ISSN: 2319-6505

Available Online at http://journalijcar.org

International Journal of Current Advanced Research
Vol 4, Issue 10, pp 409-414, October 2015

International Journal of Current Advanced Research

ISSN: 2319 - 6475

REVIEW ARTICLE

OVERVIEW OF LYME DISEASE: A CRITIQUE OF AN IGNORED PANDEMIC

K Paul Stoller

Hyperbaric Oxygen Clinic of San Francisco

ARTICLE INFO

Article History:

Received 20th, September, 2015
Received in revised form 30th,September, 2015
Accepted 15th, October, 2015
Published online 28th, October, 2015

Key words:
Lyme, Borrelia, pandemic, infection, disease, Alzheimer's disease

ABSTRACT

A seemingly stealthy pandemic of epic proportions, causing untold misery and suffering for millions, thrives amidst a culture of politics, greed, corruption, incompetence and arrogance. Endemic in many parts of the world Lyme disease and its associated co-infections doesn't even exist in the minds of some in the medical community, can't be easily diagnosed, treatment regimens are often confusing and not evidence based. When treatment is attempted it is often inadequate or substandard leaving many with chronic persistent infections. While not fitting a vaccine model, paradoxically the quest for finding a vaccine seems to have superseded all other priorities. The evidence of neuroborreliosis being causative in Alzheimer's disease has been sidelined presumably because that information is financially threatening to some controlling faction of our civilization. To complicate matters further there are many pathogenic Borrelia, some that may even rival Borrelia burgdorferi in causing illness, and almost nothing is being done to significantly advance either our ability to diagnose and treat this group of infections that are as problematic as they are pernicious.

INTRODUCTION

Lyme disease (or Lyme borreliosis) is a multi-organ inflammatory disorder caused by both the infection and the immune response to the pathogenic genomic species of *Borrelia burgdorferi (Bb)* sensu lato (sl) that causes it. The *Bb* sl complex is the umbrella for at least 18 other genospecies. In Europe, several of these are pathogenic to humans: *B. afzelii*, *B. garinii*, *Bb* sensu stricto (ss), *B. bavariensis* (also known as *B. garinii* [OspA serotype 4]) and *B. spielmanii*, while the pathogenicity of others in the Bb sl complex is not yet fully understood. In ticks, *B. afzelii* and *B. garinii* are the most common European circulating genospecies, followed by *B. burgdorferi* ss and *B. valaisiana*. There are focal distributions, for example, in the San Francisco Bay area almost half the infected ticks will have *B. miyamotoi*, whereas *B. lusitaniae* is seen more around the Mediterranean basin.

There is no question the disease in endemic in the United States, but it has been reported from Europe, Middle-East, South-East Asia, former Soviet Union, Australia and India where some areas are seropositive at a rate near 18% (Praharaj 2008). Diagnostic procedures that rely only on the antigens relevant to the North American strain could cause serious false negative rates even within the United States itself.

B. burgdorferi ss is most often associated with arthritis and neuroborreliosis, *B. garinii* with neuroborreliosis, and *B. afzelii* with the chronic skin condition acrodermatitis chronica atrophicans. Of course, it is possible to have been infected with multiple species at the same time, and these organisms are found all over the planet – it is unclear why many think strains such as *B. garinii* are not present in the Western hemisphere, when in fact they are present (Sulene 2002).

Without a telltale cough, disfiguring lesions, blood oozing from multiple orifices, Lyme disease has stealthily passed under the radar for a very long time causing everything from arthritis, cardiac disease, dementia and mental illness all unnoticed where even today acknowledging that Lyme is a real infection is still steeped in controversy. The disease is named after the town of Lyme, Connecticut, where the first cases were found in 1975. The organism causing the disease was isolated in 1981 by entomologist Dr. Willy Burgdorfer, who passed at the end of 2014.

In the documentary Under our Skin, Dr. Burgdorfer can be heard saying: "The controversy in the Lyme disease research is a shameful affair, and I say this because the whole thing is politically tainted. Money goes to the same people who have for the last thirty years produced the same thing-nothing."

Lyme disease, if treated early is thought to be curable with a single antibiotic given for a few weeks a few weeks in this case should be about six to eight weeks. However, most humans are bitten by nymphs and go unrecognized and an accurate diagnosis can take years. Co-infections are often not looked for and there is no diagnostic test that confirms clearance of the Lyme organism even if adequately treated.

Add to this a lot of bad science or lack of science, which has led to poor recommendations, such as the advice that a tick must be attached for at least 48 hours before the infection can be transmitted. In fact, there are other ways to get Lyme

besides getting bitten by an infected tick, such as blood transfusion, sex and trans-placental transmission. Lyme that is treated late or only partially, or that is chronic, can leave humans and animals extremely sick, often disabled, mentally ill, or dead. Also, those who come down with Lyme disease often have a co-infection that may not respond to the short course of a single antibiotic just given for a few weeks.

The controversy with the *Bb* sl starts immediately with diagnosis. *Bb* and its brothers and sisters all can cause disease, and some are very virulent, such as *B. hermsii.* Indirect testing of an immune response to the organism (*Bb*) is currently the only "recognized" means of diagnosis (the CDC does not recognize PCR testing for DNA as diagnostic), which is inherently problematic because the testing is calibrated to *Bb* not the other species. For example, *B miyamotoi* is probably as common as *Bb* in the San Francisco Bay area - that will not produce a positive Western Blot (WB) test. *C*urrent testing procedure also produces results that vary with the stage of the disease and the immune status of the patient.

Why are we letting this disease run amok, because now there is more than Lyme disease to worry about… now there is *B. miyamotoi* disease (BMD), which may be as common as Lyme in some areas (Molloy *et al*. 2015). *B. miyamotoi* wasn't even recognized as a human pathogen until 2011.

In 1994, the CDC (Center for Disease Control [USA]) and FDA (Food and Drug Administration) gave their blessing to the diagnostic protocol that continues to be used today (2015) a two-tier diagnostic test based on measurement of antibody response in the blood. In the first step, enzyme-linked immunoabsorbent assay (ELISA), antibodies responsive to a mixture of whole-cell *Bb* spirochetes are evaluated. Because other microbes share some of these proteins, a more refined test, the Western Blot, was implemented that detects a more specific set of antigens.

The most contentious part of the two-step diagnostic standard some refer to it as the Dearborn criteria was elimination from the Western Blot of two *Bb* proteins: outer surface protein A (OspA), from which the first vaccine was made, and outer surface protein B (OspB), the envisioned component of next-generation vaccines. But antibodies to OspA and OspB are sometimes the only biomarkers present in those with late-stage disease.

The CDC dealt with this problem by saying that their standard for someone testing positive on the now truncated diagnostic criteria was only to meet the benchmark for case reporting (to a local Health Department) but not for diagnosis.

The Spirochete and the Vaccine

When LYMErix® (the first approved Lyme vaccine ~circa 1998) was given the green light by the FDA, Allen Steere was then chief of the rheumatology and immunology department at Tufts School of Medicine. But even though Steere was in charge of the research that developed the vaccine, he seemed to have some reservations about LYMErix® if given to individuals with the HLA-DR4 gene, which is present in roughly 30 percent of the population. Published in the journal *Science,* (Gross 1998) a few months after the vaccine was approved, Steere's evidence revealed a striking resemblance between a portion of the OspA molecule and the human protein LFA-1. LFA-1 is an *integrin* and brings together white blood cells called T cells and cells presenting the immune system with molecules, or antigens, the immune system needs to do something about.

However, it is more than just Velcro: it helps activate and program the immune system. The concern was that T cells primed to attack OspA would also attack human cells lined with the "molecular mimic," LFA-1. The result, Steere suggested, might be autoimmune disease, in which T cells continued their attack on the mimic even when OspA was gone. (Trollmo 2001)

This is the mechanism involved in many untoward reactions that other vaccines seem to have as well. That is, something in the vaccine too closely resembles proteins or molecules in the human body, and the vaccine causes the immune system to attack parts of the body it should not be attacking. This is why vaccines are "unavoidably unsafe" even if they were "clean"-free of mercury, aluminum, squalene, oncogenic retroviruses and human DNA from aborted fetal cells (clean vaccines don't exist).

So, the LYMErix® vaccine caused immune-toxicity in those who received it, but the vaccine was given to many individuals who had previously had Lyme disease and were assumed to have been treated completely, and upon vaccination not only did all their old symptoms reappear, but antibody counts soared to most of the bands in the WB test . . . bands that have nothing to do with the OspA antigen. In all likelihood, injecting the OspA antigen signaled Lyme persister cells to wake up and the previous Lyme patients who thought they were free and clear of the illness found out otherwise. Borrelia is not the only family of bacteria that create these metabolic dormant sleeper cells that wait for right conditions to reactivate. The presence of bio-film and persister cells (Sharmam 2015) make treating Lyme a challenge for many.

Vaccine proponents were very upset that the vaccine was pulled in 2002, stating there was no justification for not using it (Abott 2006). To proponents of vaccines there is never a bad vaccine, and any strange and untoward reactions are caused by anything other than a vaccine.

Injecting patients with the OspA antigen who had incompletely treated Lyme may have reactivated a latent infection that either remained undiagnosed or was not dealt with in the first place, but the truth is it could have been a combination of events-setting off an immune-toxicity reaction and activation of a latent infection, and if the latter is even half the truth, it means there are far more Lyme cases out there than previously realized.

Medical History Repeating Itself

In 2011, *Alzheimer's disease-a neurospirochetosis: Analysis of the evidence following Koch's and Hill's criteria*, (Miklossy 2011) was published a meta-analysis on the autopsied brains of Alzheimer's disease (AD) patients. What was found was that spirochetes were observed in the brain in

more than 90 percent of AD cases. *Bb* was detected in the brain in 25.3 percent of AD cases analyzed, and it was thirteen times more frequent in AD compared to controls. The brains also had more than one species of spirochete.

A century ago, the bacteriologist Noguchi Hideyo demonstrated the presence of *Treponema pallidum* (syphilitic spirochete) in the brains of patients with a progressive paralysis, proving that the spirochete was the cause of the disease. Penicillin therapy eradicated General Paresis Dementia, as it was called, after it was made available to civilian physicians at the end of World War II. It is history repeating itself but matters are more serious as no one was denying the existence of syphilis a hundred years ago, but many deny the existence of Lyme disease.

The CDC, the NIH (National Institutes of Health), and the DOD (Department of Defense) own partial rights to revenue from many of the United States patents identified as especially significant for Lyme disease vaccines and tests. That approach means what is best for science; medicine and patients with Lyme may not be best for the financial interests of the government and the pharmaceutical companies they are in bed with. In 2013, the *Journal of Law, Medicine & Ethics* published a paper discussing the fallout from the deep institutional corruption in pharmaceutical companies (Light 2013).

It has only been a couple of years since the CDC revised its estimate from 30,000 new cases of Lyme per year to 300,000+, a move that raised cynical suspicions that this sudden ten-fold increase would herald a new marketable intervention. Pharmaceutical interests aren't going to invest in translational clinical research unless they have something to market, and so a more accurate head count was needed to prepare the market.

The Diagnostic Controversy

There is a two-tier antibody-testing algorithm where first-line screening test (ELISA) fails to detect up to 60 percent of infections. Those patients who do test positive will be subjected to the second, more sensitive test, the Western Blot (WB), but the criteria are so narrow that up to 90 percent of infected patients are excluded from being told they are positive or that maybe they might have a non *Bb* Borrelia infection.

Based on the revised CDC estimate of 330,000 new cases of Lyme per year, the prevalence of *Borrelia* infections in the United States may be between 18 percent to 30 percent of the adult population -18 percent is the seropositive rate in the Arunachal Pradesh region of India (Praharaj 2008) – that is about a quarter of a million people just in that one region. Unlike syphilis, Lyme disease does not cause obvious ulcers on genitalia, nor does it have any dramatic tell-tale symptom. There are certain similarities between the Lyme epidemic and the Tuberculosis (TB) epidemic, but since TB and coughing went hand-and-hand, it was not an epidemic that could be hidden in plain sight. The TB epidemic is alive and well outside of the USA and Canada, it is just that over 90% of infected individuals are asymptomatic. Many with Lyme can remain fairly asymptomatic as well if their immune systems are in good shape, but immune systems have good days and bad days so eventually most Lyme victims will become symptomatic one way or another.

Diagnosis is nontrivial. It is possible to have not one single band positive on the conventional (CDC criteria) WB test and still have Lyme, because regardless of testing viability, the organism is capable of suppressing one's immune system to not respond, so ultimately Lyme, is a clinical diagnosis until the day that there is a reliable and acceptable direct test – hopefully a test that will look at multiple Borrelia species. That puts a lot of responsibility on clinicians who are, at the time of this writing, pretty clueless about Lyme and other Borrelia caused infections.

Whether it is denial or nihilism, many physicians believe Lyme is limited to a few select areas, and most areas are free of Lyme-transmitting organisms. From Australia to Canada many in healthcare ignore or avoid the reality of this infection altogether. It has been over two decades that knowledge of the extent of the Borrelia infestation in the tick population in Australia has been public (Wills and Barry 1991), and yet almost the entire medical community in Australia is in complete denial. *"These findings indicate that some species of tick often responsible for human and animal tick bites in this country commonly harbour Borrelia species spirochetes. On structural and antigenic grounds these microbes are likely to be the aetiological agents of Lyme disease in Australia."* Wills and Barry found 42% of the ticks they evaluated had Borrelia in their guts.

One has to assume public health authorities didn't want to hear that for reasons unknown and it would be safe to assume they didn't want to hear there are congenital and gestational transfer cases or there is confirmed evidence Lyme can be an STD (transferred between sexually active couples). And don't even hope to dare that the blood supply is screened for *Borrelia* . . . It isn't. Infectious Diseases Society of America (IDSA) set the guidelines for diagnosis and treatment of Lyme disease. These guidelines have not been updated to reflect the existence of chronic Lyme disease, of which evidence has been found in well over 300 peer-reviewed articles.

The CDC benchmark for reporting Lyme to Health Departments was adopted as the benchmark for diagnosing Lyme, which is why the positive diagnoses of up to 90 percent of patients are missed. To add to the complication of underreporting, the IDSA's guidelines recommend substandard care, which in turn creates a horde of chronic Lyme patients because the two to four weeks of the single antibiotic they received based on those guidelines did not cure them. There is some good news, the guidelines that have been adopted by another group, the International Lyme and Associated Disease Society (ILADS) based on the published work of Cameron, Johnson and Maloney (Cameron 2014), were accepted by the National Guidelines Clearinghouse (NGC) website.

The National Guidelines Clearinghouse (NGC) is an initiative of the Agency for Healthcare Research and Quality (AHRQ), under the umbrella of the U.S. Department of Health and Human Services. The NGC recently adopted the IOM

standards for developing trustworthy guidelines, which define the highest level of excellence that a guideline can achieve. Guidelines posted on the NGC website must now satisfy these standards. Thus, the inclusion of ILADS's peer reviewed guidelines on the NGC website demonstrates that they meet the level of excellence called for by the IOM. Bottom-line is it makes what the IDSA has to say even more irrelevant.

In 2001, the white paper *Conflicts of Interest in Lyme Disease: Laboratory Testing, Vaccination and Treatment Guidelines by the Lyme Disease Association* (LDA 2001) documents how a handful of well-placed researchers, with serious conflicts of interest, have corrupted the process of guideline development. In 2013, Mary Beth Pfeiffer of the *Poughkeepsie Journal* found "Ties That Bind" (Pfeiffer 2013) between government health officials and outside scientists in a disinformation campaign to steer the nation's perceptions and response to Lyme disease.

Senator Richard Blumenthal as the Attorney General of Connecticut launched an investigation of IDSA, based on allegations of abuses of monopoly power and exclusionary conduct, in violation of antitrust law. In May 2008, Blumenthal (State of Connecticut 2008) said: "My office uncovered undisclosed financial interests held by several of the most powerful IDSA panelists. The IDSA's guideline panel improperly ignored or minimized consideration of alternative medical opinion and evidence regarding chronic Lyme disease, potentially raising serious questions about whether the recommendations reflected all relevant science." From the article *Lyme Disease: The Next Decade* (Stricker and Johnson 2011): "The review panel held a public hearing that featured more than 300 peer-reviewed articles and 1600 pages of analysis supporting the concept of persistent infection despite short-course antibiotic therapy of 2 to 4 weeks in patients with persistent Lyme disease symptoms. Despite this extensive evidence, the IDSA review panel voted unanimously to uphold the flawed Lyme guidelines."

It took 5 years for the CDC to comply with journalist Kris Newby's Freedom of Information Request (FOIA) for emails and resumes from three CDC employees. The emails, when they finally showed up, revealed that there is a shadow group setting Lyme disease policy and a national research agenda without public oversight or transparency. This is the subject of Mary Beth Pfeiffer's "Ties That Bind" article already mentioned. The group convened regularly online and during government-funded closed-door meetings. Aside from their significant ties to commercial interests in Lyme disease tests and vaccines, this group made sure its members received the lion share of government grants. The fact that it took five years to comply with the FOIA requests speaks to the level of collusion the CDC has with this shadow group.

How Did This Get So Out of Control?

There is no question that humans have encroached into areas that in the past were left untouched, and so our exposure to certain aspects of Nature have increased, but there may have been some assistance in this area. The US Department of Agriculture opened the secret Animal Disease Center on Plume Island-a mere 10 miles from Lyme, Connecticut in 1954, "Lab 257," and while they denied for decades that they were attempting to create a livestock-based bioweapons program, *Newsday* magazine found the documents that proved otherwise (Newsday 1993). The Department of Homeland Security took control in 2003. In 2008, an interesting article was published: *Wide Distribution of a High-Virulence* Borrelia burgdorferi *Clone in Europe and North America*. Somehow (Qiu 2008), a *Bb* with an unnaturally strong virulence never seen before arose in the last two centuries that is as close as the researchers could temporarily narrow it down. But when you understand that there seems to have been an attempt to weaponize *Bb* at the animal research facility on Plume Island, the timeline was closer to the last fifty to sixty years. Knowing that there is evidence of the emergence of a more virulent strain of *Bb* gives all the more credibility to the history of Lab 257 (Carroll 2004).

Diagnostic Dilemma

While it won't catch every case of Lyme, using a lab that places back the WB bands that were removed by the CDC the ones connected to OspA and B is a must. The yield on the PCR test is only about 30 percent even if Lyme organisms inhabit the victim, and while it is certainly confirmatory (except to the CDC) that one has Lyme if DNA is found, 70 percent false-negative results makes getting the PCR problematic because of the expense. It is not clear why the CDC does not recognize the importance of a positive PCR assay, even while they acknowledge the poor yield from doing the test. A qualitative epitope immunoassay test on band 31, if positive, indicates a better than 98 percent chance they have chronic Lyme disease (caused by *Bb*). A form of ELISA testing called Multi-Peptide ELISA (MPE) seems to solve many of the inherent problems with the WB (Vojdani 2009) by utilizing *in vivo* induced antigen technology (IVIAT) and enzyme-linked immunosorbent assay (ELISA).

There is a broad heterogeneity of the immunodominant antigens from pathogenic causing *Borrelia* strains and the utilization of a single strain in the whole-cell lysate immunoblot assay is obviously going to cause false negative results. IVIAT is a technique that identifies pathogen antigens that are immunogenic and expressed *in vivo* during human infection. Many Lyme patients who have previously been infected with EBV (the virus that causes mononucleosis and is responsible for a great deal of chronic fatigue) will reactivate in the presence of Lyme and other infections. This is not a co-infection, but should be taken into consideration when formulating treatment.

Treatment Issues

By the time a neuroborreliosis victim gets diagnosed they are often so cognitively compromised or prone to any number of psychiatric symptoms that executive function is impaired and being compliant with a treatment protocol is a nontrivial matter. Lyme can give patients everything from mild cognitive impairment to full-blown schizophrenia, in which case almost all hope for moving through treatment is lost unless there is a very strong family member willing to work with such an individual.

The Jarisch-Herxheimer (Herx) Reaction (die-off) is a cytokine storm in which the immune system can't distinguish

between needing to set off an inflammatory reaction because it thinks there is a massive attack going on, or the death of millions of pathologic organisms. If only treating Lyme were as simple and straightforward as treating syphilis but it is not. Herx reactions are perhaps the most problematic aspect for patients undergoing therapy making the treatment worse than the disease. Biofilms are responsible for a number of chronic infections from many types of organisms. They are not exclusive to Lyme. Antibiotics can't always get into biofilm. Even hyperbaric oxygen, lethal for a facultative anaerobe, can't always penetrate biofilm. It is biofilm that has, in part, been responsible for Lyme patients requiring years of antibiotics and often only to suppress the infection, not eradicate it.

There are several antimicrobial agents that when used, specifically in combination and when pulsed, show considerable promise for efficacious treatment. One of those drugs is nitazoxonide approved to treat diarrhea from protozoal infections. It is capable of treating worms, protozoa, influenza, Hep B virus, and many intestinal bacteria (Clostridia and *H. pylori*, to name two). It has also been found to have some effect treating ovarian cancer and colon cancer. This drug could be an important, although still underappreciated, intervention in treating chronic infections that rely heavily on biofilm to survive.

The co-infections of *Babesia*, *Bartonella*, *Erlichea*, and Rickettsia rickettsia should be tested for, and it is always possible that one can have *Bartonella* or *Babesia* without getting Lyme. For example, there are some parts of the USA where almost all the cats have *Bartonella*, and that infection can be transmitted by fleas (no ticks required). There are also antibiotic resistant cases of Bartonella where drugs like mycobutin or diethylcarbamazine can be helpful. Doxycycline, like nitazoxonide, has no trouble crossing the blood-brain barrier and it has some antiprotozoal activity as well. Some prefer minocycline, but if a patient is over the age of seven one member of this class should be used.

Azithromycin is another frequently utilized antibiotic that is synergistic with doxycycline and nitazoxonide. The issue of persistent cells that become dormant waiting for antibiotics to cease calls for pulsing these agents – be they the three mentioned above or other combinations. The point is aggressive long term treatment is often required for many who have been harboring Lyme disease for some time. Proteolytic enzymes, such as serratiopeptidase (*Serratia* E-15 protease), are possible additions to the protocol in the hope that enough gets absorbed, and that what gets absorbed is enough to affect the Lyme biofilm.

When a patient gets Lyme, rarely is it only Lyme . . . often one is dealing with multiple infections, and multiple organs are involved, and using multiple antimicrobial agents, increases the chance of yeast (*Candida albicans*) overgrowth in the gut and it is often prudent to use an antifungal agent to keep potential yeast complications at a minimum. Treatment for Lyme has to be individualized, and it can get complicated, given there is no objective laboratory measures available for determining when therapy can be terminated. Most physicians are neither aware of the penetration of this infection in the population nor how to treat this is a real conundrum. Now, once the monoclonal antibody for OspA comes on the market with the potential to prevent infection in the previously uninfected, assuming the administration of these monoclonal antibodies on a yearly or twice a year basis, the marketing required to scare the uninfected to get this intervention will undoubtedly advance both diagnostic and treatment options for the infected, but this is still to be seen.

There are several variations on treatment beyond choosing among antimicrobial agents, for example hyperbaric oxygen, previously mentioned, not only enhances the immune system but also acts directly as an antibiotic with organisms that can't stand too much oxygen. In 2013, Woods Hole Oceanographic Institute discovered that the *Borrelia* organism is manganese-dependent, because it substitutes in manganese in place of where almost all other organisms use iron. By doing so, manganese helps the *Borrelia* evade the immune system. A second-tier antituberculosis drug called *para-aminosalicylic acid* (*PAS*) can bring down manganese levels in the human body and make life very tough for the Lyme bacteria, but there have been no clinical studies in this area. Recently it was found that that the common antihistamine Claritin® (loratadine) inhibits manganese transport into the Lyme organism. However, the study in vitro and many things work in vitro and have zero application in a living organism (in vivo).

There are other answers out there, the future will reveal perhaps even more aggressive protocols for antibiotic resistant Lyme, for example an "in vitro" study was published (Feng 2015) that suggested that a combination of daptomycin in conjunction with doxycycline and cefoperazone might be efficacious for treating persistent Lyme. But this is a test tube study and still needs to be tested clinically in human bodies. Last but not least, to help decrease the tick population, something should be done in a very proactive way, and that would be a liberal introduction of foxes into high tick-infested areas, as they will eat a lot of rodents that the ticks feed off. Where possible, the introduction of opossums, which will just dine on the ticks themselves but these ecologically correct interventions will have to wait till there is an honest accounting for how many are actually infected.

CONCLUSION

Ultimately, Lyme disease is still a clinical diagnosis and not just based on lab findings, and while there are some interesting biomarkers that can guide one in determining length of treatment required they are far from reliable. The level of uncertainty about how and when to treat, how and when to stop treating, and the confusing dearth of reliable diagnostic testing is not conducive to having most in medicine want to step into this mess even if they were acutely aware of the extent of Lyme Disease in the population, which they are not.

Lyme disease clearly meets the definition of a pandemic and yet the silence about what to do, given the extent of the damage it is doing, is a conundrum that does not reflect well on humanity's ability (at least the part of humanity that deals with treating disease) to deal with infectious issues that have some moderate complexity to them. Modern medicine has

failed miserably to meet the needs of the millions infected and the woefully inadequate response implies modern medicine is oblivious to what is taking place. If all the obfuscation surrounding Lyme disease has something to do with the lack of some big company being able to take financial advantage of the crisis then that would be sad and a reflection of our dysfunctional approach to reality, hopefully that is all that this is about.

References

1. Cameron, DJ Johnson, LB, Maloney, EL (2014): Evidence assessments and guideline recommendations in Lyme disease: the clinical management of known tick bites, erythema migrans rashes and persistent disease, Expert Review of Anti-infective Therapy, 12:9, 1103-1135.
2. Carroll, MC (2004): Lab 257: The Disturbing Story of the Government's Secret Plum Island Germ Laboratory (New York: William Morrow, 2004).
3. Feng J, Auwaerter PG, Zhang Y, (2015): Drug Combinations against *Borrelia burgdorferi* Persisters *In Vitro*: Eradication Achieved by Using Daptomycin, Cefoperazone and Doxycycline. PLoS ONE 10(3): e0117207.
4. Gross *et al*. (1998): Identification of LFA-1 as a Candidate Autoantigen in Treatment-Resistant Lyme Arthritis, S*cience* 281 703-706.
5. LDA, Inc., "Conflicts of Interest in Lyme Disease: Laboratory Testing, Vaccination, and Treatment Guidelines" Conflict report, for the Lyme Disease Association, Inc. (Jackson, New Jersey, 2001), http://www.lymediseaseassociation.org /images/ pdf/ Conflict Report.pdf.
6. Light DW, *et al*. (2013): Institutional Corruption of Pharmaceuticals and the Myth of Safe and Effecitive Drugs. *Journal of Law, Medicine and Ethics* 14:590-610.
7. Miklossy, J (2011): "Alzheimer's disease-a neurospirochetosis. Analysis of the evidence following Koch's and Hill's criteria," *Journal of Neuro inflammation.* 8:90.
8. Molloy, P. *et al* (2015): *Borrelia miyamotoi* Disease in the Northeastern United States: A Case Series. *Ann Intern Med.* 163(2):91-98
9. *Newsday* (1993). "Plum Island's shadowy past: Once-secret documents reveal lab's mission was germ warfare."
10. Pfeiffer, MB, (2013), "Lyme, the ties that bind," *Poughkeepsie Journal*, May 17, 2013, http:// archive. poughkeepsiejournal.com/interactive/article /2013 0517 /WATCHDOG/ 305160051/ INTER ACTIVE- Lyme-ties-bind.
11. Praharaj (2008) Seroprevalence of Borrelia burgdorferi in North Eastern India. *MJAFI*, Vol. 64, No. 1, MJAFI, Vol. 64, No. 1.
12. Qiu, WG *et al*. (2008): "Wide Distribution of a High-Virulence*Borrelia burgdorferi* Clone in Europe and North America," *Emerging Infectious Diseases* 14 1097-1104.
13. Sharmam B, *et al* (2015): *Borrelia burgdorferi*, the causative agent of Lyme disease, forms drug-tolerant persister cells. *Antimicrob. Agents Chemother*.(ACC)
14. State of Connecticut Office of the Attorney General, May 1, 2008, "Attorney General's Investigation Reveals Flawed Lyme Disease Guideline Process, IDSA Agrees To Reassess Guidelines, Install Independent Arbiter," http://www.ct.gov/ag/cwp/view.asp?a=2795&q=414284
15. Stricker, RB, Johnson, L. (2011): Lyme disease: the next decade, *Infection and drug resistance* 4:1-9.
16. Sulene P, *et al*. (2002). "Serological reactivity to Borrelia burgdorferi, Borrelia afzellii, and Borrelia garinii antigens in patients afflicted by peripheral facial paralysis in Brazil." *Otol. Neurotol*. 23:S33
17. Trollmo, C *et al*., "Molecular Mimicry in Lyme Arthritis Demonstrated at the Single Cell Level: LFA-1α_L Is a Partial Agonist for Outer Surface Protein A-Reactive T Cells," *The Journal of Immunology* 166 (2001): 5276-5291.
18. Vojdani, A *et al* (2009): Novel Diagnosis of Lyme Disease: Potential for CAM Intervention. *Evid Based Complement Alternat Med.* 6(3): 283–295.
19. Wills MC, Barry RD. (1991) Detecting the cause of Lyme disease in Australia. *Med J of Aust*. [Letter]. Aug 19;155:275.

****