LYME BORRELIOSIS: A HOMEOPATHIC PERSPECTIVE

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Lyme borreliosis is a challenge to scientists, homeopathic and allopathic clinicians, and their patients. As a clinical entity, this syndrome defies tidy identification, nomenclature, diagnosis, temporal staging, treatment, or even description. As of the fall of 2006, branches of the human medical community formally proposed two entirely separate definitions of chronic Lyme disease. The issue is now under litigation.

There are great differences in the approach to treatment and the understanding of the Lyme disease process within the homeopathic community as well as within the allopathic community, not to mention the divide between "conventional" and "alternative" veterinarians and doctors. In the recently published ACVIM Small Animal Consensus Statement of Lyme Disease in Dogs, there was frankly acknowledged lack of consensus on several key points regarding diagnosis, treatment and prevention. The issues on the table are much more complex than "antibiotic" vs. "homeopathic remedy". Faced with the large and growing concern regarding Lyme disease as it impacts animals and their human caretakers, it behooves homeopaths to have a basic understanding of the biology of the organisms involved, the standard tests available, and the conventional modes of treatment. When we can communicate clearly and knowledgeably with our clients and their conventional veterinarians, we are in a strong position to treat Lyme disease curatively within the context of our understanding of acute and chronic disease processes.

In this presentation today, I plan to review the basics of Lyme disease as understood by the conventional veterinary community, then consider Lyme disease within the context of Hahnemann's work, the concepts of miasms, genera epidemica, and acute and chronic disease. Establishing common vocabulary and nomenclature will bring clarity to what can be a confusing and misunderstood condition and will build a foundation for integrating homeopathic understanding and approach with current scientific knowledge. We will explore the model of Lyme disease as a regional bacterial endemic influence that can lead to acute disease or precipitate chronic disease. Finally, we will review some cases, which will ground our discussion in practical approach.

What is Lyme Disease?

Read almost any standard medical reference, and you will ascertain that Lyme disease is caused by a spirochetal organism of the genus Borrelia, transmitted by the bite of an Ixodes tick. (The "deer tick" of the Northeast United States is Ixodes scapularis). However, depending on what you read beyond that and whether you study borreliosis in humans or animals, you will find that Lyme disease is rampantly over or under-diagnosed; arises as an easily recognized acute disease and is responsive to treatment, or accounts for a large number of chronic problems and is extremely refractory to treatment; progresses to a life-threatening stage if un-treated, or resolves on its own; may be considered another "great imitator" disease like syphilis, or has a clearly defined set of symptoms; exists everywhere in the United States and is spreading world-wide, or

is restricted to a few suburban hot-spots; or is a creation of advanced diagnostic capability and does not really exist as a disease at all.

First, What is Disease?

Before attempting to clarify this seemingly conflicting body of information and apply it theoretically and practically, let's review some basic concepts and nomenclature. For example, how may we define disease in general, and Lyme disease in particular? Samuel Hahnemann wrote in the *Organon* that "disease [is] a state of being in which the organism is dynamically altered by a morbidly mistuned life force" (footnote 8, §8), that "*diseases* are nothing other *than alterations of condition in healthy people* which express themselves through disease signs ..." (§19). His understanding of a diseased state of being does not disavow the role of causative agents, however he distinguishes clearly between any physical initiating cause and the potential response by a susceptible life force, without which no disease results (§31):

> "The – partly psychical and partly physical – inimical potencies in life on earth (which we call disease malignities) do not possess an absolute power to morbidly mistune the human condition. We become diseased by them only when our organism is just exactly and sufficiently disposed and laid open to be assailed by the cause of disease that is present, and to be altered in its condition, mistuned and displaced into abnormal feelings and functions, hence they do not produce disease in everyone, nor at all times."

About 70 years later, when the existence of microbes was well accepted, Stuart Close wrote in *The Genius of Homeopathy* "the first proposition is that disease is not a thing but only a condition of a thing; that disease is only a changed state of health, a perverted vital action; and not in any sense a material or tangible entity to be seen, handled, or weighed, although it may be measured." Again, the emphasis is on the "terrain," as Pasteur is said to have eventually appreciated, not the microorganisms.

It is helpful to appreciate the excitement in the scientific community around the time of Hahnemann (who largely predated the field of microbiology) and shortly after. Pasteur, Leeuwenhoek, Koch and others were discovering the world of microbes, experimentally taking cholera by the spoonfuls for the greater good, and for a time it seemed that the easy answer to all disease conditions was to eradicate the bugs. Now, more than 100 years later, the scientific community is again giving great consideration to individual susceptibility, the immune system, the terrain. Apart from the purely cause-and-effect mechanistic explanations of, for example, Lyme disease, it is possible to find a more subtle and dynamic definition based on current clinical research: "immunological responses by the host to infection produces the clinical manifestations in the dog . . ." (Troy, 2003). Although at face value this would seem a simple and obvious statement, it is not such a stretch from this to the following:

> **The mistunement of the vital force, in response to impingement by imimical potenc(ies) on a susceptible individual, results in disease expressed by the individual as a unique totality of symptoms.**

Susceptibility is a key concept to appreciate in the context of understanding Lyme disease. Why is it that so many seropositive animals and people exhibit no clinical disease? We can say these individuals were exposed to Borrelia spp. but lack susceptibility. In Kent's discussion of Hahnemann's §31 (above), he reminds the reader that "Because of . . . varying degrees of susceptibility some are protected from disease cause and some are made sick; the one who is made sick is susceptible to the disease cause in accordance with the plane he is in . . . The degree of the disease cause fits his susceptibility at the moment he is made sick" (*Lectures on Homeopathic Philosophy,* Lecture XIV). Only susceptible individuals are vulnerable to impingement. Susceptibility may be determined by many factors including heredity, lifestyle, and immunological challenge.

Let's consider one more seemingly simple concept, "infection." We are all familiar with the over-diagnosis of inflammatory processes as infection, so let us clarify that by "infection" we are speaking about bacterial involvement. But what is active infection vs. sub-clinical infection? How about exposure that leads to seroconversion, the presence of antibodies? If the antibodies persist, do we infer that infection (i.e. the bacterial antigenic challenge) is still present? Is there a difference between a healthy individual with a persistent sub-clinical infection, and a host? How are we to understand bacterial colonists that cause disease only in immuno-compromised individuals?

There exists a spectrum of interaction between any organism and its bacterial milieu, from balanced cohabitation, to death and decomposition (contrary to western mass marketing efforts to convince otherwise, there is no healthy life in the absence of bacteria). For instance, early in life one must acquire a healthy population of bacteria for normal function; one can also be exposed to pathogenic bacteria and develop protective immunity; be exposed to pathogenic bacteria and address the exposure without developing disease symptoms; acquire a bacterial population which may be variably termed a latent, sub-clinical or chronic infection; lose one's balance with a normally benign co-existing bacterial population; or be overwhelmed in some devastating way. We can consider these variations within the context of our understanding of bacterial and viral diseases. Some examples are tetanus and polio, for which immunity may be acquired naturally and without incident; demodecosis, a non-communicable disease in which a normal fauna becomes overwhelming; shingles, in which a latent or sub-clinical virus acquired in childhood as chickenpox erupts; salmonellosis, which may be a carrier state for many healthy individuals; anthrax, which is invariably fatal; and cholera and malaria, discussed extensively by Hahnemann.

"Infection" may be defined conventionally as "invasion and multiplication of microorganisms in body tissues, especially that cause local cellular injury due to competitive metabolism toxins, intracellular replication, or antigen-antibody response" (Dorland's). The homeopathic concept of impingement, rather, encompasses all subtleties of interaction. Impingement may be defined as the non-material action of an external force against an individual, therefore, individuals may be infected without resultant impingement. It must be remembered that Hahnemann, before the establishment of microbiology, presaged germ theory (which posits that microorganisms are the cause of disease) with his model of dynamic disease transmission. Had he known of the existence of bacterial agents, his distinction between the *prima causa morbi* (first cause of disease) and the inner *wesen* [essence] of the disease itself

(Introduction, *Organon*) would still stand, because he so clearly distinguished between the disease and any physical agent.

Ticks and Borrelia spp.

Let's briefly consider the biology and history of the organisms relevant to Lyme disease, Borrelia spp. and Ixodid ticks, which are arthropods (eight legs), not insects (six legs). Borrelia spp. are spirochetal bacteria, as are Treponema spp., the bacterial agent of syphilis. Borrelia spp. are capable of existing in many different dormant and active stages, in many different areas of the body, and in many different vertebrate hosts. They are not associated with clinical disease in many of their hosts. Borrelia spp. have evolved a complex relationship with their equally complex vectors, ticks, which require one or more blood meals to reach maturity and reproduce. Ixodid ticks, the most common vector of Borrelia spp., do not need to feed on deer, although they are commonly known as "deer ticks". The tick larvae are not infective when they hatch, even when laid by an infected female. They become carriers for Borrelia spp. if their first meal is on an infected warm-blooded host (usually a small mammal or bird). As they molt into newly infected tiny nymphs, they attach to another warm-blooded host for another meal and so transmit Borrelia spp. or acquire it. The nymphs molt to adults, which attach to a third host before mating and laying eggs. Here are some take-home facts about Ixodid ticks and Borrelia spp.:

- Tick-borne diseases usually require at least a day of attachment to be transmitted.
- Not every deer tick is infected with Borrelia spp.
- Only the female ticks transmit tick-borne disease.
- Cold weather does not kill ticks.
- The entire tick life-cycle takes two years.
- Many ticks are becoming resistant to prescription pesticides.
- Borreliosis is not directly communicable between host species.
- Not all inflamed tick bites indicate transmission of Borrelia spp.
- Unlike humans, dogs do not usually develop *erythema migrans* (bulls-eye rash) at the site of a Borrelia-positive bite; however, Borrelia can easily be biopsied from that area for some time.
- The incidence of Borrelia positive ticks does not correspond directly with tick population.

Can Lyme Disease be Experimentally Studied?

Robert Koch formulated what are known today as Koch's Postulates in 1890 to define causative agents of disease, shortly after the existence of bacteria was discovered. Although the Postulates have undergone considerable revision (including by Koch himself) and exception as scientific understanding of disease has increased, they remain as a standard by which infectious organisms are measured. They are:

1. The microorganism must be found in all organisms suffering from the disease, but not in healthy organisms.

2. The microorganism must be isolated from a diseased organism and grown in pure culture.

3. The cultured microorganism should cause disease when introduced into a healthy organism.

4. The microorganism must be re-isolated from the inoculated, diseased experimental host and identified as being identical to the original specific causative agent.

Certainly, Lyme borreliosis does not fit tidily into this model. Many animals (and people) are seropositive by various tests for Borrelia spp. without any clinical signs: this phenomenon is true however for other diseases as well, including well-characterized ones. Asymptomatic carrier states are well documented. Borrelia spp. can be difficult (although not impossible) to isolate and culture. The disease caused by direct inoculation with Borrelia is not clinically the same as vector-borne Borreliosis. Although in some previous studies dogs were immunosuppressed in order to be experimentally infected with Borrelia spp., Lyme borreliosis is now easily transmitted under experimental conditions using infected ticks (this phenomenon reflects the complex interaction of the bacteria with its vector, a factor which confounds testing as well). Borrelia organisms can be isolated and cultured from experimentally infected animals.

Where did Lyme Disease Come From?

Reports of rashes and arthropathies following tick bites date back to the turn of the last century. Borrelia burgdorferi have been identified from tissues of white-footed mice preserved 100 years ago. In the 1970s, a cluster of rashes and arthritic conditions in children in Old Lyme, Connecticut, gave rise to the eventual isolation and identification of Borrelia burgdorferi (by Willy Burgdorfer in 1982) and subsequent labeling of Lyme disease. Ticks and their hosts flourish on the edges of disrupted habitats. Although deer are not a required host, they are a common one, and explosions in the deer population with the expansion of suburbia are well recognized. The Northeast and areas of the West Coast report a high incidence of human and animal Lyme borreliosis, however Lyme disease has been reported in almost every state of the United States (the lower frequency of Lyme disease elsewhere in the country may be due differences in optimal host populations). Lyme disease is also being reported with increasing frequency in Europe also, with its own subsets of tick and Borrelia species.

Other tick-borne diseases continue to be recognized as new entities. Currently under investigation is a tick-related rash disease, STARI (Southern Tick-Associated Rash Illness), for which no causative agent has been defined as transmitted by its host, Amblyomma americanum

(lone star tick). It has not been associated with the same depth of pathology as human Lyme disease and appears to be antibiotic responsive. Another increasingly diagnosed tick-borne illness affecting animals and people is Ehrlichia equii, also known as anaplasmosis (Anaplasmosa phagocytophila).

Diagnostic Tests for Lyme Disease

A positive Lyme test is why many clients come to us for help, saying "my dog has Lyme's!" (Note: the correct terminology is Lyme disease, not Lyme's disease.) Commonly performed first-tier tests include ELISA (enzyme-linked immunosorbent assay) tests such as the C6 tests. Second-tier Western Blot tests, which are more sensitive and specific, qualitatively and quantitatively examine a wide spectrum of immunoreactivity. One of the most important efforts we can make is to help these clients understand what the tests really mean:

- Standard screening tests for Lyme disease are antibody (immune response) tests, not antigen (bacteria) tests, and indicate exposure only.
- The tests do not diagnose or predict active disease.
- The antibody titer does not equate with level of infection.
- In some areas of the United States, an estimated 50-90% of dogs are seropositive. A very small fraction of these dogs (less than 10%) are ever clinically affected.
- It is difficult to distinguish between persistent borreliosis and reinfection, even in winter months when ticks are dormant.

Problems interpreting Lyme tests may arise from animals symptomatic for Lyme disease with a history of tick bites, but negative titers; persistent titers in the face of treatment, with or without persistence or recurrence of clinical signs; cross-reactivity with other diseases; and timing of immune response. A valid application for the common Lyme disease screening is testing prior to any consideration of Lyme vaccination. Seropositive individuals are at a higher risk for fulminant Lyme-like disease that is documented but not yet well understood.

Antigen tests that examine for the Borrelia spp. organisms directly, such as the PCR (Polymerase Chain Reaction), are not in common use. These tests are invasive, costly, and problematic. Borrelia like to hide in inaccessible parts of the body, exist in many forms and life stages, and are difficult to culture. False negatives as well as false positives (Borrelia spp. fragments) are possible. Antigen tests are usually performed in research studies or as later stage diagnostic tests in severely or chronically affected individuals.

So, in the face of problematic testing, how can we help our clients, and our patients who may or may not have Lyme disease? It is common for clients to bring concerns with them not only for their pets, but for themselves, friends, family members; these concerns are not always overtly expressed but the subtext is nonetheless important to understand. Clients often acquire information and misinformation from a wide range of sources, and sometimes assume that their

pets have diseases the way people do. For example, many more seropositive humans have clinical symptoms of Lyme disease than do dogs. This may be so because clinically affected people are usually the ones tested. General screening of the healthy human population for Lyme disease is not the norm as it is now in dogs. But since for people a "positive" test for Lyme often confirms disease, clients may view the "positive" screening test of their dogs with equal significance. Remember to ask your clients if their animal companions are sick.

Theoretical Homeopathic Models for Understanding Lyme Disease

There is an extensive and well-documented body of clinical homeopathic work and discussion on a wide variety of conventionally recognized diseases. Acute infectious diseases of bacterial and viral etiologies, epidemics, emotional and physical trauma, as well as recurring conditions and chronic deep-seated ailments have been treated and analyzed in great depth by past masters of homeopathy as well as more recent homeopaths. Where does Lyme disease fit among these categories? How can understanding its behavior aid in our treatment? Let's examine Lyme borreliosis within the context of acute vs. chronic disease, and miasmatic theory. We can then apply our theoretical understanding to a review of some disease conditions that were prevalent at the time of Hahnemann.

Acute or Chronic Disease

Is Lyme disease acute or chronic? On the surface this would seem a very straightforward question. Hahnemann writes "*Acute disease*s are rapid illness-processes of the abnormally mistuned life principle which are suited to complete their course more or less quickly, but always in a moderate time. *Chronic diseases* are those which (each in its own way) dynamically mistune the living organisms with small, often unnoticed beginnings . . . [they] arise from dynamic infection by a chronic miasm" (§72). Examples of acute diseases, which are generally considered either self-limiting or fatal, include: canine parvovirus, feline and canine distemper, and in humans measles, whooping cough, and scarlet fever. These diseases are considered "acute fixed miasms" because they recur in a similar way and persist so in the environment. (However, Hahnemann is careful to observe that most "acute febrile disease" is not a true acute disease at all, rather a flare-up of chronic disease (§73).) Acute disease refers to its temporal course, not its severity. There are well-recognized acute outbursts of chronic disease that are very dire clinically.

Can an "acute" presentation of Lyme disease be recognized? Yes: there are case examples in both the animal and human literature in which previously asymptomatic individuals were bitten by Borrelia spp. positive tick(s), and became seropositive, acutely febrile and lame, with or without an accompanying rash. The consistent rheumatic presentation may perhaps be considered the disease *wesen*. Can the argument be made that the "acute" presentation was actually flare-up of chronic Lyme disease or chronic psora? Yes, although the argument weakens in individuals that were previously seronegative or exhibit seroconversion of a type consistent with recent exposure, and in individuals with no previous similar symptomatic history. We understand that there are few if any individuals free from psora or other chronic miasmatic influence, especially pure-bred animals; however this does not negate the effect of an external influence on the susceptible individual.

Is a "chronic" form of Lyme disease recognized? Certainly this is a presentation with which we are very familiar. Animals suffer repeated episodes of fever and lameness, or more vague symptoms **depending on the individual**, which may or may not be associated with repeated exposure. There is much debate within the conventional community about whether the recurrent symptoms in such cases represent resurgence of residual Borrelia organisms, re-infection, or some unspecified autoimmune process. There are also varying descriptions within the homeopathic community of Lyme disease as an acute, half-acute, or chronic disease. In an effort to examine the nature of Lyme disease, the following questions may be posed:

> **Is Lyme borreliosis self-limiting?** *Once disease symptoms are present, sometimes - but not always. There are many animals with persistent sub-clinical infection but no impingement.*
>
> **Does the onset of symptoms occur promptly after infection?** *No, there is usually a lag time of several weeks.*
>
> **Is there a clearly characterized acute manifestation of Lyme disease?** *Yes, a febrile rheumatic state with or without an eruption (but with documented local reaction in the absence of a rash).*
>
> **Are there variable expressions of chronic Lyme disease?** *Certainly. Rheumatic symptoms may recur, but many more vague or variable chronic disease patterns may emerge as well, with or without the presence of Borrelia organisms or detectable antibodies.*
>
> **Is there evidence of persistent borreliosis with or without treatment?** *Yes, in clinically affected animals that are treated with antibiotics as well as those who are not, and also in animals that do not develop clinical disease.*

So we can see that, while Lyme borreliosis fulfils some criteria for an acute or half-acute disease, our discussion would be incomplete without acknowledging the pertinacity of this organism and the patterns of disruption that can ensue over months and years.

When we treat a patient in the initial presentation, are we addressing true acute disease, or the initial stage of a chronic disease? Is it chronic disease? Not yet. The sequelae of acute infection may include either awakened miasmatic disease or a new chronic disease: "It can also happen that the *new disease*, after impinging for a long time on the organism, *joins the old one that is dissimilar to it,* and they form a *complicated* disease. Each disease takes in its own region in the organism, that is, it takes the organs especially appropriate for it" (*Organon,* §40).

Hahnemann writes in *The Chronic Diseases* that even syphilis, if treated properly in the acute stage, is easily cured as long as the treatment is not suppressive or merely palliative: "In this state, and especially when it is not yet complicated with psora . . . *there is on earth no chronic miasma, no chronic disease springing from a miasma, which is more curable and more*

easily curable than this." Could an individual easily be cured of an initial Lyme infection? Yes, although how many of our patients are free from psora? Not many.

As homeopaths, we are very familiar with the scenario of chronic symptom expression and understand that such expression may not be contingent on the persistence of any one infectious organism (where applicable) but rather that the individual has gotten "stuck" (sometimes for years) in an effort to repel the organism and achieve health. Homeopathic treatment of a chronic condition is not contingent on the presence or absence of the initial causation.

When we see cases of symptomatic Lyme borreliosis in the initial dramatic throes, it is appropriate to address the state with remedies that correspond to acute disease. This approach is indicated in prescribing for both acute disease as well as acute manifestation of any chronic disease. It is then prudent to follow up with constitutional treatment and/or remedies that take into account the etiology of the causation. Theoretically, treatment at this next stage is being directed at either the individual's underlying miasmatic predisposition to Lyme disease (if one holds that Lyme disease is acute) or at staving off the chronic entrenchment of Lyme disease (if one holds that Lyme disease is its own chronic miasm). Patients with a susceptibility to joint disease, for example, may be more vulnerable to chronic infection, since Borrelia spp. have an affinity for joint tissues. Their disease will be expressed with chronic symptoms in the joints referable to their unique underlying constitutional miasmatic state, or with symptoms referable to the complicated disease that may result if the Lyme borreliosis and the patient's underlying miasmatic disease are of equal strength and have joined forces.

Homeopathic treatment is based on an assessment of the patient's symptoms, both present and historical. It is not necessary to definitively categorize Lyme borreliosis as an acute or chronic disease in order to treat it homeopathically. By understanding how it behaves, however, we are in a position to consider remedies that are most appropriate in the initial acute presentation, and are alert to the need for chronic treatment both to address underlying susceptibility and assist the patient in eradicating the disease. Consider Hahnemann's therapeutic recommendation in *The Chronic Diseases*: " . . . during the treatment of chronic disease by antipsoric remedies we often need the other non-antipsoric store of medicines in cases where epidemic disease or intermediate diseases arising from (various) causes attack our chronic patients, and so not only temporarily disturb the treatment, but even interrupt it for a longer time."

Miasmatic Disease

Acute and chronic diseases are considered miasmatic influences. The term "miasm" is variably applied and the terminology can be confusing. A "miasm" has been used to describe a specific acute disease as above (infectious, epidemic or endemic), a category of disease (acute vs. chronic), or a pattern state of chronic disease. The three primary chronic miasms as described by Hahnemann are psora, sycosis and syphilis. These are pervasive disruptive influences with an originally infectious basis (scabies, gonorrhea and syphilis, respectively) that result in characteristic patterns of disease expression. In recognizing the pattern, it is possible to consider a family or category of homeopathic remedies with affinity for that state.

In brief summary, psora may be regarded as resulting in disorders of function and regulation, sycosis may result in patterns of excess (behavior, discharges, growths) and syphilis may be considered in diseases that are destruction on a emotional and/or physical level. Psora is considered by Hahnemann and homeopaths since to be the fundamental miasm that exists as a base for infection by other miasms. It should be noted that the presence of one of the miasms is not the same as infection with the associated organism; one can treat a patient for "syphilitic disease of a miasmatic nature" without assuming that patient is infected with Treponema bacteria, the original infectious agent of syphilis. Hahnemann recognized that the original infectious causes became entrenched chronic miasms in a population after much passage of time, and in the absence of curative treatment.

Do specific disease conditions always result in a single miasmatic response? Not necessarily. John Saxton, in *Miasms as Practical Tools*, describes how each individual has a balance, in perfect health, between "Production, Removal, and Control". Under challenge, one or another functional aspect is provoked to express symptoms. Individuals may have a predominant miasmatic predisposition, but are not restricted to a single pattern of disease. Since more than one disease miasm can be present in a single patient, the therapeutic key is to recognize which one is uppermost at the time.

Since the time of Hahnemann, other diseases have given rise to other named miasmatic states. Henny Heudens-Mast, in *The Foundation of the Chronic Miasms*, discusses how an individual can acquire the "tubercular miasm":

1. The patient contracted the disease.

2. The patient received the miasmatic influence from someone in the family—someone in the family's history at some time in the past contracted the actual disease for which the miasm is named.

3. The patient contracted the miasmatic influence through vaccination against the disease.

Disruption in the primary patterns of normal body function and the resultant miasmatic expression are sufficient to explain chronic disease states. John Saxton writes: "It cannot be emphasized too strongly that in the author's opinion, although (homeopathically) all chronic disease is miasmatic, not every chronic disease is a new miasm." With specific regard to Lyme disease, he writes: "Thus in the case of Lyme disease (as with any other infection), the forces present in the causal agent are reflections of the three basic forces of nature in their correct balanced format for that organism, and it is better to think of them as physiological for that organism, rather than pathological or miasmatic" (personal communication).

We have some theoretical context now in which to begin to look at how Lyme borreliosis behaves clinically, and we are not obligated to designate a Lyme miasm (yet). Although, should we wish to establish a persistent Lyme miasm, we might continue to do the following: strengthen the endemic infective agent by selecting for antibiotic and pesticide resistance while decreasing the strength of our patients by applications of the same; repeatedly administer vaccines with the documented ability to precipitate the symptoms and pathology of the disease; continue to disrupt

habitats in a way that favors vectors and their hosts; continue to inbreed purebred animals with an increased susceptibility to Lyme disease. David Little writes "Miasms like Lyme disease are endemic because they depend on a zoological host." He does classify Lyme disease, along with syphilis, as a chronic infectious miasm. Eventually, zoological hosts would not be necessary for an inherited form of originally infectious Lyme miasm to be transmitted.

Comparative Disease

What diseases studied by Hahnemann correspond to Lyme disease? Hahnemann discussed the transmission and treatment of cholera in great detail, and other acute epidemics as well. His extensive discussion of "swamp fever" (malaria) is clinically reminiscent of present-day Lyme disease. He writes in *The Chronic Diseases*:

> "Endemic diseases, with their striking pertinacity, depend almost wholly on a psoric complication, or on psora modified by the peculiarity of the nature of the locality (and the especial mode of life of the inhabitants), so that, *e.g.*, in intermittent fever originating in a marshy region, the patients, even after removal into a dry region, often remain uncured despite of all their use of china, unless the antipsoric treatment is especially used. The exhalation from swamps seems to be one of the strongest physical causes of the development of the psora latent within so many persons and this most of all in hot countries . . . Wherever psora lies latent within (and how frequently is this the case?) it is developed into Chronic disease of every kind . . . through stagnant water and the gases that emanate from damp soil and from swamps . . ."

And in the *Organon* (§244):

> "Intermittent fevers endemic to marshy regions and places where flooding is frequent have given the old school physicians a lot of trouble. Yet, a healthy young person can accustom himself even to marshy regions, and remain healthy in such surroundings, provided his regimen is faultless and he is not oppressed from deprivation, fatigue, or destructive passions. The intermittent fevers endemic to the place will, at most, only seize him when he is a newcomer. One or two *of the smallest* doses of highly potentized cinchona solution would, along with an orderly way of life, soon free him of the disease. If people cannot be free of intermittent marsh fever . . . then this always means that psora, striving to develop itself, lies at the base of their malady."

Hahnemann expresses sympathy for the patients suffering from marsh fever receiving large material doses of cinchona (quinine): "Larger, frequently repeated doses of cinchona can indeed free such patients from what is typical of intermittent marsh fevers . . . but such people, deceived into believing they are cured, are not. They are left suffering in a different manner . . . from a cinchona wasting sickness which is occasionally incurable." In *The Lesser Writings* he also vividly describes cinchona bark and other herbs as suppressive agents. In comparison, Lyme disease is also often refractory to treatment by antibiotics. Even when antibiotics ameliorate clinical disease, individuals often remain seropositive for a long time (with or without clinical recurrence). We can recall our earlier discussion of acute impingement leading to chronic disease and consider Hahnemann's caution in the *Organon* (§242):

> "If the first attacks of such an . . . intermittent fever are left uncured, or if the patient is debilitated by allopathic mistreatment, then the indwelling, dormant psora (which unfortunately is already in so many human beings) develops and takes on the typus of the epidemic intermittent fever; that is, the psoric disease, to all appearances, plays out the role of the epidemic intermittent fever. In these cases, the medicine that would have been helpful for the initial paroxysms is no longer fitting and can help no more. We now have a case of psoric intermittent fever only."

So we can see that although a specific infectious agent (i.e Plasmodium) had not yet been identified for swamp fever, this is irrelevant within the context of the disease pattern. Hahnemann had recognized a pervasive, stubborn, regional influence. A morbific influence capable of causing acute disease in many individuals, except those either with a completely wholesome lifestyle, or those free of psora or other inherited chronic disease; A morbific influence with a troublesome predilection for triggering latent chronic disease or becoming entrenched in the individual; A morbific influence with some affinity, in the acute stage, for a single remedy, *Cinchona (China) officinalis;* A morbific influence which almost always requires anti-psoric management. Note his prophetic words vis-a-vis malaria in *The Lesser Writings*:

> "Probably some other diseases, which we cannot show to depend on a peculiar miasm, as gout, marsh-ague, and several other diseases that occur here and there endemically . . . also arise either from a single unvarying cause, or from the confluence of several definite causes that are liable to be associated and that are always the same, otherwise they would not produce diseases of such a specific kind, and would not occur so frequently."

We can infer that Hahnemann was leaning towards consideration of malaria, which he considered an endemic fever, as one of the (acute) miasmatic maladies, e.g. "hydrophobia [rabies], the venereal diseases, small-pox, measles". He reminds us in this context that "No alteration occurs without a cause." If we evaluate Lyme disease as an analog to malaria, we can see how it meshes with both Hahnemann's understanding of "swamp fever" and current clinical knowledge regarding Lyme borreliosis:

- Both organisms (Borrelia spp. and Plasmodium spp.) are regionally vector-transmitted.
- Infection is widespread; disease expression is highly variable.
- Individuals may possess protective premunitive immunity which may be adversely affected by conventional treatment.
- In the susceptible individual, acute infection often leads to individualized expression of chronic disease.
- Infection may be refractory to treatment unless underlying health is optimized.
- *Cinchona* has an affinity for malaria in the acute presentation, as *Ledum* does for Lyme disease (to be discussed below).

- The initial treatment of Lyme disease (and malaria) frequently requires follow-up treatment.

- Patients may have long-term symptoms without detectable antibodies or organisms present.

Hahnemann expressed a deep respect for "swamp fever" as a worthy adversary, as should we for Lyme borreliosis. He even counseled against inhabiting swampy areas. Remember that he encouraged seeking out etiology: "It will help the physician to bring about a cure if he can find out the data of the most probable *occasion* of an acute disease, and the most significant factors in the entire history of a protracted wasting sickness, enabling him to find out its *fundamental cause* . . . [which] mostly rests upon a chronic miasm" (§5). His caveats were that the agent of disease not be confused with the disease itself, and that practitioners not direct their therapeutic efforts at ineffectively (and sometimes harmfully) attempting to eradicate any physical inciting cause or agent. It is interesting to note that in the human literature there are recent reports of people with Lyme disease even injecting themselves with malarial blood products in what may be a desperate effort to displace their borreliosis through drastic introduction of a similar disease. Syphilis was also historically treated with "malariotherapy."

Two other well-described diseases, syphilis and rabies, are discussed in the conventional and homeopathic literature in connection with Lyme disease. There are some biological similarities between borreliosis and syphilis, although the latter is transmitted through direct sexual contact, not a vector. The infective agent in each case is a highly morbific spirochete with a predilection for latency. There can be serological cross-reactivity. Some histopathological lesions are similar. There are superficial (skin) infective stages recognized as well as deeper forms. Both diseases have been called "great imitators" because of their highly variable and extensive expression of symptoms. In its more destructive manifestation, borreliosis is certainly an expression of the syphilitic miasm. However, it is not necessary to default to predominantly syphilitic remedies in order to address Lyme disease. The variability of disease expression can be understood within the context of our understanding of how disease develops at a deeper level in the individual with a weaker vital force, or when disease is suppressed. If one reads a list of symptoms of chronic Lyme disease and chronic syphilis, every ailment under the sun is represented. These ailments may be described as the totality of chronic disease sufferings which can be triggered by infection with these organisms. The biological similarities between the two organisms may affect morbidity, but are not necessarily predictive of miasmatic behavior.

In *The Chronic Diseases*, Hahnemann refers to rabies as a "half-acute miasm" without further elucidation of the terminology in this context, and details its insidious and often fatal course in humans and animals. The term "half-acute" may refer to the delay between the instant of infection and the development of symptoms, which can be greatly prolonged and occur without local eruption. Once symptoms emerge, rabies acts like an acute miasm, i.e. the patient dies or recovers fully. David Little describes half-acute miasms as follows: "Half-acute miasms are sub acute in nature and reach their crisis after a moderate period of time. . . . diseases such as tetanus, Rocky Mountain spotted fever [a rickettsial tick-borne entity] may be included in this

category. Lyme disease has been specifically posited as a fellow half-acute miasm by Will Taylor, along with malaria:

> "I suspect that we can reasonably add malaria and perhaps typhoid to this list. Reflecting on the nature of Lyme disease, I believe it most reasonably belongs in this category - as an obstinate and excessively tedious acute disease that, although not technically chronic in the manner of psora, sycosis, syphilis and tuberculosis, may play out over a lengthy period of time and express in highly varied and changeable ways."

Whether Lyme disease is more accurately described as an acute, half-acute, or chronic miasmatic disease, this does not obviate our knowledge that Borrelia spp. are a pernicious influence. Their influence can precipitate acute disease which often subsequently awakens the pre-existing miasmatic disease of the individual, or establishes chronic infection with subsequent impingement. In *The Chronic Diseases*, Hahnemann describes how the "great epidemic diseases . . . leave the organism so shaken and irritated, that . . . the psora which was before slumbering and latent now awakes . . ." Let us summarize thus far as follows:

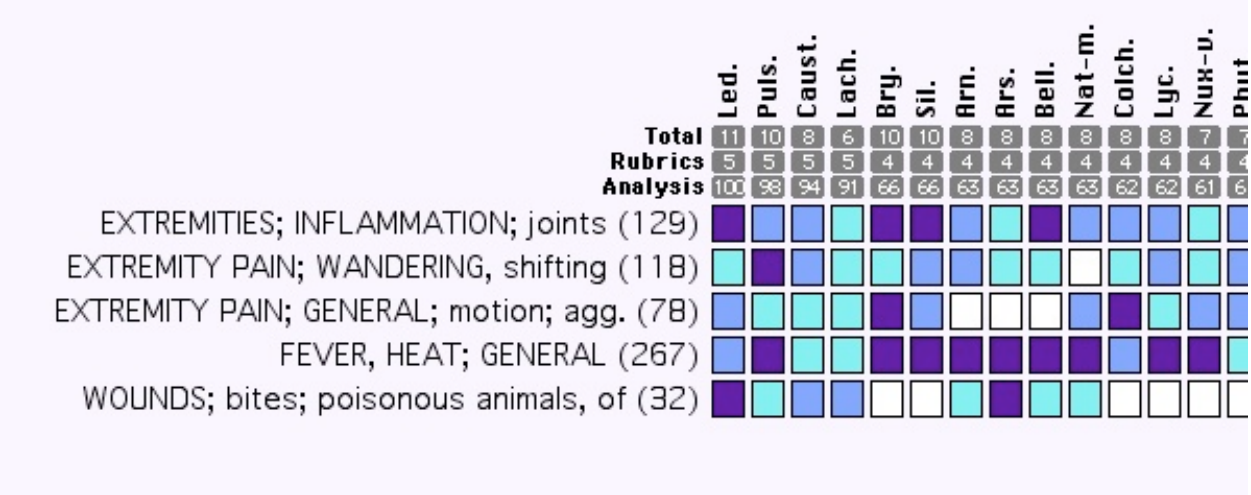
> **Lyme disease is the expression of symptoms in susceptible individuals following infection by Borrelia spp. usually transmitted by a tick bite. Borrelia spp. are agents of disease with a high morbidity and are regionally pervasive. As inciting causes, Borrelia spp. may precipitate uncomplicated acute disease; if incorrectly treated, or in individuals with pre-existing immune compromise, chronic disease commonly results. Chronic disease is individually expressed as psoric, sycotic, or syphilitic in its pattern of symptoms, depending on the constitutional make-up and vital force of the individual. The chronic disease set in motion may or may not be accompanied by persistent borreliosis.**

Is *Ledum palustre* the Answer?

Acute infectious disease may lend itself to cure in many individuals with a single remedy, according to Hahnemann (§241):

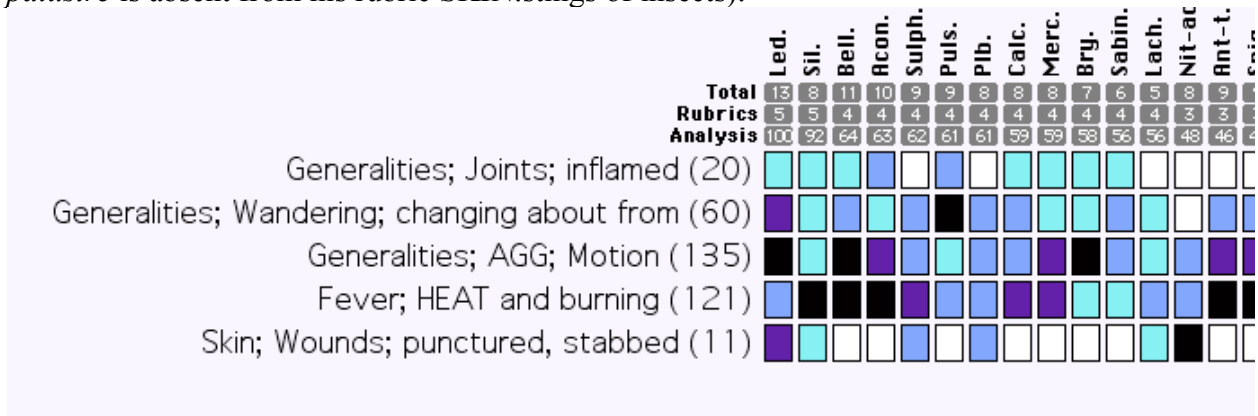
> "If the character of the epidemic disease is discovered according to the symptom complex common to all the patients [i.e., the genus epidemicus], this will point to the homeopathically fitting (specific) remedy for the totality of the cases."

Ledum palustre, a homeopathic remedy derived from a plant in the Ericaceae (heath) family, has frequently been cited as a specific for Lyme disease. It has an affinity for rheumatic conditions and also for puncture wounds and insect bites, which correspond to broad outlines of Lyme symptomatology. If we examine a brief repertorization based on the common presenting symptoms of acute Lyme borreliosis, the remedy emerges clearly:



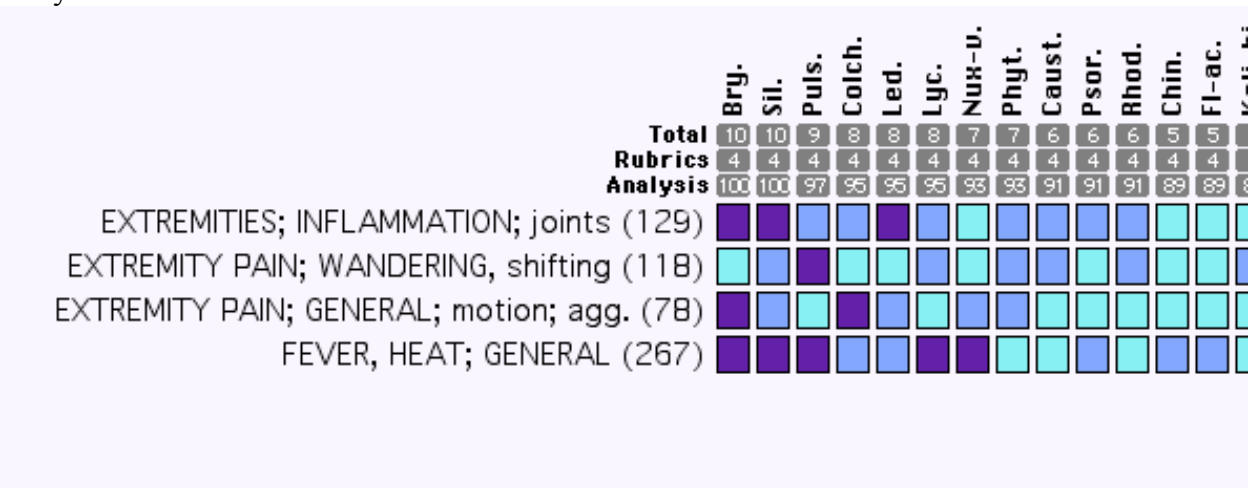
from the Complete Millenium Repertory

And a similar analysis using Boenninghausen's Repertory (although interestingly *Ledum palustre* is absent from his rubric SKIN:stings of insects):



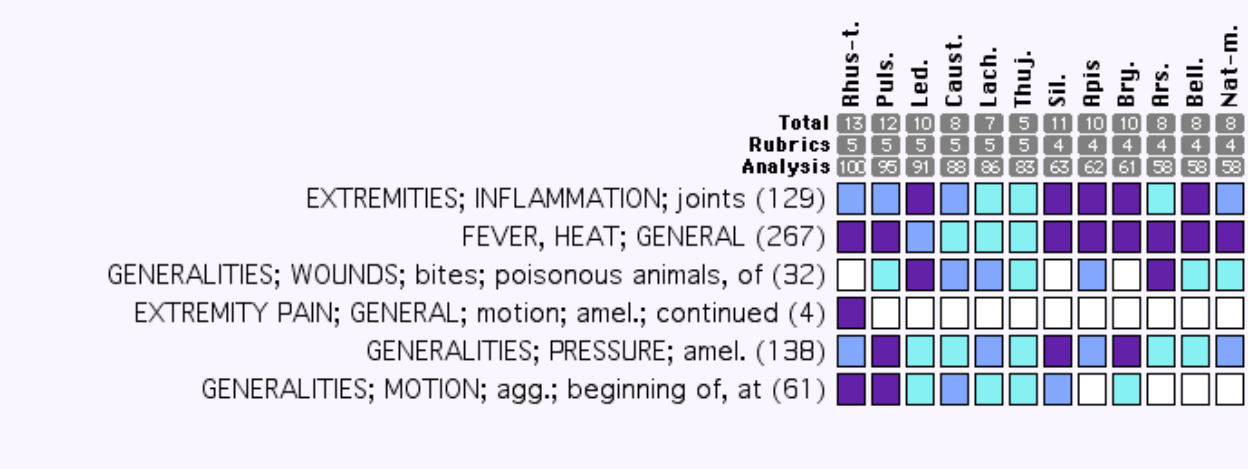
from Boenninghausen's Repertory

Even if we remove the etiological factor of the tick bite, *Ledum palustre* still ranks high in this analysis:



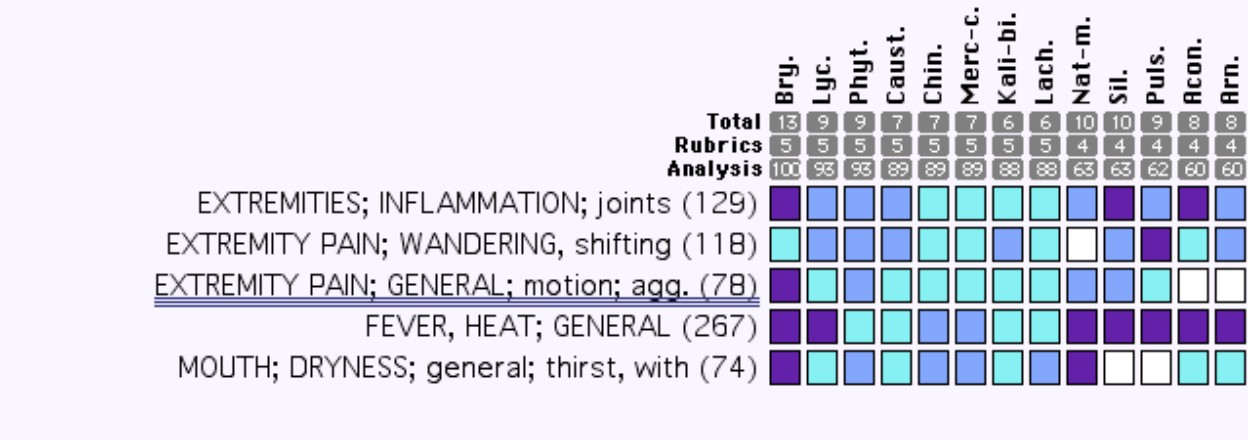
from the Complete Millenium Repertory

But perhaps your patient is exhibiting clear symptoms of acute inflammatory disease that can help you make a more individualized prescription, for example, feels better once up and moving, and prefers to lie with the affected body parts on a firm surface:



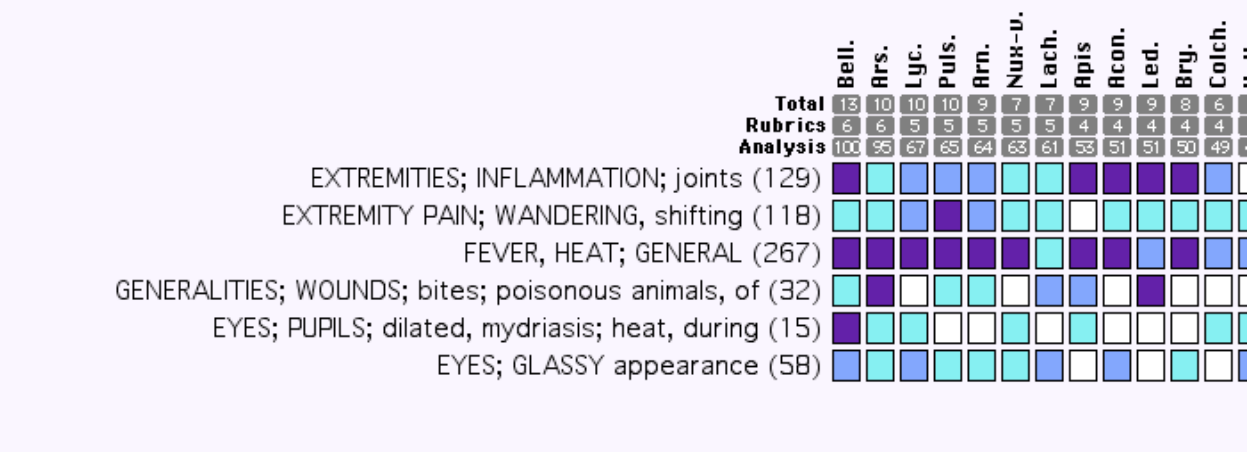
from the Complete Millenium Repertory

Or maybe your patient is exquisite pain with any movement, and you are struck by how dry the gums are, even though there is tremendous thirst:



from the Complete Millenium Repertory

Or maybe your patient is wild and excitable, with glassy eyes and dilated pupils:



from the Complete Millenium Repertory

So you can see how in the absence of highly individualized prescribing or concomitant symptoms, *Ledum palustre* may be appropriate, however, it is by no means a foregone conclusion. It may certainly come into play as an intercurrent remedy, and may be appropriate in some cases as a first-aid remedy given after a suspect tick bite just as for otherbothersome insect (or arthropod) bites.

Another homeopathic approach to treatment is prescription of a Lyme nosode, which may be variably prepared from the ticks, from a Borrelia spp. itself, or some other disease product. It can be difficult in Lyme-endemic areas to administer the nosode prophylactically; the continued exposure may require such frequently repeated dosing that an overdose, or proving, of the remedy may result. It is possible that the nosode might have some benefit as an intercurrent prescription, or in the absence of a clear constitutional prescription, however this presupposes a "Lyme state" in the individual to which the nosode would be homeopathic or isopathic. Lyme nosodes await published provings.

Other homeopathic remedies of interest have been described in conjunction with the treatment of Lyme disease at various temporal stages. Peter Alex, in *The Homeopathic Treatment of Lyme Disease*, cites extensive clinical success with humans using *Aurum arsenicosum*. A proving of the remedy is detailed in his book. In his experience, *Aurum arsenicosum* addresses what he regards as a syphilitic component of Lyme miasm he describes as endemic in his region of Germany in the human population.

Because the conventional treatment for Lyme borreliosis (with or without clinical disease) is antibiotics, and because many patients acutely affected with Lyme disease respond clinically at least initially to antibiotics, veterinary homeopaths often do not see Lyme disease in the initial presentation. In fact, due to the increased prevalence of antibiotic administration based on positive serology but no clinical disease, by the time we see these patients they may have not only chronic disease but possibly induced drug miasm as well. Antibiotics have many well-documented effects; they may reduce (but rarely eliminate) bacterial numbers and have anti-inflammatory properties (e.g. doxycycline), but may potentially have adverse effects recognized

as side effects (conventionally) or the results of palliation and suppression (homeopathically). It is interesting to note that doxycycline (the most commonly prescribed antibiotic for Lyme disease) is bacteriostatic, not bacteriocidal. The conventional therapeutic goal therefore depends not on primary eradication of the bacteria, but relies squarely on the disease-fighting capabilities of the patient, hoping the Borrelia spp. have been weakened sufficiently by the antibiotic.

Both the short-term and long-term prescription of antibiotics for Lyme disease are hotly debated in the conventional community. Some prescribe a short course of antibiotics initially, sometimes after a tick bite but before any symptoms have a chance to emerge (assuming the tick was even infective). Some insist that two weeks of antibiotics is useless and four to six weeks are the minimum effective course. Some adhere to repeated "pulse" dosing with antibiotics. It is difficult to know how to compare the effects of antibiotics on this disease, because there is evidence that the symptoms resolve on their own, without treatment, in an experimental setting. The dogs remain seropositive, but asymptomatic. Although antibiotics may hasten clinical improvement, questions must be asked: are antibiotics merely affecting the weaker Borrelia spp. population, leaving the more resistant organisms to establish a latent population? Does the administration of antibiotics affect the individual's ability to develop premunitive immunity? Are antibiotics ever successful in completely clearing an infection? How therapeutically effective are repeated courses of antibiotics? These are questions openly asked by the conventional community.

Let's consider the recommendation of early, aggressive antibiotic treatment after acute infection of Lyme disease. The goal is to eradicate the bacterial infection before it has a chance to disseminate locally or become entrenched distantly. Borrelia spp. have been shown to multiply and spread rapidly from the site of a tick bite. Dogs which are not clinically affected or yet seropositive have numerous Borrelia organisms around the tick bite, and in short order develop local changes in lymph nodes and nearby joints, whether or not they go on to develop clinical disease. This is what Hahnemann says in *The Chronic Diseases* about infection and subsequent development of disease:

> "With respect to the origin of these three chronic maladies [scabies, syphilis, gonorrhea] as in the acute miasmatic eruptional diseases, three different important moments are to be more attentively considered than has hitherto been done: *First,* the time of infection; *secondly*, the period of time during with the whole organism is being penetrated by the disease infused, until it has developed within; and *thirdly,* the breaking out of the external ailment . . . The infections with miasmas, as well of the acute as of the above-mentioned Chronic disease, takes place, without doubt, in *one single moment,* and that moment, the one most favorable for infection."

It certainly seems like a therapeutic challenge to expect antibiotics to definitively resolve an infection at the first or early second period of time. How do we know when to treat? Ticks are prevalent. Once symptoms are demonstrated, infection has resulted in impingement and disease is established dynamically, and infection established physically. It is not practical to keep asymptomatic animals on antibiotics all the time, nor is it established that periodic administration of antibiotics, based on serology only, resolves infection or has any protective effect.

So when homeopaths don't have access to the golden window of acute borreliosis, how do we treat "chronic Lyme disease?" We remember that the borreliosis at this point is incidental and part of a larger picture, that we are treating chronic disease, not just chronic borreliosis; by understanding that the *wesen* of Borrelia spp. acts on the dynamic level of impingement on the life force instantaneously; by looking at our patients, and considering etiology without being restricted by it; by taking our case and making the homeopathic prescription that fits that case.

Cases

AMBER

A Small Remedy in (Alleged) Lyme Disease in a Dog. *New England Journal of Homeopathy,* volume 7 No. 1, 1998. Reprinted by permission. Other cases may be accessed through www.nesh.com.

Jeff Levy, DVM

Signalment: Amber, a 2 year female Rhodesian Ridgeback, belongs to a homeopath

History: previously, one episode of false pregnancy and mastitis (tx *Belladona*). One episode of severe urticaria (tx *Rhus tox*).

Presenting symptoms: watery, bloody, projectile diarrhea.
Initial prescription: *Lachesis* 30C (the author questions acute vs. chronic disease)

First follow-up, 24 hours: diarrhea cleared, appetite and energy improved. New symptoms:heat, swelling and tenderness in the left carpal joint. High Lyme titer, titer type not specified.

Allopathic diagnosis for diarrhea and lameness: Lyme disease.

Assessment: *Lachesis* palliative, similar enough to produce the carpal inflammation, a never-seen-before symptom in this dog.

New prescription: *Euphorbium* 30C. This small remedy covers carpal inflammation, and also watery diarrhea with tenesmus, and prostration. It has an affinity for mucous membranes.

Second follow-up: within 24 hours, rested and "back to her old self" with increased mobility. Mild limp resolved within 2-3 days. At the time of publication, no symptoms had returned within the year.

Third follow-up, 2007, personal communication: Dr. Levy reports that this dog never had a recurrence of Lyme disease symptoms and remained symptom-free until the present time (at age 11). He did not treat her constitutionally at any time although the owner may have done so.

Discussion: We can see in this dog's history, and in Dr. Levy's discussion, that there may have been chronic disease in this dog as evidenced by the episodes of false pregnancy and urticaria.

So there certainly may have been a psoric susceptibility to an acute infection, although chronic disease never appears to have developed further as a result. Good clinical results were achieved with a single dose of a homeopathic remedy in a modest potency.

In order to arrive at his prescription, Dr. Levy studied the affinities of the remedy and gave considerable weight to the concurrent symptoms of diarrhea with joint inflammation. He also evaluated remedy relationship; *Euphorbium* follows *Lachesis* well, which had some similarity to this case.

Note: At the time the article was written, Dr. Levy questioned the validity of Lyme disease, for many of the reasons we have discussed:

> "I have yet to be convinced that Lyme disease is a true disease entity in dogs. Certainly there is a test of antibody titer to *Borrelia burgdorferi*, and dogs with suspicious symptoms often have a high titer. On the other hand, in endemic areas . . . more than half of asymptomatic dogs also have high titers. Furthermore, I have found such a diversity of symptoms in cases that have been diagnosed . . . that I have difficulty seeing this as a single disease entity."
>
> In Amber's case, I believe that the clinical diagnosis is irrelevant. I believe that this was an acute disease . . . But whether we call it Lyme disease or something else, the only certain label for her conditions is a *Euphorbium* state, since that was the simillimum.

Open Questions:

What was Amber's Lyme titer subsequently?

Did she have a positive titer before this episode?

Would she have gotten better on her own?

Would her clinical course have looked different if she had taken antibiotics? Better or worse?

APRIL

Anne C. Hermans, DVM, CVH

Signalment: Female, spayed, golden retriever, date of birth 12/04. On a home-prepared raw meat based diet with some processed food, modest nutritional supplements.

History: Rescued from a shelter, where she received one multivalent modified live vaccine (DHLPP), Rabies vaccine, and was dewormed. Mild episode of contagious cough in March 2005, self-limiting, not treated.

June 2005

Initial presentation. Mild ear/head shaking, owner concerned with allergies. Some scabs from tick bites. No other symptoms.

Assessment: the mild ear symptomatology is characteristic of latent psora; favorable time for constitutional treatment. Owner elects to wait and watch.

April 2006
Negative titers (Snap Idexx at conventional veterinarian) for Heartworm and tick-borne disease. Owner giving monthly heartworm preventative but not applying pesticides.

Very occasional blobs of mucoid eye discharge.

Assessment: further development of latent psora. Owner elects to give 3 year Rabies vaccine (no others), not to treat symptoms at present.

August 25, 2006
Phone consultation: Eyes dramatically symptomatic and bloodshot, with swelling of the lids, mucopurulent discharge, tearing, photophobia. Conventional veterinarian does not see any corneal damage. No recent head shaking or ear symptoms. Occasional slight diarrhea.

Assessment: flare-up of chronic disease. Not acute disease.

Treatment: *Pulsatilla* 200C, single dose, dry. No conventional eye medications given. Follow-up in 3-4 days and again in 1 week.

August 28, 2006
Follow-up physical exam: Eyes greatly improved after *Pulsatilla* and continuing to improve. Other symptoms: small waxy sebaceous cyst on top of head with black waxy discharge. Owner reports tenacious whitish mucoid eye discharge on and off. Behavior steady and overall doing great.

Assessment: acute flare-up resolving, will be necessary to follow up with treatment of chronic disease, which is probably primarily psoric but possibly sycotic in nature. Owner is holding *Sulphur* 200C and *Thuja* 200C.

September 1, 2006
Owner reports the eyes look "pretty good" but are not continuing to improve. She has noticed that April seems itchy—ears, skin.

Assessment: psoric miasm.

Prescription: *Sulphur* 200C. single dose, dry. Follow-up in 4-6 weeks, sooner if needed.

September 18, 2006
E-mail report: cyst has shrunk dramatically and the black discharge has all but vanished. Eye discharge decreased initially but recently has increased again. The discharge overall is looser.

April's eyes are occasionally red. No increase in symptoms was noted within the first few days after the remedy. No new symptoms.

Assessment:
Overall improvement that has lasted almost three weeks. Without confirmatory observed counteraction there is a small chance the prescription is palliative rather than curative, but 200C is considered on the low end of the potency scale for this dog's vital force, in which case a counteraction is less likely to be observed. *Sulphur* probably curative.

Prescription: Repeat *Sulphur* 200C, single dose, dry. Follow-up in 4-6 weeks, sooner if needed.

October 6, 2006:
Responded by second day to above prescription but symptoms have not cleared 100%. A little occasional head shaking, no redness or odor in ears. Sclera are very mildy injected and milky eye dicharge is rare. Skin cyst had regressed almost 100% but is beginning to return.

Assessment:
Probably curative but insufficient potency, possibly palliative. Note the helpful "barometer" of the local lesion (skin cyst). In view of improvements:

Prescription: *Sulphur* 1M, single dose, dry. Follow up in 6-8 weeks, sooner if needed.

October 24, 2006
E-mail report: Incomplete resolution of symptoms. Some eye discharge most mornings.

Assessment: Sluggish response. Psoric miasm blocked by sycosis?

Prescription: *Thuja* 200C, single dose, dry. Follow up in 4-6 weeks, sooner if needed.

November 22, 2006
E-mail report: No apparent response to *Thuja*. Energy seems low and owner has noticed increased fearfulness and sensitivity to noise. Eye discharge is mild to moderate. There is occasional head shaking and there is slight odor to ears but no redness. Cyst on top of head is minimal.

Assessment: *Thuja* was not the correct prescription, as evidenced by the persistence of pre-existing symptoms and the early development of some mental/emotional ones. Better progress with *Sulphur*.

Prescription: *Sulphur* 1M (which the owner had on hand), single dose, dry. Office follow-up soon.

December 1, 2006
Office visit: Eyes look great. No conjunctivitis. Owner reports variable discharge; some days clear, some days more opaque; mild to moderate in frequency. Head shaking increased for 2 days

post remedy and has stopped; no odor or redness. Skin cyst is palpable but small and not discharging. Energy and mood are excellent.

Assessment: Slow and steady curative response. Some observable counteraction this time (temporarily increased head shaking). Will probably need *Sulphur* 10M or another remedy in the future.

December 15, 2006
Positive screening tests (Idexx Snap) at conventional veterinarian for Lyme and anaplasmosis, negative for heartworm and ehrlichiosis. No symptoms referable to tick-borne disease at present or in her history. Treatment with antibiotics recommended. After extensive discussion, owners elect additional blood tests:

Blood Chemistry—normal
Complete Blood Count—normal, no blood parasites detected
Urinalysis—normal, no proteinuria
Lyme C6 Quantitiative Antibody Elisa (Idexx)—191, considered a high titer
No Western Blot performed.

Assessment: April has been exposed at some point to Borrelia burgdorferi and Ehrlichia equi. One could conventionally term this state exposure, sub-clinical infection, or latent infection, depending on one's perspective. However, there has (to date) been **no impingement** on her vital force by these organisms, since there has been no development of disease. There is also no evidence that the organisms are still present. The positive titers are not predictive of future disease.

Plan: April is already under constitutional treatment and her nutrition and lifestyle are optimal. Her owners are doing everything possible to decrease her susceptibility to disease. Given her breed's predisposition to Lyme disease sequelae, we will follow up with diagnostics periodically and the owners will monitor for symptoms closely.

It is also imperative that her homeopathic case management be vigilant. As she has been obliging enough to present us with some responsive external symptoms with which to assess her chronic disease, we can follow her closely, assuming that if we are seeing strong curative responses, we are also treating her for whatever level of bacterial infection she may or may not have.

February 21, 2007:
Called for update, owner reports April is "doing fine." Eyes and ears are not symptomatic.

Assessment: Curative response to *Sulphur* 1M given 11/22/06, or else psora has become latent.

March 2, 2007:
Office visit: Energy excellent. No itching. No head shaking at all. Cyst completely regressed. Eye discharge very occasional, usually none (owner thinks she saw it twice in the past two months).

Assessment: Curative response. Wait, watch, and monitor closely for return or increase in symptoms. Recheck CBC and urinalysis in a few months, with or without rechecking titers.

April 30, 2007:
No symptoms either previous or new. Energy excellent. Owner elects not to repeat titers due to confusing interpretation and expense.

CBC—normal
Urinalysis—normal (no proteinuria)

Assessment: No reason to alter course or increase level of concern. Will likely see symptoms return at some point.

June 26, 2007:
E-mail report: Owner has noticed cyst on head returning slightly, and some occasional eye discharge. Ears are fine. No other symptoms.

Assessment: Gradual return of symptoms as expected.

Prescription: *Sulphur* 10M, single dose, dry. Report in 1-2 months, sooner if needed.

Discussion: On the surface this would seem to be a very straightforward situation, but it bears closer consideration. April's health has been optimized from the start with good nutrition, and she had begun constitutional treatment for minor problems before her exposure to Borrelia spp. (and anaplasmosis). Her resistance to disease is as high as possible. If she had been heavily vaccinated, fed poorly, treated with toxic topical pesticides, and her latent psora ignored, it is more likely that she would have developed clinical disease as a result of her infection. Her continued good care improves the likelihood that she will clear an infection if there is one still present, and be resistant to re-infection. Antibiotics have not been introduced into her system, which reserve them as a fresh tool should the owners elect to treat this way in the future.

This has not been a case of Lyme disease, but Lyme borreliosis. However, April's case management may be taken as an example of an approach to health that supports and strengthens the individual, without disregarding potential problems.

Compare April's current level of health, at the age of 2 1/2 , with the many golden retrievers who start young with a life-time of eye, ear (and skin) problems, cysts, glandular dysfunction, weight problems, and so forth. She is in excellent health and has not needed or received anything other than homeopathic treatment, to which she has responded in the short and long-term. Her health history is not completely uneventful, but by proceeding thoughtfully, we can chart a safe course for her. Her owners are confidently enjoying her robust health while taking a prudent course of action to safeguard it.

Open Questions:

Would placing April on antibiotics lessen her chance of developing disease?

Would antibiotics confer any lasting benefit?

Could April develop Lyme disease symptoms in the future?

ARCHIE

Judy Herman, DVM, CVH (assessments by Anne C. Hermans, DVM, CVH)

Signalment: male golden retriever, date of birth 4/5/2006

Past history: Several vaccines (not for Lyme) in May and June of 2006. Mild yeasty odor in ears.

January 29, 2007
Per-acutely lame and shivering, especially on the right side. Temp 102.4. Extremely painful hindquarters and RF, joints. Ticks removed 2 weeks before.

Western Blot for Lyme: High level of antibody to infection.

Assessment: An acute infectious episode. Most likely some psora underlying. Cannot rule out an acute flare-up of chronic disease, although no previous symptomatology.

Prescription: owners elected doxycycline.

January 31, 2007
After antibiotics were started Archie become more painful, Temp 103, stopped eating. Began vomiting after doxycycline dose was increased.

Assessment: Suppression/side effects/Herxeimer reaction. Antibiotics discontinued.

Prescriptions: *Bryonia* 30C in water, with no change initially reported within the day. Then *Causticum* 30C in water. However, after the fact, the owner said that after the *Bryonia* Archie got up and ate a little, and his temp went down to 102.

February 1, 2007
Temp went up to 102.6 then 106. Refuses/unable to get up. Wants to eat but doesn't. Painful in all limbs and spine, including neck. Lying on right (more painful) side. Cries if approached.

Assessment: progression of disease.

Prescription: *Lycopodium* 1M in liquid. Temp decreased slightly but no change in pain.

Assessment: incorrect prescription. Possibly better initial results with *Bryonia* previously.

Prescription: *Bryonia* 200C in liquid. Within an hour, temp was 102, getting up on his own, ate, was "more himself".

Assessment: palliative or curative response.

February 2, 2007
Temp back up to 102.6, not eating. Rather than repeat the *Bryonia*, another remedy was given. *Bryonia* was considered to have been palliative, although it is possible that since Archie was febrile, another dose of *Bryonia* would have been effective as his fever rose again (repetition being more frequently indicated in acute febrile episodes).

Prescription: *Calcarea carbonica* 200C in liquid, one time. Consider that *Calcarea* follows *Bryonia* well.

February 3, 2007
Within an hour of receiving *Calcarea,* Archie got up and ate, and started playing with the other dog. Temperature now normal. Still slightly lame on the LH. Barking and playful.

Assessment: curative reaction. Wait.

February 5, 2007
"Totally back to himself."

Assessment: acute episode resolved with anti-miasmatic remedy, after initial response to an "acute" remedy.

March 30, 2007
No further symptoms or treatment. Ears smell slightly waxy. Small red line on gums.

Assessment: Some degree of chronic disease still present. Watching and waiting is appropriate. Returning symptoms may include joint pain and fever, or an increase in ear or gum problems.

June 30, 2007
Dr. Herman reports that Archie has had no further symptoms. This spring he lost two of his canine companions, a significant emotional stress, but a crisis was not precipitated.

Assessment: No active chronic disease.

Discussion: Can we infer that this acute episode was actually an acute flare-up of chronic disease, since an anti-miasmatic remedy was required for complete resolution? Not necessarily. Acute infectious disease often requires anti-miasmatic follow-up. *Calcarea carbonica* is Archie's constitutional remedy at present and probably corresponded to his psoric state prior to impingement by Borrelia spp.

If Archie has a flare-up of Lyme disease symptoms in the future, he could be considered to either have "chronic Lyme disease" or "chronic disease triggered by acute Lyme disease". Either way,

the homeopathic treatment would be the same; prescription based on totality of symptoms, with an evaluation of the miasm(s) involved.

Open Questions:

If Archie had received constitutional treatment prior to exposure, would we expect the disease to appear differently?

Is Archie more or less susceptible to re-infection than if he had received a full course of antibiotics, or is there no difference?

Did Archie have acute disease, chronic disease, or both?

If Archie's titer persists, does he have "chronic Lyme disease?"

PROPOSED SUMMARY OF LYME DISEASE

Term	**Description**
Lyme borreliosis	Infection with Borrelia spp. Requires compatible species. Regionally endemic depending on vector/host biology. Assume repeated exposure in endemic areas.
Lyme disease	Clinical symptoms arising from impingement secondary to infection. Requires susceptible species and individual (pre-existing immune compromise, miasmatic disease, chronic inherited disease).
Temporal miasm	Acute or half-acute.
Treatment of initial episode	Consider non-antimiasmatic remedies.
Subsequent treatment	Consider anti-miasmatic remedies, even if clinical recovery is complete.
Miasmatic sequelae	Untreated or ineffectively treated disease may lead to persistent recurrent miasmatic expression of symptoms, or a new complicated chronic disease. Variably termed: chronic Lyme disease, post-Lyme syndrome.
Prevention of infection	Check for ticks twice daily, if possible. Avoid leaf litter and tall grass. Employ non-toxic deterrent shampoos, sprays, and dips.
Prevention of disease	Maximize individual vitality and resistance to disease through non-toxic lifestyle, optimal species-appropriate nutrition, and constitutional homeopathic treatment as indicated. Avoid Lyme vaccine in animals that are seropositive or have any underlying chronic disease to any degree. Consider *Ledum palustre* judiciously after suspicious tick bite.
Diagnostic testing	Antibody tests assess individual immunoreactivity only. Tests do not correlate with disease severity. Tests are not predictive of future disease.

REFERENCES

Alex, Peter. *The Homeopathic Treatment of Lyme Disease.* (2005) HomeopathyWest Publishing, El Cerrito, CA.

Aronowitz, Robert. When Do Symptoms Become a Disease? *Annals of Internal Medicine.* (2001) 134:9 803-808.

Brooks, Wendy. Lyme Disease. *The Pet Health Library, Veterinary Information Network.* (2003) http://www.veterinarypartner.com/Content.plx?P=A&A=1588. Retrieved on June 6th 2007.

Bushmich, Sandra. Lyme Borreliosis in Domestic Animals. *Journal of Spirochetal and Tick-Borne Diseases.* (1994) 1:1.

Bushmich, Sandra. Personal communication. June 5th 2007, Department of Pathobiology, University of Connecticut, Storrs CT.

Center for Disease Control. Appendix Methods Used for Creating a National Lyme Disease Risk Map. *Morbidity and Mortality Weekly Report.* (1999) 48:RR07 21-24.

Chou, Jaime et al. Detection of Borrelia burgdorferi DNA in tissues from dogs with presumptive Lyme borreliosis. *Journal of the American Veterinary Medical Association*. (2006) 229:8 1260-1265.

Close, Stuart. *The Genius of Homeopathy; Lectures and Essays on Homemopathic Philosophy.* Reprinted 1997, B. Jain Publishers, New Delhi, India.

Croke, Cindy *et al.* Occurrence of Severe Destructive Lyme Arthritis in Hamsters Vaccinated with Outer Surface Protein A and Challenged with *Borrelia burgdorferi. Infection and Immunity.* (2000) 68:2 658-663.

Dorland's Pocket Medical Dictionary. Twenty-third ed. (1982). W.B. Saunders Company, Philadelphia, USA.

Epidemiologic Notes and Reports Imported Malaria Associated with Malariotherapy of Lyme Disease – New Jersy. *Morbidity and Mortality Weekly Report.* (1990) 39(48):873-875. http://www.cdc.gov/mmwr/preview/mmwrhtml/00001850.htm, retrieved July 2, 2007.

Fritz, Curtis and Anne Kjemtrup. Lyme Borreliosis. *Journal of the American Veterinary Medical Association*. (2003) 223:9 1261-1270.

Gray, Bill. Ledum Palustre. [reprinted with permission from] *the homeopath, as cited by ReferenceWorks Pro 3.3.1.* 1994(52).

Hahnemann, Samuel. *Organon of the Medical Art.* (1842). Sixth edition edited and annotated by Wenda Brewster O'Reilly (1996). Birdcage Books, Redmond, Washington, USA.

Hahnemann, Samuel. *The Chronic Diseases; Their Peculiar Nature and Their Homeopathic Cure.* (1828) Reprinted 1997, B. Jain Publishers, New Delhi, India.

Hahnemann, Samuel. *The Lesser Writings.* (1841) Collected and translated by R. E. Dudgeon. Reprinted 2001, B. Jain Publishers, New Delhi, India.

Herman, Judy. Case Report. *Personal communication.* Spring 2007.

Heudens-Mast, Henny. *The Foundation of the Chronic Miasms in the Practice of Homeopathy.* (2005) Lutea Press, LLC, Tallahassee, Florida, USA.

Hoskins, Johnny. Lyme Borreliosis remains a laboratory diagnosis. *DVM Newsmagazine.* (2001) May.

Johnson, Lorraine. Lyme disease: two standards of care. *International Lyme and Associated Diseases Society*. (2005) http://www.ilads.org/insurance.html. Retrieved on June 6th 2007.

Kent Homeopathic Association. Lyme. *ReferenceWorks Pro 3.3.1.*

Kent, James Tyler. *Lectures on Homeopathic Philosophy.* (1900). Reprinted 1979, Homeopathic Educational Services, Berkeley, California, USA.

Khasnis, Atul A. and Mary D. Nettleman. Global Warming and Infectious Disease. *Archives of Medical Research.* (2005) 36: 689-696.

Kolata, Gina. Lyme Disease is Hard to Catch and Easy to Halt, Study Finds. *The New York Times.* June 13th, 2001.

Levy, Jeff. A Small Remedy in (Alleged) Lyme Disease in a Dog. *The New England Journal of Homeopathy.* (1998) 7:1 62-65.

Levy, Jeff. *Personal communication,* January 25, 2007.

Little, David. Miasms in Classical Homeopathy. *Homeopathic Online Education.* http://www.simillimum.com/education/little-library/constitution-temperaments-and-miasms/mch/article.php. Retrieved June 6th 2007.

Little, David. Miasms as Contagion and Their Effects. *Hahnemann's Advanced Methods*. (2001) Chapter 3, Lesson 2. *http://www.hoemembers.com/members/lessons/Hahnemanns-Advanced-Methods/Volume-3/Chapter-3/C3-L2.asp*. Retrieved June 30, 2007 by Dr. Jeff Feinman.

Littman, Meryl *et al.* ACVIM Small Animal Consensus Statement of Lyme Disease in Dogs: Diagnosis, Treatment, and Prevention. *Journal of Veterinary Internal Medicine.* (2006) 20:422-434.

Marshall WF 3rd *et al.* Detection of Borrelia burgdorferi DNA in museum specimens of Peromyscus leucopus. *Journal of Infectious Diseases.* (1994) 170:4 1027-1032.

Mogilyansky, Elena *et al*. Comparison of Western Immunoblotting and the C6 Lyme Antibody Test for Laboratory Detection of Lyme Disease. *Clinical and Diagnostic Laboratory Immunology.* (2004) 11(5):924-929.

Pachner, AR. Borrelia burgdorferi in the nervous system: the new "great imitator". *Annals of the New York Academy of Sciences. (1988)* 539:1 56-64.

Saxton, John. *Miasms as Practical Tools: A Homeopathic Approach to Chronic Disease.* (2006) Beaconsfield Publishers Ltd., Beaconsfield, Bucks, UK.

Saxton, John. The 'Miasmatic' nature of Lyme Disease. *Personal communication.* E-mail February 21st 2007.

Sigal, Leonard. Misconceptions about Lyme Disease: Confusions Hiding behind Ill-Chosen Terminology. *Annals of Internal Medicine.* (2002) 136:5 413-419.

Sternbach, George and Charles L. Dibble. Willy Burgdorfer: Lyme Disease. The Journal of Emergency Medicine. (1996) 14(5):631-634.

Straubinger, Reinhard *et al.* Persistence of *Borrelia burgdorfei* in Experimentally Infected Dogs after Antibiotic Treatment. *Journal of Clinical Microbiology.* (1997) 35:1 111-116.

Summers, BA et al. Histopathological Studies of Experimental Lyme Disease in the Dog. *Journal of Comparative Pathology.* (2005) 133:1-13.

Taylor, Will. An Anamnesis of Lyme Disease; Applying the process of determining the Genus Epidemicus to a Subacute Miasm. *Whole Health Now.* (2002) http://www.wholehealthnow.com/homeopathy_pro/wt11.html. Retrieved on June 6th 2007.

Troy, Gregory. Canine Lyme disease still raises debate on definitive diagnosis. *DVM Newsmagazine.* (2003) March.

Wasmoen, Terri L *et al*. Examination of Koch's postulates for Borrelia burgdorferi as the causative agent of limb/joint dysfunction in dogs with Borreliosis. *Journal of the American Veterinary Medical Association*. (1992) 201:3 412-418.

Whitmont, Ronald D. Homeopathy and Lyme Disease. *http://www.homeopathicmd.com/articles_3.html*. Retrieved on June 6th 2007.