

LYME BRAIN

WHAT TO DO WHEN
YOU'RE GOING CRAZY



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Lyme Brain

What to do when you're 'going crazy'

Section 1

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*If any of you lacks wisdom,
let him ask God, who gives
generously to all without
reproach, and it will be
given him.
-James 1:5*

This book is not going to explain everything you need to know about brain-based issues associated with Lyme disease but it is our hope, above everything else, to give you HOPE. There IS a reason you may feel like you're going crazy; there IS an explanation for your memory loss, your focus issues, brain fog, depression and anxiety. If you never should walk in our door, please find some door where a doctor seeks your best, desires to find the cause and is less apt to blame a label. Always know that God made you and He loves you! Feel free to contact us for help professionally or send us your prayer requests:

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“You are never too old to set another goal or to dream a new dream.”

- C. S. Lewis

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Chapter One

Understanding the LYME Stages



I don't like ticks. We live on 10 acres and have purposely cut our grass to the edge of the adjacent cornfield and keep our dogs out at night to keep deer away. Since having Lyme twice and seeing patients struggle with the consequences of late-stage Lyme, I choose to view my love for deer on a television screen. Many of you also have a love-hate relationship with nature.

Since the advent of Lyme disease several decades ago, countless people have suffered far worse symptoms than I. I was lucky enough to catch it in Phase 1 and successfully annihilate it with antibiotics. Those who were misdiagnosed in this early stage or who never had the vicious early symptoms were left to discover the culprit of their chronic disability much later.

Why is this? What actually happens in the body when bacteria tries to inhabit and multiply against a defense dedicated to protect? Isn't the body meant to kill such pathogens?

We humans have multiple defense mechanisms against invasive organisms. We've made allies with microbes as fetuses and allow certain bacteria to flourish in exchange for their symbiotic help in nutrient absorption as well as keeping 'bad guys' out.

Beginning immediately at birth, humans are colonized by a myriad of microorganisms that assemble into complex 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, 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.

Why is your Microbiota important in Lyme?

By being the primary stimulation of immune system function, a healthy flora helps create our epithelial boundary and integrity, as well as to “educate” our innate immune defenses. It also provides “colonization resistance” against pathogen invasion (keeps Lyme from growing), regulates intermediary metabolism, and processes ingested chemicals.

What are the distinguishing characteristics of microbes like Lyme that desire to make us their host? A successful pathogen or commensal must do the following:

- Enter the human host (through a tick, passed through saliva...);
- 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 be transmitted to a new susceptible host, though we seem to be Lyme’s final host.

Smart Bugs

It has long been but a hypothesis that Lyme pathogens 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 health of the human host plays as important a role as the pathogen in determining outcome.

An initial step required of Lyme bacteria is to gain access to the host in sufficient numbers. Such access requires that the microorganism not only enter the bloodstream but also then reach its *unique* niche or microenvironment on or within the host. To accomplish this goal, Lyme may make use of motility, chemotactic properties, and adhesive structures (or *adhesins*) that mediate binding to specific cell receptors or to other microorganisms (piggy-backing on other microbes).

Lyme pathogens that persist usually rely upon multiple adhesins and adherence mechanisms. Preexisting microorganisms (the host's existing microbiota) will hopefully 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 Lyme pathogens against 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 Lyme 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 are complicated and are just now being better understood but it

is safe to say that Lyme and its co-infections are some of the best enemy spies ever trained! More on this later.

The above dissertation into microbiology was simply to say that Lyme is difficult to kill. Its ultimate purpose is to survive and reproduce; your ultimate purpose is to keep it from doing so.

It is during the initial invasion of a pathogen that we have the best opportunity to destroy it. As soon as it begins to reproduce it begins to learn how to produce ingenious ways to dodge defenses. Like a well-coached team, the speed of the replication cycles of the bacteria enables it to make genetic changes to survive its environment. This initial phase, when the pathogen remains extracellular is what I have termed Phase 1.

As an undetermined amount of time goes by, the bacteria's ability to move intercellular is simply one disguise it is capable of. It can also meld itself to indigenous microbiota and exchange DNA to 'buddy-up' and bypass immune attack. Other times it may attract interleukin 10, a Th2 cytokine to its cell surface thereby repelling a Th1 (killer cell) attack.

Regardless of its mechanism, I define a Phase 2 pathogen as one that can no longer be killed by a normal immune response or by an antibiotic medication as it goes intracellular. Continued use of antibiotic at this stage will only be fruitful if continued long-term (3-6 years) and carries with it greater morbidity.

As the normal immune response attempts to kill the invading organism it shifts from a Th1, killer response to a B-cell (Th2) antibody response. This teeter-tottering back and forth is normal in an infectious disease as B-cells are looking for that which the killer cells have attempted to kill so they can make antibodies and 'tag' the bad guys. Over multiple, fruitless cycles where B-cells fail to locate hiding bacteria, the B-cells begin to create antibodies against self-cells in the vicinity of the invasion. THIS is the very definition of an autoimmune disease and what I term, Phase 3.

Summary of Lyme disease progression

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, i.e. once one moves into Phase 2, the ability to completely kill Lyme with antibiotic therapy is greatly reduced!

2. PHASE 2 = Chronic Lyme – Chronic Lyme, Phase 2 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 and, in the case of long-term antibiotic use, there will be considerable damage to the Gut and other cell membranes. Though it is better than Phase THREE, this phase is still horrible.

3. PHASE 3 = Autoimmune Lyme - When the patient's condition continues to linger, the immune system is constantly trying to kill it – this is normal. However, in doing so, the “killer” side of the immune system, the Th1 response, fires to kill the pathogen and is unable to enter the cell to destroy those bacteria that have entered. This will eventually calm the Th1 response and set-off a B-cell (Th2) immune response in an attempt to find the bacteria (the antigen) and make antibodies against the bug, thereby “tagging” the bacteria (with the antibody) allowing quick detection and destruction.

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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