 years old. I had just graduated for college and even though Bill was ready for a family right away, I wanted to wait a few years to make some money and get some of my student loans paid. When I turned 28, I thought I'd better listen to my mother regarding my 'biological clock'. I was ready to start a family so I got off the pill. After a year of trying and nothing was happening, we made an appointment with my regular doctor. He did a bunch of tests on both Bill and I and I only remember him saying that there was no reason that I couldn't get pregnant, so we just kept trying. Another year passed and we were both starting to get a little worried. My doctor referred me to a fertility specialist and we went through a lot of expensive testing to be offered even more expensive procedures that had no guarantee. A friend referred me to her doctor, a chiropractic neurologist who specialized in autoimmune diseases and fertility problems. I thought, “you're kidding right?” I could not, for the life of me, think that he was going to be able to do anything

for me! Well, I gave it a shot, he offered a 100% guaranteed plan and Bill said we had nothing to lose. Bill was wrong; I lost my infertility! I was pregnant in 6 months and have a beautiful baby girl. It turned out that I had Hashimoto's disease, which is an autoimmune problem with the thyroid. That is why I couldn't get pregnant. None of my other doctors caught it."

The adrenal cortex also makes some of our sex hormones and a very important percentage of them in the postmenopausal female. The ability of the adrenal glands to secrete these reproductive hormones postmenopausal has a very important impact on the intensity of menopausal symptoms. If a woman is going through hot flashes and sweating, anger issues, and an inability to handle stress, it has to do with the fluctuations of estrogen from a fatigued adrenal system. The adrenal glands are supposed to regulate the estradiol levels in the blood as the ovaries are down-regulated with age, giving the woman a smooth, symptom-less transition into menopause.

When a woman becomes perimenopausal, the pituitary is decreasing its production of follicle-stimulating hormone, the hormone that functions in release of estradiol. The adrenals take over at this point to balance the decreased production. If the adrenals are exhausted, they don't make up that balance in estradiol and the woman has strict, strong fluctuations in estradiol levels, and end up with huge swings in energy, hot flashes, sweating, etc., that go with perimenopausal symptoms.

Cortisol is the primary glucocorticoid secreted by the adrenal cortex and increases the blood glucose concentration. A person in a high-stress situation will release more cortisol, and that cortisol increases the blood glucose. This makes a lot of sense from a sympathetic, fight or flight mechanism; when a giant grisly bear jumps on our path, we need a lot of glucose in our bloodstream for muscle cells to make ATP, so we can run away from our predator. But it doesn't make a lot of sense when our job is just extremely stressful causing our adrenals to put out vast amounts

of cortisol on a daily basis. Our sympathetic nervous system can't tell the difference between a real grisly bear encounter and stock market crash; the results will be the same. The real or perceived exogenous stress source causing a hyper-adrenal output causes a hyper-cortisolemia leading to a hyper-glucose reaction and a hyper-insulinemia. This is what we call a negative cycle; it spins us downward towards greater sickness and death.

High adrenal output causes a lot of damage to other tissues and may, in itself, be a source for an autoimmune response. It certainly is a component in the autoimmune system and, again, an absolute must in treating concurrently with an autoimmune disease. Treatment is aimed at supporting the organ with chemicals called adaptogens, herbal formulas that are known to balance high and low adrenal outputs. We also use a compound called phosphatidylserine, which has a balancing affect on the hippocampal formation, the part of the brain that senses these stressful situations. It's this hippocampal-hypothalamus-pituitary-adrenal link that we want to calm down.

Common signs and symptoms of adrenal problems are going to be fatigue, headaches, and multiple allergies. Because a person's stress response is constantly firing, it fatigues their immune system. Stomach ulcers are also common as is cravings for sweets or caffeine. They'll have addictions to food, alcohol, drugs, or cigarettes. Other signs and symptoms can be dizziness, asthma issues, varicose veins, blood disorders, and blood pressure issues.


Adrenal dysfunction is also tied to the midbrain and the frontal lobes which can lead to anxiety and depression which may be the chief symptom that brings the patient to the office. We see a high number of patients today that are on brain-altering, mind-changing medication due to stress issues that are really adrenal issues affecting the frontal lobes; all these could be addressed from a functional medicine perspective, if you address all the systems that are tied together. By the time the person has symptoms that bring them into a doctor's office, whether it's a

medical doctor or an alternative practitioner, there has been down regulation of multiple systems.

Though it's impossible to discuss every system and the cellular response necessary to thoroughly analyze a patient with autoimmune disease in this book, we would be amiss if we didn't discuss the gastrointestinal component because it always exists in autoimmune disorders and is essential for us getting the nutrients in order to regulate all the systems.

The spectrum of autoimmune disease

Organ Specific Autoimmune Diseases

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- ◆ Graves Disease (Thyroid: TSHR Abs, TPO Abs)
 - ◆ Hashimoto Thytreoiditis (Thyroid: TPO Abs, Tg Abs)
 - ◆ Diabetes Type I (Pankreas: GAD II Abs, IA2 Abs, ICA)
 - ◆ Goodpasture Syndrome (Kidney: GBM Abs)
 - ◆ Pernicious Anemia (Stomach: Parietal Cell Abs)
 - ◆ Primary Biliary Cirrhosis (Liver, Bile: AMAbs)
 - ◆ Myasthenia Gravis (Muscles: AChR Abs)
 - ◆ Dermato-/Polymyositis (Skin / Muscles: Jo 1 Abs)
 - ◆ Vasculitis (Vessels: ANCA)
 - ◆ Rheumatoid Arthritis (Joints: CRP, RF, RA33 Abs, Sa Abs)
 - ◆ MCTD (RNP Abs)
 - ◆ Scleroderma (Scl 70 Abs, CENP Abs, PM/Sci Abs)
 - ◆ SLE (ANA, Cardiolipin Abs, Beta 2 GP I Abs)

Multi-systemic Autoimmune Diseases

Other Named Autoimmune Diseases:

Amyotrophic lateral sclerosis (ALS)

Many autoimmune processes are fatal. ALS, also named Lou Gehrig's disease after the famous Baseball player who died due to ALS, is a progressive, fatal neurological disease that attacks the nerve cells (neurons) responsible for controlling muscles. In ALS, both the brain and the spinal neurons degenerate and die. ALS is triggered by diverse antigens; some are toxic exposures, injuries and infections. One study in Guam looked at the pathogenesis of the disease revealed high aluminum in the water and a plant excitatory neurotoxin as possible causes. In Italy, an increasing number of soccer players have developed ALS; some have connected this with the use of illegal toxic substances or exposure to pesticides used on playing fields.

Symptoms of ALS include muscle weakening, muscles waste away and twitch. Patients with ALS lose their strength and the ability to move their arms, legs, and body. Muscles in the diaphragm and chest wall fail, and then patients lose the ability to breathe. In most cases the disease does not impair a person's mind, a small percentage of patients may experience problems with memory or decision-making, and there is growing evidence that some may even develop dementia.

Bell's palsy

Bells Palsy is a sudden onset of inflammation in the Facial Nerve (Cranial Nerve 7). This is often temporary but left untreated, can become permanent. Most noticed as a facial droop caused by inflammation and paralysis of the facial nerve. The inflammation in the facial nerve is the result of autoimmune processes and can involve the either side. A swollen facial nerve can be compressed in the facial canal. Facial palsy can be an early manifestation of Lymes disease.

Usually the causative factor is thought to be herpes virus, cytomegalovirus (CMV) or Borrelia by most standard medical professionals, but any antigen can be at cause. Facial palsy may be triggered by exposure to cold air draft, high amounts of stress and everything else that may fire an immune response.

Symptoms range in severity from mild weakness causing a facial droop to total uni-lateral paralysis and may include twitching, weakness, drooping eyelid or corner of the mouth, drooling, dry eyes, impairment of taste, and excessive tearing in the eye. Bell's palsy can occur on both sides when accompanied by Guillain Barre syndrome.

Cardiovascular Autoimmune Diseases

Some speculate that the leading cause of death in the world, heart disease, is due to inflammatory reactions in the capillary wall at the site of atherosclerosis. It is the plaguing, caused by a high homocysteine level which then etches the arterial wall. This 'scratch' is then healed with a cholesterol patch. The high homocysteine levels are tied to inability to absorb vitamin B12 and folic acid – a problem endemic in autoimmune disorders. These nutrients are essential in converting homocysteine to its harmless substrate. Some have termed this celiac disease myocarditis, which is a misnomer since celiac disease accompanies few of the coronary conditions. It can be ANY autoimmune disorder that can lead to cardiovascular autoimmune disease. However it is a treatable condition and anti-gliadin and other antibodies as well as cytokines can be checked in patients who are unresponsive to conventional treatments. These patients respond to dietary management and TH1/TH2 control, antigen removal, sublingual B12/Folic Acid supplementation, and diet guidelines that are in the Gluten Diet section.

Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)

CIDP is an acquired immune-mediated inflammatory disorder of the peripheral nervous system. The disorder is sometimes called chronic relapsing polyneuropathy. CIDP is closely related to Guillain-Barré syndrome and it is considered the chronic counterpart of that acute

disease. Its symptoms are also similar to progressive inflammatory neuropathy. An asymmetrical variant of CIDP is known as Lewis-Sumner syndrome.

The pathologic hallmark of the disease is loss of the myelin sheath (the fatty covering that protects nerve fibers) of the peripheral nerves. Chronic inflammatory demyelinating polyneuropathy is believed to be due to an autoimmune response from a foreign infection attacking the peripheral nerves. As a result, the affected nerves fail to respond, or respond only weakly, to stimuli causing numbing, tingling, pain, progressive muscle weakness, loss of deep tendon reflexes, fatigue, and abnormal sensations. The likelihood of progression of the disease is high.

Dermatitis, Autoimmune Atopic or Eczema

Atopic dermatitis (AD) is a chronic, itching, inflammatory skin disease which is associated with asthma or hay fever and a familial occurrence of these conditions. The disease comes in attacks that seem to get better then worse in cycles. There are a number of different eczemas – rashes which cause the skin to become inflamed and itchy. AD is also called 'atopic eczema' or 'infantile eczema'. It affects people with dry and rough skin and may be caused by a variety of antigens.

Patients with atopic dermatitis often have elevated serum IgE levels and a hyper-sensitization to a variety of environmental allergens quite possibly due to an autoimmune response elsewhere in the body. This 'remote' autoimmune response leaves the patient hyper-Th1 or hyper-Th2, making them hyper-sensitive to a variety of allergens they would not normally react to.

Guillain–Barré syndrome (GBS)

GBS is an *acute inflammatory demyelinating polyneuropathy* (AIDP), an autoimmune disorder affecting the peripheral nervous system, usually

triggered by an acute infectious process. The syndrome was named after the French physicians Guillain, Barré and Strohl, who were the first to describe it in 1916. It is sometimes called Landry's paralysis, after the French physician who first described a variant of it in 1859. It is included in the wider group of peripheral neuropathies. It is frequently severe and usually exhibits as an ascending paralysis noted by weakness in the legs that spreads to the upper limbs and the face along with complete loss of deep tendon reflexes.

The disorder is characterized by symmetrical weakness which usually affects the lower limbs first, and rapidly progresses in an ascending fashion. Patients generally notice weakness in their legs, manifesting as "rubbery legs" or legs that tend to buckle, with or without dysesthesias (numbness or tingling). As the weakness progresses upward, usually over periods of hours to days, the arms and facial muscles also become affected. Frequently, the lower cranial nerves may be affected, oropharyngeal dysphagia (drooling, or difficulty swallowing and/or maintaining an open airway) and respiratory difficulties.

Guillain–Barré syndrome is due to an immune response on the peripheral nerves and damage to the myelin, the fatty insulating layer of the nerve, leading to a muscle paralysis that may be accompanied by sensory or autonomic disturbances.

Lyme disease

SEE MY COMPLETE BOOK ON LYME below:

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But briefly: Lyme disease was first recognized in 1975 after a number of cases occurred in the same town in North America. It subsequently took its name from this town, which was called Old Lyme, in Connecticut. Lyme disease, which is spread to humans by a small bug called the deer tick. This bug passes a spirochete called *Treponema Pallidum* to the

human. Lyme borreliosis is due to infection with the spirochete *Borrelia burgdorferi*, and is associated with persistent infection unless treated with antibiotics within the first several weeks. The persistent nature of infection by *B. burgdorferi* can lead to development of chronic autoimmune disease. Lymes transforms into multiple autoimmune conditions.

Klempner did a study in chronic Lyme and found that antibiotics did not change the course of disease once it became chronic. Then NIH (National Institutes of Health) recommended that autoimmune basis of Lyme disease needs to be explored. Currently *Borrelia* antibodies have been associated with remitting relapsing Multiple sclerosis, Thyroiditis, carotid artery disease, epilepsy and arthritis.

Usually the first sign of Lyme infection is a circular ‘bulls-eye’ skin rash at the point of entry. This can easily be overlooked and missed if the bite is in the scalp. This follows with symptoms of tiredness, headache, joint pains, and flu-like symptoms. If not treated these symptoms may last for weeks, months, years and even decades. As the disease progresses then shortness of breath, chest pains, weakness, and tingling numbness in the legs and arms starts. Some may start to notice memory problems, difficulty concentrating and fatigue as well as joint swelling and arthritis. Usually the blood tests for Lyme show false negatives after the first 30-60 days. As with all autoimmune diseases, the first course of action is to locate and eliminate the antigen.

Migraine autoimmune headaches

Migraine affects 35 million Americans, most of whom are women. Migraine is preceded or accompanied by a sensory warning signs called a (aura), such as flashes of light, blind spots, smell or tingling in your arm or leg. A migraine headache can follow with signs and symptoms, such as nausea, vomiting and sensitivity to light and sound. Migraine pain is usually throbbing and can last for hours or even days.

Inflammatory markers go up rapidly in an attack of Migraines, CRP is elevated, the spinal fluid protein becomes elevated and more white cells are seen in the spinal fluid during a migraine attack. Migraine and epileptic seizure disorders are interrelated and like other autoimmune diseases migraines happen more in women. There may be associated epilepsy with migraine.

Migraine often comes in remissions and relapses just like autoimmune disease. Migraine is associated with women just like autoimmune disorders. Some women with Lupus present with migraine, as their first symptom. Following anti-inflammatory treatment their migraine attacks usually resolve. Many patients with lupus present with migraines secondary to severe vasospasm. These patients have anti-phospholipids antibodies and at times the migraine will only respond to steroids or cyclophosphamide. MRI scans obtained during a migraine have shown dramatic thickening of brain folds called (gyral) with enhancement which suggests inflammation.

General symptoms of Migraine

- One-sided throbbing head pain which worsens with physical movement.
- Nausea, Vomiting
- Twisted shining lines in front of the eye sometimes without a headache.
- Weakness or numbness in a hand or leg
- Sensitivity to light (Photosensitive headaches respond to magnesium)
- Sensitivity to sound, smell, and light, (patients prefers a dark room)

Multiple Sclerosis

Multiple sclerosis (MS) is a chronic, autoimmune disease in which the immune system attacks the Myelin covering the nerves in the brain and

spinal cord. This is similar to CIDP neuropathy where the attack is against peripheral myelin. The myelin in the Brain and spinal cord is made by cells called oligodendrocytes and in the peripheral nerves by Schwann cells.

Multiple sclerosis can develop after exposure to Epstein-Barr virus (EBV), Chlamydia, STD's, other parasitic infestations, as well as environmental toxins and foods. The body then incorrectly directs antibodies and white blood cells against the myelin sheath, which surrounds nerves in the brain and spinal cord. This causes inflammation and injury to the myelin-sheath. This damage results in multiple areas of scarring (sclerosis). Eventually, this damage can slow or block the nerve signals that control muscle coordination, strength, sensation and vision. This damage can be visualized by a M.R.I. scan as multiple white spots in the brain.

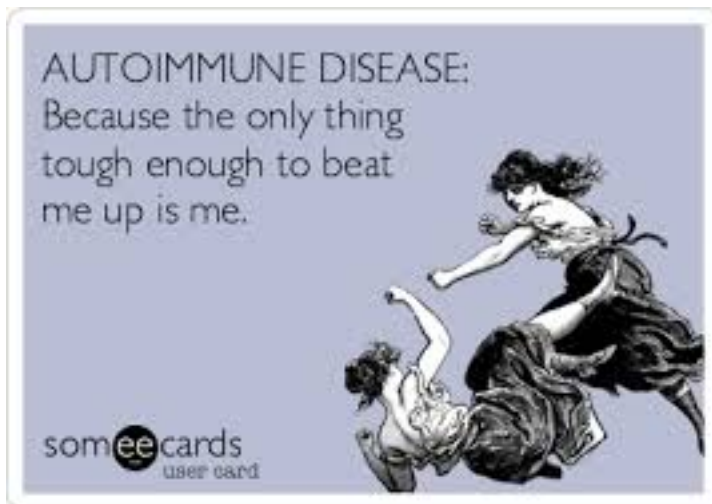
Different Types of MS:

- **Relapsing remitting** type of MS is seen in 90% of the cases characterized by relapses (disease flare-ups), followed by periods of remission. This is the most common type. I have seen many cases where the patient was labeled as progressive MS only to find they had clear history of remissions and relapses.
- **Primary progressive** form of MS, which shows a gradual decline, without periods of remission. People with this form of MS are usually older than 40 when symptoms begin.
- **Secondary progressive.** About half the people with relapsing remitting MS eventually enter a stage of continuous deterioration referred to as secondary progressive MS.
- **Progressive M.S.** Not a good medical prognosis as the disease progresses rapidly.

Symptoms include:

- Numbness or weakness which typically occurs on one side of the body.

- Double vision, blurring of vision or sudden loss of vision (optic neuritis).
- Tingling numbness or pain one half of the body.
- Electric-shock sensations that occur with certain head movements
- Tremor, lack of coordination or unsteady gait and weakness.
- Fatigue specially after exposure to heat, or exercise.
- Dizziness or feeling of spinning.



Myasthenia Gravis

Myasthenia Gravis is a chronic autoimmune, neuromuscular disease characterized by weakness of the voluntary (skeletal) muscles of the body. The name myasthenia gravis, means "grave muscle weakness." The hallmark of myasthenia gravis is muscle weakness that increases during activity and improves after of rest.

Certain muscles such as those that control eye and eyelid movement, facial expression, chewing, talking, and swallowing are often, involved in the disorder. The muscles that control breathing, neck movements and limb movements may also be affected when a nerve impulse travels down the nerve; a chemical neurotransmitter called acetylcholine is released in the nerve ending and travels to acetylcholine (Ach) receptors

located on the muscle side of the synapse, causing the muscle to contract. Among people with myasthenia gravis, this normal impulse transmission of Ach is disrupted by autoantibodies that target the body's own Ach- receptors and block them. If enough receptors are blocked by autoantibodies, then the muscle contraction will be weak, causing the symptoms of myasthenia gravis.

Many pesticides contain organophosphorus chemicals that can inhibit the acetyl cholinesterase enzyme and make myasthenia worse. Halides (like **chlorine** and **fluorine**) may pose additional risk for myasthenia gravis patients. In one case report, a patient was exposed to chlorine gas and subsequently developed generalized myasthenia gravis). Fluoride is also implicated, and fluoridated water may trigger a myasthenia gravis crisis or contribute to long-term deterioration, with extreme exhaustion and muscle weakness, so please avoid fluoride containing toothpaste.

Reflex sympathetic dystrophy R.S.D or Complex regional pain syndrome:

Complex regional pain syndrome or RSD is essentially inflammation of the autonomic nerves in a localized area. RSD has been associated with injury dating back to the Civil War. We have already described the association of autoimmune disorders with injury. In general, patients who have complex regional pain syndrome suffer from pain, sensory changes, edema, sweating, and temperature disturbance in the afflicted extremity. Chronic changes can involve the skin, nails, and bone. Persistent inflammation, of the sympathetic nervous system and the central nervous system causes this condition.

Symptoms include increased sweating, skin color changes, skin temperature changes, weakness of the affected area, swelling, as well as symptoms outside the affected dermatome.

Restless Leg Syndrome

People with restless leg syndrome, or RLS, have a creepy-crawly feeling in their legs. This causes an irresistible urge to move the legs. It's a major cause of sleep loss, as the symptoms are most likely to occur at night. It has been found that brain cells need iron, oxygen carried by hemoglobin, and activation. They get nutrients from transport molecules that carry iron from the blood. Normal brain cells have doorways that let these transport molecules into the cell. Patients with restless leg syndrome lacked these portals, known as transferrin receptors. This means in spite of adequate amounts of iron in the blood not enough of it can enter the brain to prevent molecular damage.

Previous studies have shown that bacterial overgrowth in the small intestine causes inflammatory cells to increase production of IL-6. This cytokine, in turn, is known to boost levels of hepcidin, a protein that decreases iron absorption and transport. Bacterial overgrowth in the gut could be causing the problems and natural, anti-parasitic therapy targeting the stomach and small intestine might be the solution. RLS behaves differently from other autoimmune diseases, as this condition will increase during pregnancy. RLS is seen commonly, in patients with Fibromyalgia and Irritable Bowel syndrome.

Rheumatic Autoimmune Disorders

Rheumatoid arthritis is triggered by Mycoplasma. RA is two to three times more common in women than in men and generally strikes between the ages of 20 and 50. Rheumatoid arthritis can also affect children. The diagnosis is based upon clinical examination and elevated ESR or CRP along with x-rays showing early damage in the joints.

Investigators have shown that Mycoplasma which is a small bug without a cell wall causes arthritis in humans. Mycoplasma antigens have been found in all grains and their products, they are ubiquitous in our storehouses and impossible to avoid.

In 1949 at the International Congress on Rheumatic Diseases reported the relationship between Mycoplasma and joint disease. National Institutes of Health (NIH) issued a research grants in 1950, to Thomas Brown, M.D., who reported an immunologic reaction of antigen and antibody (with Mycoplasma as the suspected antigen) as the cause of rheumatoid disease. Further support of Mycoplasma as a causative agent and antigen was proven in 1964, when a high incidence of Mycoplasma antibodies in the blood of rheumatoid arthritis patients and lupus patients was found. Also recognized was a 4:1 higher incidence of Mycoplasma antibodies in females suggesting a correlation with the higher incidences of rheumatoid arthritis in females. In 1989, NIH requested grant applications for the controlled clinical trials of tetracycline therapy for rheumatoid arthritis. The preliminary results of the clinical trials, known as MIRA or Minocycline in Rheumatoid Arthritis, were promising and the NIH requested grant applications for studies of Mycoplasma as causes for rheumatoid diseases in 1993 and for a study for intravenous antibiotics for rheumatoid arthritis in 1994. The result of the MIRA clinical trial stated, that Patients who suffer from mild to moderate can benefit from Minocycline. A review of ten randomized controlled trials involving 535 patients were reviewed, reviewers reported Minocycline was associated with a clinically improvement in disease activity in RA with no absolute increased risk of side effects.

Symptoms of RA:

- Pain and swelling in joints, especially in the smaller joints of your hands and feet
- Generalized aching or stiffness in joints after sleep or after periods of rest
- Reduced motion of the affected joints; deformity of joints over time
- Weakness in muscles attached to the affected joints
- Fatigue, which can be severe during a flare-up , Low-grade fever
- General sense of not feeling well (malaise)

In RA, the joints in the wrists, hands, feet and knees are most often affected. Later in the disease, shoulders, elbows, hips, jaw and neck can be involved. It generally affects both sides of your body at the same time. Small lumps, called rheumatoid nodules, may form under the skin at pressure points and can occur at elbows, hands, feet and Achilles tendons.

Rosacea

Rosacea is a common autoimmune allergic condition characterized by symptoms of facial flushing and a spectrum of clinical signs, including erythema, telangiectasia, and coarseness of skin, edema, papules, pustules, ocular lesions and an inflammatory papulopustular eruption resembling acne. Rosacea affects mostly adults, usually people with fair skin, between the ages of 30 and 60. About 16 million Americans have this skin condition. Although it's more common in women, men may develop the disorder. Left untreated, rosacea tends to be progressive, which means it gets worse over time. Rosacea is remitting and relapsing, it flares up for a period of weeks to months and then signs & symptoms lessen for a while before rosacea flares up again.

Rosacea fulminans is a sub type of rosacea, occurs exclusively in women well past adolescence.



There is a high prevalence of *Helicobacter pylori* (Hp) infection seen in patients with rosacea, with evidence of dermatological improvement in patients treated with antibiotics for this infection. In a study done on Rosacea patients after eradication of Hp, 51 out of 53 treated rosacea patients became Hp negative. The symptoms of rosacea disappeared in 51 patients, markedly declined in one and remained unchanged in one patient. Conclusion from this study is that Hp eradication helps a majority of patients with Rosacea.

Scleroderma & C.R.E.S.T

Scleroderma is an autoimmune disease that can cause thickening, hardening, or tightening of the skin, blood vessels and internal organs. Scleroderma is chronic, which means it can last a long time. This is one disease in which the patients never gain weight, due to the effect it has on tightening their skin they all look skinny. They also have very hard hands, with skin around the fingers tight. They have difficulty swallowing as the esophagus is tight, so is the stomach wall tight and they cannot tolerate large meals. There are two types of scleroderma localized and systemic.

A) Systemic Scleroderma (SS) also called systemic sclerosis, the immune system causes inflammation in the small blood vessels and the collagen-producing cells located in the skin and throughout the body. SS causes the small blood vessels in the fingers to be inflamed; this causes injuries on the hands and fingers to heal slowly. In severe cases, ulcers form on the hands and fingers. People with Systemic Scleroderma are usually cold-sensitive. The inflamed small blood vessels and the reduced blood supply cause cold temperature sensitivity. Systemic Scleroderma patients also have problems with their heart, lungs and gastrointestinal tract. These problems occur as tissue builds up in the skin and organs due to inflammation.

B) Localized Scleroderma called Morphea affects the collagen-producing cells in just some areas of the body, and usually does not

affect the internal organs and blood vessels. Localized Scleroderma can be seen as patches of thick skin or as a line of thick skin. The line may extend down a leg or arm.

C) A sub type of scleroderma is called CREST which has a distinct set of characteristics that give the syndrome its acronymic name. These characteristics include:

- **Calcinosis:** Tiny calcium deposits form under your skin, on elbows, knees and fingers; and can occur almost anywhere, in the body.
- **Raynaud's phenomenon:** The hands and forearms become cold and blue due to inflammation in the blood vessels the upper extremity.
- **Esophageal dysfunction:** Inflammation in the stomach and esophagus can cause swallowing problems and retention of fluids in the stomach.
- **Sclerodactyly:** Thick hard patches of skin start to calcify. This bone-like skin can even be seen on X-ray.
- **Telangiectasia:** Small, spider-like blood vessels start to form on lips and fingers.

Transverse Myelitis

The National Institutes of Health list Transverse myelitis as a neurological disorder caused by inflammation across both sides of one level, or segment, of the spinal cord. The term *myelitis* refers to inflammation of the spinal cord; *transverse* simply describes the position of the inflammation, that is, across the width of the spinal cord. Attacks of inflammation can damage or destroy myelin, the fatty insulating substance that covers nerve cell fibers, much like that in MS. This damage causes nervous system scars that interrupt communications between the nerves in the spinal cord and the rest of the body.

Symptoms of transverse myelitis include a loss of spinal cord function over several hours to several weeks. What usually begins as a sudden onset of lower back pain, muscle weakness, or abnormal sensations in the toes and feet can rapidly progress to more severe symptoms, including paralysis, urinary retention, and loss of bowel control. Although some patients recover from transverse myelitis with minor or no residual problems, others suffer permanent impairments that affect their ability to perform ordinary tasks of daily living.

Autoimmune in nature, TM has many causes because there are so many possible antigens. The inflammation that causes such extensive damage to nerve fibers of the spinal cord may result from viral infections, abnormal immune reactions, or insufficient blood flow through the blood vessels located in the spinal cord. Transverse myelitis also may occur as a complication of syphilis, measles, Lyme disease, and some vaccinations, including those for chickenpox and rabies. Cases in which a cause cannot be identified are called *idiopathic*.

Trigeminal Neuralgia (TN)

Inflammation of the 5th Cranial Nerve with pain in the cheek or head, called Trigeminal Neuralgia or *tic douloureux*. The pain causes sudden, twitching, burning or shock-like face pain that lasts a second or two followed by a pain free interval for a few minutes and can continue to reoccur in episodes. The intensity of pain can become incapacitating. TN pain is typically felt on one side of the jaw or cheek. Episodes last for days, or weeks at a time and then can reoccur later. In the days before an episode begins, some patients may experience a tingling or numbing sensation or a somewhat constant and aching pain. The attacks often worsen over time. The pain can be triggered by vibration or contact with the cheek (such as when shaving, washing the face) brushing teeth, eating, drinking, talking, or being exposed to the wind. TN occurs in people over age 50, and is more common in women than in men.

A more complete list of Autoimmune Diseases:

- Achlorhydra Autoimmune Active Chronic Hepatitis
- Addison's Disease
- Alopecia Areata
- Amyotrophic Lateral Sclerosis (ALS, Lou Gehrig's Disease)
- Ankylosing Spondylitis
- Anti-GBM Nephritis or anti-TBM Nephritis
- Antiphospholipid Syndrome
- Aplastic Anemia
- Arthritis
- Asthma
- Atopic Allergy
- Atopic Dermatitis
- Autoimmune Inner Ear Disease (AIED)
- Autoimmune Lymphoproliferative Syndrome (ALPS)
- Balo Disease
- Behcet's Disease
- Berger's Disease (IgA Nephropathy)
- Bullous Pemphigoid
- Cardiomyopathy
- Celiac Disease
- Chronic Fatigue Immune Dysfunction Syndrome (CFIDS)
- Churg Strauss Syndrome
- Cicatricial Pemphigoid
- Cogan's Syndrome
- Cold Agglutinin Disease
- Colitis
- Cranial Arteritis
- CREST Syndrome
- Crohn's Disease
- Cushing's Syndrome
- Deigo's Disease
- Dermatitis
- Dermatomyositis

- Devic Disease
- Diabetes, Type 1
- Diabetes, Type 2
- Dressler's Syndrome
- Discoid Lupus
- Eczema
- Essential Mixed Cryoglobulinemia
- Eosinophilic Fasciitis
- Epidermolysis Bullosa Acquisita
- Evan's Syndrome
- Fibromyalgia
- Fibromyositis
- Fibrosing Alveolitis
- Gastritis
- Giant Cell Arteritis
- Glomerulonephritis
- Goodpasture's Disease
- Grave's Disease
- Guillian-Barre Syndrome
- Hashimoto's Thyroiditis
- Hemolytic Anemia
- Henoch-Schonlein Purpura
- Hepatitis
- Hughes Syndrome
- Idiopathic Adrenal Atrophy
- Idiopathic Pulmonary Fibrosis
- Idiopathic Thrombocytopenia Purpura
- Inflammatory Demyelinating Polyneuropathy
- Irritable Bowel Syndrome
- Kawasaki's Disease
- Lichen Planus
- Lou Gehrig's Disease
- Lupoid Hepatitis
- Lupus
- Lymes Disease

- Meniere's Disease
- Mixed Connective Tissue Disease
- Multiple Myeloma
- Multiple Sclerosis
- Myasthenia Gravis
- Myositis
- Ocular Cicatricial Pemphigoid
- Osteoporosis
- Pars Planitis
- Pemphigus Vulgaris
- Polyglandular Autoimmune Syndromes
- Polymyalgia Rheumatica (PMR)
- Polymyositis
- Primary Biliary Cirrhosis
- Primary Sclerosing Cholangitis
- Psoriasis
- Raynaud's Phenomenon
- Reiter's Syndrome
- Rheumatic Fever
- Rheumatoid Arthritis
- Sarcoidosis
- Scleritis
- Scleroderma
- Sjogren's Syndrome
- Sticky Blood Syndrome
- Still's Disease
- Stiff Man Syndrome
- Sydenham Chorea
- Systemic Lupus Erythmatosis (SLE)
- Takayasu's Arteritis
- Temporal Arteritis
- Ulcerative Colitis
- Vasculitis
- Vitiligo
- Wegener's Granulomatosis

- Wilson's Syndrome

How do you know if a patient is autoimmune? Are there some clinical 'hints' that should tip the practitioner or signs that should make a person question? I've listed a few signals that, if you notice you fit into one or more of these, it may be a good idea to get some testing done:

1. You already suffer from a known autoimmune disorder (RA, psoriasis, ulcerative colitis, Type 1 diabetes, Sjorgen's syndrome, scleroderma, sarcoidosis, lupus, Hashimotos....you get the picture). If you already have another autoimmune disease, the chances are higher that current symptoms in a seemingly unrelated area may be from an attack to that tissue as well.
2. Your symptoms wax and wane. This is classic with autoimmune disease. Remember, it is when the immune attack occurs that you usually feel the worse so when your Th1 or Th2 system is 'ramped up', the inflammation is highest and your symptoms worsen. As time passes, your immune system may fatigue and ironically, when your immune system is completely 'pooped out' is when you feel better, you think that you may be on the road to recovery only to be knocked back down once your body has rested and gotten 'back to the fight'.
3. You take a boatload of supplements. I've had patients bring in bags of supplements that they've tried, are trying, or read about and plan to start. Usually autoimmune patients are desperate, they are searching, have not received much support or have run into a salesman who peddled them stuff they just don't need. There is a serious danger here as well. Remember that certain supplements stimulate a Th1 response and others stimulate a Th2 response. If you are Th1 dominant and you are taking Th1 stimulants, you are feeding the fire! You may as well drink poison!
4. Life fell apart for you after _____. This is a very common finding in the history of the case; events in life ramp the immune system and can cause it to recognize a latent antigen that has lain

dormant for years. I liken it to your home in a quiet neighborhood. You like your neighbors and never noticed anything wrong or unusual until the day that the city doubled the police force and added security for the upcoming political event next month. When you came home from work there were four police cars next door and they were hauling thugs out in handcuffs from the crack house no one knew was there. Events in life, whether emotional, physical or spiritual, can cause a rise in the immune response, an increase in security that may flush out things lodged in tissues for years. Well, if this added security recognizes an antigen that isn't alive and won't die, the immune response is 'turned on' and the autoimmune disease is set in motion.



5. Following pregnancy. A pregnant woman normally will be Th2 dominant in her third trimester and then Th1 dominant post partum. I often hear comments like, "I always felt best being pregnant, if I could only stay in a pregnant state, my life would be great;" or, "I love my kids but pregnancy just killed me, it was the worst I ever felt." The one who felt great during pregnancy was the autoimmune patient who was Th1 dominant. When in the third trimester their body swung to Th2 dominant, it was a temporary balancing that dramatically improved symptoms and they felt great. Usually this same patient suffered post partum depression due to the violent swing in the other direction after giving birth. The opposite was true for the mother who hated being pregnant.

She was Th2 dominant already and the Th2 swing in the third trimester just made her worse; boy was she a happy momma once the baby came and she just couldn't figure out why those other moms struggled with depression and exhaustion.

6. Positive testing via immune panels. (see below) Ultimately you want to get tested.



I. Autoimmune testing. The typical testing for autoimmune diagnosis is antibody testing. If Hashimoto's is suspected, protocol dictates we run TPO antibodies and if positive, it would be a definite confirmation of our diagnosis. The only problem with antibody testing is that if a patient is Th1 dominant, they will be suppressing the Th2 system that makes the antibodies. Many patients that truly are autoimmune patients have negative antibody tests due to Th1 dominance and the diagnosis is missed! A more accurate testing is Cytokine tests. These will prove an autoimmune reaction AND show which side is dominant!

This is why we do NOT rely solely on standard blood antibody tests. A more unique approach is often needed and there are specific, quality functional labs that will pinpoint difficult cases.

II. Antigen Testing. There are many sources for testing antigens. Again, standard blood tests will only reveal what is circulating in the blood at the time of the draw so they tend to be unreliable. Hair analysis

for heavy metals is somewhat reliable but samples must be done correctly without coloring, harsh shampoos and other hair products, etc.

A technique called Applied Kinesiology is most reliable and one we use for screening toxins, but it necessitates a professional with a lot of experience in the art – something I've studied, practiced, and taught for 30 years.

We also utilize specialty labs that measure specific functional lab values. Enterolab is a laboratory that created the most a very reliable analysis for several food-based toxins and genetic testing for such – it is a stool test. We will often run the Enterolab test for gluten, soy, casein, egg, and yeast on a patient with a suspected autoimmune disorder. We run a Stool Microbial Ecology Profile as well. This test, by Genova/Metametrix Labs, will reveal intestinal parasites.



One of our favorite labs is Cyrex. They have a multitude of panels to choose from including an Intestinal Antigenic Permeability Screen (Actomyosin IgA, Occludin/Zonulin IgG, Occludin/Zonulin IgA,

Occludin/Zonulin IgM, Lipopolysaccharides (LPS) IgG, Lipopolysaccharides (LPS) IgA, Lipopolysaccharides (LPS) IgM), Wheat/Gluten Proteome Reactivity & Autoimmunity measuring over 24 possible antigens, Gluten-Associated Cross-Reactive Foods and Foods Sensitivity measuring dozens of cross-reacting antigens, and many more unique, specific tests.

Array 11 Chemical Immune Reactivity Screen	IN RANGE (Normal)	EQUIVOCAL*	OUT OF RANGE	REFERENCE (ELISA Index)
Aflatoxins IgG+IgA			2.06	0.4-1.8
Aflatoxins IgM	1.30			0.1-1.9
Formaldehyde and Glutaraldehyde IgG+IgA	0.89			0.3-1.4
Formaldehyde and Glutaraldehyde IgM	1.22			0.1-1.8
Isocyanate IgG+IgA		0.96		0.1-1.1
Isocyanate IgM	0.59			0.1-1.2
Trimellitic and Phthalic Anhydrides IgG+IgA		1.08		0.1-1.3
Trimellitic and Phthalic Anhydrides IgM	1.45			0.1-2.0
Benzene Ring Compounds IgG+IgA	0.99			0.2-1.3
Benzene Ring Compounds IgM	1.01			0.1-1.6
BPA Binding Protein IgG+IgA	0.78			0.2-1.8
BPA Binding Protein IgM	0.70			0.1-1.8
Bisphenol A IgG+IgA		1.43		0.1-1.8
Bisphenol A IgM	1.43			0.1-2.0
Tetrabromobisphenol A IgG+IgA	1.16			0.1-1.6
Tetrabromobisphenol A IgM	0.73			0.2-2.0
Tetrachloroethylene IgG+IgA	1.41			0.4-2.0
Tetrachloroethylene IgM	0.96			0.1-2.6
Parabens IgG+IgA		1.53		0.2-1.7
Parabens IgM	0.48			0.1-1.8
Mercury Compounds IgG+IgA		1.47		0.1-1.5
Mercury Compounds IgM	0.94			0.1-2.1
Mixed Heavy Metals IgG+IgA	1.07			0.2-1.8
Mixed Heavy Metals IgM	1.04			0.1-1.8

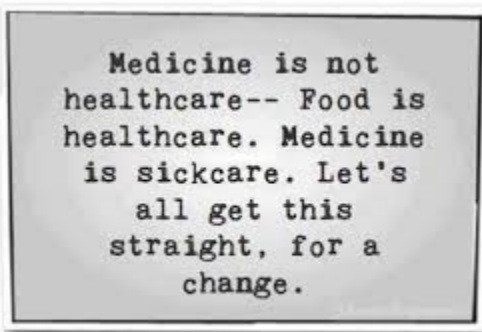
III. Complete Blood Panels (LabCorp or your local lab)

We need a Complete Metabolic Panel, a Lipid panel, a Thyroid panel (TSH, free T3, Free T4, and Total T3), a CBC with auto differential, C-reactive protein, homocysteine, TIBC, and 25-OH Vitamin D as well as 1, 25-OH Vitamin D levels. We recommend that you run the TPO and

TGB antibodies or other antibodies specific to the area of attack even though they may not be positive if the patient is Th1 dominant.

The main priorities when looking at the blood work are:

- Autoimmune diagnosis, antigen detection and immune system dominance
- Anemias present: Iron, B12, Vitamin D, Pernicious, and Folic Acid
- Blood sugar/Insulin balance within functional ranges
- Adrenal function and hypothalamus-pituitary axis health
- Liver congestion, and health of detoxification pathways
- Gastrointestinal tract health, Leaky Gut, Metabolic Toxic Bowel, Probiotic health, Stomach health, Hypochlorhydria, H Pylori infections, ulcerations
- Cell membrane health, Bio-Impedance test, fatty acid metabolism
- Thyroid health – complete thyroid panels
- Inflammatory states, possible cancer markers, toxicities
- Other pathologies, genetic markers, genetic predominance



IV. Adrenal Stress Index (ASI) from www.diagnostechs.com:

We run this test on everyone simply due to the fact that stress is ubiquitous in this country. If there is any chronic fatigue, brain imbalances, hormone issues, blood sugar problems, etc., adrenal

fluctuations may be evident. The ASI measures cortisol output throughout the day.

V. Immune Panels:

1) Immune Dysregulation: Th1/Th2 out of balance

Th1 and Th2 → which is dominant? If NO autoimmune disorder → neither is!

Th1 Dom = high IL-2, IL-12, NKC and TNF-alpha

Th1 is T-cells. T-cells are the police force that attacks and cleans up afterward. (Helper T-cells, Suppressor T-cells, NKC, regulatory T-cells, and macrophages).

Th2 Dom = high IL-4, IL-13 and IL-10

Th2 is B-cells. B-cells make anti-bodies. They tell T-cells what to kill. If the testing comes back with a high B-cell count, the patient is Th2 dominant.

2) Active Antigen vs. Dysregulation.

Active antigens are BIOTOXINS → parasites, bacteria, virus, mold, yeast, fungi, or protozoan → that your body is trying to KILL right NOW, NOT an autoimmune response to them; this is a BIG DIFFERENCE. If your immune system is simply killing an Antigen, then aid the high immune pattern (if high Th1, help increase it). BUT, if it is autoimmune toxicity, then treat as an autoimmune condition, NOT a normal physiologic response!!!!

The best indicator for an active antigen as the cause of the patient's abnormal dominance is the "Helper/Suppressor" ratio on the T & B cell panel. (Also called "CD4:CD8" ratio).

The closer to 2.5 the ratio is (or if above that), the more likely it is that you're dealing with an Active Antigen. If the ratio is below 1.2, then you are most likely dealing with a dys-regulation problem.

If ACTIVE ANTIGEN = treat accordingly!!

To test for active antigens use the following tests:

- 1) Metametrix.com = Stool Microbial Ecology Profile #2105
- 2) Enterolab.com for food antigens
- 3) Urea/H.Pylori breathe test from Metsol.com

VI. Intestinal Permeability from www.genovadiagnostics.com:

LGS or Leaky gut syndrome describes a condition of altered or damaged bowel lining, caused by antibiotics, toxins, poor diet, parasites or infection can lead to increased permeability of the gut wall to toxins, microbes, undigested food, waste or larger than normal macromolecules. It has been proposed that these substances affect the body directly, while others postulate an immune reaction to these substances.

VII. HORMONE PANELS (we use the DUTCH panel):

We can check hormone panels to determine if the patient suffers from low testosterone in males or low/hi estrogen/progesterone levels in females. Symptoms related to decreased hormone levels may include depression, fatigue, mental fogginess, mood swings, hot flashes, sweating attacks, weight gain, and decreased physical stamina. We ONLY run what are called Expanded Panels. These are panels that take a 'movie' over an entire cycle for the female. It does little good to see a snapshot of hormonal activity; we need the entire cycle to measure fluctuations.

VIII. Urea / H. Pylori from www.metsol.com:

We run the only reliable test for H. Pylori bacteria to determine any problems related to the gut function. This is a breathe test, not a blood test.

IX. Homocysteine and C-reactive protein:

Testing for inflammation. Tested through LabCorp or your local lab. We cannot overemphasize the importance of homocysteine and C-reactive protein testing. Homocysteine levels are toxic in functionally high concentrations and responsible for the etching of the arteriole walls that lead to atherosclerosis. C-reactive protein is a measurement of inflammation in the body and may be the first indicator of an autoimmune response.

X. Additional Thyroid Testing:

Understanding Thyroid Markers and Panels

TSH: Thyroid Stimulating Hormone (TSH) is also called thyrotropin. The pituitary releases this hormone after the hypothalamus releases TRH (thyrotropin-releasing-hormone). This is the most common marker used to assess thyroid function and it is also the most sensitive. The TSH levels increase when the T4 levels drop, and the TSH falls when T4 levels increase. This is the only test performed in the traditional health care model as a means to screen the patient for thyroid disorders; this is because they are only concerned for screening the thyroid for hormone replacement and not optimal physiological function. A TSH test alone does not consider thyroid-pituitary feedback loops, peripheral thyroid metabolism, or potential or active risk factors as identified by antibody testing. A high TSH with or without changes in T4 or T3 is diagnostic to determine hypothyroidism. If the thyroid is not making enough T4 the pituitary will pump out TSH to stimulate its production. A low TSH is used to determine hyperthyroid activity. If the thyroid is overactive, such as in Grave's disease, the antibodies bind to active thyrotropin

(TSH) receptors on the thyroid cells and stimulate T4 production without the influence of TSH. Please note that some antibodies may inhibit thyroid function by inactivating instead of stimulating thyrotropin receptors. This is called an autoimmune hypothyroid. These patterns will demonstrate a hypothyroid pattern (elevated TSH) with elevated thyroid antibodies.

Laboratory Reference Range: 0.5 – 5.5 (varies from one lab to another)

Functional or Optimal Reference Range: 1.5 – 3.5

Total Thyroxine (TT4): The TT4 test measures both bound and unbound Thyroxine levels. Therefore, it does not give the activity of T4 when measured alone. This test is best completed with a T3 uptake. The free Thyroxine index (FT4) can be calculated by using the T3 uptake and demonstrate a level of T4 activity. Total T4 levels can be altered by many drugs (see Category of drugs that interfere with thyroid activity).

Functional Reference Range: 6-12 ug/d

Free Thyroxine Index: As stated earlier, the total Thyroxine and the T3 uptake must be used together to calculate the FT4. The index is measured by multiplying the TT4 levels by the T3 uptake levels. The result is the FT4 and it determines the amount of active T4 available. The impact of drugs, as will be discussed, will always impact T4 and resin T3 uptake levels in opposite directions due to their impact on binding sites. If the TT4 level is depressed, then the T3 uptake is high; if the TT4 is elevated, the resin uptake is low. Please note that even if you are taking drugs that may impact thyroid binding, the free Thyroxine index should be within the normal range if your thyroid is functioning normally.

Functional Reference Range: 1.2 -4.9 ml/dl

Free Thyroxine (FT4): The free Thyroxine test is used to measure the amount of free or active T4 in the blood. All the factors such as drugs and physical conditions that may impact the TT4 do not impact the FT4. The level of T4 in the blood is high with hyperthyroidism or low with hypothyroidism. Please note that even a TSH with normal T4 is enough to diagnose hypothyroidism. A rare pattern is an elevated T4 without hyperthyroidism which may be related to a hereditary condition of thyroid resistance. Elevated free T4 may also be caused by patients taking heparin or by an acute illness that may briefly cause the binding protein levels to suddenly fall. If an illness becomes severe and chronic it may decrease the FT4 levels but it is not a thyroid disease.

Functional Reference Range: 1.0 -1.5 ng/dL

Resin T3 Uptake: The resin T3 uptake measures the amount of sites for active (unbound) T3 to bind with Thyroxine binding proteins. This test is performed by mixing the blood with radioactive thyroid hormones. These radioactive hormones then combine with binding sites on Thyroxine-binding proteins. The blood is then exposed to a substance called a resin which will bind the unbound thyroid hormones and measure for radioactivity. The result can be expressed as the percent of radioactivity found on the resin, compared to the original radioactivity that was added. The more binding sites that are open on the proteins, the lower the resin uptake result will be, and vice versa. For example, anything that reduces the binding sites, such as elevated testosterone or testosterone replacement therapy, can cause a low T4 measurement because it leaves very few binding sites for any thyroid hormone to bind to. If T3 is added to the sample of the blood, little T3 will be bound. This pattern would have low TT4 levels and high resin T3 uptake levels. On the other hand, anything that raises the binding sites such as estrogen or birth control pills would cause a pattern of high TT4 and low T3 uptake.

Functional Reference Range: 28-38 mg/dl

Free Triiodothyronine (FT3): This test measures the free T3 hormone levels. This test is rarely completed in traditional endocrinology. It is typically only used in a situation when a patient has hyperthyroid, yet the FT4 levels are normal. However, the FT3 test is the best marker for measuring the amount of active thyroid hormones available for the thyroid receptor sites.

Functional Reference Range: 300-400 pg/ml

Reverse T3 (rT3): This test measures the amount of reverse T3 that is produced. The production of rT3 typically takes place in cases of extreme stress, such as major trauma, surgery or severe chronic stress. It appears that the increased production of reverse T3 is due to an inability to clear rT3m as well as from elevated cortisol.

Functional Reference Range: 90-350 pg/ml

Thyroid Antibodies: Thyroid auto-antibodies indicate that the body's immune system is attacking itself. Production of thyroid auto-antibodies may create a hypothyroid or a hyperthyroid state. Some antibodies attach to the TSH receptors but do not cause a response; therefore, the patient will complain of low thyroid symptoms. However, the serum TSH may not be altered. It is just not able to cause a cellular change. On the other hand, some antibodies will bind to the receptor sites and cause over activation of the thyroid. This will present as elevated T4 levels, a low TSH, and elevated thyroid antibodies.

Epigenetic Gene Testing:

We now always test for gene SNPs, which are defects on specific genes that can speed or slow different metabolic pathways. These help us understand WHY different people have more difficulty detoxing, making energy, handling stress, etc. This knowledge can really change the course of treatment.

See the example test below:

DNA Methylation Pathway Profile; Blood Spot

RESULTS				
Gene Name / Variation	Mutation Not Present	Mutation(s) Present	Call	
SHMT / C1420T	-/-		G	Minus "-" represents no mutation
AHCY / 1		+/-	Hetero	Plus "+" represents a mutation
AHCY / 2		+/-	Hetero	"-/-" indicates there is no mutation
AHCY / 19		+/-	Hetero	"+/-" indicates there is one mutation
MTHFR / C677T		+/-	Hetero	
MTHFR / A1298C	-/-		A	"+/+" indicates there is a double mutation
MTHFR / 3	-/-		C	
MTR / A2756G	-/-		A	
MTRR / A66G		+/-	Hetero	
MTRR / H595Y		+/-	Hetero	
MTRR / K350A		+/-	Hetero	
MTRR / R415T	-/-		C	
MTRR / S257T	-/-		T	
MTRR / 11		+/-	Hetero	
BHMT / 1		+/-	Hetero	
BHMT / 2	-/-		C	
BHMT / 4	-/-		A	
BHMT / 8	-/-		C	
CBS / C699T	-/-		C	
CBS / A360A		+/+	T	
CBS / N212N	-/-		C	
COMT / V158M		+/-	Hetero	
COMT / H62H		+/-	Hetero	
COMT / 61	-/-		G	
SUOX / S370S	-/-		C	
VDR / Taq1		+/-	Hetero	
VDR / Fok1	-/-		C	
MAO A / R297R		+/-	Hetero	
NOS / D298E		+/-	Hetero	
ACAT / 1-02	-/-		G	

What WE typically do:

Since we see patients from all over the world (most never step foot in our office) we had need to develop a testing protocol based on specific testing:

1. We always begin with a Doctor phone/Skype CONSULTATION. This enables the doctor and potential patient to communicate and see if our method would be a “good fit”. For this Consultation, we require our initial paperwork (available for download on our website www.ConnersClinic.com) be completed.

2. Should both decide to continue, we require even more detailed History/Questionnaires be completed to prepare for what we call an EXTENDED CONSULTATION. This usually includes an hour with the doctor (usually on phone/Skype).

3. When we complete our EXTENDED CONSULTATION with a new patient, we make some clinical decisions based upon our communication as well as the paperwork completed. We then order testing to determine the answers to our FOUR NECESSARY QUESTIONS. The patient is sent specific test kits with detailed collection instructions.

4. When ALL test results have come in, another DETAILED PROTOCOL CONSULTATION will be scheduled where the patient will receive a complete

PLAN OF ATTACK with all of the doctor's recommendations.

5. Every patient is then enrolled in our PATIENT FORUM where answers to questions, supportive information, and relationships with like-minded, fellow patients can be found.

Simply contact our office for more information – 651-739-1248.



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Final Remarks

Regardless of what you choose about healthcare, I pray that you make wise, rational decisions based on facts (though often hidden) and not fear. You need to take responsibility and not hand it over to any practitioner, conventional or alternative. Get advice from many, weigh it all against their biases, and pray for peace about your decisions.

Kevin Connors, Pastoral Medical Association, Fellowship in Integrative Cancer Therapy and Fellowship in Anti-Aging, Regenerative and Functional Medicine, both through the American Academy of Anti-Aging Medicine.

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