The 3 Phases of Lyme

Dr. Conners CLEAR-LYME Protocols

Dr. Kevin Conners

Section 2

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The History of Lyme disease

According to Adams Medical Encyclopedia, “Lyme disease was first reported in the United States in the town of Old Lyme, Connecticut, in 1975. In the United States, most Lyme disease infections occur in the following areas:

- Northeastern states, from Virginia to Maine
- North-central states, mostly in Wisconsin and Minnesota

- West Coast, particularly northern California

Risk factors for Lyme disease include:

- Doing outside activities that increase tick exposure (for example, gardening, hunting, or hiking) in an area where Lyme disease is known to occur
- Having a pet that may carry ticks home
- Walking in high grasses

Important facts about tick bites and Lyme disease:

- In most cases, a tick must be attached to your body for 24 - 36 hours to spread the bacteria to your blood.
- Blacklegged ticks can be so small that they are almost impossible to see. Many people with Lyme disease never even saw a tick on their body.
- Most people who are bitten by a tick do not get Lyme disease."

See videos on the linked page below for further clarification:

http://connersclinic.com/lymes-disease/

Advanced Information: How Lyme HIDES

First, Our OWN Microbes

Beginning immediately at birth, humans are colonized by a myriad of microorganisms that assemble into complex stereotypic communities, creating a beneficial indigenous microbiota (our flora). The result is a “supra-organism” in which our microbial partners outnumber our human cells by 10-to-1. Most currently available information about the human microbiota concerns the bacterial component, although they are by no means the only important members. However, bacteria will be the focus of this discussion.

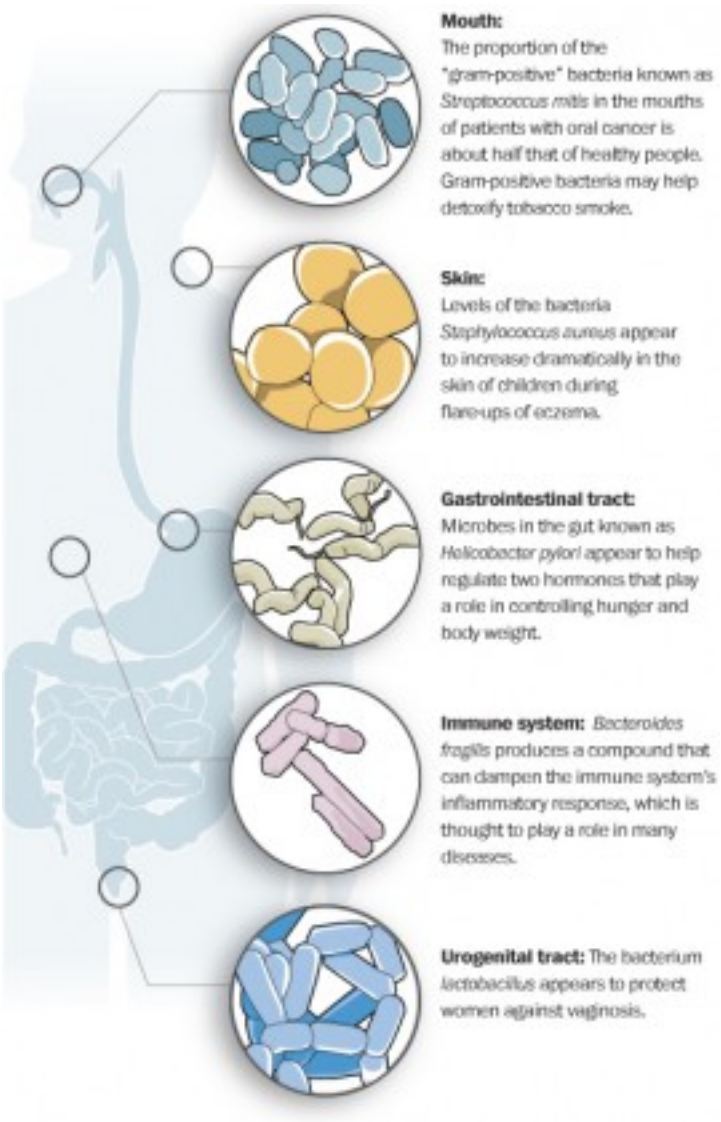
In contrast to the relatively rare harmful encounters with pathogens (like Lyme), indigenous human-microbe relationships are the dominant forms in which we interact with microbes and are fundamentally important to human physiology. Co-adaptation and co-dependency are features of our relationships with these friendly bugs.

This we now know to be true:

- The human microbiota facilitates nutrient acquisition and energy extraction from food,
- It promotes terminal (postnatal) differentiation of mucosal structure and function, and
- It stimulates both the innate and adaptive immune systems.

By so being the primary stimulation of immune system function it helps to create an epithelial boundary and integrity, as well as to “educate” our innate immune defenses. It also provides “colonization resistance” against pathogen invasion, regulates intermediary metabolism, and processes ingested chemicals.

It also is extremely important in the resistance of Lyme disease. Symbiotic microbiota resist all pathogenic species simply by competing with territory. Unfortunately, Lyme bacteria migrate to places our ‘normal’ microbes dare not go. Intracellular (within the cell) penetration is one such place but, by all means, it is not the only way Lyme evades immune destruction.



Precisely how our microbial community is assembled is still just being better studied. In the neonatal period, the community assembly process is especially dynamic and is influenced by early environmental (in particular, maternal) exposures. It is believed that the composition and functional capabilities of the indigenous microbiota evolve in a generally orderly fashion, as diet, hormonal environment, other environmental factors, and occasional ecologic disturbances play out their effects on a distinct, albeit diverse, human genetic background.

Differences in the capability of strains may explain variation among individuals in the metabolism of drugs such as digoxin and other exogenous chemicals as well as an individual's ability to fight off virulent pathogens. Differences in the capability of strains to tolerate normal inflammation may also influence the composition of the microbiota. Although there is evidence for shared functional

capabilities among the intestinal microbial communities of different healthy humans, host genetics is a source of variation in the makeup of the human indigenous microbiota.

What IS an Infection?

Infection (or *colonization*) is simply the establishment of a microorganism on or within a host; it may be short lived, as in our encounters with “transients”, or be persistent and may result in only low gain or harm to either participant. *H.pylori* would be another example. While it *may* cause immediate disease (stomach ulcers), it may also lead to chronic, insidious, subclinical effects with long-term consequences (cancer, heart disease). Many microorganisms with a capacity for sustained multiplication in humans, including members of the indigenous microbiota, cause disease more readily in individuals with underlying chronic disease or in those who are otherwise compromised. The common term *opportunist* suits this category of pathogen well.

What are the distinguishing characteristics of microbes that live in humans? A successful LYME pathogen must do the following:

- Enter the human host;
- Become established, which includes successful competition with indigenous microbes;
- Acquire nutrients;
- Avoid or circumvent the host’s innate defenses and a powerful immune system;
- Above all, replicate;
- Disseminate if necessary to a preferred site; and

Eventually, ALL pathogens desire to be transmitted to a new susceptible host.

The Genes

Whether a pathogen or a commensal, a microorganism must also possess an interactive group of complementary genetic properties that promote its interaction with the human host. For a given microorganism, the genetic traits define unique attributes that enable it to follow a common sequence of steps

used in establishing infection or, in some cases, subsequent disease.

Genetic testing techniques now permit the identification, isolation, and characterization of many of these genes. The availability of the host (e.g., human) genome sequence also enables multiple synergistic approaches for understanding virulence, including the identification of host susceptibility traits, genome-wide assessments of host response, and clues about the mechanisms of host defense and pathogen counter-defense.

Smart Bugs

It has long been but a hypothesis that pathogens (take *Borellia* from Lyme for example) breach intact host anatomic, cellular, or biochemical barriers that ordinarily prevent entry by other microorganisms. Thus, pathogens "go where other microbes dare not." In addition, many pathogens, such as *Borellia*, *Mycobacterium tuberculosis*, *Treponema pallidum*, *Chlamydia trachomatis*, and *Salmonella typhi,* have the capacity to establish persistent (often subclinical) infection in the human host and have evolved the extraordinary capacity to live in the inner sanctums of our innate and adaptive immune defenses or, in general, to compete well in the face of otherwise hostile host conditions.

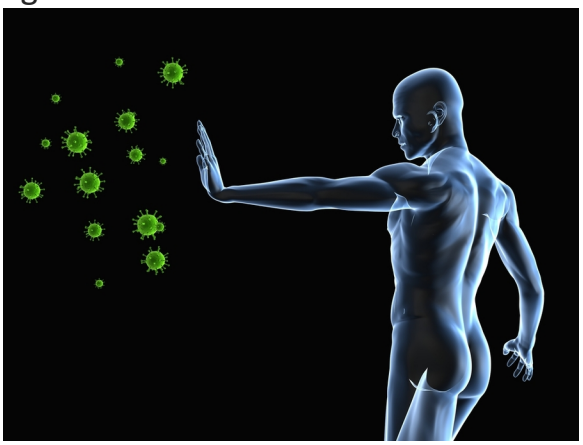
Some may even evoke human macrophage activity to defend itself against immune attack!

For example, *Salmonella* profits from the inflammatory response that it provokes in the gut by using the oxidized form of a locally produced host factor for a selective growth advantage against commensals. A distinction, then, between a primary pathogen and opportunist is that the pathogen has an *inherent* ability to breach the host barriers that ordinarily restrict other microbes, whereas the opportunist requires some underlying defect or alteration in the host's defenses, whether it be genetic, ecologic (altered microbiota), or caused by underlying disease, to establish itself in a usually privileged host niche. Clearly, the nature of the host plays as important a role as the pathogen in determining outcome.

An initial step required of a pathogen is to gain access to the host in sufficient numbers. Such access requires that the microorganism not only make contact with an appropriate surface but also then reach its *unique* niche or

microenvironment on or within the host. This requirement is not trivial. Some pathogens must survive for varying periods in the external environment. Others have evolved an effective and efficient means of transmission. To accomplish this goal, the infecting microbe may make use of motility, chemotactic properties, and adhesive structures (or *adhesins*) that mediate binding to specific eukaryotic cell receptors or to other microorganisms (piggy-backing on other microbes).

Pathogens that persist at the surface of skin or mucosa usually rely upon multiple redundant adhesins and adherence mechanisms. If the adhesin is immunogenic, expression is usually regulated; in addition, antigenic variants may arise. Preexisting microorganisms (the host's existing microbiota) provide competition against establishment of the newcomer so long as it is healthy and abundant.



Normal inherent host defense mechanisms should pose the most difficult set of obstacles for pathogens and commensals in establishing themselves in a host. For any set of specific host defenses, an individual pathogen will have a unique and distinctive counterstrategy. Some of the best-known mechanisms that pathogenic microbes use for countering host defenses include the use of an antiphagocytic capsule and the elaboration of toxins and microbial enzymes that act on host immune cells and/or destroy anatomic barriers. These are smart bugs, after all.

Microorganisms also use subtle biochemical mechanisms to avoid, subvert, or, as we now increasingly understand, manipulate host defenses. These strategies include the elaboration of immunoglobulin-specific proteases, iron sequestration mechanisms, coating themselves with host proteins to confuse the immune surveillance system, or causing host cells to signal inappropriately, leading to dysregulation of host defenses or even host cell death. It really is quite amazing!

Examples of these mechanisms include the production of immunoglobulin A1

protease by the meningococci, the use of receptors for iron-saturated human transferrin and lactoferrin by *N. gonorrhoeae*, and the coating of *T. pallidum* with human soluble fibronectin.

Yersinia, *Mycobacterium*, and *Bordetella* stimulate a TH2 response and diminish the killer cells by inducing host cell production of interleukin-10, which is a potent immunosuppressive cytokine so it can slip past defense like a cunning spy. Antigenic variation and intracellular invasion are other common strategies used by successful pathogens to avoid immune detection.

Their Ultimate Purpose

The ability to multiply is a characteristic of all living organisms since, ultimately, reproduction and survival is its goal. Whether the pathogen's habitat in the relevant host is intracellular or extracellular, mucosal or submucosal, within the bloodstream or within another privileged anatomic site, pathogens have evolved a distinct set of biochemical tactics to achieve this goal. The ultimate success of a pathogen, indeed, of any microorganism, is measured by the degree to which it can multiply and to the extent that it succeeds is to the demise of the host.

An emerging concept of microbial disease causation, with origins in the field of ecology, is the notion of "community as pathogen," in which a conserved broad feature of the microbial community contributes to pathology, rather than any one specific member or component. This concept may be relevant to a wide variety of chronic inflammatory processes of skin and mucosa, including inflammatory bowel disease and chronic periodontitis. This concept answers questions many clinicians may have encountered as to the difficultly in both isolating and treating an individual pathogen.

In addition, microbial pathogenesis involves synergies between organisms, as well as between gene products, each of which may be insufficient alone in causing disease. For example, several members of the human health-associated nasopharyngeal microbiota, including *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Streptococcus pyogenes*, regularly cause well-defined, well-known human diseases. One may develop antibodies to individual organisms yet

such commensal pathogens persist in a significant proportion and can be associated with both acute and chronic disease.

Summary

Lyme avoids being killed by your immune system (phagocytosis) by:

1. Lyme **HIDES**. The pathogens may invade or remain confined in regions inaccessible to phagocytes inside of cells and/or in certain internal tissues (e.g. the lumens of glands, the urinary bladder) and surface tissues (e.g. unbroken skin) that are not well patrolled by phagocytes.

2. Lyme bacteria may be able to **avoid provoking an overwhelming inflammatory response**. Without inflammation the host is unable to focus the phagocytic defenses.

3. Lyme bacteria or their products **inhibit phagocyte chemotaxis**. For example, they suppress neutrophil chemotaxis, that is, they prevent an immune cells ability to be chemically attracted to it. This is an extremely handy spy technique that basically fools your immune system into thinking that the Lyme bacteria are the ‘good guys’.

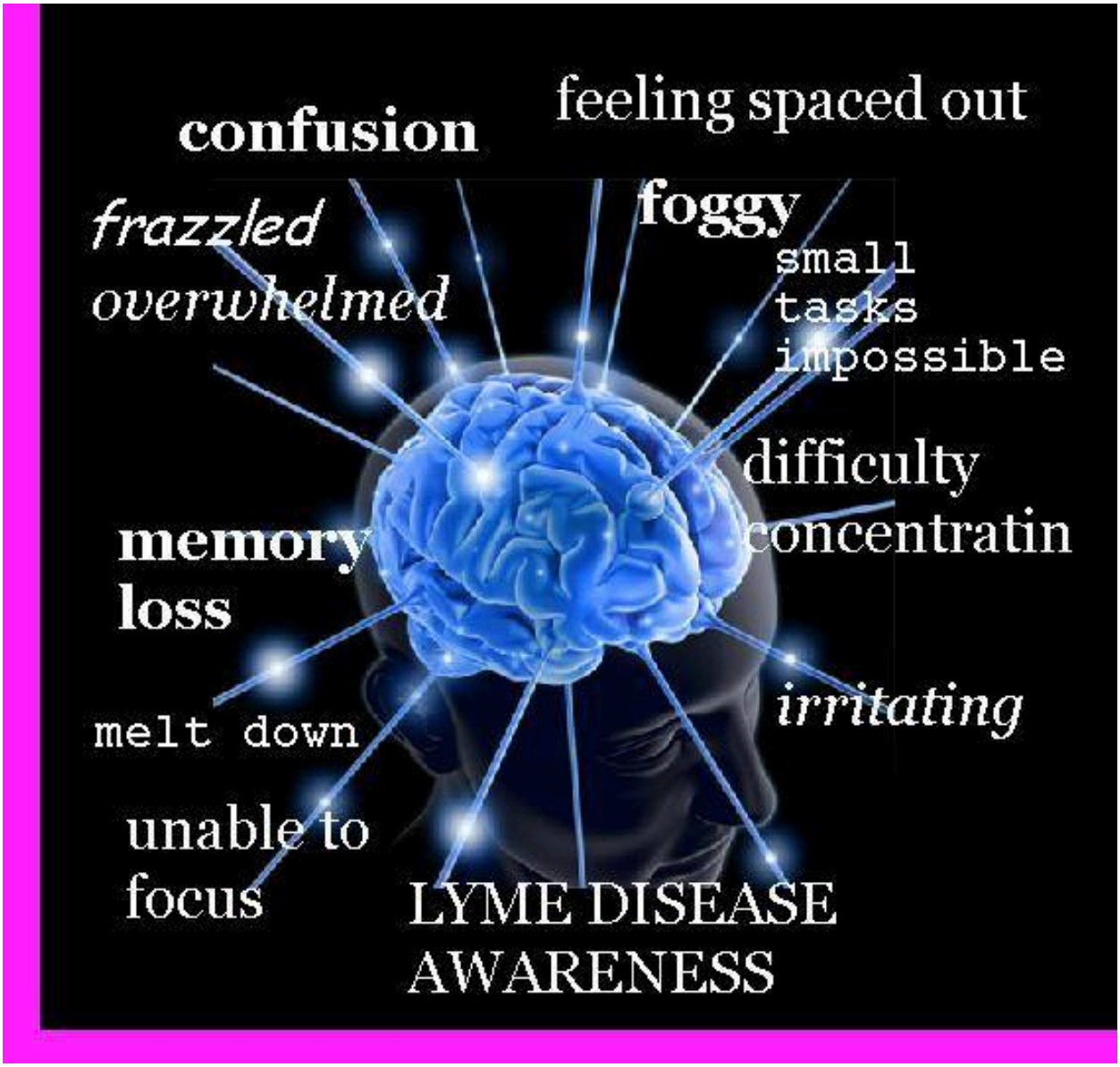
4. Lyme **DISGUISES** itself. Lyme pathogens can cover their surface with a component which is seen as "self" by the host immune system. Such a strategy hides the antigenic surface of the bacterial cell. Phagocytes cannot recognize it upon contact

and the possibility of the B cells creating antibodies to enhance phagocytosis is minimized.

5. Lyme bacteria may employ strategies to **avoid engulfment** (ingestion) if immune cells do make contact with them. This is not an uncommon strategy as many important pathogenic bacteria bear on their surfaces substances that inhibit phagocytic adsorption or engulfment. Classical examples of antiphagocytic substances on bacterial surfaces include polysaccharide capsules and biofilms with different proteins.

6. Lyme bacteria may possess the cunning ability to **meld their DNA** with symbiotic microbiota to completely morph its frequency and appear as a part of the host's microbiota.

Bottom line – Lyme stays alive through ingenious methods to replicate and make your life miserable.

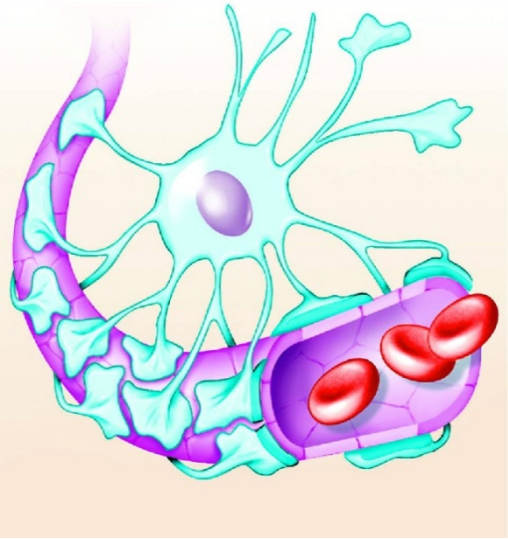


How Lyme Attacks the BRAIN

Understanding the Blood-Brain Barrier

Because some of the most damaging and debilitating symptoms of Lyme affect the nervous system, particularly the brain, it is important to understand how this happens. Note: see my book **Lyme Brain** for more detail on this subject.

The blood-brain-barrier (BBB) is a feature of our anatomy and physiology. There are specialized cells in our bodies that act as a blocking wall or filter, which prevents many substances from getting into our brains and spinal cord. The blood-brain-barrier makes it impossible, or at least very tough for medications to reach the brain (which is mostly a good thing—we don't want chemicals in our brains!). Throughout our bodies, we have capillaries (our smallest blood vessels), which have a lining of specialized cells (endothelial). These endothelial cells are tightly fitted together to form a filter, which protects the brain by preventing large molecules from passing through to it. Your blood-brain-barrier can be weakened by various illnesses, radiation, infection, and trauma. (4)



The BBB, which is formed by the endothelial cells that line cerebral micro vessels and specialized glial (brain) cells called astrocytes, have an important role in maintaining a precisely regulated microenvironment for reliable neuronal signaling in the brain. This means that when an organism like Lyme, infiltrates the brain by passing through a damaged blood-brain barrier, all sorts of bad things can happen. Most assuredly, the patient will have a local (in the brain) inflammatory process that, depending on the precise location, will alter normal

neuronal conduction and mimic gross lesions. Long-term inflammation is at cause of gross lesions that then are commonly diagnosed as the disorder displayed. (1)

For example, at present there are approximately 2.1 million people that have a diagnosis of Multiple Sclerosis in America. Symptoms of MS are unpredictable; vary from person to person, and from time to time in the same person. For example: One person may experience abnormal fatigue and episodes of numbness and tingling. Another could have loss of balance and muscle coordination making walking difficult. Still another could have slurred speech, tremors, stiffness, and bladder problems.

Sometimes major symptoms disappear completely, and the person regains lost functions. In severe MS, people have symptoms on a permanent basis including partial or complete paralysis, and difficulties with vision, cognition, speech, and elimination. (2)

Just like many of our “disease diagnoses”, a diagnosis of MS tells you NOTHING of the cause! Can Lyme be the cause of MS? Of course it can! Is Lyme ALWAYS the cause of MS? Of course NOT!

Furthermore, brain inflammation from Lyme is not dependent on the Lyme spirochete itself crossing the BBB. Inflammation elsewhere in the body is defined by the release of specialized chemicals called cytokines. There are specific cytokines that are highly inflammatory and are proven to cross the BBB.

Antibiotic drugs are typically too large to cross the BBB. And, if they do get through, it is thought that they cannot penetrate in large enough quantity to have the desire effect. This makes infections of the brain difficult to treat. Although weakening of the blood-brain-barrier may make it possible for some antibiotics to break through, it is highly questionable whether or not it is safe for them to get there!

If the patient is NOT Phase 3 (autoimmune), herbal remedies such as teasel, cat's claw, or samento, may be good for Lyme. While some people have found them symptomatically helpful, their molecules cannot cross the blood-brain-barrier

either and can leave the patient frustrated from their ongoing neurological symptoms.

NOTE: The blood-brain-barrier is NOT a factor when it comes to homeopathic whole-body, RIFE frequency healing and some other wellness-based approaches to healing.

Comprehensive homeopathy and RIFE are 'energy medicine', not chemical approaches. Many highly reputable medical sources concur that energy medicine is a big part of the future of health care. The truth is that it has already been widely available, but not accepted or pursued by the masses in traditional Western medicine.

If you have been on antibiotics for years, in an effort to recover from Lyme, perhaps you will want to consider this information wisely. If Lyme Borrelia bacteria, as well as Bartonella, Ehrlicia, and Babesia are living in your brain, can antibiotics likely kill them? This question does not even take into account the antibiotic resistant nature of many bacteria species.

1. *Nature Reviews Neuroscience **7**, 41-53 (January 2006) | doi:10.1038/nrn1824*
Astrocyte–endothelial interactions at the blood–brain barrier
N. Joan Abbot, Lars Rönnbäck & Elisabeth Hansson
2. *http://www.nationalmssociety.org/about-multiple-sclerosis/what-we-know-about-ms/faqs-about-ms/index.aspx#howmany*
3. ***Passage of Cytokines across the Blood-Brain Barrier***
Banks W.A..[a] · Kastin A.J.[a] · Broadwell R.D.[b]
4. ***THE CELL BIOLOGY OF THE BLOOD-BRAIN BARRIER***
Annual Review of Neuroscience, Vol. 22: 11-28 (Volume publication date March 1999)DOI: 10.1146/annurev.neuro.22.1.11 ***L. L. Rubin*** *Ontogeny, Inc., Cambridge, Massachusetts 02138-1118;*

H. pylori a Common Co-Infection

Your GUT is a barrier. When we eat food and pathogens that may accompany it hit the first barrier, the stomach, acid present to digest also prevents infection. Here, the pH is extremely acidic for two main reasons: it break down food to be absorbed and kills pathogens trying to hitch a ride. This second purpose of HCl (stomach acid) is of vital importance as it is the first line of defense against potential destroyers of other barriers like the blood-brain barrier.

Helicobacter pylori, for example, are ubiquitous bacteria that should be easily killed by a normal acid balance in the stomach. If you have an imbalanced HCl supply, H. pylori infiltrates and can either cause a stomach or duodenal ulcer or will pass through attacking other organs. It is estimated that 95% of H. pylori infections are chronic, insidious and subclinical – meaning it is rarely diagnosed as a disorder itself and usually the culprit of many other named diseases. H. pylori is such a common cause of vessel damage that leads to both heart disease and blood-brain barrier disruption that we must address it in a book about Lyme since one hypothesis states that one way Lyme survives is to bind itself (through the DNA) to H. pylori.

If Lyme Co-Hides with H. Pylori, How does it enter the BRAIN? It does so though the blood vessels. More specifically, through the cells that line the vessels called endothelial cells.

Under normal conditions, chemicals released by tissues knock on the endothelial cells door looking for permission to enter. For instance, a sympathetic nervous system response (fight or flight) in the brain to a perceived stress causes the release of a chemical that will enter a gate in the endothelial fence to cause the smooth muscle layer to contract and narrow the lumen of the vessel. This increases the speed of blood flow and increases the blood pressure so you can run away from the danger. It is a normal response, but like any normal response, we can get ‘stuck’ in an ‘on’ position from chronic source of stimulation.

There are really an endless number of possible insults that could 'breach the gates' of the endothelial wall – Lyme is just ONE of these. Chemical toxicity, heavy metal toxicity, food additives, flavorings, colorings, infections, and endotoxins are just a few of the other things that can break the gates and cause damage to the endothelial layer, the small, smooth muscles and tissue underneath, and interact with the astrocytes that act as the next (and special) barrier in the brain. You may have heard about the damage that Homocysteine, glucose, or oxidized LDL cause, but by far, the worst culprit for damage is infection.

Subclinical (silent) infections (like H. pylori and Lyme) are the number one 'bad guy' causing endothelial disease, which leads to blood-brain barrier disruption (among other things). "Subclinical" means the patient doesn't know they have it! It's a silent disorder that can cause mild, insidious vasculature damage for years (and yes, it can start at birth) until the victim has symptoms of ADHD, anxiety,

depression, memory loss and dementia, just to name a few. I know this is a lot of info so I'll sum this up:

1. BBB disruption really starts with damage to the endothelial layer – the single-celled barrier that lines the vessels.
2. If the endothelial layer is 'breached', several bad things occur that lead to inflammation in the vessel wall, the tiny muscles underneath the wall, and the astrocyte cells that are meant to keep larger molecules of things out of the CNS.
3. Many possible sources of endothelial damage exist due to poor diet, environmental exposure to toxins, and ubiquitous infectious organisms but subclinical infections (unknown to the patient) are the most common and least diagnosed cause of endothelial disease and hence, brain disorders.

Endothelial disease is always the start of BBB disruption and usually never addressed by the any doctor. Heck, most doctors don't even address the fact that there is a disruption in the BBB. Worse, many doctors are still blaming the patient's depression on a chemical imbalance as if it was a disease that poor victim contracted when they were caught out in the rain without a jacket. Medications can change a person's mood, they can numb symptoms, and dull hyperactivity. Medications cannot cure because they do nothing for 'cause'.

The Three Responses in the Endothelial Cells

There are three possible responses that occur when vascular endothelium is damaged by the infinite number of possible insults:

- a local inflammatory response,
- an oxidative stress response,
- and an autoimmune reaction.

All three possible may eventually occur and all include inflammation, which is the more damaging aspect of each response. Like every tissue, the endothelium maintains a fine balance between injury and repair. It's like a teeter-totter that tips gently back and forth; vessels are damaged by endless assaults and

then healed by a collection of innate physiologic responses that viewed as a whole, over time, we call health. If an individual has the unfortunate event of continual and prolonged damage, the repair can actually bring about problems that we shall soon see.

For fear that I've already bored you to tears, I'll just summarize my point here:

Over time, a ramped-up immune response involved in chronic Lyme does a multitude of things:

1. It destroys endothelial tissue (both receptors and entire cells) due to collateral damage in its attempts to kill the antigen, and

2. It starts to mistake self-tissue for the enemy and begins direct destruction of self-tissue, and

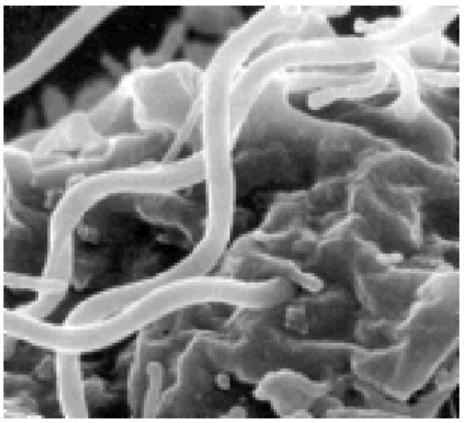
3. An immune response can destroy the astrocyte barrier further allowing antigens to enter the CNS and further the inflammatory spread INSIDE THE BRAIN!

REVIEW of the THREE PHASES of Lyme:

1. Phase 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. Phase 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 Nutraceuticals 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. Phase 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. When it cannot find the hiding Lyme, the B-cell (Th2) response fires to create antibodies to "tag" the bacteria in order to make them easier to recognize by the killer (Th1) cells. After months of teetering back and forth from a Th1-Th2 reaction, the B cells begin to create antibodies against your OWN cells. This is the very definition of an autoimmune disease and is exactly what takes place in Phase 3 Lyme.

Now that you have antibodies to self-tissue, every time you fire an immune response for ANY reason, or take immune stimulants to BOOST your immune system you are KILLING YOURSELF FIRST!

These patients are miserable and it is the autoimmune phase of Lyme that is deadly.

The question on EVERYONE's mind:

"How do I know what PHASE I am in?

See this link and take the quiz:

http://connersclinic.com/lyme-determining-your-phase/

Note: In my desire to help everyone regardless of his or her ability to pay for care, I've assembled a quiz (option 3) on the above page link to help you determine your phase.

Neurological Symptoms and CLD

Phase 3 Lyme patients, due to inflammation in the brain from either systemic inflammation or direct infiltration of spirochetes across the blood-brain barrier, may experience a number of neurological symptoms.

Initially, most people think of swollen and painful joints when they think of Lyme disease, if they think of anything at all. However, when you look at the symptom list below, you can see that every part of the body can be affected. The frightening collection of neurological symptoms experienced by many Lyme-disease patients is frequently called “neuro-lyme”, but in fact only represents a portion of the illness.

Do you have any of these symptoms?

Head, Face, Neck:

- Unexplained hair loss
- Headache, mild or severe
- Twitching of facial or other muscles
- Facial paralysis (Bell’s palsy)
- Tingling of nose, cheek, or face
- Stiff or painful neck, creaks and cracks
- Jaw pain or stiffness
- Sore throat

Eyes/ Vision:

- Double or blurry vision
- Increased floating spots

- Pain in eyes, or swelling around eyes
- Oversensitivity to light
- Flashing lights
- Ears/Hearing
- Decreased hearing in one or both ears
- Buzzing in ears
- Pain in ears, oversensitivity to sound
- Ringing in one or both ears

Digestive and Excretory Systems:

- Diarrhea
- Constipation
- Irritable bladder (trouble starting, stopping)
- Upset stomach (nausea or pain)

Musculoskeletal System:

- Any joint pain or swelling
- Stiffness of joints, back, neck
- Muscle pain or cramps

Respiratory and Circulatory Systems:

- Shortness of breath, cough
- Chest pain or rib soreness
- Night sweats or unexplained chills
- Heart palpitations or extra beats

- Heart blockage

Neurological System:

- Tremors or unexplained shaking
- Burning or stabbing sensations in the body
- Weakness or partial paralysis
- Pressure in the head
- Numbness in body, tingling, pinpricks
- Poor balance, dizziness, difficulty walking
- Increased motion sickness
- Lightheadedness, wooziness

Psychological Well-being:

- Mood swings, irritability
- Unusual depression
- Disorientation (getting or feeling lost)
- Feeling as if you are losing your mind
- Overemotional reactions, crying easily
- Too much sleep or insomnia
- Difficulty falling or staying asleep

Mental Capacity:

- Memory loss (short or long term)
- Confusion, difficulty in thinking
- Difficulty with concentration or reading

- Going to the wrong place
- Speech difficulty (slurred or slow)
- Stammering speech
- Forgetting how to perform simple tasks

Reproduction and Sexuality:

- Loss of sex drive
- Sexual dysfunction

Females only:

- Unexplained menstrual pain, irregularity
- Unexplained breast pain, discharge
- Pelvic pain

General Well Being:

- Unexplained weight gain or loss
- Extreme fatigue
- Swollen glands
- Unexplained fevers (high- or low-grade)
- Continual infections (sinus, kidney, eye, etc.)
- Symptoms seem to change, come and go
- Pain migrates (moves) to different body parts
- Early on, experienced a flu-like illness, after which you have not since felt well

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 Conners, 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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You will be receiving the next section in a few days!