

# LRA by ELISA/ACT® CLINICAL UPDATE # 16

## *Autoimmune Thyroiditis*

### AUTOIMMUNE THYROIDITIS

Autoimmune thyroiditis is the most common undiagnosed autoimmune disease. This will make patients resistant to a variety of therapies, while often going undiagnosed as the cause of fatigue or other problems (i.e., failure to heal or respond to other therapies).

**Autoimmune thyroiditis is challenging to diagnose. Hashimoto's thyroiditis involves most actively the attack on the thyroid gland associated with increased cell-mediated delayed allergies.**

Patients often have other autoimmune and delayed hypersensitivity conditions that progress, making them multiple system autoimmune disease patients. This is one of the reasons for a delayed diagnosis as other disease processes are diagnosed, and the thyroid component is overlooked in the process of trying to heal conditions such as RA, IBS, DM, and migraines. A systemic approach to reset the immune system and repair the immune process allows sustained improvement.

Patients with thyroiditis often have antibodies to thyroid tissue produced locally inside the thyroid gland. This is an indication of an increase in permeability of the gland. The blood thyroid barrier is being breached, so thyroid tissue, which should be restricted and isolated to do its job of producing hormones and various other

energetically important compounds, is getting exported to the body systemically rather than leaving the thyroid tissue isolated as it should be to do its job. This connective tissue that should be isolating the gland becomes more permeable because repair has been deferred in light of the increase in defense that the immune system needs to do. When this increase in permeability occurs, indicating that the thyroid is breached, lymphocytes enter, and cell-mediated responses occur. In more advanced cases, anti-thyroid antibodies or anti-thyroid globulin antibodies are produced as markers of that thyroiditis.

Comprehensive and careful survey reveals auto antibodies to many other structures in the body, which can help confirm that there are multi-system conditions for most patients with thyroiditis.

### ELEMENTS OF THYROIDITIS

**Thyroiditis shows increase in tissue permeability, an accumulated repair deficit, and over engagement of the body due to digestive remnants that are causing an increased defense workload that has predisposed the patient to the condition.**

Palpation is an art. A healthy thyroid gland can be felt; midpoint in thyroiditis there is swelling in the gland. This swelling occurs in order to hypertrophy or produce more thyroid tissue. It also takes on water, and this

increase in size can be detected. At a more advanced stage, the thyroid gland shrinks due to lack of its ability to compensate and hypertrophy, and at that point, the gland cannot be palpated, although you actually have a more advanced form of thyroiditis. The corollary component is whether the gland can restore itself - and it can.

With reestablishment of an appropriate blood thyroid barrier because protein synthesis and connective tissue repair synthesis goes on, the thyroid follicles themselves are able to regenerate. The gland will expand and is now able to be palpated, not due to thyroiditis, but because there is regeneration going on. Then, when repair is complete, it will be the size of when it was the largest. During the mid-stage of degeneration and regeneration, the thyroid gland can be palpated as 'enlarged'. This is difficult to distinguish. Infrastructure must be repaired before follicles can repair themselves, and so palpation is an indirect way of looking at thyroiditis.

### INDEX

Background on autoimmune thyroiditis and LRA by ELISA/ACT tests .....	1
The immune system's role: A summary .....	1
LRA by ELISA/ACT in diagnosis .....	2
Thyroiditis and delayed "allergy" .....	3
Clinicians report on LRA by ELISA/ACT ..	4

A patient with thyroiditis has an autoimmune disease that is cell-mediated with T-cell activation against the thyroid tissue. Cell-mediated responses as well as antibody responses need to be measured, and it is not uncommon when there are multiple systems involved to have immune complexes as a component of the condition. It is important to measure what these immune complexes are recognizing as foreign in order to lower the load systematically so we can stimulate the repair process.

**LRA by ELISA/ACT® PLANS YIELD CLINICAL SUCCESSES**

Case Example:

Erin presented as a 36 yo P5G3 obese white female (18 Kgm over lean weight). She first presents with an 8-year complaint of ‘lack of energy, easy fatigability, inability to keep up with her children or friends, and ‘little joy in living’.

Prior lab values had been unremarkable including:

- Total T3 of 120 (80-180 µg/dl) and
- Total T4 of 5.2 (4.6-12 µg/dl).

From her history and because routine thyroid lab tests are so insensitive, the following additional tests were done:

- Test Value Usual Range
1. Free T3 170(230-620 pg/dl)
  2. Free T4 0.5(0.7-1.9 ng/dl)
  3. TSH 12(0.5-6 µU/ml)
  4. TRF 92(9-30 µIU/ml)
  5. Autoantibodies:  
 microsomal>2048 not detected  
 thyroid>4096 not detected  
 receptorND not detected
  6. 2° PP Glu186(90-140 mg/dl)
  7. 2° PP Ins 80(20- 64)
  8. FCortisol 44( 4- 27 mg/dl)
  9. FDHEA 2( 4- 9 mg/dl)

Erin was diagnosed with Hashimoto’s thyroiditis, insulin insensitivity (syndrome X), and hyperadrenalism.

On her **LRA by ELISA/ACT tests** she was found reactive to the:

1. cow dairy group,
2. MSG,
3. arsenic,
4. sulfite
5. carrots, and
6. chloroform.

She substituted for these items following an Alkaline Way repair diet, supplemented with high activity nutrients to correct deficits and enhance repair capability.

She was given 2 grains of desiccated thyroid daily when her first morning temperature was below 98°F. She used a dichromatic green light for 20 minutes twice daily as phototherapy. She also used baking soda and epsom salt baths as part of a stress adaptation program.

After following this program for 6 months, she was asymptomatic. She had lost 10 Kgm of weight. Her free thyroid hormone values were within healthy ranges. Anti-microsomal and anti-thyroglobulin antibodies were weakly positive (8:1). Fasting and postprandial glucose and insulin were within healthy levels. Free cortisol and DHEA had normalized. Follow-up LRA by ELISA/ACT tests showed loss of all reactions except for arsenic and sulfite.

**Fig. 1. Clinical tests of immune responses. LRA by ELISA/ACT tests replace 3 or more assays by measuring all delayed pathways in a functionally meaningful way. This avoids the clinically false positive tests that ELISA IgG tests measure because they don’t distinguish reactive from protective antibodies.**

- Autoimmune illnesses include most rheumatoid connective tissue diseases (arthritis, Sjogren’s syndrome, lupus/SLE, scleroderma), asthma, inflammatory bowel disease (IBS, Crohn’s disease, and ulcerative colitis), diabetes (DM), multiple sclerosis (MS), migraine headaches, chronic fatigue syndrome, immunotoxic infertility, ITP, Grave’s hyperthyroidism, Hashimoto’s thyroiditis, pemphigus, eczema and psoriasis, fibromyalgia, myofascial and related chronic pain syndromes.

What Measured	Reactions	Humoral	Immune Complex	Cellular
IgE (‘RAST’)	Y	N	N	N
IgA	N	Y (only IgA)	N	N
IgM	N	Y (only IgM)	N	N
IgG (‘Generalist’)	N	Y (only IgG)	N	N
IgG1	N	Y (IgG1)	N	N
IgG2	N	Y (IgG2)	N	N
IgG3	N	Y (IgG3)	N	N
IgG4	N	Y (IgG4)	N	N
Immune Complex	N	N	Y	N
Raji Cell Assay	N	N	Y	N
Lymphocyte Cytokine release	N	N	N	Y
LRA by ELISA/ACT	N	Y	Y	Y



Further follow up at 36 months found Erin at her lean weight, symptom free, and with all lab values within healthy ranges. LRA by ELISA/ACT delayed allergy to sulfite persisted. At 60 months Erin was again asymptomatic with lab values within healthy ranges and no LRA by ELISA/ACT reactivities.

In autoimmune endocrinopathy, there is an organ-specific component and a general component to it. This is the rule more than the exception, so thyroiditis is not only an indicator of an autoimmune process, but is also a systemic depletion of the infrastructure of connective tissue structure and increase in permeability of multiple tissues that should be isolated from the normal traffic of cells that occurs.

Along with immune and hormonal dysregulation, there is an autoimmune endocrinopathy that is common, hyperreactivity to delayed immune response that is common, along with increased risk of dysplasia and cancer, with increased frequency and increased hospitality to *Candida* locally in the gut and potentially systemically.

## CROSS REACTIVITIES

Antibodies can also crossreact with hormones and their receptors that lead to a decrease in sensitivity to that hormone. Just as there are anti-insulin antibodies occurring in DM, you can have anti-thyroglobulin antibodies in thyroiditis and anti-T3 hormone receptor antibodies occurring in thyroiditis. Any of these can be markers of immune activation, and all of these respond to down regulating the immune response by lowering the burden and decreasing the amount of activation that is occurring from digestive remnants and systemic tissue debris. When the system is reset to tolerance, the body is then able to regulate these lymphocyte clones. Auto antibodies that can block adrenergic receptors can occur, and this will increase asthma, allergic rhinitis, and various other hypersensitivity conditions that can occur concurrently with thyroiditis.

The clinical picture includes morning fatigue, short-term memory loss, allergic conditions and asthma, difficulty with weight regulation, bladder disorders, temperature change intolerance, and irregular menses in women. This is seen because in order to get the body going in the morning multiple hormones are activated. Cortisol is at its peak, and along with cortisol, what brings up the body's temperature in the morning is the release of thyroxin, T4, and its more active cousin, T3, triiodothyronine hormone. If the thyroid gland is being activated by lymphocytes, then its capacity to produce a healthy hormone output is reduced. Due to these conditions, a patient can be fatigued and have lower body temperatures in the morning.

It used to be said that a low morning body temperature was a marker for hypothyroidism or underactivity of the thyroid gland. It is more complex than that. There are stress hormones in the neurotensin family that mimic the lack of thyroid hormone. The activation of neurotensin, both in the brain and potentially systemically, may be to compete with thyroid hormone for receptors. Excess of neurotensin stress hormone can mimic all of the effects of hypothyroidism. The clinical way to determine the difference is to see what the response is to the equivalent of 1, 2, or 3 grains given to the person the night before so that in the morning he/she can have the benefit of having that supplemental thyroid tissue. If the body temperature comes up 1 degree or more on therapeutic thyroid hormone therapy, that is first order recognition of hypothyroidism or thyroiditis. In contrast, if the first morning body temperature remains low in the face of appropriate thyroid hormone therapy, that's a reasonably good indication that the problem is a stress response in the CNS - excess of neurotensin. So, what we want to do is reduce the stress hormones and not focus on the thyroid gland for those conditions.

## CLINICAL CHALLENGE

### The thyroid hormone challenge:

**Use 1 grain for one week**

**Use 2 grains for 1 week**

**Use 3 grains for 1 week ...**

**taken in the evening before bed**

Look for an increase in the first morning temperature of more than 1 degree. Do not give more than 3 grains without doing special studies that support the need. If 3 grains do not increase the basal temperature, than that's an indication of the neurotensin stress hormone excess.

This challenge is done by the more progressive physicians. One of the best of thyroidologists, Dr. Bruce Weintraub (he was at NIH and now is at Mass General Hospital in Boston, Massachusetts), feels that it is very important to give a therapeutic challenge to patients with unexplained fatigue that might be at risk for functional hypothyroidism. His elegant studies have shown that even when the body produces reasonable amounts of the thyroid hormone, if the receptor is altered by metabolic acidosis, or by toxins that bind to the receptor and distort it, or by antibodies to the receptor, then the hormone will be less effective, and people can have functional hypothyroidism because of receptor defects even when they have what seems to be reasonable amounts of circulating thyroid hormone. This happens in the same way that patients can have reasonable amounts of circulating insulin, but because of anti-insulin antibodies, have that insulin not be active, or because of distortion of insulin receptors, that insulin may not be active and functional for them as so it is with any of the endocrine glands and receptor systems in the body ... and so a therapeutic challenge is important in these conditions. The response of 1 degree increase in first morning basal temperature is an indication of functional response in patients with fatigue that look functionally hypothyroid. Overall, approximately 30% of patients with "normal" blood work, but functional indicators of fatigue and low body temperature, make them appear



hypothyroid on the basis of neurotensin stress hormone, not thyroid hormone lack.

## ALLERGY CONNECTIONS

Patients with thyroiditis often initially see allergists because they have rhinitis and various other allergic conditions or asthma and airway resistance conditions. They often go to pulmonary and or allergy pulmonary specialists, or they have hives, itching, or urticaria, and that is what they present with at the doctor. They may have eczema or they have headaches and go to a headache center or migraine specialist, or they will have arthralgias or IBS symptomatology, and that symptom complex brings them to a specialist who looks for a targeted related cause to the problem rather than systemic underlying causes to the problem. This is a very important part of why the thyroiditis is so often late in the diagnosis and should be thought of early on in any of the populations of folks with an increase in sensitivity type presentation.

Patients with changes in short-term memory, especially with intermittent or episodic fatigue associated with that, and with a sense of foreboding, hopelessness, or depression should have a therapeutic trial of thyroid and a look at their immune regulation system as a first order part of their management rather than as a last resort in their diagnostic therapy as so often happens. These folks are often the so-called night people. They do better as the day goes on. They have difficulty getting themselves started early in the day, but they are ready for a full life at night. This is often because the phase shifting in their essential nervous system is itself a response to the hyperadrenergic and hypercortisolemic responses of the stress and distress hormones. It is important not to fuel that process, but use it as an indicator for looking further for multiple endocrine causes and immune, hormonal, and neurochemical dysfunction as the underlying condition.

The folks with vascular headaches may respond to symptom-reactive medication therapy, but that very often doesn't address the underlying causes which, not uncommonly, are related to thyroiditis and other

autoantibodies and cell-mediated immune responses that underlie the vascular headaches.

The way to properly address these folks is with a careful history, looking at the multiple ways in which the immune, hormonal, and neurochemical systems may be dysfunctional, and bearing in mind that systemic causes for specific conditions are really more the rule than the exception today. It's not clear that this was true 100 years ago, but in our complex society and in our chronically ill society, it is becoming more the rule.

Thyroiditis is diagnosed more often in women than in men. Most men with thyroiditis get other diagnoses and are never properly assessed for their predisposing thyroiditis. The stress of puberty and young adulthood is often common in the history of patients with thyroiditis. Often thyroiditis can occur in association with changes at puberty or the stress of college life and may not be properly diagnosed for years or decades, leading folks to feel that they're not able to keep up with life when in fact it is just because their battery has become so uncoupled, and their energy system has become reversibly depleted-but nonetheless depleted. These are women that typically have difficulty with their menses and cycling of their period. They often have difficulty with mood swings in adolescence and adulthood, often have postpartum depression after giving birth, and may benefit from counseling, but will benefit substantially from having their immune, neurochemical, and hormonal systems rebalanced.

These are women who tend to not be tolerant of oral contraceptives. They have more side effects than the average population; they frequently get infections - not surprising to us as their immune systems are doing so much defense work that its resilience and capacity to neutralize foreign infectious invaders is less. Because of the repair deferral, the increase in tissue permeability also makes them more susceptible and hospitable to pathogens that another healthier person, when exposed to,

will neutralize easily and readily before that agent is able to proliferate and cause an infection.

## MITRAL VALVE PROLAPSE

Mitral Valve Prolapse or Barlow's Syndrome is common in these folks. This is a sign of magnesium deficiency from the underlying metabolic acidosis that occurs as a consequence of the immune reactions and very often acid-forming diet these folks are on, and not uncommonly when you correct that underlying metabolic acidosis and correct that hypomagnesemia, the annulus of the mitral valve shrinks, the connective tissue in the mitral valve supporting ring increases in its tensile strength, and the prolapse disappears. Mitral Valve Prolapse is an indication of magnesium deficiency, an indicator of connective tissue permeability, repair deficit in the connective tissue, and decrease in tensile strength in the ring of connective tissue that's the supporting structure for the mitral valve. In lots of studies that have been done, the leaflet of the mitral valve itself is quite normal, but the supporting connective tissue structure has relaxed and that's what has allowed for the valve leaflet to prolapse in a retrograde fashion and provoke this Mitral Valve Prolapse or Barlow's Syndrome.

The patient that has these so-called multiple treatment-resistant conditions can benefit from proper diagnosis and proper stimulation of their repair competence, immune defense and repair system, and the concomitant neurochemical and hormonal regrouping and repair that occurs. These are folks that are often more sensitive to perfumes and odors, and more sensitive to cigarette smoke, car exhaust, and other kinds of similar irritative exposures that provoke prominent responses in the form of rhinitis or increased airway resistance. When the acidosis/immune overload is corrected, so the increased tissue permeability is corrected, and the person loses their increased sensitivity to these irritants. Seasonal allergies also often spontaneously go into remission.



There was a period of time where medical management of thyroiditis included surgical excision. That was in part because of the lack of understanding of the underlying mechanism and concern about the increased risk of thyroid cancer that attends to untreated thyroiditis. And so indeed, untreated thyroiditis at its end stage can have an increase in dysplasia and eventually neoplasm and cancer, but properly managed so that the blood thyroid barrier is reestablished and the thyroid gland is able to repair itself, then the dysplasia risk essentially vanishes. These are also patients who often spend lots of time trying to figure out why they feel so helpless and hopeless in life, and the underlying predisposition is an immune, hormonal, neurochemical one, not a lack of life experience or the result of life traumas. When the polyendocrine autoimmune conditions are properly managed, then the challenges of living become more manageable, and these folks are able to get on with their productive lives.

For a variety of reasons, there's a risk of alcoholism in these patients, and it can be for any or all of the following: the lack of ability to mobilize in response to the challenges of life can lead people to look for an escape, the pain that occurs with the polyendocrinopathy conditions can lead people to look for an escape, the lack of being able to find an effective solution to mood disorders and related conditions can lead people to look for an escape, and so the increase in the risk of consequential alcoholism. Alcoholism as a consequence of underlying polyendocrinopathy and autoimmune conditions is understandable when you tease apart the components. These patients often do well when their immune, hormonal, neurochemical systems are rebalanced, and then they are able to set the bottle aside.

## ANTIBODY TYPES

There are laboratory tests for looking at specific thyroid components. You can have **anti-thyroid microsomal antibodies, anti-thyroid globulin antibodies, anti-thyroid receptor antibodies, and in order to look at all the possible variance of thyroiditis,**

**you really need to look at all of those different antibody pools.** So simply doing a test for microsomal antibodies and finding that negative does not rule out thyroiditis. It rules out one variant form of thyroiditis. In the same way, because the antibodies can distort the functionality of the hormones, doing a routine assay for T4, T3, and TSH, the thyroid stimulating hormone, and finding those in the normal, usual range, does not rule out thyroiditis. Indeed, what the ranges of these hormones are and what these thyroid stimulating hormones should be for healthy people is usually not available from the laboratory, and so these assays are standardized on patients who are not known to have thyroid disorders. But, since underlying thyroid dysfunction is so commonly undiagnosed, and early expressions of the condition are so common, the usual laboratory ranges include some subpopulation of folks who actually have some thyroid disorder on a functional basis and are included because they don't have diagnosed disease when the laboratories standardize their assays.

In those laboratories that have tried to look only at highly healthy individuals, the variance range of thyroid hormone tends to be at the top third of the usual laboratory range, so it may well be that we gain too much comfort and have too little reason for confidence when we look at the usual laboratory studies for thyroid hormone, find them within usual ranges when we don't really know what the healthy range is and we're not really getting a functional measure of that thyroid hormone activity. We're just quantifying how much thyroid hormone activity or thyroid stimulating hormone activity is present at that moment in time.

There are periods of time when thyroiditis can be associated with excess of thyroid gland activity or hyperthyroidism, Grave's Disease, or fulminant Hashimoto's thyroiditis (p.785). There are times when you want to give thyroid hormone and soluble iodine to help block some of the excess thyroid hormone that can rage through the body at periods of activation of the gland in the thyroiditis process. Concurrently, though, you want to stimulate the repair of the basement membrane that

should keep the cells and unnecessary elements away from the thyroid gland. That's possible only with the use of proper diagnostic tools to find out what's loading up on the immune system and avoiding those foods and chemicals for a period of time while repair goes on.

The thyroid gland is really quite forgiving and quite remarkable when you stop pummeling it and stop attacking it with the cellular and other constituents that overstimulate it that the body is able to forgive and forget.

Well over three-fourths of the folks with thyroiditis who have followed the **LRA by ELISA/ACT program** have been able to restore normality to their thyroid activity, and many of these patients have decades of thyroiditis under their belts before they start the program, so even in extended stage or late stage thyroid disease, in thyroiditis, you can rest the gland and allow it to restore itself, and the follicles are able to reestablish themselves. There is an endpoint where there is essentially no remaining thyroid tissue to proliferate. At that point when the cells are dead, the cells are dead, and then you need continuing hormonal support for those folks for a lifetime. That's probably 1 in 20 of the patients with thyroiditis.

## FAMILY LINKAGES

Thyroiditis does tend to run in families, and this has given rise to the question of "is this a genetically predisposed population?" It may be, but some of the very habits and patterns of learned distress and acid-forming diets and maldigestion processes as well as those habits of living run in families, and so it's not at all clear that there's a strong genetic component as there is a learned adaptation response that folks have that predisposes them to the thyroiditis and the multiple system endocrinopathies and autoimmune endocrinopathies that are seen.

In terms of thyroid replacement there are some who favor giving T3 (Cytomel™) - the active form of thyroid hormone



(typically anywhere from 50 - 500 mcg/d). There are those who favor desiccated thyroid as a restorative source of thyroid hormone and natural thyroid constituents that T3 lacks. You can have folks that favor one over the other. Desiccated thyroid seems to work well for most people, but some people do better on T3.

If you are not going to strengthen the immune system, then reducing the load from fungal organisms like Candida, through the use of Nystatin and similar treatments still makes sense. From experience, Nystatin can select for resistant yeast organisms in the same way that antibiotics can select for antibiotic-resistant strains of those bacteria or organisms. Better clinical results have been seen by:

1. reducing the intake of immunoreactive foods and chemicals,
2. increasing ascorbate to adequate amounts based on ascorbate calibration, and
3. magnesium intake to adequate amounts based on a magnesium loading test or clinical assessment including choline citrate to facilitate magnesium uptake in people with magnesium uptake block, and
4. the glutamine and PAK (pyridoxal alpha-ketoglutarate) to stimulate intestinal tissue repair.

Proper and sufficient supplementation so that you achieve a healthy transit time of 12 to 18 hours, rather than a 48 to 132-hour transit time with which typical Americans are burdened.

The shorter, healthy 12-18 hour transit time does not allow for proliferation of the yeast organisms, and if you don't have fermentable digestive remnants for the yeast to grow on, then candida and similar fungi don't proliferate.

While Nystatin can be used, its best clinical results occur when you are also addressing the underlying causes of:

1. long transit time,
2. antioxidant deficits, and
3. immunoreactive digestive remnants that load up the body's immune system and can lead to fermentable, digestible remnants in

the colon that the yeast organisms require for growth.

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#### Contact

If you have any questions or would like more information about LRA by ELISA/ACT testing, please contact ELISA/ACT Biotechnologies' Client Services Department at 800-553-5472.