

Holly Ahern, MS, MT(ASCP), Associate Professor of Microbiology, SUNY Adirondack  
ORAL TESTIMONY TO THE NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEES

Senator Hannon, Senator Serino, and members of the Senate Lyme and TBD Task Force and Health Committee

I am here today because my beautiful, smart, and athletically talented daughter was once bitten by a tick.

The tick was an adult-stage deer tick. It was attached to her for less than 12 hours. There was no typical bull's-eye rash, or any type of rash, or any other signs of illness. According to the prevailing medical wisdom, since the tick wasn't a nymph-stage, and it wasn't attached for more than 36 hours, she was at low risk of disease. So the tick bite went untreated.

We now know that the tick infected her with two remarkably insidious bacteria named *Borrelia* and *Bartonella*, which stopped her body from mounting an immune response, stealthily moved out of her blood, set up residence in other parts of her body, and gradually triggered inflammation.

Months passed. She came home from college one weekend complaining of aches and pains, which escalated to searing headaches, a heart arrhythmia, feeling like she had electricity running up and down her arms and legs and that her muscles were on fire, and overwhelming fatigue. She went to bed and didn't get up to return to her normal life for almost a year. In between, we traveled a long road from doctor to doctor, trying to get an accurate diagnosis, find an effective treatment, all the while fighting with our insurance company to cover any of it. It was a nightmare NO ONE should have to experience.

But every year, hundreds of thousands of people in the US develop Lyme disease and experience this same nightmare. According to CDC statistics from 2015 ([cdc.gov](http://cdc.gov)), nearly 400,000 NEW CASES of Lyme disease occur every year. New cases occur so frequently that Lyme disease is now the SECOND MOST COMMON infectious disease in the United States (Chlamydia is #1, Lyme disease is #2, and Gonorrhea is #3). These statistics alone should be enough to trigger a public health challenge to the outdated model of Lyme disease as being "hard to catch."

A significant proportion of people infected with the bacteria that cause Lyme disease go on to suffer from debilitating arthritic, cardiac and neurological symptoms that may last months to years and profoundly impair their quality of life. Eight years after her ordeal began, my daughter has regained much of what she lost, because I was able to put my knowledge and experience as a scientist to good use and be an advocate for her. But most people don't have microbiologist mothers to help them, and the medical community is still deeply conflicted over what to do about people like her, who don't fit the model deemed appropriate for this disease.

And as a microbiologist and her mom, I have to live with the knowledge that she may have recurring health challenges as a result of that tick bite for the rest of her life.

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In New York State, the “official” public health position on Lyme disease is that it is a “high incidence, low impact” disease. Calling this disease “low impact” is demeaning and marginalizing to the majority of Lyme disease patients who, like my daughter, have had their lives completely upended by this illness.

The tactic of “gaslighting” Lyme disease patients, to make them question their reality and doubt the existence of their crippling symptoms, has been employed by the medical and public health community since the disease was first conceived in 1977. In my daughter’s case, we were told by one medical provider that she was, after all, a “girl” and that her symptoms came on because she couldn’t handle the pressures of college. It didn’t matter that she was a low maintenance kid, getting A’s in her classes, had just achieved All-American in swimming, and was generally loving her college experience -- right up until that fateful day when she went to bed and couldn’t get back up.

The prevailing medical view of Lyme disease flows from a relatively small number of clinical studies, which are notable because (1) they share the same authors and (2) they share the same basic design flaws. Early studies were plagued by confirmation bias -- which is the tendency to interpret new evidence as confirmation of one's existing beliefs or theories. Subsequent studies are marred by anchor bias—which is the process of relying too heavily on early studies that are often later proved to be flawed or incorrect.

Many errors have been compounded because every research study that investigated the clinical course of Lyme disease in humans since the late 1970s, used the appearance of a bull’s-eye rash as the main criteria for either inclusion in the study, or as an end point of the study.

Why is this a problem? A single 1984 study anchored the belief that a bull’s-eye rash occurs in 70-80% of Lyme disease cases. Yet there are a dozen or more scientific, peer-reviewed studies which REPEATEDLY and REPRODUCIBLY show that the actual rate of the bulls-eye rash in Lyme disease is only 10-40%.

In other words, the existing medical understanding of Lyme disease is based on clinical research studies that EXCLUDES 60-90% of people who actually have the disease. It’s hard to imagine that it would be acceptable to exclude from research studies 60-90% of those who had HIV, diabetes, cancer or ANY ILLNESS OTHER than Lyme disease.

It is safe to say that NEXT TO NOTHING is known about how to diagnose and appropriately treat people who, like my daughter, never got a rash and simply just got sick after being bitten by a tick.

If her blood test for Lyme disease had not been barely positive, it is likely that my daughter’s health would have gotten considerably worse. As difficult as it was to identify effective treatments, at least we knew our enemy – two tick-borne bacteria, named *Borrelia* and

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ORAL TESTIMONY TO THE NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEES

*Bartonella*, which are not like other bacteria and don't respond as expected (meaning, they don't die) with standard antibiotic treatment.

The inaccuracy of existing diagnostic tests is a significant roadblock to treatment for many Lyme disease patients. A thirty year-old test called "two-tier serology" is considered the "gold-standard" of diagnostic tests for Lyme disease, despite scientific evidence that REPEATEDLY AND REPRODUCIBLY shows this test to have a rate of FALSE NEGATIVES greater than 50%.

Using the "gold standard" diagnostic test, 50% of those who actually have Lyme disease, are dismissed as NOT having Lyme disease. Those without a diagnosis go without treatment, and this is a major reason why many develop severe, persistent illness.

Would a test with a 50% failure rate be tolerated for any other infectious disease like HIV or Hepatitis C? This lack of reliability has led a number of state legislatures to pass laws requiring that patients be informed by their medical providers about the inaccuracy of this test. Similar legislation should be considered in this state as well, to protect New York residents from the consequences of falsely negative blood tests.

While acknowledging that cases of Lyme disease are "under-reported," New York currently performs statewide Lyme disease surveillance by taking a 20% random sample of positive serological test reports from across the state. Therefore, surveillance data on Lyme disease, which is used to allocate public health resources in New York State, is based on the results of a diagnostic test that is wrong over half the time.

New York is home to arguably the best public health laboratory in the world. So why isn't Wadsworth working to develop a better diagnostic test for Lyme disease, as they have for other vector-borne diseases like West Nile virus and more recently, Zika virus?

Frustration with the lack of progress on practically every aspect of Lyme disease science and medicine, patients, advocacy organizations like ours (the Lyme Action Network), and private philanthropic organizations such as the Focus on Lyme Foundation, are pulling together to fund new research and break through the Lyme disease status quo on diagnostic testing and treatments.

Thanks to funding provided by the New York State Senate Lyme and Tick-borne Disease Task Force and Focus on Lyme, we are filling a research void by building a biorepository of human blood samples. The blood will first be characterized by routine laboratory testing for evidence of tick-borne infections, and then used to advance development of a molecular detection assay called LymeSeq, which will directly identify DNA and RNA from multiple species of *Borrelia* and other tick-borne microbes.

Diagnostic tests such as LymeSeq will allow physicians to determine the INFECTION STATUS of their patients, to not only increase the probability of an accurate diagnosis, but to guide the

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ORAL TESTIMONY TO THE NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEES

course of treatment as well. LymeSeq is currently in the analytical validity stage of development and will soon be ready for clinical validation studies, starting this fall using samples from the Focus on Lyme biorepository.

In the future, the biorepository will be available to other researchers to advance the state of the science for all tick-borne diseases. Along the way, we hope to add significantly to the body of knowledge about the rate of tick-borne co-infections in humans as well.

This is truly a patient-powered project. Participation has been overwhelming, with some people travelling from other states and Canada to participate. I believe this speaks volumes about the level of frustration and how marginalized and gaslighted the Lyme community feels, and how strong the need for change.

The World Health Organization states that "everyone has the RIGHT to the highest attainable standard of health," which includes access to medical care. The majority of Lyme disease patients are DENIED this basic right, simply because the illness they experience does not fit within a set of incomplete, conflicted, and contested medical "rules."

Lyme disease is MUCH MORE than a bull's-eye rash and rapidly resolving symptoms. It can be a long term, life altering or life ending illness. Lyme disease patients and the brave physicians that put themselves on the line to treat them, deserve to have their narrative heard.

In the interest of time, I have submitted as written testimony a review of the scientific underpinnings of the current medical guidelines for Lyme disease in humans, coauthored with a bioethicist. I ask that you take the time to read this review as a supplement to my oral testimony today.

Thank you for hearing my testimony and for your leadership on this issue.

## A Critical Reappraisal of the Science and Bioethics of Lyme Disease Medicine

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The Centers for Disease Control and Prevention (CDC) holds as its mission the protection of U.S. citizens from disease regardless of whether the disease threat is “chronic or acute, curable or preventable, human error or deliberate attack.” The CDC includes in its “Pledge to the American People” that all public health decisions will be based on the highest quality scientific data that is derived openly and objectively; to place the benefits to society above the benefits to the institution; and to treat all persons with dignity, honesty, and respect (1).

However, in the case of Lyme disease, the CDC is not fulfilling its mission or its pledge to protect the American public from a disease this governmental agency readily acknowledges it has been underestimating for years (2).

Based on epidemiological reports of notifiable diseases maintained by the CDC, Lyme disease is overall one of the top three notifiable infectious diseases in the United States. Although commonly referred to as the leading vector-borne disease in the United States, a review of CDC surveillance data on all infectious diseases on the “Nationally Notifiable” list of diseases, shows that Lyme disease is second only to Chlamydia (a sexually transmitted disease) in terms of overall number of new cases annually.

The public health response to this high incidence infectious disease is not commensurate with the significant disease burden experienced by people who acquire the infection, nor to a society which must support those left sick and disabled by Lyme disease symptoms. This disease burden stems directly from fundamental misconceptions about the nature of the bacteria, the biology of the infection they cause, and the symptoms patients experience as a result.

### Seminal research studies created and perpetuated Lyme disease misconceptions

In 1977, Steere et al. (3) published the first description of an “epidemic” of arthritis occurring in patients clustered in the vicinity of Lyme, Connecticut. By 1982, Wilhelm Burgdorfer, a research scientist employed by the Epidemiology Branch of the National Institute of Allergy and Infectious Diseases (NIAID), had identified a spiral-shaped bacterium in the genus *Borrelia* as the agent responsible for the disease symptom (4). The bacterium was named for him shortly thereafter, and *Borrelia burgdorferi* was firmly established as “the cause” of Lyme disease.

In epidemiology research, it is common practice to seek a unique characteristic (a clinical “sign”) that can be used to definitively distinguish one condition from all others. In his search for the perfect clinical “sign,” Steere noted that a small proportion (25%) of his original group of patients had developed a very unusual rash resembling a target or “bulls-eye.” Steere focused on this rash, medically referred to as an

WRITTEN TESTIMONY TO NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEE

"erythema migrans" or "EM" rash, because he presumed it to be the tell-tale sign he was seeking. His errant presumption has influenced scientific research related to tick-borne disease ever since.

All subsequent studies on the clinical aspects of Lyme disease in human subjects included a recruitment process heavily skewed toward the population of patients who developed an EM rash as a sign of "early" infection with *Borrelia burgdorferi*. While we now know that only a minority of patients with Lyme disease develop the EM rash, this fundamental study design flaw introduced a strong and pervasive bias into later studies on the clinical manifestations of Lyme disease. This is readily evidenced in a second paper published in 1977, which focused on patients with the EM rash and arthritis with joint swelling as the primary indicators of infection. Not surprisingly, by the time the second study was published, the purported incidence of the EM rash in Lyme disease patients had been increased to 70% (5).

In the earliest epidemiological investigations of Lyme disease in New York (6) and Minnesota (7), the EM rash was used as the primary defining criteria for a "case" of Lyme disease. To assess the risk and burden of Lyme disease in these states, physicians were "urged" to report patients presenting to them with a "case" of Lyme disease, which for the purpose of both studies was defined as a person with Lyme disease symptoms who had the "presence of erythema chronica migrans (EM) rash," or secondarily, systemic symptoms of meningitis, facial palsy, or large joint arthritis, appearing during the months of May, June, or July (6, 7).

By using this study design, investigators biased the data toward those persons who presented to physicians with an EM rash during the summer months. Consequently the data appeared to show a relative increase (to nearly 80%) in the proportion of Lyme disease cases associated with an EM rash. These investigations also gave rise to another misconception, which is that new Lyme disease cases occur in only a few regions of the United States (particularly the northeast), during the months of May, June and July. These months coincide with the feeding activity of the second life stage (called a nymph) of *Ixodes scapularis* ticks.

The enzootic cycle of *Borrelia burgdorferi* requires an infected host animal to serve as a "reservoir" that maintains the bacteria in nature, and a tick to serve as the "vector" that transmits the bacteria from the reservoir to other animals, including humans. Ticks have three life stages (larvae, nymph, and adult), each requiring a blood meal to fuel transition to the next stage. Entomological research indicates that nymph ticks feeding on an infected reservoir host (such as mice and migratory birds) is central to perpetuating the enzootic cycle (8).

Although all three stages of *Ixodes* spp. can feed on humans, past and present entomological and ecological studies on Lyme disease transmission have focused almost exclusively on the feeding activities of the nymph stage tick on mice. To date, there have been no studies done to determine which tick life stage is most involved in transmission of *Borrelia* or any other tick-borne pathogen to humans. According to disease ecologists, this is because humans are considered "dead end" hosts in the enzootic cycle of the disease-causing agent.

The overreliance on the EM rash as a clinical sign and the presumption that nymph ticks are the primary vector in human cases of Lyme disease has biased scientific thought on this topic for many years. For example, in an investigation of the efficacy of a single dose of doxycycline administered shortly after a tick bite as a preventative treatment for Lyme disease, the “primary end point” for defining a case of Lyme disease “was the development of erythema migrans at the site of the tick bite” (9).

This study, published in the influential *New England Journal of Medicine* in 2001, is notable for two reasons. Although the authors concluded that a “single 200 mg dose of doxycycline administered within 72 hours” of a tick bite prevented Lyme disease, the study design excluded persons who did not develop an EM rash as evidence of Lyme disease. Therefore, the data shows only that treatment prevented the development of an EM rash. Additionally, the entomological data presented in the paper showed that an EM rash appeared at the tick bite site only when the person had been bitten by the nymph stage tick. Therefore only patients bitten by nymph ticks met the inclusion criteria for the secondary determination of whether the prophylactic treatment was a success.

A more probable scenario that can be derived from the data is that persons bitten by an adult tick do not develop an EM rash. These people were excluded from the rest of the study, but may have still developed Lyme disease. An alternative hypothesis that can be derived from the data is there may be as yet unexplored differences between the nymph and adult tick life stages in terms of the mechanics of the biting process and possibly the range of pathogens and/or interactions among pathogens carried by each. The role of adult ticks in the transmission cycle of Lyme disease in humans is therefore still unknown.

To date, this study has never been repeated, and all other studies on tick bites and disease transmission have involved animal models and nymph stage ticks. Although completed over 15 years ago, there has been no followup assessment of the efficacy of administering a single dose of doxycycline at the time of a tick bite to prevent the development of other Lyme disease symptoms, or if this approach would work to prevent Lyme disease if a person is bitten by an adult stage tick. Because it was published in an influential journal, however, this single study is the only “evidence” that can be cited to support the recommendation to physicians that treating a patient with a tick bite with a single prophylactic dose of doxycycline will prevent them from developing Lyme disease.

Other published clinical studies have provided conclusive evidence that in clinical practice, the EM rash is present in only a minority of Lyme disease cases (10), and that the EM is only weakly associated with any Lyme disease symptom other than arthritis. Despite this, the EM rash continues to be used as a diagnostic standard and a treatment end point, with the CDC standing behind its oft-repeated public health recommendation that an EM rash will be the primary sign of Lyme disease 60-80% of the time.

**Little scientific evidence to support “Post-treatment Lyme disease syndrome”**

August 29, 2017

WRITTEN TESTIMONY TO NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEE

Since their discovery and development as drugs in the 1950s, antibiotics have been thought of as a “magic bullet” capable of curing infectious diseases caused by bacteria. Antibiotics had been used so successfully against bacterial diseases that Dr. William H. Stewart, the U.S. Surgeon General from 1965–1969, has been quoted as having said, “It is time to close the book on infectious diseases, and declare the war against pestilence won.” In 1982, this was still a strongly held belief.

Once a bacterium was determined to be the cause of the epidemic of “Lyme arthritis” in the early 1980s, the next step was to determine which antibiotics should be used for treatment. The first investigation of the efficacy of antibiotic treatments for Lyme disease was published in 1983 (11).

The data presented in this seminal study indicated that nearly 50% of antibiotic treated patients continued to experience debilitating symptoms post-treatment. However, the study authors surprisingly concluded that for patients with “early” Lyme disease, 10 – 14 days of tetracycline was an effective treatment. After reviewing the data, it should be asked how the researchers were able to reconcile data showing a high rate of treatment failure with their conclusion that 10 – 14 days of an antibiotic was an effective treatment for Lyme disease.

At the time this study was conducted, infectious diseases caused by bacteria were no longer considered to be a significant threat to human health. While that simplistic view has been shown over the past two decades to be false and short-sighted, in 1984 research that appeared to show an antibiotic failing to effectively treat a bacterial infection deeply conflicted with prevailing medical dogma.

Because the research findings did not support the precept that antibiotics never fail, the data was rearranged in such a way to create the artificial impression that the vast majority of Lyme disease patients recovered fully after completing a standard antibiotic regimen of 10-14 days.

Specifically, the data on patient outcomes was broken down into two groups – patients who experienced “Major” symptoms after the antibiotic treatment vs. those with “Minor” symptoms. “Major” symptoms were defined as those a physician would be trained to perceive as clinical “signs” of Lyme disease, including a recurrence of the EM rash and/or severe and potentially life threatening meningitis, carditis, or arthritis with noticeable swelling of the joint. Patients who experienced post-treatment “Major” symptoms were considered treatment failures. Few people in any of the treatment groups developed these “Major” conditions.

“Minor” symptoms were defined as those a physician would be trained to perceive as disease “symptoms” as opposed to a “sign.” In medical practice, symptoms are considered more “subjective” because they were based on a patient’s description of their personal experience. In the “Minor” symptoms group, patients showed symptoms of arthritis without apparent joint swelling, tachycardia, cranial nerve palsy, peripheral neuropathy, severe fatigue, headaches, and changes in mental function. While these symptoms greatly impaired the patients’ quality of life, they were interpreted as a treatment success, not antibiotic treatment failures.



A critical analysis of the data presented in this paper clearly shows that nearly half of the patients enrolled in this study were left with post-treatment symptoms. The study authors justified their conclusion that 10-14 days of an oral antibiotic was an effective treatment for Lyme disease by discounting the symptoms they quite subjectively and arbitrarily determined to be "Minor," with no regard for the degree to which these symptoms impaired the patient's quality of life.

Other studies have yielded similar results. In one such study titled "Failure of Tetracycline Therapy in Early Lyme Disease," 100% of tetracycline-treated Lyme disease patients were left with the same painful and debilitating post-treatment symptoms deemed to be "Minor" (but not treatment failures) by Steere, et al. in 1983 (12). The authors of this study concluded that more research was needed to assess the true efficacy of the standard antibiotic treatment paradigm on Lyme disease patients. Yet, the CDC continues to support the recommendation that Lyme disease at any stage in the infection is curable by a short treatment with doxycycline.

An unfortunate but entirely avoidable outcome of the bias introduced and perpetuated in those early studies is that Lyme disease patients who continue to suffer debilitating joint pain, peripheral neuropathy, severe fatigue, tachycardia, and other symptoms after the recommended 10 – 14 days of antibiotic treatment, are now relegated to a nebulous medical state called "Post-treatment Lyme disease syndrome" (PTLDS), or sometimes just, "Medically Unexplained Symptoms." This moniker establishes the baseless implication that the original infection was successfully treated, and that symptoms are due to some other cause. As there is no diagnostic code among the International Classification of Diseases (ICD) medical coding system for this particular medical state, patients seeking medical help for their ongoing symptoms often must do so without insurance reimbursement.

Current competing hypotheses to explain so-called PTLDS include: 1.) continuing infection by bacteria that survive antibiotic treatment and precipitate chronic inflammation; 2.) the presence of remnants (including DNA) of dead bacteria in tissues precipitating chronic inflammation; 3.) an autoimmune type of reaction; or 4.) the overactive imaginations of people with nothing better to do than complain to their doctors about the pain of their daily lives (13).

The preponderance of the scientific evidence strongly points to persisting infection by antibiotic tolerant forms of several different *Borrelia* genospecies, along with comorbid infections caused by other tick-borne microorganisms, as the underlying cause of the chronic disease symptoms seen in both untreated and treated Lyme disease patients. Past and present research on the biology of *Borrelia* provides considerable insight into how these bacteria are able to cause chronic disease in humans.

#### **The remarkable biology of *Borrelia***

Although the CDC insists the proportion of Lyme disease patients who have continuing disease after short term antibiotic treatment is in the range of 10-20%, the scientific evidence, including that which

WRITTEN TESTIMONY TO NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEE

was presented in the earliest published studies, indicate this number to be significantly higher (30-50%). That many Lyme disease patients experience new onset or recurring symptoms after treatment is explainable within the context of the natural biology of infection by *Borrelia* and other tick-borne pathogens.

*Borrelia* are a type of bacterium called a spirochete, based on their appearance as a slender, twisted rod when observed under the microscope. Spirochetes are known to have a drill-like motility enabling easy movement through viscous solutions and penetration through collagen-rich tissues.

The biology of *Borrelia* is vastly different from other bacteria. They have an exceptionally complex genome. Because they evolved to be totally dependent on a host animal, *Borrelia* lack genes for many metabolic traits common in their free-living counterparts. Their reproductive strategies do not include rapid growth to large numbers, followed by a release of toxins to facilitate quick dispersal to new hosts. As such, they also lack genes for the known classic bacterial virulence factors, such as exotoxins and endotoxins (14). The pathogenicity of *Borrelia* is associated with slow growth and periods of no growth, which is more akin to the pathogenic approach taken by *Mycobacterium tuberculosis*, the cause of tuberculosis and a bacterium which is known to persistently infect as much as one-third of the world's population, according to the World Health Organization.

The *Borrelia* genome evolves rapidly, with substantial genetic variation even within a single generation. There are hundreds of different genospecies of *Borrelia*, each known to prefer different host tissues (such as skin or joints, cardiac or nerve tissue), which may help explain the broad range of clinical symptoms observed in Lyme disease patients (14). For example, genospecies of *Borrelia* that localize to the skin (and produce an EM rash) may also localize to joint tissue due to the collagen-rich nature of these tissues, causing symptoms of arthritis. Other genospecies localize to regions of connective tissue associated with the membranous linings of the heart or nervous systems, leading to carditis or neuropathology.

In nature, *Borrelia* is at home as a "commensal" living in relative peace within small mammal hosts such as mice. *Borrelia* infection is permanent in their natural hosts, and although mice develop antibodies against the bacteria, the infection generally does not lead to disease. Ticks pick up the bacteria when they feed on infected mice or birds, and then transfer the bacteria to humans when they take their next blood meal (15).

Ecological relationship between the bacteria, ticks, and host are important considerations in Lyme disease for several reasons. To establish a permanent infection, the bacteria must communicate with their host to suppress the parts of the immune response that would lead to their destruction. In their natural host, this communication is largely successful and the bacteria are permitted to establish permanent residence and tap into host resources to survive (16).

WRITTEN TESTIMONY TO NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEE

Humans are not a natural host for *Borrelia* or other tick-borne microbes. As longer-lived animals, bacterial infections trigger a more complex immune response and attempts to establish a commensal relationship are not entirely successful. As a result, presence of the bacteria in a human host triggers inflammation and other “innate” responses (16). Baumgarth and others have provided conclusive evidence that *Borrelia* have the ability to disable the switch from the innate to the more specific “adaptive” immune responses, which includes the production of antibodies capable of sterilizing the infection (17, 18). The net result is a type of “frustrated commensalism” between *Borrelia* and a human host, one in which long term infection is established but accompanied by a waxing and waning state of immune system activation (16).

The inherent ability of *Borrelia* to suppress the production of antibodies in humans is highly significant. In the U.S., the most widely used blood tests for Lyme disease diagnosis are the ELISA and the Western blot. These are assays that detect antibodies produced by a human host to neutralize specific proteins (called antigens) located on the surface of *Borrelia burgdorferi* spirochetes. The inherent design flaw in a test that relies on an infected human producing sufficient quantities of specific antibodies, when the bacteria suppress antibody production, is obvious.

The specificity of the interaction between the antigens used in development of Lyme disease blood tests and the antibodies detected in those tests, presents an additional problem. Different genotypes of *Borrelia* capable of causing disease symptoms in humans do not all have the same surface antigens as *Borrelia burgdorferi*. Even within the same generation, individual cells of *Borrelia* have the genetic ability to vary expression of surface antigens to further thwart antibody production (19). These biological characteristics provide an additional reason why Lyme disease tests based on detecting high levels of specific antibodies against *Borrelia burgdorferi* in a person’s blood are simply inadequate as a diagnostic tool.

One additional factor is the biological ability of *Borrelia* to take different cellular forms. Best known and commonly portrayed in the spirochete form, *Borrelia* also develop rounded forms called “round bodies” or RBs, which have been observed in studies dating back several decades. Recently it has been shown that the surfaces of the spirochete and RB forms of *Borrelia* are different (20). The existing diagnostic tests for Lyme disease find antibodies in blood produced against cell surface antigens found on the spirochete form of the cell, only.

The remarkable biology of *Borrelia* contributes greatly to the inadequacy of antibody-based tests as a diagnostic tool for Lyme disease. The experimentally determined sensitivity (probability of detecting a disease) of the ELISA test, applied as a “screening” or “first tier” test that must be positive before the more specific Western blot is even done, is less than 50% (21). Due to test unreliability, Virginia (Va. Code Ann. § 54.1-2963.2) and Maryland (Md. Code Ann., Health Law § 20-1701) have passed laws stipulating that physicians must inform their patients that a negative result on a blood test does not mean they do not have Lyme disease.

WRITTEN TESTIMONY TO NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEE

As previously mentioned, *Borrelia* responds to adverse environmental changes (such as when exposed to antibiotics) by changing from motile spirochete form into RB forms. RBs are dormant with little or no metabolic activity, such as protein synthesis or DNA replication. Antibiotics work by disrupting cell metabolism, and therefore the dormant RBs are antibiotic tolerant “persister” cells. As the name implies, RBs persist in their dormant state but are capable of reactivation when conditions improve, leading to resurgence in bacterial numbers and disease symptoms (22).

Notably, a significant recent research finding shows that antibiotic tolerance due to *Borrelia* persists is actually induced by exposure to the antibiotics routinely used as front-line treatment approaches for Lyme disease (23).

An additional biological factor that contributes to antibiotic tolerance in *Borrelia* is biofilm growth. Biofilms are sessile communities of bacteria, and biofilm-based bacterial cells are biologically different from the “planktonic” (motile and metabolically active) forms.

Biofilms of *Borrelia* have been directly observed in studies conducted on culture-grown bacteria, and now also have been shown to form in human tissues (24). *Borrelia* is known to preferentially localize to collagen containing tissues – skin, the joints, the linings of the heart, and the membranes of both the central nervous system (from where they sometimes spill into the highly protected interior and cause meningitis) and peripheral nervous system. Biofilms of *Borrelia* in these protected tissue sites may serve as a source of the bacteria detected during resurgence events.

Persistent infection by *Borrelia*, relying on biofilm growth and persistent round body forms, is the rule in nature and not the exception for all of the genospecies of this bacteria discovered to date. This has been repeatedly, and conclusively, shown in numerous studies conducted on animal models (mice, hamsters, dogs, and non-human primates) and recently, in humans (25).

Combined, these research results provide an explanation for why a few weeks of antibiotics does not always result in complete resolution of disease symptoms, or prevent a recurrence of disease symptoms weeks, months, or years after the initial infection occurred.

The sizeable body of research on the microbiology of *Borrelia*, the disease as it occurs in animal models, (particularly non-human primates which are excellent models of human disease), and the variability of the clinical disease manifestations in humans reveals the false dichotomy exhibited by defenders of the current Lyme disease status quo who proclaim there is “No Evidence” in support of the hypothesis that 10-14 days of an antibiotic is not a curative treatment. This false and circular argument is an “appeal to ignorance” that attempts to shift the burden of proof in the contentious debate over whether differing treatment approaches (longer therapy, pulsed dosing, different antimicrobial agents) would provide better patient outcomes in Lyme disease.

August 29, 2017

WRITTEN TESTIMONY TO NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEE

There is abundant scientific evidence supporting the hypothesis that persisting infection by antibiotic tolerant forms of *Borrelia*, and/or comorbid tick-borne infections may be the underlying cause of the chronic inflammation that precipitates persisting, recurring, or post-treatment Lyme disease symptoms. A lack of research, or over-reliance on evidence derived from poorly designed studies that have been interpreted with extreme bias, are not the same as “No Evidence.”

**The medical construct of Lyme disease must be revised to match the science**

The current medical construct of Lyme disease, rooted in the earliest published studies from 1977, describes a disease that is entirely inconsistent with the disease as it is experienced by patients. Clinically, the current medical construct for Lyme disease describes an infectious disease caused by one single genospecies of a specific bacterium (*Borrelia burgdorferi*), in which the initial infection leaves behind a tell-tale EM rash as a clear objective sign, for which there is a reliable, antibody-based blood test. Lyme disease is additionally considered to be easily and fully treatable with routine doses of antibiotics.

A complete and unbiased review of the scientific literature shows clearly that the above Lyme disease construct is only one manifestation of a complex, systemic disease. In actuality, the disease rarely begins with an EM rash, and nearly 50% of the time evolves into a disabling chronic disease with a myriad of disabling symptoms. The current “gold standard” blood tests for Lyme disease are only effective when high levels of specific antibodies are produced, but *Borrelia* employ immune evasive strategies to skew or suppress antibody production. In addition, conclusive scientific evidence shows that *B. burgdorferi* is only one of many disease-causing genospecies of *Borrelia*, that *Borrelia* cells can exist in nearly “invisible” forms, and that Lyme disease may involve other comorbid infections, none of which are directly detected by the existing diagnostic blood tests for Lyme disease (26).

While the patient narrative for Lyme disease matches the existing medical construct about half the time, the other 50% of patients describe a completely different disease process. With nearly 400,000 new cases of Lyme disease diagnosed each year, the number of people in the U.S. with long term debilitating symptoms and chronic disease precipitated by a tick-borne infection can be estimated at over 150,000 people per year. Compounded over many years, there are millions of people in the U.S. with chronic disease stemming from diagnosed and undiagnosed Lyme disease. Studies show that people with chronic Lyme disease symptoms have significantly lower life functioning ability (27), and this comes at considerable cost to the U.S. healthcare system (28).

Existing and emerging research shows that in nature, most genospecies of *Borrelia* cause persistent infections in animals, including humans. This same body of science also strongly implicates coinfections with other tick-borne microbes as the cause of many of the problems associated with Lyme disease diagnosis and treatment (29, 30).

WRITTEN TESTIMONY TO NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEE

From the meager scientific data accumulated from studies done with human subjects treated with antibiotics, the only conclusion that can be legitimately reached is that short-term antibiotic treatment makes an EM skin rash subside. There is currently no existing published research studies done with human subjects that addresses the efficacy of antibiotic therapy on any other manifestation of Lyme disease, particularly those in which patient complaints are not considered "signs" in the medical sense. The so-called "subjective" symptom – heart issues, joint and muscle pain, overwhelming fatigue, and cognitive disruptions -- significantly impair the quality of life for Lyme disease patients. More research on treatment approaches for all forms of the disease are desperately needed.

Lack of leadership on the part of the NIH and CDC has left a void in the knowledge needed to successfully lessen the disease burden on millions of past, present and future Lyme disease victims. Because of a recent influx of funding from private philanthropic individuals and groups, microbiological and clinical research that should have taken place 30 years ago is currently underway at several leading research universities across the country. There is hope that this research will lead the way to a better understanding of the microbiology and pathology of Lyme disease, along with a broader recognition of the health burden Lyme disease places on both individuals and society. With this knowledge must come a long overdue commitment of publically funded initiatives to address the public health disaster that Lyme disease has been allowed to become.

#### **An ethics construct**

Ethical concerns in Lyme disease medicine are glaring, particularly regarding both evident bias in the prevailing guidelines and issues surrounding informed consent. These are certainly pertinent in the broader context of patient beneficence and autonomy. As for the moral principle of non-maleficence, the other side of beneficence, its relevance was recently addressed in Jariwala et al. (31). The time-honored principle of beneficence requires that physicians act in their patients' best interests. Patients' well-being overrides any interests of others – physicians, institution, insurance, etc. The modern notion of autonomy insists that patients are the primary decision-makers regarding their healthcare. Autonomy has become a cornerstone in medical ethics due to the recognition that patients are persons in the full moral sense, that is, by virtue of being persons they possess moral status and therefore certain inviolable moral rights, principally the right of self-determination. These principles incorporate the moral rule of informed consent. Failure to properly inform patients by not acknowledging the clear controversy surrounding Lyme disease diagnosis and treatment and excluding viable treatment options violates both beneficence and autonomy.

#### **Unfair Bias**

As the start, none of this suggests that science and politics must never mix. Indeed, they do. Good science and ethics drives morally sound policy. However, policy-grounded on skewed science is bad policy. Consider the issue of undue bias. Increasingly evident is the deliberate selectivity of data to fashion policy that sustains the orthodox paradigm regarding Lyme disease, a paradigm squarely

outdated when considering accumulating scientific evidence as to the biology of infection caused by *Borrelia* and downplaying numerous patients' symptoms affecting their qualities of life.

There are clearly dissenting positions regarding diagnosing and treating Lyme disease. Yet guideline panels appear to have stacked the deck rather than pursuing the imperative to seriously examine the evidence. While knowledgeable and respected scientists represent the orthodox position regarding Lyme disease diagnosis and treatment, there are equally competent and experienced scientists as well as clinicians on the front lines with first-hand clinical exposure to patients suffering from symptoms of Lyme disease and familiar with patients' histories and symptom trajectories. These clinicians hold positions at odds with the official dogma. Yet their voices are quelled when not allowed at the table in addressing guidelines. Eliminating dissent shows little respect for acknowledging diverse positions. In the process, it short-circuits the reasonably sufficient information patients need in order to consent to treatment.

This explains why, in November 2006, Connecticut Attorney General Richard Blumenthal launched an anti-trust investigation into the influential Infectious Diseases Society of America (IDSA) guidelines that established the medical standard of care regarding Lyme disease diagnosis and treatment. This was the first instance of antitrust inquiry into treatment guidelines' process (32). The power that IDSA wields is apparent on multiple levels. Not only do its members act as peer reviewers for various medical journals, but the organization itself publishes *The Journal of Infectious Diseases* and *Clinical Infectious Disease*, both considered preeminent journals specializing in infectious diseases.

In their 2010 analysis, attorney Lorraine Johnson, JD, MBA, and hematologist Raphael Stricker, MD carefully lay out specific problems with those guidelines (33). Since both authors are affiliated with societies opposing the guidelines, notably the International Lyme and Associated Diseases Society (ILADS), their study itself is not without bias. Nonetheless, they cite how Blumenthal's office noted problems regarding conflicts of interest, particularly financial, over-dependency on certain expert opinion already favoring the official status, false appearance of unanimity, and others. But the most striking flaw in the guidelines lies in the non-acknowledgement of the fact of clear controversy, thereby minimizing any efficacy to alternative treatment. As described later, this limitation impairs the legitimacy of informed consent.

Spurred by Blumenthal's anti-trust inquiry and after close review of the IDSA guidelines and hearing arguments supporting and opposing them, an IDSA review panel issued a final report in April 2010. In fairness to IDSA, bear in mind that not all its members support the official line. Some who opposed requested seats on the review panel. Their requests, however, were refused on the tenuous reason that seating was already filled, even though panel membership later increased (32). Moreover, two months prior to the final report, Blumenthal's office expressed concern over the review panel's voting procedures. Nonetheless, the concluding report, chaired by former IDSA president Carol Baker, upheld the earlier guidelines with a nearly unanimous vote. The decision strongly rested its case on the argument that there was no solid evidence indicating absence of benefit to Lyme disease patients from

standard antibiotic treatment after one month. In the face of counter arguments from ILADS, this conclusion is a classic example of the fallacy of appeal to ignorance, the wrongheaded argument that lack of evidence to the contrary claim constitutes evidence, thus begging the question regarding the diagnosis of Lyme disease.

Clustered together, biases described earlier -- that the EM rash and arthritis with joint swelling are clear "signs" of Lyme disease, that the disease is regionally limited to the northeast and only during summer months, and that it is inflicted by ticks in lymph stage -- seem to indicate a more serious underlying disposition, referred to as "confirmation bias." Confirmation bias arises when professionals with pre-established premises and conclusions and, particularly if sitting on committees or panels to design clinical practice guidelines, with obvious or subtle conflicts of interest, examine and interpret data in ways that strengthen and support their own presuppositions. Surely, human bias is natural, but clearly hazardous when it determines standards of medical care, thereby influencing public opinion, determining insurance coverage, and, in turn, impacting patients' health and well-being, breaching patient beneficence.

Confirmation bias is more than simply the "inattentional blindness" that can beset us when we assume we'll notice what stands out from the ordinary, the unexpected "gorilla" on the basketball court (34). Confirmation bias is tainted with a certain quality of deliberateness and intention, perhaps unconscious, to behold and fashion evidence in ways that confirm our strongly held beliefs. It is this disposition to see what one seeks that reflects confirmation bias, a process counter to scientifically sound research that demands examining evidence as impartially as possible. The science on the biology of *Borrelia* infection and intricacies of symptoms described earlier offers an up-to-date scientific base that offers more solid ground for policy.

### **Informed Consent**

Controversy in the Lyme debate fundamentally revolves around whether or not the disease persists after standard treatment to become chronic Lyme disease. That is, does chronic Lyme disease exist? It appears that the weight of scientific studies supports that it does. Why then are other treatment approaches not alluded to or explained when informing patients of treatment options? If these are excluded from discussion, there are insufficient grounds for properly informed consent.

Issues regarding informed consent, the cornerstone of modern healthcare ethics in reflecting the vital moral principle of patient autonomy, typically arise in the context of patients about to undergo a specific procedure, often radical or invasive. Nonetheless, it applies more generally to diagnosis and proposed treatment. Lyme disease is no exception. Hence, the central concern lies in whether sufficient grounds for informed consent exist when the informing process excludes competing views of the condition and accompanying treatment options. The notion of informed consent therefore needs further unpacking.



WRITTEN TESTIMONY TO NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEE

Genuine informed consent is more than merely fulfilling the institutional requirement of signing a consent form. It is a complex process that entails a rich communicative dynamic requiring that patients be sufficiently informed before they can offer consent, both voluntarily and competently. In the case of Lyme disease, the first ingredient of being sufficiently informed is directly relevant.

Whether the patient is sufficiently informed pertains to the *what*, the *how*, and *who* of a proposed medical procedure. Surely this does not necessitate conveying all information – this is not possible – but information directly germane to the patient’s decision-making is required. Moreover, it is vitally important to bear in mind that properly informing a patient of the procedure, rationale, risks, benefits, and reasonable options means that the informing is *not simply content-driven*. In their illuminating *Rethinking Informed Consent in Bioethics*, philosophers Neil Manson and Onora O’Neill underscore the original meaning behind “to inform” -- to give form and shape, as in sculpting (35). Informing, therefore, is not merely a matter of transmitting content, but unavoidably involves process and context. The authors carefully discuss how information solely as content rests upon a strict mathematical, misguided model of information and communication, and this conduit model of the physician simply delivering information as data is incomplete.

Disclosure as strictly content distorts both information and communication since it obscures context, assumed norms, claims, and inferences. Manson and O’Neill examine the process of disclosure linguistically as consisting in speech acts, more especially truth claims. In applying all this to Lyme disease, consider treating patients bitten by a tick and possibly having the disease. Examples of truth claims would be “You may have Lyme disease,” “One sure sign of the disease is a small circle with a bulls-eye,” “Our standard way of treating the disease is to give you a 10-14 day regimen of antibiotics,” “This should be enough to prevent the disease from spreading.”

Here is where context is crucial. For the patient, and each patient is unique, truth claims like these are meaningful within the framework of his or her desires, beliefs, values, and expectations. To properly inform the patient, the claims in themselves must be *both* true (accurate) and truthful (honest). And being honest demands that one avoids being evasive. Even if the information is accurate, information can still be evasive, therefore dishonest.

To repeat, truth claims in the disclosure process must be both accurate and honest. If not, then there are no legitimate grounds for consent, since informed consent only makes sense if one is so informed. All this revives ways of re-examining the complex interactive dynamic of informed consent. Regarding Lyme disease, informing patients of the standard diagnosis and treatment protocol without acknowledging the unmistakable controversy over diagnosis and treatment and precluding any information as to alternative treatments offers accurate information to a degree, but remains evasive, untruthful, and therefore constitutes no ground for consent.

The patient is the center of moral concern in healthcare. Shifting the center of interest to other stakeholders violates patient’s personhood, dignity, well-being, and autonomy. Consider three

fundamental questions when patients seek treatment. First, what *is* wrong? What is the condition from which the patient suffers? Next, what *can* be done about it? Third, what *should* be done about it? This latter reflects the patient's own value systems and moral priorities. It ought to be the patient herself who decides this. As for the first two questions, they remain in the realm of medical expertise, and it is the physician who must address them. Given the clear controversy surrounding Lyme disease, it is imperative that physicians be honest with their patients to convey the complexity as well as possible and reasonable treatment options. Anything short violates the sacred covenantal relation between doctor and patient.

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August 29, 2017

WRITTEN TESTIMONY TO NYS SENATE TBD TASK FORCE AND HEALTH COMMITTEE

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