Laboratory Tests and Diagnosis for Lyme Disease and Co-infections

by Armin Schwarzbach, M.D., Ph.D.

It is important to detect Borrelia infections at an early stage. The earlier the diagnosis, the easier and more effective the treatment measures (usually antibiotics) and the shorter the ordeal of the infected patients.

This is how you can diagnose Lyme Disease - a Lyme disease involves progressions in stages depending on symptoms and ailments (time specification after the tick bite):

1. Stage I (after days up to weeks): "bull's eye rash" ("Erythema chronicum migrans"), only in 40-70% of all cases), Borrelia lymphocyto-ma, headache, fever, sweats. "Summation peak" (app. 20% of all cases), exhaustion and dizziness, facial palsy (especially with children/pupils).

2. Stage II (after weeks up to months): inflammation of the brain, meninges, spinal cord, any nerve in the body, inflammation of the joints ("arthritis"), joint and muscle pain, inflammation of the eye, liver and kidneys, myocarditis, pericarditis, cardiac arrhythmia.

3. Stage III (after months up to years): Thinning of the skin at the back of the hand, "Acrodermatitis chronica atrophicans", Borrelia lymphocyto-ma, fatigue, lassitude, depression, arthritis, cognitive dysfunctions, cardiac insufficiency, joint inflammations, swelling, tendon inflammations, inflammation of the bursa, vasculitis, myocardial diseases, depression.

Symptoms of Lyme disease occur in bursts with alternating intensity and appearance in contrast to a classic organic illness. Many patients also suffer from slightly higher temperature during the bursts. Co-infections with other bacteria and viruses have been increasing over the past years and often lead to a complicated course of the disease. Often the tick bite is not detected early enough or the acute treatment by the attending physician is not sufficient. The chronic Lyme disease patients often go through a phase of "clinical remission" as they do not only suffer from actual physical and mental ailments, but also from not receiving a reliable diagnosis and the fact that their illness is not taken seriously.

There are three main reasons why a Lyme infection is not immediately detected in daily practice, so that the disease can be cured at an early stage:

1. There is no bull's eye rash ("Erythema chronicum migrans"). Research has proven that this classic symptom of Lyme disease only appears in 40% to a maximum of 70% of all cases.

2. No tick bite detected! Such a bite can be induced even by very small ticks (larvae, nymph). It was not detected because there was no specific skin reaction. In addition, the scientific community now assumes that Borrelia bacteria can also be transmitted by infected insects, feather, louse, mite, tick, and mosquito.

3. Only conventional blood tests have been performed. This was either too early (antibodies can be tested positive only after a period of up to six weeks after the tick bite) or there has been no antibody production in the body, or the cellular stage was not or not sufficiently tested (necessary test: Borrelia IgM- and IgG-ELISA).

First the laboratory diagnosis is crucial for the diagnosis of Lyme disease. There is a difference between the humoral (antibodies) and cellular level (lymphocytes, NK cells). Both must be examined at the same time when a Lyme disease or an essential illness is suspected.

1. Laboratory testing of the humoral level (antibodies): Borrelia IgM- and IgG-EIA (Enzyme Immunoassay) as well as Borrelia IgM- and IgG-Immunoblot

2. Laboratory testing of the cellular level: Borrelia IgG-ELISA (Erythema Chronicum Migrans), CD57+ cells should have been tested positive results of Borrelia IgM-ELISA and Borrelia IgG-ELISA in all. The Borrelia IgM- and IgG-Immunoblot along with the Borrelia IgM- and IgG-ELISA (even with a negative ELISA) is important: The laboratory has to test for VlsE (Variable major protein-like sequence Expressed) in EIA and Immunoblot. VlsE describes the characteristics of the Borrelia as a "chameleon", since it can permanently change the surface structure VlsE in vivo to resist the detection via the immune system. VlsE has the highest sensitivity for the antibody search!

2. Cellular level: a) The Borrelia IgG ELISPOT®-LTT test is informative about the current activity of the Borrelia bacteria and is 20 to 200 fold more sensitive than an ELISA antibodies test.

b) The CD57+ cells indicate the extent of immune response during or after a chronic Lyme disease and are the prognostic factor during and after the antibiotic treatment.

The complete laboratory diagnosis is very complex and - considering the co-infections - has to be put together like a mosaic. This demands experienced Lyme disease analysts who can diagnose and evaluate all other infections as well.

However, the complexity of a Lyme disease infection is not sufficiently covered by this and needs additional laboratory testing. Therefore further internationally accepted methods are applied which are of considerable importance for the therapy both before and after the therapy (e.g. for the cellular level: Borrelia IgG-ELISA and the CD57+ cells).

In practice the following combinations of blood tests have proven useful: Laboratory diagnosis stage I

1. Borrelia IgG- and IgM-EIA incl. VlsE

2. Borrelia IgG- and IgM-Immunoblot incl. VlsE

3. Borrelia IgG-ELISA incl. VlsE

4. Borrelia IgM-ELISA

Necessary "Staging" (evaluation of progression) of Lyme disease during and after an antibiotic or holistic approach to therapy: The above mentioned parameters also have to be checked during Lyme therapy.

Recommended laboratory diagnosis during the therapy:

Stage I

Performance of the 3 tests mentioned above for 4 weeks after beginning of therapy and 8 weeks after the end of therapy.

Stage II and III

Performance of the 4 tests mentioned above every 8 weeks after the beginning of therapy as well as 8 weeks after the end of therapy for 6 months.

For explanation: Borrelia antibodies or titers cannot be "eliminated", but they can exist in the blood for months or even years. After a successful antibiotic therapy Borrelia IgG-ELISA as well as CD57+ cells should have come to a "normal" level 8 weeks after the end of therapy. But - in spite of a symptomatic status after treatment there might still be positive results of Borrelia IgG-ELISA and/or CD57+ cells. Therefore the patient should be considered for a "monitoring" of the activity tests and possible future symptoms. Otherwise there might be the risk of a relapse, a new infection or a co-infection.

“Lyme”... cont’d pg 4
by Shelley White

Missy Mossor has given birth to five infants. Out of those five, she has buried four. Her fourteen year old daughter MacKenzie survived, but is now fighting for her life alongside Missy. The underlying culprit? Lyme disease. This is an exceptionally intricate and heartbreaking story that every mother must hear. When beginning her story, Missy opened by saying, “I could not believe how heartless and cruel people could be.”

Told it was rare for doctors, Missy said became pregnant again within a few months. This time she gave birth to her baby girl, MacKenzie. She was six and a half weeks early, and weighed six pounds and fifteen ounces. Missy could not believe it. MacKenzie was the healthiest looking baby in the unit, with little fat rolls and long dark hair. Unfortunately, this did not prove to be Missy’s happy ending.

Last year Missy found out she has had Lyme disease for the whole time, resulting in all her previous pregnancies complications. Without knowing, she passed Lyme to her daughter. MacKenzie, who is now fourteen, is unable to go to school due to the aggressive progression of her disease. Both her and MacKenzie are debilitated and spend their days within walls struggling to win their battle against Lyme.

MOTHER BURIES FOUR BABIES

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Medical Perspectives

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Please help Support Kim!
She is so close to her goal of raising the needed funds for her Lyme disease treatment and medical expenses.

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**Overview of the most frequent co-infections of Lyme Disease**

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Transmission</th>
<th>Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlamydia</td>
<td>Ixodes ricinus</td>
<td>Fever, headache, muscle pain, fatigue</td>
<td>Doxycycline, Azithromycin</td>
</tr>
<tr>
<td>Ehrlichia</td>
<td>Ixodes ricinus</td>
<td>Fatigue, fever, rash, joint pain</td>
<td>Doxycycline, Minocycline</td>
</tr>
<tr>
<td>Anaplasma</td>
<td>Ixodes ricinus</td>
<td>Fatigue, headache, muscle weakness</td>
<td>Doxycycline, Minocycline</td>
</tr>
</tbody>
</table>

**Diagnosis:**
- **Indirect ELISA:** IgM and IgG antibodies
- **Western Blot:** Confirmatory test

**Therapy:**
- Doxycycline, Minocycline
- Azithromycin, Clarithromycin
- Ceftriaxone, Cefotaxime

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**Chlamydial Pneumonia**

- **Symptoms:** Fever, cough, chest pain
- **Diagnosis:** IgM and IgG antibodies
- **Therapy:** Doxycycline, Azithromycin

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**Ehrlichial Diseases**

- **Symptoms:** Fever, headache, rash, muscle pain
- **Diagnosis:** IgM and IgG antibodies
- **Therapy:** Doxycycline, Minocycline

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**Anaplastic Lymphoma**

- **Symptoms:** Fatigue, fever, rash, joint pain
- **Diagnosis:** Immunohistochemistry
- **Therapy:** Doxycycline, Ceftriaxone

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**Rickettsial Diseases**

- **Symptoms:** Rash, fever, headache, muscle pain
- **Diagnosis:** IgM and IgG antibodies
- **Therapy:** Doxycycline, Minocycline

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**Babesial Diseases**

- **Symptoms:** Fever, headache, fatigue, nausea
- **Diagnosis:** PCR or BARNA in blood
- **Therapy:** Chloramphenicol, Atovaquone

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**Mycoplasmal Pneumonia**

- **Symptoms:** Fever, cough, chest pain
- **Diagnosis:** IgM and IgG antibodies
- **Therapy:** Doxycycline, Azithromycin

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**Lyme Disease**

- **Symptoms:** Fatigue, muscle pain, joint pain
- **Diagnosis:** PCR or BARNA in blood
- **Therapy:** Doxycycline, Minocycline

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**Lyme Disease**

- **Symptoms:** Fatigue, fever, rash, joint pain
- **Diagnosis:** PCR or BARNA in blood
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**Ftreatment**

- **Therapy:** Doxycycline, Minocycline
- **Diagnosis:** PCR or BARNA in blood
- **Therapy:** Doxycycline, Minocycline
Lyme is a Brain Disease

by Virginia T. Sherr

Lyme borreliosis is a brain disease as well as a multisystemic disease caused by spirochetal bacteria.* Quite frankly, it is an infection that has been burdened with a thousand inaccurate medical diagnoses. The manner in which the current pandemic of tertiary Lyme disease, neuroborreliosis, has usually been handled—either angrily dismissed or strangely misdiagnosed—throughout the 30 years following its “discovery,” has blemished the historic excellence of modern American Medicine. After all the years, neuroborreliosis is still actually considered rare by a majority of physicians, most of whom are spirochetically naive. Officially tallied patients (the numbers showing a dip down to 19,804 cases in 2004 after flawed reporting styles were instituted), when combined with uncounted cases may approach upward of an annual quarter million new borreliosis infections in the USA alone. And Lyme infections have been verified as present on all but one continent, globally. The disease is more common than not accompanied by several of a half dozen or so of the other serious tick-borne co-infections that currently have been identified. Losses of acuity in the human brain’s visual cortex have been observed as early as 6 hours following the toxic bite of an infected tick. Lyme may persist after too brief a period of treatment if or if there has been no treatment, and may result in chronic infections whereupon Lyme borreliosis becomes a potential cause of every symptom in medical and psychiatric lexicons. It is the “Great Imitator” of this Millennium, spirochetal paresis (neuro-syphilis) having its precursor and its model. Chronic or persistent Lyme disease—neuroborreliosis—seldom is identified by the symptoms of its most frequent form—manifest encephalitis—an infected inflamed brain as well as an infected nervous system. However, this is the form in which it most commonly exists. Unfortunately, the tendency that is falsely considered typical—a bull’s eye rash, fever, positive Elisa test, and/or a swollen large joint—occurs in fewer then half of proven cases. Instead, Lyme borreliosis confirms itself in subtle to profound neuro-psychiatric symptoms such as overdriving confusion, loss of organizational skills, decreased concentration, memory loss, mood disorders, irritability, and uncompromised rages—to mention just a few. These symptoms can be very obvious to an experienced professional practicing in a Lyme-endemic area. However, cerebral-behavioral symptoms of neuro-Lyme remain invisible to those whose diagnoses are solely based on old-fashioned concepts limited only to the aforesaid doctor-viewed rashes, swollen knees with positive Elisa blood tests. Blood tests completed by local labs most frequently show false negatives due to general laboratories’ inadequate understanding of proper diagnostic technique and choices of poor quality spirochetal samples on which to base tests. Of course, insurance companies prefer their negative tests. As mentioned, Lyme can rapidly go from Stage One (Early borreliosis) to Late (Tertiary) Stage disease following attachment of an infected deer tick’s or other vector’s bite so that quick and competent treatment are of the greatest importance. Later, accurate findings by sophisticated laboratories may be helpful, especially if Late Stage symptoms appear many years after the infection. Over the years, I have been asked to come a compendium of my published and unpublished works on the subject of Borrelia’s neuro-psychiatric epidemic. These literary contributions advocate for correction of medical neglect—the usually inadequate, sometimes cruel, diagnostic and treatment neglect experienced by victims of chronic Lyme borreliosis and its co-infections. I also have had articles published in an effort to attract attention from Organized Medicine—attention badly needed on behalf of a nearly invisible but serious epidemic that is more significant by far than anything this country has experienced since the Spanish Flu of 1918, the causative spirochete being less immediately deadly than was the virus of that epidem- ic, but deadly, nonetheless, cerebrally. Sadly, Organized Medicine has mostly ignored or deserted the field of neuro-Lyme’s immense pro-

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**Methylcobalamin Treatment in Lyme Patients (Methyl Vitamin B12)**

by Erin Era, PharmD

Candidate at the UMES School of Pharmacy Class of 2013

Lyme disease is a potentially devastating condition which can perpetuate many concurrent clinical abnormalities, including a deficit in Vitamin B12. Lyme disease is caused by the spirochete Borrelia burgdorferi which is most commonly transmitted by a bite from an infected tick, but recent studies do show that there is a chance that the spirochete may be transmitted by infected mosquitoes as well. Once this spirochete has entered the bloodstream, it can take as little as 12 hours for it to enter the central nervous system. There is now evidence that Borrelia burgdorferi produces potent neurotoxins that can cause the symptoms of encephalopathy and cause chronic inflammation, causing neurologic degeneration. There have also been studies that show Borrelia burgdorferi inhibits and kills B and T cells and decreases the count of the CD-57 subset of the natural killer cells, all of which are imperative for an adequate immune response, not only to Lyme disease which is then allowed to perpetuate, but also to other opportunistic infections that may arise. Along with vital immune system components, other nutrients become depleted in the patient with chronic Lyme disease, among these is Vitamin B12.

Vitamin B12 is a supplement that has been used in the treatment of neurodegenerative diseases such as peripheral neuropathy, diabetic neuropathy, multiple sclerosis, and for the preliminary treatment of amyotrophic lateral sclerosis (ALS or Lou Gehrig’s disease). A recent look Lyme disease treatment includes the addition of Vitamin B12 to help heal problems within the central and peripheral nervous system, increase immune function, regulate sleeping patterns and increase energy levels.

Vitamin B12 is available as Cyanocobalamin, Hydroxycobalamin, and Methylcobalamin. Cyanocobalamin is available as an over-the-counter supplement or as a prescription intramuscular or subcutaneous injection. This form of Vitamin B12 is artificially manufactured by a process that includes bacterial fermentation and the addition of potassium cyanide for stability; so, as the name would indicate, Cyanocobalamin contains cyanide. The cyanide is an appropriate form of Vitamin B12 for most people looking to supplement their B12 intake; however, this is not the active form in the body. In order for our body to be able to use Cyanocobalamin, it needs to be converted to Methylcobalamin in the liver, the active “Vitamin” and the most highly reduced form of Vitamin B12. If this conversion does not happen, our brain cannot reap the benefit of methylcobalamin. For those affected by Lyme disease, using the activated methylcobalamin assists a body already in distress by avoiding hepatic conversion.

Vitamin B12 in its active form, Methylcobalamin, along with folic acid (L-methylfolate), are both used to methylate the amino acid homocysteine to methionine. Since adequate amounts of methionine cannot be achieved through diet alone, increasing the conversion of homocysteine to methionine, through Methylcobalamin supplementation helps to supplement the amount of methionine that can be used for SAM-e (S-adenosyl methionine) production. SAM-e production is accomplished by the condensation of methionine and ATP. Some of methylcobalamin’s neuroprotective properties have been proposed to be dependent on this increased production of SAM-e which seems to aid in nerve regeneration and protection. Along with the neuroprotective aspects of methylcobalamin, it has been shown to not only increase sleep quality, but also concentration and feeling refreshed after sleep.

Methylcobalamin is the most highly reduced form of Vitamin B12 and is already in its active form, this makes it a potent antioxidant. A patient with Lyme disease is undergoing oxidative stress, which may inhibit the ability to produce Methylcobalamin. Using methylcobalamin and L-methylfolate in their “methylated/active” forms is also beneficial for a small population of patients who have a mutated MTHFR gene. A patient with a mutated MTHFR gene does not methylate well and would not be able to synthesize in vivo active methylcobalamin from cyanocobalamin or activate folic acid to L-methylfolate. For these reasons the use of methylcobalamin amin already reduced and in its active form may be preferred over cyanocobalamin. Methylcobalamin is a prescription medication that will need to be compounded. It is not absorbed when taken orally, so intramuscular or subcutaneous injections are the preferred delivery routes. Although injection is the preferred route of administration, sublingual tablets as well as intranasal sprays are available. Algae is expected to turn red shortly after each dose. If red urine or red stool occurs, the current dose may need to be evaluated and possibly lowered. For more information on how Methyl B-12 might be beneficial for you, call Community Compounding Pharmacy at 1-855-LymeRx or send your inquiry to compounding specialist, Melissa Ruark at melissa@communitypharmacy.com.

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### Sex & Lyme Disease

**by Dr. Robert C. Brillnsfield**

How does chronic Lyme disease affect sexual functioning, and how can it be treated? Lyme can affect several systems, including the nervous system, which can have a direct effect upon the central nervous system, the endocrine system, the peripheral nervous system, the gastrointestinal system, and/or the body. It is well recognized that B. burgdorferi (BB) causes depression, obsessive-compulsive disorder, and psychosis that are functions of the emotional averse pathway of the brain. However, we can also see dysfunction of the reward pathways as well as the desire for pleasure, feeding, bonding and sex. Since Lyme disease alters the afferent pathways which affect what and who we are repelled from, it is understandable that Lyme can also alter sexual attraction and behavioral patterns as well. In this area, we shall begin with some patient accounts and observations.

### Sexual arousal:

Some patients report a decline in both libido and overall sexual functioning. Some patients report that activity in sex and sexual functioning remain normal while a few notice only that they exist in pursuit of seeking help for their ail-

### Children:

To their everlasting shame, medical authorities have stood by while inno-

### Risk factors:

- **PCR on Rickettsia in blood (EDTA-blood):** direct detection
- **Antibodies Rickettsia IgM and -IgG:** indirect detection - control of pro-
- **Arthritis, tendovaginitis**
- **Chlamydia trachomatis**
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- **Pathogen:** Chlamyphila pneumoniae (gram-negative, intracellular)
- **Transmission:** sexual contact, human to human
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“The new delivery system and pleasant taste make Tri-Fortify™ Orange an outstanding product.”

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