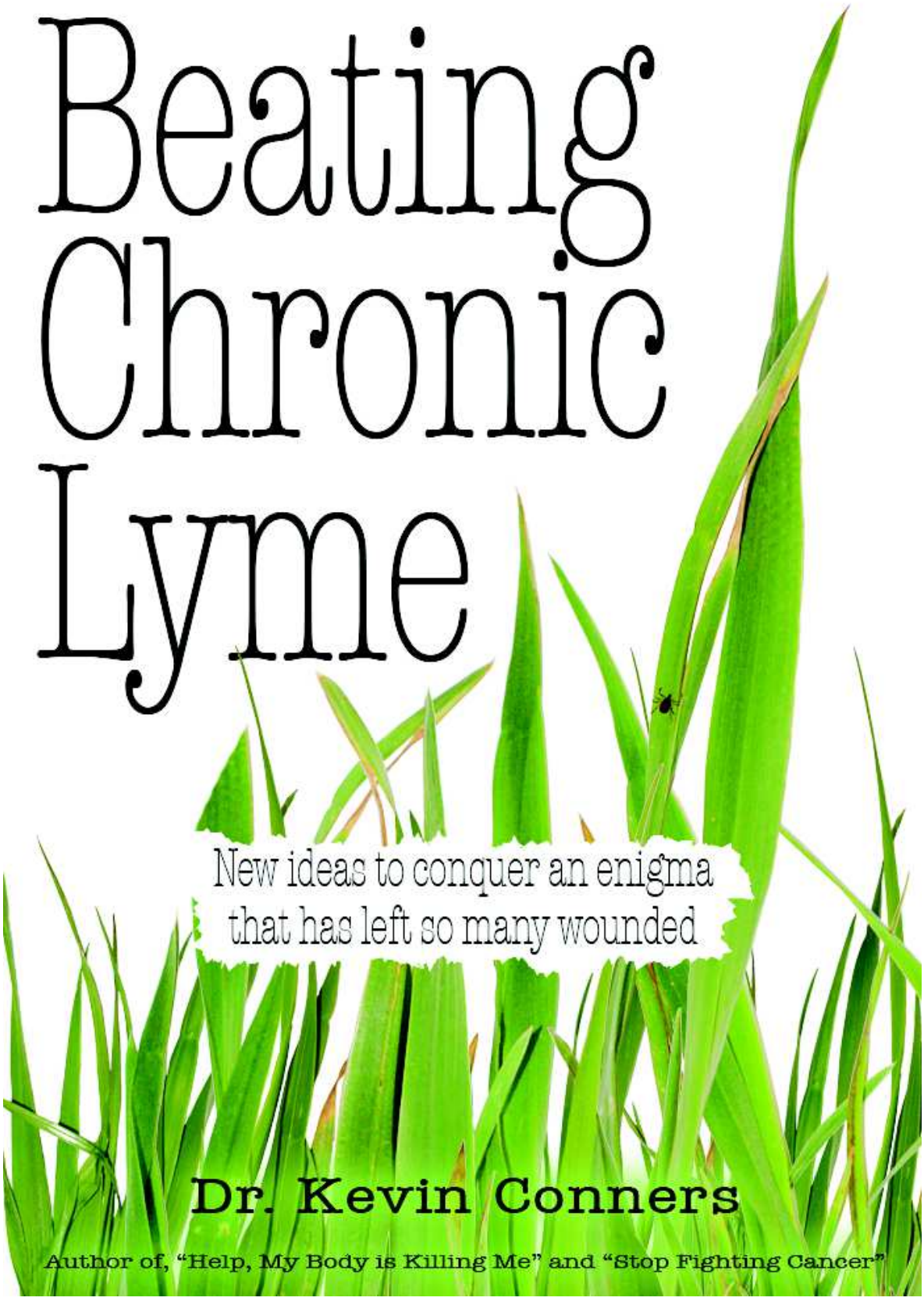
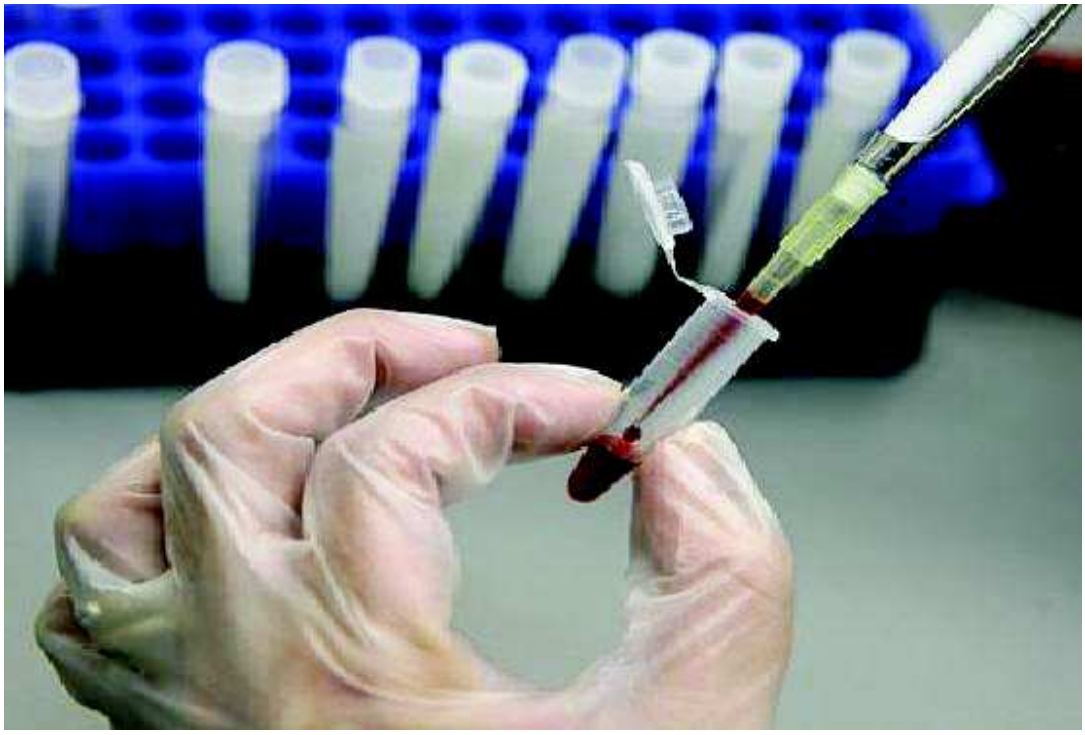
Beating Chronic Lyme



New ideas to conquer an enigma that has left so many wounded

Dr. Kevin Conners

Author of, "Help, My Body is Killing Me" and "Stop Fighting Cancer"



Understanding the Immune Response

"When wealth is lost, nothing is lost; when health is lost, something is lost; when character is lost, all is lost."

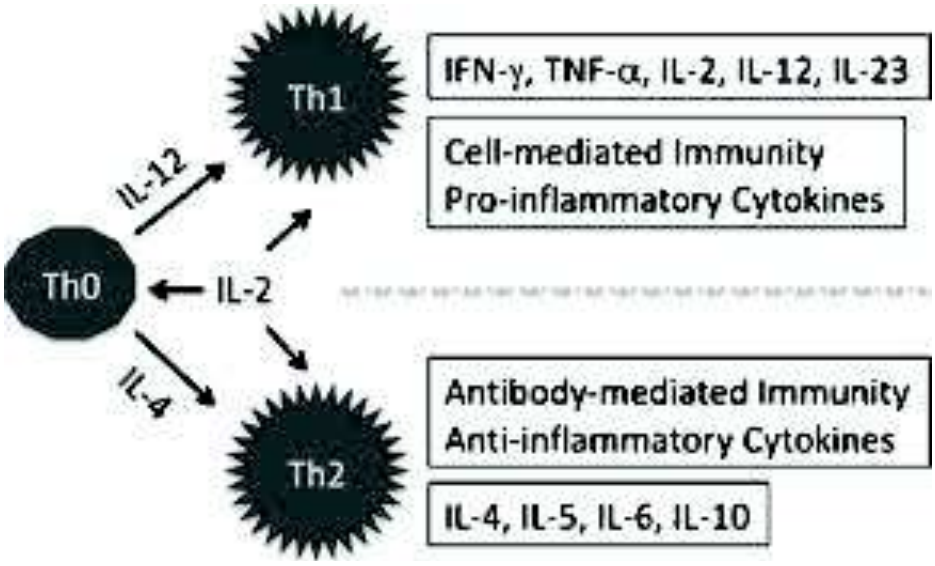
Billy Graham

What is Autoimmune Lyme?

Once you've missed the "window of opportunity" of killing the pathogen with an antibiotic, things turn south. Chronic Lyme disease is, after all, what this book is supposed to be about, so what does one do?

First, one must remember that CLD usually becomes an autoimmune disorder. So it is necessary to begin with an understanding of what an autoimmune disease really is.

It is important to understand that an autoimmune disease is a 'state' that the immune system is in. It is NOT a disease of an organ; and even though it is given a multitude of names depending on the tissue currently affected, it is a STATE of the immune system attacking the tissue it was meant to protect.



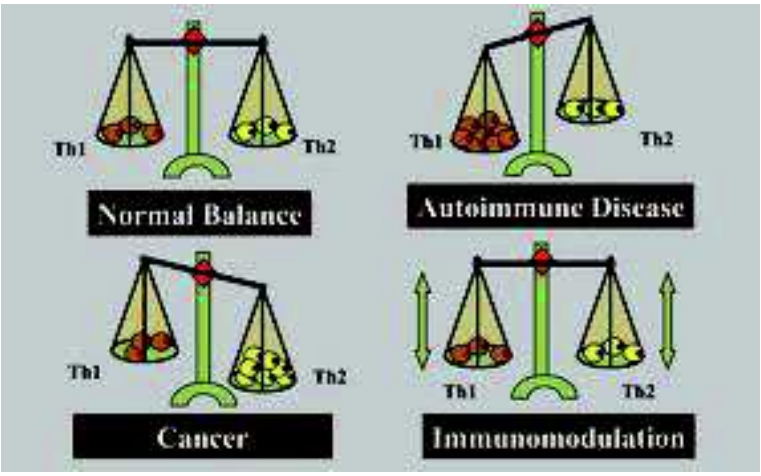
Some highlight points to know about your immune system:

- Your immune system does one thing and only one thing – it KILLS things.
- Your immune system may be separated into two responses – Th1 and Th2 (simplistically, there are more but we'll leave it at that for now)

- Your immune system is supposed to only 'turn on' against bio-toxins (living organisms like bacteria, virus, parasites…that is, things that it can kill)
- The Th1 response is the immediate, killer cell response (think of it as the Marine Corps) against the enemy and is the primary killer of antigens like Lyme pathogens and its co-infections. *What* it 'turns on' against is called an **antigen** in the immune response.
- The Th2 response is sent out secondarily and is mainly responsible for making antibodies against the antigen that the Th1 system 'turned on' against. The antibodies 'tag' that antigens and the Th1 system can then more easily find and kill them.
- Your immune system assists in the cleaning up of old cells necessary for cancer to NOT develop in the first place. This is primarily a Th1 function.
- Both Th1 and Th2 responses are named such because they carry a slurry of different chemicals (immune cells, chemokines and cytokines) that make up such a response.

An AUTOIMMUNE disorder happens when your immune system starts attacking self-tissue. Really, an autoimmune disease develops because your immune system has 'turned-on' against something IT FOUND lodged in self-tissue and now is destroying self-tissue as well.

Let's expand that definition a little more so you can fully understand it: If my immune system fires a response against a flu virus I just picked up and it's a particularly virulent virus, a strong Th1 response is released in an attempt to kill the foreign invader and bring me back to health. My 'strong Th1 response' is really a collection of different cells that are looking for a battle; they are seeking an enemy with guns loaded. Let's say they find the flu virus and recognize that it is the enemy they were commissioned to kill, they attack it, kill it and then retreat in victory. The Th1/Th2 system goes back into balance and life is good.



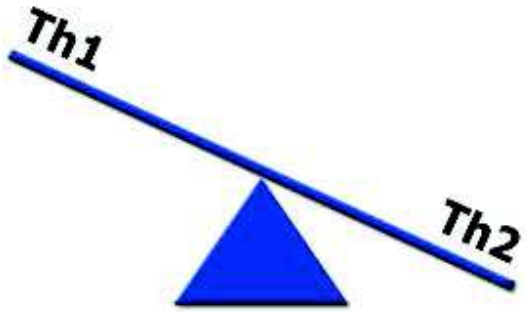
An autoimmune disease begins when, for a multitude of reasons we won't breach here, stray cytokines from a Th1 response didn't recognize the flu virus as the enemy but recognized something that they were never supposed to recognize as an enemy – let's say a heavy metal toxicity in my thyroid.

Because I was exposed to a great amount of mercury from amalgams, vaccinations, and just living in a toxic world, mercury had lodge in the fat cells surrounding my thyroid and other tissues. My liver, unable to clear out that which I was exposed to, caused my system to shunt the toxicity to fat storage cells for safe keeping. Never was my immune system supposed to 'turn-on' against such chemical toxicity!

Is my immune system ever going to be able to kill mercury? Of course not; mercury is an element on the periodic table, not a living organism. If my immune system inadvertently turns-on against something that *cannot or will not die*, there will be a lot of collateral damage and I might even begin to start making antibodies against the tissues surrounding the attack. This is an autoimmune disease; it isn't really a disease at all, it is an immune attack on self-tissue because my immune system is firing against something it never should have fired against! Remember, when the immune system turns-on against something, it does so until it achieves victory, until it kills it.

In the case of Lyme: Lyme disease in its acute state is theoretically 'killable' by one's immune system. Because it is very virulent, it may take your un-aided

immune response some time to make a dent in it. This is a problem because after an indiscriminant amount of time (from a matter of a few days to several weeks), Lyme disease morphs into its spirochete (viral-like) phase and can move intracellular (within the cell).



Normally, when a virus attempts to hide from one's immune response by infiltrating a cell, the cell gives off a marker on the outside of the cell membrane to alert the immune cytokines that it has been breached. Immune killer cells then engulf the entire cell, killing both the cell and the invading pathogen and protecting the whole in the process.

However, there are certain bacterial and viral organisms (Lyme being one of these) that has the capability to disarm the cell by disabling the marker that informs the immune system it has been attacked. I like to describe it this way: Think of a bank robber entering a bank and demanding the teller to fill up his bag with money. The teller pushes a secret button under the counter that summons the police and the thief is apprehended. But a smart thief (Lyme) cut the phone line outside of the building before entering the bank and the secret button does nothing to inform the police car that passes by completely unaware. Tricky little beastie isn't he?

Summary: An autoimmune disease is when one's immune system is firing against something (either predominantly Th1 dominant or predominantly Th2 dominant) that it found lodged in self-tissue that either cannot or will not die (as in Lyme) and is destroying self-tissue in the process.

So, what is the CAUSE of an autoimmune attack? It is not really an "immune system gone wrong" as it is an immune system thinking it is doing "right" but firing against something that it can never kill. The only way to ultimately correct an autoimmune disorder is to remove the antigen it is making war against. This way you are essentially fooling your immune system to think that it has won and the enemy is dead. In the case of autoimmune disease against a specific organ like Hashimoto's hypothyroidism, there is little help in direct organ support without correcting the cause. The mechanism for the issue is the immune response in the first place and not that the organ is deficient in any type of nutrient; the reason the person may need hormone replacement (such as Synthyroid) in hypothyroidism is because the immune system is actually destroying the cells, but replacement without halting the destruction is missing the point.

What does this all have to do with Lyme?

What does this all have to do with Lyme? Everything! Remember, Chronic Lyme (CLD) is when the acute infection has moved into a viral-like, spirochete phase that is nearly impossible to kill as it hides inside cells and evades one's immune

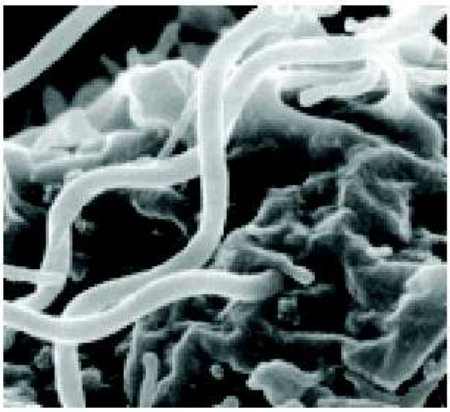
response. When Th1 cytokines cannot find the pathogen, they start to kill surrounding tissue creating an autoimmune disorder. CLD becomes an autoimmune disorder!

REVIEW of the THREE PHASES of Lyme:

1. Acute infection – in this phase, the patient STILL has the capability to KILL the disease with an antibiotic. This is why I HIGHLY recommend that those living in Lyme-infested areas have antibiotics on hand to use should they develop symptoms in Lyme season. This is ONLY open for a WINDOW of time!

The "window of opportunity" to KILL Lyme in the ACUTE PHASE can be VERY short.

2. Chronic Lyme – Chronic Lyme phase begins the moment the first bacteria EXIT the bloodstream and ENTER the intracellular space (go inside the cell and hide). This phase still may be treated with antibiotics and immune-boosting Nutriceuticals BUT it will be a LONG, drawn-out treatment plan. Though it is better than Phase THREE, Chronic Lyme is horrible.



A scanning electron microscope image of Borrelia burgdorferi penetrating a human B cell (in vitro), at a magnification of approximately 89,000.
Photo Credit: David W. Dorward, Ph.D.
NIH Rocky Mountain Labs, MT.

3. Autoimmune Lyme - When the patient's condition continues to linger, the immune system is constantly trying to kill it. In doing so, the "killer" side of the immune system, the Th1 response, fires to kill the pathogen. THIS phase is really what this book is all about! These patients are miserable and it is the autoimmune phase of Lyme that is deadly.

How does it progress?

Usually people with CLD that has now become an autoimmune disorder, involving much destruction and therefore many symptoms have a Th1 dominant autoimmune response, that is, the immune response is stuck in a Th1 (killer cell, Marine Corps) attack. This typically brings about much tissue damage, much inflammation and a greater number of symptoms that causes them to seek medical care and hopefully arrive at a diagnosis. They often are misdiagnosed as having MS, RA, Hashimoto's, etc. Actually, it may not be a misdiagnosis as all autoimmune disorders have an antigen at its core and that antigen might just be Lyme!

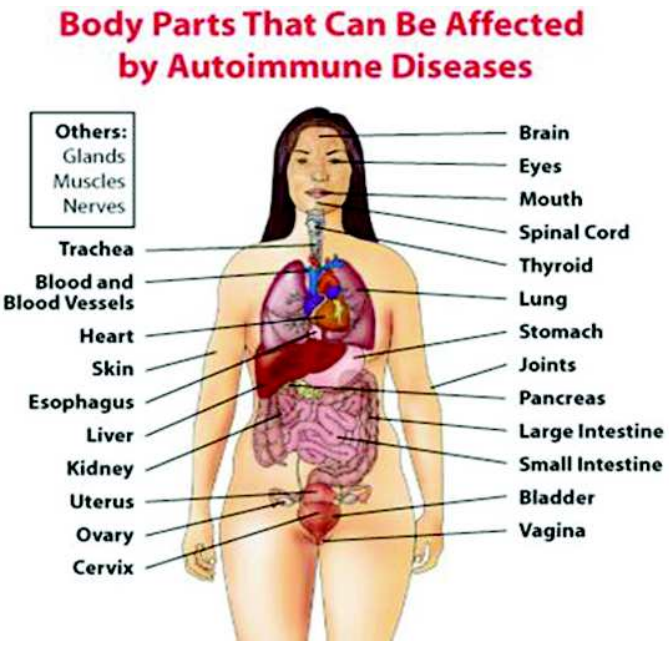
Neither the standard medical nor an alternative healthcare has adequately dealt with autoimmune conditions, including CLD, because most fail to understand the Th1/Th2 issue. Medically, the patient is given long-term antibiotics, anti-malarial drugs, steroids, anti-inflammatories and more that may temporarily relieve the symptoms but do nothing to remove the cause; alternative doctors have

supported the organs with glandulars and tried to kill the CLD with herbs or other supplements. Let's face it, if either traditional medical or the alternative models had any great percentage of success treating CLD autoimmune disorder, you wouldn't be reading this book because you probably wouldn't have any symptoms.

It is important to understand that an autoimmune disease is a 'state' that the immune system is in. It is NOT a disease of an organ; and even though it is given a multitude of names depending on the tissue currently affected, it is a STATE of the immune system attacking the tissue it was meant to protect.

It's absolutely necessary to figure out if a person is Th1 or Th2 dominant because it will dictate what type of protocols that will be most effective for dampening their immune activity. We know that typical 'immune stimulants' like Astragalus, Cats Claw, Samento, Echinacea, Garlic, Glycyrrhizin, Melissa Officinalis, Maitake mushrooms, seem to stimulate the Th1 response. We also know that things like pine bark extract, grape seed extract, green tea extract, Pycnogenol, Resveratrol, and caffeine are things that stimulate the Th2 response. So if a patient's CLD attack of their joints, brain, muscles or fatigue is a Th1 dominant response, adding Th1 stimulants will MAKE THEM WORSE! You can effectively aid in balancing a Th1 dominant individual by giving Th2 stimulants and visa, versa.

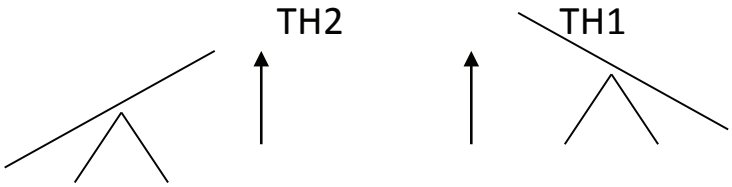
It is often that the patient's history will be obvious as to which dominance they are 'stuck' in. If they've attempted taking high amounts of Cats Claw, Garlic and Echinacea in the past only to feel horribly worse afterward, there's a pretty good chance they are Th1 dominant autoimmune. If drinking green tea or coffee takes away your major symptoms, the possibility exists that you are Th1 dominant; if it made you feel worse, you may be Th2 dominant. But do NOT rely on this; it is always wise to do the testing! I wish it were always that easy to detect dominance. Many people just don't seem to get better after giving full effort with numerous nutritional or standard approaches. This should be at least a clue that there is something deeper not being addressed.



Also, you have to be very careful stimulating a Th1 or Th2 response. People can't figure out why they still feel terrible even while taking the boatload of vitamins their nutritionist recommended. If you are stimulating the dominant, hyper-firing system, you are literally throwing fuel on the fire. Autoimmune patients CANNOT take supplements that have both Th1 and Th2 stimulants. You are helping the immune system destroy your body! Do the testing!

Th1 and Th2 Balancing

There are 2 parts of your immune system, the Th1 and Th2 response. When a person is autoimmune, one of these systems is "hyper-firing" or Dominant. Balancing this system goes far in reducing a patient's symptoms:



These SHOULD be Balanced!

TH1 or TH2

TH2 Dominance **TH1 Dominance**

There are specific dietary changes and supplements that can help and hinder the above response: NOTE: ALL AI cases need Vitamin D, Glutathione, and Omega 3 fish oils +

- Things that stimulate the Th1 response: (Take these if you are Th2 Dominant)
  - Cats Claw
  - Samento
  - Echinacea
  - Golden Seal
  - Red Clover
  - Pau D'Arco
  - Wild Bergamot (Monarda fistulosa)
  - Oregon Grape (Mahonia aquifolium)
  - Andrographis (Andrographis paniculata)
  - Lignan-vitae (Guaiacum officinale)
  - Garlic
  - Vitamin C
  - Licorice root (Glycyrrhizin)

Astragalus
Most Medicinal mushrooms
Most Chinese Herbs
All “Immune Stimulants”
Beta-glucan mushroom
Maitake mushroom (Grifola frondosa)
Lemon Balm (Melissa officinalis)

- Things that stimulate the Th2 response: (Take these if you are Th1 Dominant)
  - Caffeine (don’t add this as this does a number on your adrenals)
  - Green Tea
  - Grape Seed Extract
  - Herbal barks (Cramp Bark, Pine Bark, and White Willow Bark)
  - Lycopene
  - Resveratrol
  - Pycnogenol

This is in NO way a complete list and individuals may react differently than expected!!!

Therefore, if a patient is Th1 Dominant, they should AVOID Th1 Stimulants and may TAKE Th2 Stimulants

The K.I.S.S. Formula for Acute Lyme, CLD and Autoimmune Lyme

Phase 1 – Treating ACUTE Lyme:

Initial bacterial phase – “Symptoms of early localized Lyme disease (Stage 1) begin days or weeks after infection. They are similar to the flu and may include:

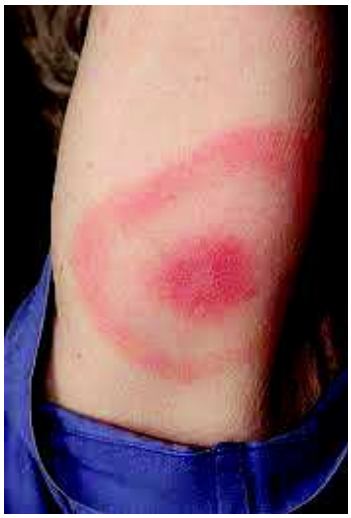
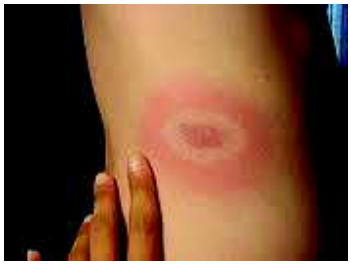
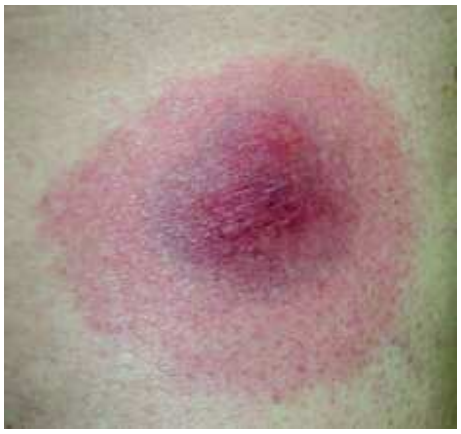
- Body-wide itching
- Chills
- Fever
- General ill-feeling
- Headache
- Light-headedness or fainting
- Muscle pain
- Stiff neck

There may be a "bull's eye" rash, a flat or slightly raised red spot at the site of the tick bite. Often there is a clear area in the center. It can be quite large and expanding in size.” (Adams)

Most commonly, no rash will be detected and the patient may never experience any of the above “acute phase” symptoms. This is most unfortunate as the patient is destined to move into a CLD state. If the person is bit in the head, the rash can hide under the hair and never be detected. Some people have a rash that last but a few hours; others never see a rash and mistakenly attribute their symptoms to a flu or food poisoning.

My RULE OF THUMB: If you live in a Lyme area and get flu-like symptoms during tick season, TREAT IT AS IF IT IS LYME and get an antibiotic!!! If you wait for the

tests to come back, it may be too late. Find a qualified Lyme-smart MD and get a prescription.

Treatment options in Phase 1:

ANTIBIOTICS

There are four types of antibiotics generally prescribed for Lyme treatment that I'd like to discuss. I am not an MD and cannot write a prescription so I'll quote Joseph J. Burrascano, M.D., *Board Member, International Lyme and Associated Diseases Society* from his 2008 work entitled "ADVANCED TOPICS IN LYME DISEASE"

1) "The TETRACYCLINES, including doxycycline and minocycline, are bacteriostatic unless given in high doses. If high blood levels are not attained, treatment failures in early and late disease are common. However, these high doses can be difficult to tolerate. For example, doxycycline can be very effective but only if adequate blood levels are achieved either by high oral doses (300 to 600 mg daily) or by parenteral administration (through an IV). Kill kinetics indicate that a large spike in blood and tissue levels is more effective than sustained levels, which is why with doxycycline, oral doses of 200 mg bid (twice per day) is more effective

than 100 mg qid (four doses per day). Likewise, this is why IV doses of 400 mg once a day is more effective than any oral regimen. (I realize IV dosing is not realistic for most patients, see below)

2) PENICILLINS are bactericidal. As would be expected in managing an infection with a gram negative organism such as Bb, amoxicillin has been shown to be more effective than oral penicillin V. With cell wall agents such as the penicillins, kill kinetics indicate that sustained bactericidal levels are needed for 72 hours to be effective. Thus the goal is to try to achieve sustained blood and tissue levels. However, since blood levels are extremely variable among patients, peak and trough levels should be measured (for details, refer to the antibiotic dosage table). Because of its short half-life and need for high levels, amoxicillin is usually administered along with probenecid. An extended release formulation of amoxicillin+clavulanate ("Augmentin XR") may also be considered if adequate trough levels are difficult to attain. An attractive alternative is benzathine penicillin ("Bicillin-LA"- see below). This is an intramuscular depot injection, and although doses are relatively small, the sustained blood and tissue levels are what make this preparation so effective.
3) CEPHALOSPORINS must be of advanced generation: first generation drugs are rarely effective and second generation drugs are comparable to amoxicillin and doxycycline both in-vitro and in-vivo. Third generation agents are currently the most effective of the cephalosporins because of their very low MBC's (0.06 for ceftriaxone), and relatively long half-life. Cephalosporins have been shown to be effective in penicillin and tetracycline failures. Cefuroxime axetil (Ceftin), a second generation agent, is also effective against staph and thus is useful in treating atypical erythema migrans that may represent a mixed infection that contains some of the more common skin pathogens in addition to Bb. Because this agent's G.I. side effects and high cost, it is not often used as first line drug. As with the penicillins, try to achieve high, sustained blood and tissue levels by frequent dosing and/or the use of probenecid. Measure peak and trough blood levels when possible. When choosing a third generation cephalosporin, there are several points to remember: Ceftriaxone is

administered twice daily (an advantage for home therapy), but has 95% biliary excretion and can crystallize in the biliary tree with resultant colic and possible cholecystitis. GI excretion results in a large impact on gut flora. Biliary and superinfection problems with ceftriaxone can be lessened if this drug is given in interrupted courses (known commonly as “pulse therapy”- refer to chapter on this on page 20 of Dr. Burrascano’s book for more info on this), so the current recommendation is to administer it four days in a row each week. Cefotaxime, which must be given at least every eight hours or as a continuous infusion, is less convenient, but as it has only 5% biliary excretion, it never causes biliary concretions, and may have less impact on gut flora.

4) RYTHROMYCIN has been shown to be almost ineffective as monotherapy. The azalide azithromycin is somewhat more effective but only minimally so when given orally. As an IV drug, much better results are seen. Clarithromycin is more effective as an oral agent than azithromycin, but can be difficult to tolerate due to its tendency to promote yeast overgrowth, bad aftertaste, and poor GI tolerance at the high doses needed. These problems are much less severe with the ketolide telithromycin, which is generally well tolerated. Erythromycins (and the advanced generation derivatives mentioned above) have impressively low MBCs and they do concentrate in tissues and penetrate cells, so they theoretically should be ideal agents. So why is it that erythromycin ineffective, and why have initial clinical results with azithromycin (and to a lesser degree, clarithromycin) have been disappointing? It has been suggested that when Bb is within a cell, it is held within a vacuole and bathed in fluid of low pH, and this acidity may inactivate azithromycin and clarithromycin. Therefore, they are administered concurrently with hydroxychloroquine or amantadine, which raise vacuolar pH, rendering these antibiotics more effective. It is not known whether this same technique will make erythromycin a more effective antibiotic in LB. Another alternative is to administer azithromycin parenterally. Results are excellent, but expect to see abrupt Jarisch-Herxheimer reactions.

Real world uses:

Again, if you even THINK that you have an acute Lyme infection; antibiotics are the way to go. I do NOT suggest playing around with alternative herbs and vitamins at this stage. They tend not to kill an acute Lyme and allow the pathogen to move into the next stage.

In my two experiences with acute Lyme, I took amoxicillin with exceptional results. However, my first incidence was easy to spot with a bulls-eye rash that appeared painted upon my belly and severe flu-like symptoms. One day of 500mg amoxicillin, taken twice daily, knocked out all of my symptoms. I only completed 3 days of antibiotics though I'd never suggest anyone to follow my lead on that. The next Lyme episode went undiagnosed for nearly 5 days as I had no rash and very little exposure to the outside. At first I thought that I had food poisoning, then the flu. After 4 days I finally tested myself Kinesiologically and sure enough, it was Lyme. Amoxicillin saved the day again.

Everyone is different and for many, doxycycline is the therapy of choice for acute Lyme. It is my opinion that messing around with any other therapy to kill acute Lyme is like playing with fire. Don't do it.

Phase 2 – Treating CHRONIC Lyme –

definition = the moment one bacterium traverses a cell membrane to enter the INSIDE of the cell.

Schematic representation of a spirochete

Again, the autoimmune response is an inflammatory response, which produces chemicals called cytokines, which are part of the body's natural defense system against outside invaders. Remember, the body's immune system may be separated into a Th1 and a Th2 response. The Th1 response may be thought of as the police force or Marine Corps, the body's initial strike force against an invader or what is called an antigen.

In a PERFECT WORLD, when an antigen (this case Lyme) is present, the Th1 system fires to try to kill the Lyme; since the bug happens to be of a nasty persuasion and strong enough to resist the Th1 response, the Th2 system kicks in, creates antibodies against the Lyme, tagging them so appropriate white blood cells can finish them off. In REALITY, this rarely happens. The Th1 response is NOT strong enough to kill the Lyme pathogens and they morph into a viral-like state and go intracellular. Your Th2 cytokines start producing antibodies to the tissues it searched in and you are in for a world of trouble because we PASSED directly to the autoimmune phase!

NOTE: This is a major reason WHY the blood tests for Lyme are so highly inaccurate – the quantities of antibodies are never created for a pathogen the immune system could not detect.

NOTE: A person with an autoimmune disease has this process stuck in the 'on' position, either hyper-Th1 or hyper-Th2, which prolonged, destroys the tissue where the antigen is recognized. MOST AUTOIMMUNE PATIENTS ARE TH1 DOMINANT, i.e., their immune system is 'stuck' in a Th1 phase!

Why is this so important? If the patient stuck in a Th1 dominant immune response, takes MORE Th1 stimulants, THEY GET WORSE (or at least no better). What is the main treatment modality for Lyme? Th1 stimulants!!! Um, HELLO…the patient is not going to get better!!!

So, REALLY IMPORTANT…

1) PHASE TWO Lyme patient CAN take immune (Th1) stimulants, but…

2) PHASE THREE (autoimmune) Lyme patients CANNOT take immune (Th1) stimulants!

Hence, if you ignore the Th1/Th2 immune response in treating a patient with CLD, both the traditional medical and the traditional alternative models of care are doomed to failure. The most important battle to fight is to calm down their immune response and stop the destruction and kill the pathogen through another route!!!

The "new model" we are proposing is simply to be more specific. If YOUR autoimmune, PHASE THREE Lyme is a hyper-Th1 attack (Th1 dominant) against the antigen and its co-infections, doesn't it make sense to do everything possible to find out how remove it while calming down the Th1 dominance? I'm no rocket scientist, but this makes sense to me. It's logical and possible to find the specific biochemical pattern perpetrating the response so we can determine how we treat them.

If you can understand this piece and the role of the immune system, you can understand how antigens (non-living toxins or nasty, hard to kill Lyme, virus,

mold, candida…) *can* be at the heart of many autoimmune disorders and even cancer.

A MAJOR part of my practice is IDENTIFYING and ELIMINATING antigens! In doing so, the body can return to homeostasis (balance) and miraculously heal itself!

Possible Treatments part 1

Nutriceuticals

Category 1 – Things that will NOT stimulate the Th1 side of the immune response. Listed below are some nutritional approaches that will NOT mess with the immune response. By this I mean, they are usually safe for those that are Th1 dominant (which is most PHASE THREE patients):

- **Lauricidin (a brand name for Monolaurin that we use)**

Monolaurin is in mother's milk and is a very powerful germ-fighter that is also in coconut oil. Many clinical studies have shown Monolaurin kills 100% of every pathogen (bad bacteria) it has been tested on – including the anti-biotic resistant *MRSA* staph infections! Even the FDA has approved Monolaurin for anti-bacterial food preparation. It also kills *energy draining pathogens and Lyme Disease borrelia strains!*

Monolaurin is a 12-Carbon fatty acid, derived from Coconut oil and prepared into what is called a mono-ester of lauric acid. Lauric Acid attacks viruses and bacteria by destroying the lipid coating that surrounds them which then causes their cell walls to lyse. However, it is not effective to simply take coconut oil. *Monolaurin is the mono-ester of lauric acid* which is far more biologically ACTIVE than simply the lauric acid (in pure coconut oil) at destroying viruses, bacteria, and fungi.

Pathogenic bacteria, viruses, molds and borrelia burgdorferi have a lipid fat exterior envelope or skin. This is by design so the bacteria and virus can easily move around, some even penetrating cells as described earlier. Monolaurin happens to have the same size lipid fat molecules so they *absorb into the pathogen's skin*. This can cause the lipid envelope to rupture and the bacteria or virus disintegrates and dies.

As Lyme dies, the blood then takes the debris to the liver where it is eliminated from the body. Like any of the Lyme-killers I describe, one MUST be careful about the RATE of die-off. Monolaurin is so good at what it does that it can kill bacteria and virus *faster* than then the liver can get rid of the dead cells. For this reason, I recommend that people slowly build up to a full daily treatment amount (1 tsp. of little pellets, 3 times a day) over the first week or so.

Monolaurin kills bacteria, Lyme's Disease and some viruses on *contact* with them (by absorption and disruption) and NOT by stimulating the immune response. This is why it is safe for Th1 dominant individuals. Like most things I recommend, one must remember that treating CLD is a PROCESS and takes many months or years. Taking Monolaurin over this entire time can also kill additional pathogens and Lyme bacteria when they emerge from cells they often hide in.

- **Colloidal Silver**

Personally, I don't recommend Colloidal silver therapy for Lyme disease very often. There is some controversy regarding this therapy regarding systemic care – meaning, many think that it is not very effective past the stomach and first part of the small intestine. However, it is a great topical antiseptic and works well for stomach infections. On the positive side, one may be able to get colloidal silver into the blood and benefit from its antiseptic properties by using it in an inhaled form through a nebulizer. The size of the colloidal particles is also important. The smaller the particles size of Colloidal Silver the better the bioavailability, the stronger the anti-microbial effect and the safer to use.

- **MMS Protocol**

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.

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