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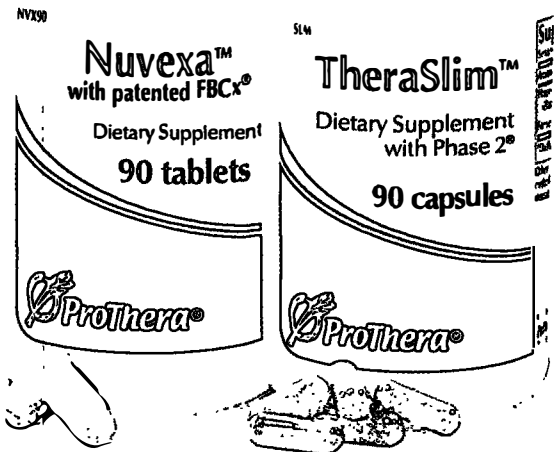
† Dyerberg, et al. Bioavailability of manne n-3 fatty acid formulations. *Prostaglandins Leukot Essent Fatty Acids* 2010 Sep;83(3):137-141

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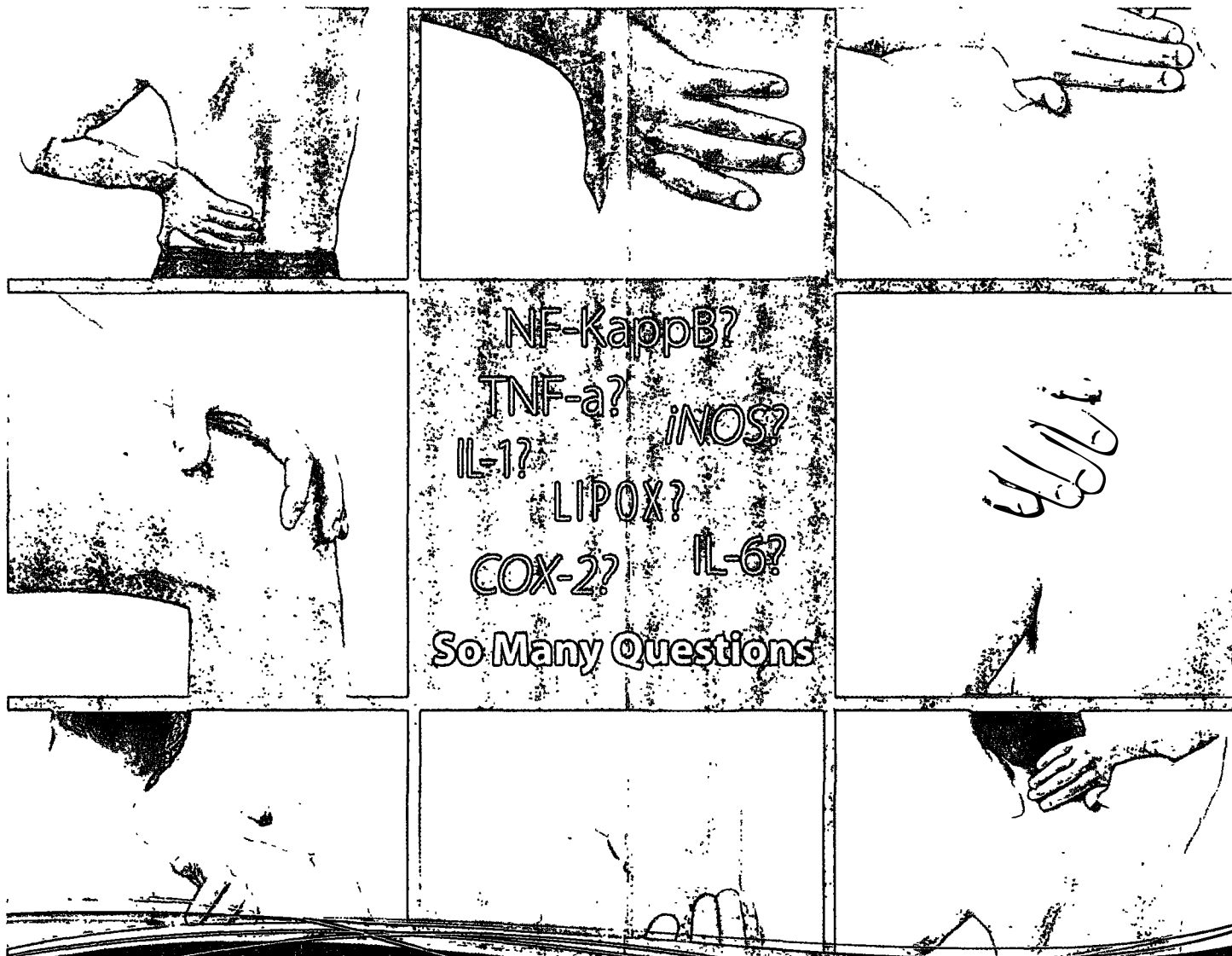
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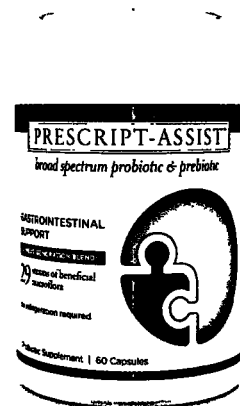
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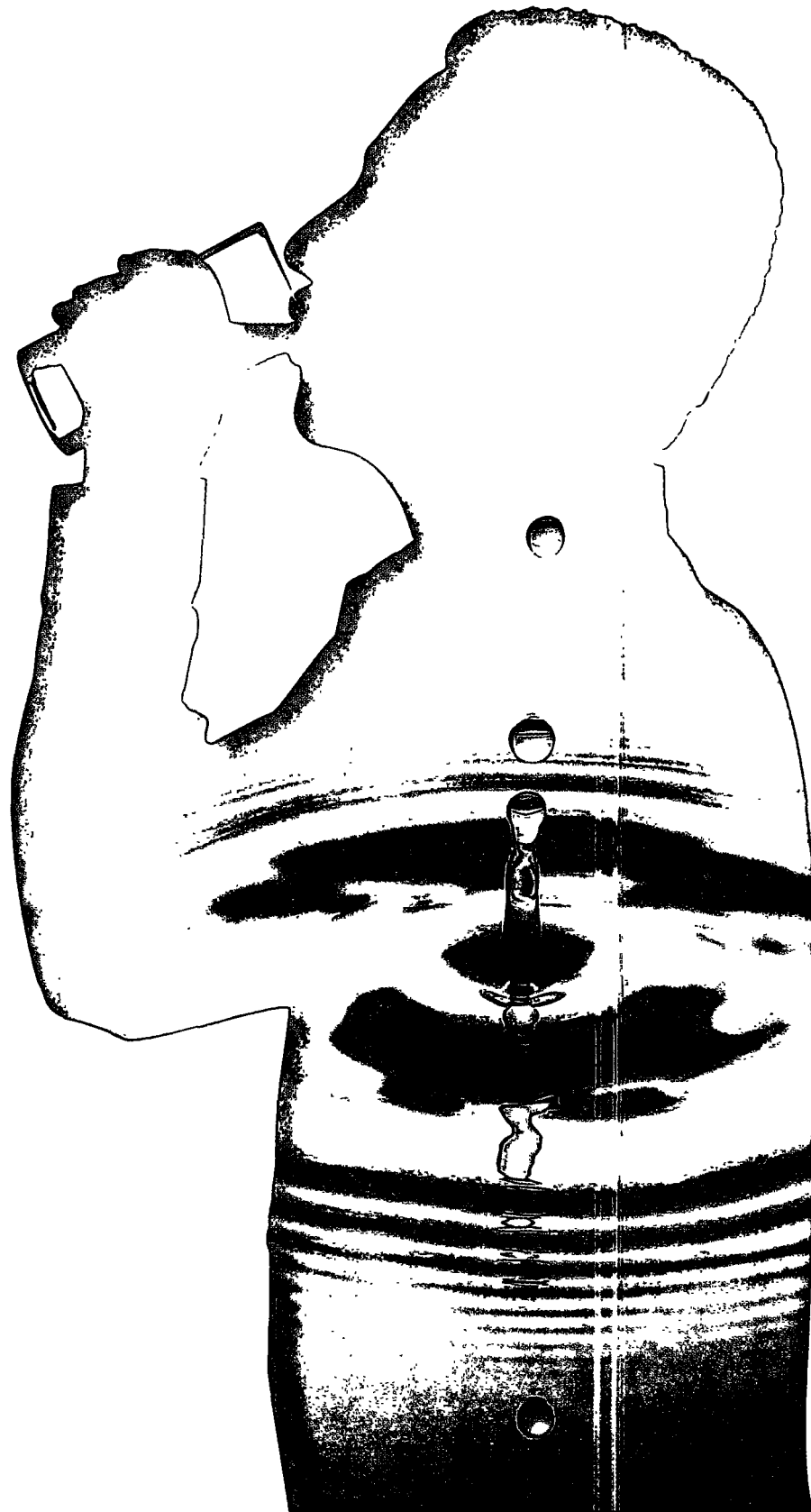
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¹ Pennis, E. (2011) Body's Hardworking Microbes Get Some Overdue Respect *Science*, 330 (December 2010), 1619





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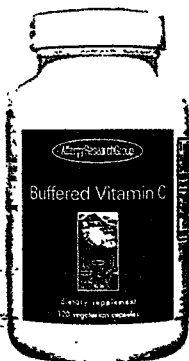
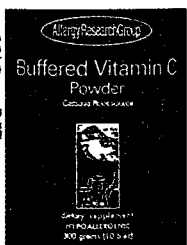
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From the Publisher

Coffee 'No-No' Advice is a 'No-No'

I'm a coffee drinker, probably a coffee "addict," so I won't claim that this is an unbiased report (however, I am not paid any fees by the coffee industry). But for all too long, we have been giving coffee drinking an unjustified bad reputation. How many lecturers on stress management have told us to just say "no" to coffee drinking? Caffeinated coffee supposedly overworks the adrenal glands, interferes with refreshing sleep, makes us jittery and agitated, and prolongs anxiety and panic attacks. A weakened adrenal system leads to excess production of cortisol, disrupting the immune system – why would we want to drink something

that is so important in disrupting our health? And as much as I love homeopathy, it has always bothered me that practitioners advise patients that drinking coffee inactivates the homeopathic remedy. Such advice has always kept me from dedicating my practice wholly to homeopathic medicine. Perhaps one of the most misguided tenets of parenting has been the prohibition of coffee drinking by teenagers and preteens – based on fear that coffee will stunt the child's growth. While the anxiety-prone may benefit from a reduction in coffee drinking, there is little evidence that a moderate consumption of coffee increases our risk

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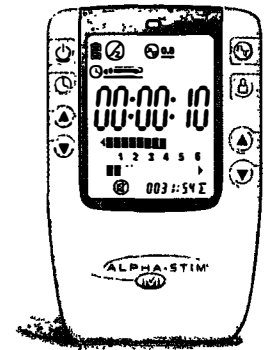


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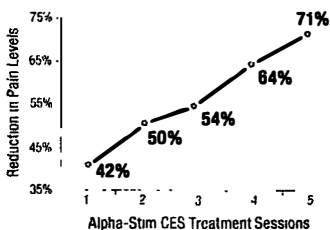
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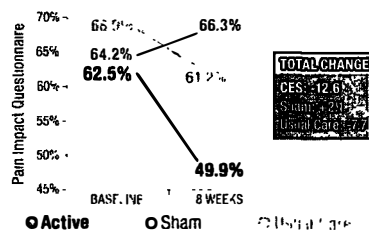
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REFERENCES

¹ Holubec JT. Cumulative response from Cranial Electrotherapy Stimulation (CES) for chronic pain. *Practical Pain Management*. 2009, 9(9) 80-83
² Taylor AG, Anderson JG, Riedel SL, et al. Cranial Electrotherapy Stimulation improves symptoms and functional status in individuals with fibromyalgia. *Pain Management Nursing*. 2013 Dec 14(4) 327-335
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Letter from the Publisher

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of developing serious illness, and the opposite actually appears to be true. As for the homeopaths, it would be nice if someone would run a study among coffee-drinkers and nondrinkers to see if there is a measurable difference in homeopathic remedy response. Aaron Carroll, MD, a professor of pediatrics at Indiana School of Medicine, provided a nice review of recent and past coffee studies in the *New York Times* (May 11), "Upshot: More Consensus on Coffee's Benefits Than You Might Think."

In a paper published by the American Heart Association in 2013, a meta-analysis of prospective cohort studies from 1966 to 2013 demonstrated a significant reduction in risk for development of cardiovascular disease in moderate coffee drinkers.¹ The study was based on 1,283,685 study participants and 47,779 cardiovascular disease cases, including 28,347 coronary artery disease cases and 12,030 stroke cases. The study found an inverse relationship between the development of cardiovascular disease and coffee drinking. Moderate coffee consumption (3–5 cups/daily) was associated with a significant reduced risk for developing cardiovascular disease; heavy coffee consumption (6 cups/daily) was not only not associated with an increased risk but showed a slightly lower risk

compared with non-coffee consumption. Other studies done previously did not show the reduction in risk as conclusively as this study.

Unfortunately, there are no meta-analyses of prospective cohort studies of the same magnitude published for cancer. However, most of the smaller cancer studies either demonstrated a reduction in the risk for developing cancer or no increase in risk. A meta-analysis done in 2007, published in *Gastroenterology*, found a 43% reduced risk of liver cancer in individuals drinking 2 or more cups of coffee daily.²

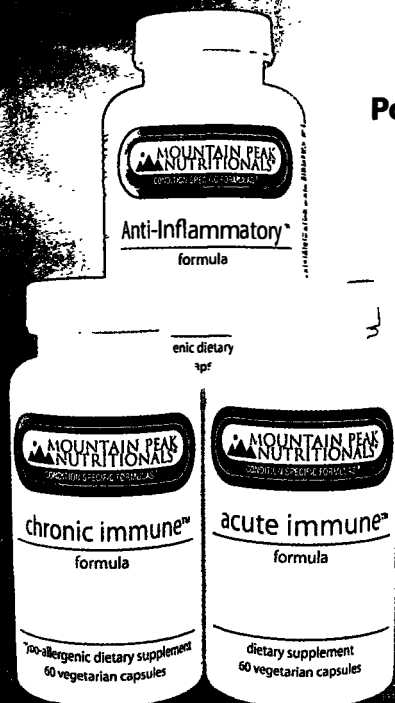
A more recent meta-analysis done in 2013 confirmed that coffee drinking confers reduced risk for developing liver cancer.³ There was a 40% reduction in the relative risk for developing liver cancer among coffee drinkers, and the risk decreased more with increased coffee drinking.

Neurologic disease, including Parkinson's disease and cognitive decline, had reduced risk with coffee consumption.⁴ The maximum reduction in risk for developing Parkinson's disease was achieved in those drinking 3 cups of coffee daily. What about overall relative risk of death from coffee drinking? Two meta-analyses each examining nearly 1 million participants were associated with reduced risk of death.^{5,6} One of the reports found that the reduction in risk was greater for women than men.⁶

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* See www.neuroscienceinc.com/references

Letter from the Publisher

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For those of us who like to advise patients to stop drinking coffee, think again.

Water Cure and Peloid Therapy

The 59th Annual NW Naturopathic Physicians Convention (NWNPC) held this year in Seattle, Washington, focused on "The Wisdom of Our Elders." While naturopathic medicine makes strides to bring "evidence-based medicine" into the clinic, there is concern that naturopathy should not abandon its roots. Indeed, some worry that the new generation of naturopathic students may not learn, much less practice, the water therapy of a century past. Susanna Czeranko, ND, faculty member at National College of Natural Medicine, lectured about the history of the older naturopathic treatments and their contemporary applications. At a time when medical doctors were still bloodletting and prescribing arsenic and mercury formulas, Father Sebastian Kneipp was successfully treating thousands of patients in Bavaria and elsewhere with his water cure. Benedict Lust, an early 20th-century New York City naturopathic practitioner, wrote about Kneipp's treatments in his journal, the *Naturopath and Herald of Health*.⁷ Lust reported that the local medical societies thought that Kneipp's treatment would fail and be decried by patients

demanding that their fees be returned. Instead doctors from Europe and the US flocked to Kneipp's institutes. Another earlier naturopath, Vincent Priesnitz, advocated the cold water cure for treatment.⁸ Priesnitz's abdominal pack was a universal remedy, appropriate for immediate treatment of acute or chronic conditions. His abdominal bandage required a wet bandage to be applied to the skin, and later the abdomen would be hand-rubbed until dry. Kneipp's abdominal compress used a cold or warm wet towel to be applied to the abdomen, covered by flannel. The treatment would last 1.5 hours. The theory was that as the body reacted to the cold compress, it would create localized heat and increase circulation. Priesnitz preferred to follow his wet compresses with friction hand-drying. Priesnitz thought the healing factor was not the application of the cold but the reaction of the body to the cold, to produce heat.

Could it be that in designing scientifically based treatment protocols, we have abandoned simple therapies that heal more powerfully and effectively?

Czeranko's second presentation, "Russian Peloid Research Revealed," introduced a more contemporary but still old-time treatment that is yielding highly successful results in gynecology applications. The term *peloid* derives from the Greek *pelos*, meaning "clay," and a peloid refers to all external applications of mud, peat, and clay.

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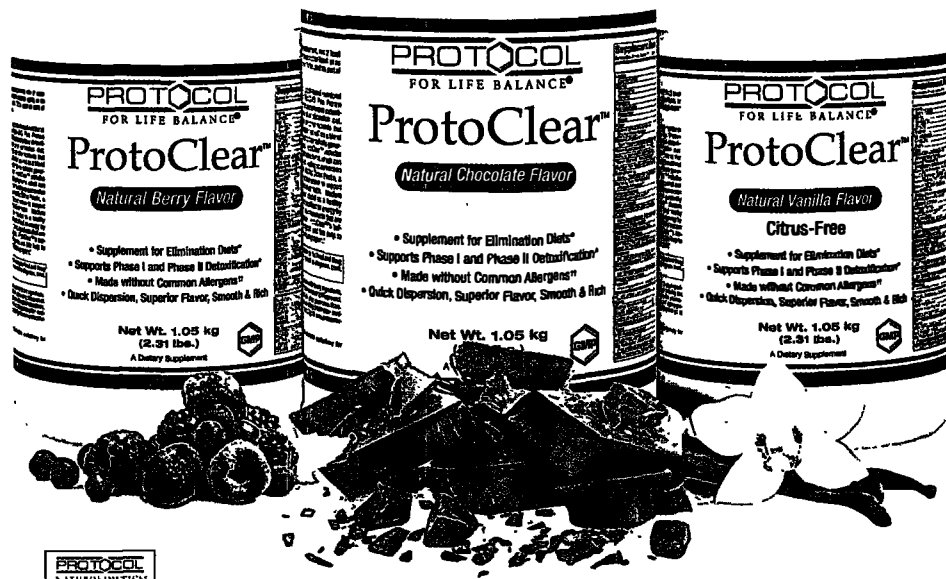
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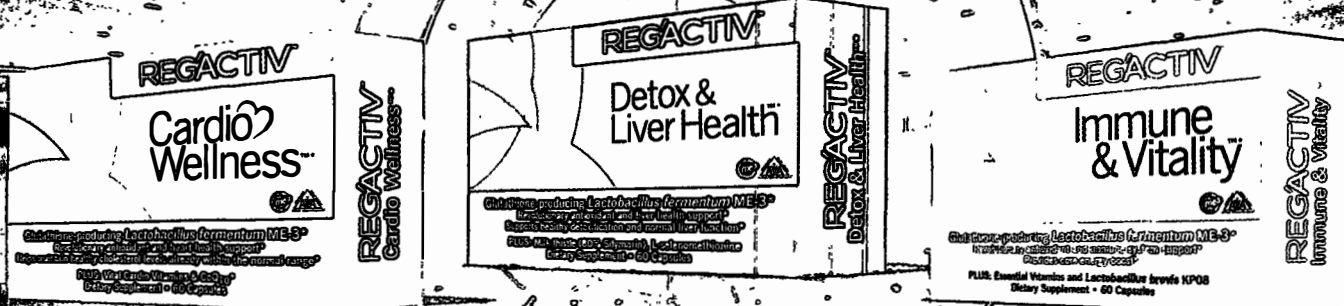
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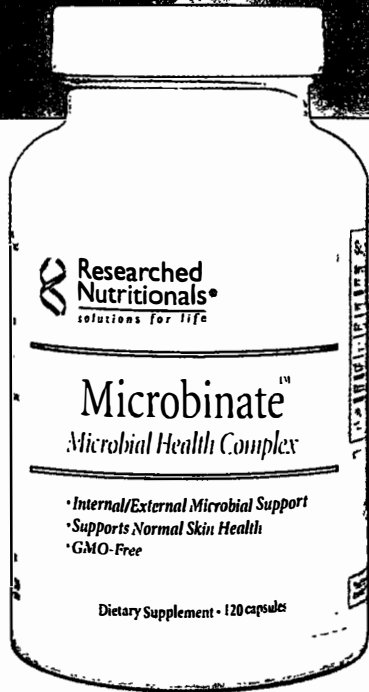
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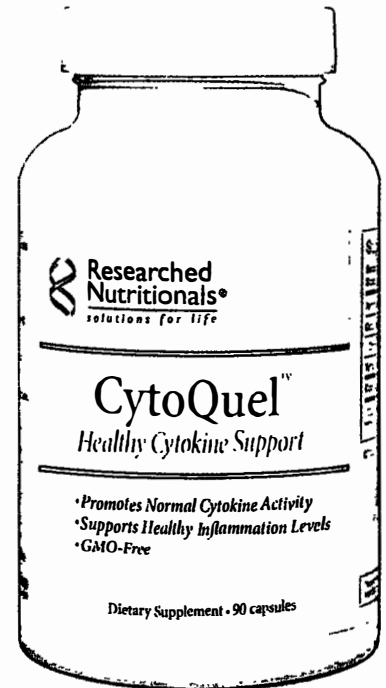
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Parasites aren't much of a concern in the developed world, right? Isn't that something you only have to worry about on vacation? This modern assumption could be causing major factors in patient health problems to be overlooked.

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One of the primary challenges in diagnosing Lyme and similar infections is the difficulty of identifying these pathogens via lab tests. Improved microscopy can help visualize these infections accurately.

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by Margo Roman, DVM, CVA, COT, CPT

Microbiome restorative therapy, commonly known as fecal matter transplants, is often pursued after the patient receives a round of antibiotics, to clear the way for new, healthy intestinal flora. But antibiotics have their drawbacks. Ozone is one possible alternative.

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Letter from the Publisher

► continued from page 10

Czeranko particularly likes the peat peloids. Peat is formed from the longtime decomposition of plants compressed under soils or marshes. In the 19th century, peat was gathered and sold as a heat source, but in Eastern Europe it was also employed in baths as health treatment. Currently it is used in spa treatments in Germany and Eastern Europe. Like the water therapies, peat offers excellent heat retention; however, it additionally contains minerals, salts, enzymes, and microorganisms. Peloids confer similar anti-inflammatory benefits as do NSAIDs, decrease abnormal cell proliferation, support joint mobility, repair mucosal permeability, and are antibacterial.

The peloid wrap is similar to the abdominal compresses described earlier; however, instead of using cold, the peloid material is placed below a heating pad, and the treatment is run for 25 minutes. Peloid wraps are done daily for a total of 10 to 15 treatments. The treatment is thought to increase the blood and lymph circulation. Peloids may be applied locally, and for vaginitis the peloid is introduced into the vagina; the material is removed after 25 minutes. Research done at the Russian Military Medical Academy and published in 2007 demonstrated greater than 90% improvement in women treated for vaginitis.⁹ Moreover, some research shows that peloids offer benefit for infertility, cervicitis, and uterine fibroids.

Peloid therapy may provide a very effective tool in women's health, based on an age-old material.

Unraveling the Enigma of Lyme Disease and Its Coinfections

Perhaps nothing more aptly represents the dichotomy between conventional "evidence-based medicine" and naturopathic medicine than the dilemma of diagnosing Lyme disease and its coinfections. Conventional medicine remains entrenched in a simple diagnostic entity, an acute infection brought about by a tick bite, leading to infection with *Borrelia burgdorferi*, diagnosed with a simple ELISA assay, and treated with an antibiotic for a period of 2 weeks. If the test is negative, then Lyme disease is ruled out. Of course, there is no screening for coinfection. The CAM medical community disagrees. Diagnosing Lyme disease remains challenging, frequently requiring testing that is more sensitive to *Borrelia* infection; coinfection screening must also be done, as other organisms such as *Babesia* and *Bartonella* are sometimes more problematic than *Borrelia*. Once the diagnosis is made, treatment for Lyme disease and coinfections is a long-term process requiring antibiotics, herbal therapies, detoxification, IV support, and more. Such lengthy treatment protocols are critical for the debilitated patient, despite conventional insurance programs' offering little assistance for expensive treatment.

The question is, when we are working up a patient, at what point do we entertain the notion that the patient

may have Lyme disease or *Babesia* or *Bartonella*? Once Lyme disease/coinfection becomes part of the differential diagnosis, do we order a simple ELISA test or do we launch into more sophisticated studies immediately? If the diagnosis is positive, do we engage in warfare, administering cocktails of oral antibiotics, antiparasitics, antifungals, and antivirals? Or do we offer a naturopathic approach, treating what was called by Claude Bernard in 19th-century France the "terrain"? Treating the terrain doesn't combat the enemy; instead the body's physiology is addressed, detoxifying the toxic buildup; reestablishing the healthful microbiome; and supporting the weaknesses of the immune, neurologic, and endocrine systems. Dan Kenner, PhD, LAc, addresses the "terrain" in this issue, discussing the work of Lyme-disease specialists Kristine Gedroic, MD, and Ann Corson, MD, as well as Jean Valnet, MD, and Bernard Christophe.

Bjørn Johan Øverbye, MD, is not only a Lyme-literate physician, he is also a Lyme disease patient who first faced his illness before Lyme disease was a working diagnosis in the 1970s. After bringing his disease under control in the 1980s, he relapsed and faced a much more difficult time in the 1990s getting treated as well as treating patients at a Lyme clinic. Stephen Fry, MD, is looking at *Borrelia* and its coinfections as not merely an IgG or IgM band; his laboratory has developed a number of innovative tests to examine Lyme disease and related microbiologic infection. James Schaller, MD, who has written numerous books on Lyme disease and coinfections, tackles babesiosis, which he defines as "one of the most deadly tick infections." Schaller is convinced that *Babesia* is very difficult to diagnose and equally challenging to treat; yet, without treating babesia, many Lyme disease patients will fail to get better. Scott Forsgren writes about another major Lyme disease coinfection, bartonellosis. Regrettably for cat lovers, nearly half of all cats may be infected with *Bartonella*, even a higher percentage of feral cats.

We are pleased to publish our 2015 update on Lyme disease and its coinfections and hope that you will find these materials useful in the office!

Jonathan Collin, MD

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Laropiprant Is the Bad One; Niacin Is/Was/Will Always Be the Good One

by W. Todd Penberthy, PhD

Orthomolecular News Service

Niacin has been used for over 60 years in tens of thousands of patients with tremendously favorable therapeutic benefit (Carlson 2005). In the first-person *New York Times* best-seller *The New 8-Week Cholesterol Cure*, the author describes his journey from being a walking heart-attack time bomb to a becoming a healthy individual. He hails high-dose niacin as the one treatment that did more to correct his poor lipid profile than any other (Kowalski 2001). Many clinical studies have shown that high doses of niacin (3000–5000 mg of plain old immediate-release niacin taken in divided doses spread out over the course of a day) cause dramatic reductions in total mortality in patients who experienced previous strokes (Creider 2012). High-dose niacin has also been clinically proved to provide positive transformational relief to many schizophrenics in studies involving administration of immediate release niacin in multi-thousand-milligram quantities to greater than 10,000 patients (Hoffer 1964; Osmond 1962). Most importantly, after 60 years of use, niacin (especially immediate-release niacin) remains far safer than the safest drug (Guyton 2007).

Bad Reporting

So why have the media suddenly presented the following niacin alarmist headlines in response to the most recent study in the *New England Journal of Medicine*?

“Niacin Drug Causes Serious Side Effects, Study Says” – *Boston Globe*; July 16, 2014

“Niacin Safety, Effectiveness Questioned in New Heart Study” – *HealthDay News*; July 17, 2014

“Doctors Say Cholesterol Drug Risky to Take” – *Times Daily*; July 16, 2014

“Niacin Risks May Present Health Risks Claim Scientists” – *Viral Global News*; July 17, 2014

“Studies Reveal New Niacin Risks” – *Drug Discovery and Development*; July 17, 2014

“No Love for Niacin” – *MedPage Today*; July 17, 2014

“Niacin Could Be More Harmful Than Helpful” – *Telemanagement*; July 18, 2014

The truth of the matter is that the study quoted and used laropiprant (trade names: Cordaptive and Tredaptive). Laropiprant is a questionable drug, and the results say next to nothing about niacin. The study compared over 25,000 patients treated with either niacin along with laropiprant, or placebo. The patients in this study had previous history of myocardial infarction, cerebrovascular disease, peripheral arterial disease, or diabetes mellitus with evidence of symptomatic coronary disease. The side effects observed in those who took the laropiprant-niacin combination were serious and included an increase in total mortality as well as significant increases in the risk for developing diabetes.

For responsible reporters, this should have raised the question of which compound, the drug laropiprant, or the vitamin niacin, is the culprit.

Such side effects have not been seen in over 10 major clinical trials of niacin involving tens of thousands of patients, not in over 60 years of regular usage of niacin in clinics across the country. However, niacin causes a warm flush on the skin. Some people find the warm niacin flush

uncomfortable, although many enjoy this temporary sensation. In this study, niacin was given in combination with laropiprant, a drug that prevents the niacin flush. By including a dose of laropiprant along with the niacin to eliminate the flush, the thought was that more patients could benefit from niacin without complaint. But in fact the niacin flush is healthy. A reduced flush response to niacin is a diagnostic for increased incidence of schizophrenia, and this assay is now widely available (Horrobin 1980; Messamore, 2003; Liu 2007; Smesny, 2007).

Problems with Laropiprant

So what about the other half of the combo, the drug laropiprant?

- Laropiprant has never been approved by the FDA for use in the US and when taken alone has been shown to increase gastrointestinal bleeding.
- Laropiprant interferes with a basic prostaglandin receptor pathway that is important for good health.
- Last year Merck announced that it would withdraw laropiprant worldwide due to complaints from continental Europe. Therefore the clinical trials in this most recent study could only be performed in the UK, Scandinavia, and China.

So why did so many media outlets and even some MDs conclude that niacin was the problem? Simple: none of the headlines mentioned laropiprant, which is quite clearly the real culprit that caused the side effects reported. The simplest way to put it is to say that sensational stories promulgated by the media are quite often completely wrong. This suggests a hidden agenda.

Confusing and fantastical headlines can increase readership for hysteria-based business models. Which headline is likely to garner the greatest attention, "Laropiprant is a Dangerous Medication that has Not Been Approved by the FDA" or "Niacin Causes Serious Side Effects"? The correct headline would be, "Niacin Doesn't Cause Serious Side Effects; Drugs Do."

Why the B Vitamins Are So Important

The B vitamins were discovered due to terrible nutritional epidemics: pellagra (niacin/vitamin B3 deficiency) and beriberi (thiamine/vitamin B1 deficiency). We are very sensitive to a deficiency of niacin. Over 100,000 people died in the American South in the first two decades of the 20th century due to a lack of niacin in their diet. It was perhaps the worst nutritional epidemic ever observed in modern times, and was a ghastly testament to how vulnerable the human animal is to niacin deficiency. The pellagra and beriberi epidemics took off shortly after the introduction of processed foods such as white rice and white flour. Poor diets, mental and physical stresses, and certain disease conditions have all been proved to actively deplete nicotinamide adenine dinucleotide (NAD) levels, causing patients to respond favorably to greater than average niacin dosing.

How is it possible that niacin can be useful for many different conditions? It seems too good to be true. The reason is that niacin is necessary for more biochemical reactions than any other vitamin-derived molecule: over 450 different gene-encoded enzymatic reactions (UniproKB database of the Swiss Institute of Bioinformatics; Penberthy 2013). That is more reactions than any other vitamin-derived cofactor! Niacin is involved in just about every major biochemical pathway. Some individuals, who have a genetically encoded amino acid polymorphism within the NAD binding domain of an enzyme protein, will have a lower binding affinity for NAD that can only

be treated by administering higher amounts of niacin to make the amount of NAD required for normal health. Genetic differences such as these are why many individuals require higher amounts of niacin in order for their enzymes to function correctly (Ames 2002).

It is a deadly shame that the media so often ignores this information. Fortunately, many physicians will see through the recent headlines that give misinformation about niacin, having already personally witnessed how effective high-dose niacin therapy is for preventing cardiovascular disease.

Nutrients are the Solution, Not the Problem

So what is the solution? At the end of the day, the data on patients with problem cholesterol/LDL levels still support 3000 to 5000 milligrams of immediate-release niacin as the best clinically proven approach to maintaining a healthy lipid profile. Niacin in 250 mg to 1000 mg doses can be purchased inexpensively from many sources. Extended-release niacin is the form of niacin most frequently sold by prescription, but it has more side effects than immediate-release (plain old) niacin ... and it costs much more.

Tangential to niacin but pointed to cardiovascular disease, conventional medicine is finally beginning to respect chelation therapy as an approach owing to the recent unparalleled positive clinical results for cardiovascular disease patients with diabetes – up to 50% prevention of recurrent heart attacks and 43% reduction in death rate from all causes (Avila 2014). Sometimes chelation therapy can be expensive. However, there are other inexpensive approaches include high-dose IP6 therapy that are yet to be conventionally appreciated. Other supplements desirable for any ideal cardiovascular disease: a nutritional regimen including additional vitamin C, magnesium, coenzyme Q, fat soluble vitamins (A, D, E, and K2), and grass-fed organic butter. Your ideal intake varies with your individuality.

Nutrients such as niacin you need.
Media misinformation you don't.

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Shorts

briefed by Jule Klotter
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Hyperbaric Oxygen Therapy for Chronic Lyme

Since the 1990s, hyperbaric oxygen therapy (HBOT) has been used as an effective adjunctive treatment for chronic Lyme disease, according to Chien-Yu Huang and colleagues. HBOT is the administration of 100% oxygen to patients in pressurized chambers. Breathing oxygen at atmospheric pressure above sea level (sea level = 1 ATA) increases the oxygen level in body tissues. Higher oxygen levels boost white blood cells' ability to kill pathogens and inhibit the growth of anaerobic bacteria. In 2014, Chien-Yu Huang et al. published a case report detailing the use of HBOT in a 31-year-old man with symptoms of chronic Lyme disease.

The subject of the case report displayed erythema migrans lesions (bull's-eye rash characteristic of Lyme) on his legs in January 2004. He also reported joint pain in his knees, shoulders, and temporomandibular joints. The man's *Borrelia* serology IgG was positive. His symptoms partially resolved after taking 500 mg amoxicillin twice daily for one month, but he continued to experience numerous symptoms for the next three years, including poor concentration, short-term memory loss, sleep disturbance, numbness in extremities, periorbital twitch, and migrating joint pain. These symptoms failed to clear despite further antibiotic treatment over a four-year period. The patient sought hyperbaric oxygen treatment in October 2011.

The man received 30 sessions of HBOT at 2.5 ATA for 1½ hours. His cognitive function and sleep improved within the first 10 sessions. Other nervous system symptoms, including numbness and eye twitching, disappeared with the second 10 sessions. Migrating joint pain resolved with the third set of 10 sessions. "Overall, completion of 30 sessions of HBOT caused noted longstanding Lyme-disease-related symptoms affecting most of the previously affected bodily areas to disappear," according to the authors. The study does not say if the improvement was permanent.

Author-journalist Jill Neimark and Byron White, ND – both of whom suffered with chronic Lyme disease – have

benefited from using portable hyperbaric oxygen chambers in their homes. "These home chambers are approved by the FDA, are available by prescription from your doctor, and pressurize to low, safe levels," says Neimark. Using the portable chamber in conjunction with an oxygen concentrator and a medical pass-through can raise tissue oxygen levels up to 400%. Neimark and White have found that infrared sauna therapy works well with HBOT: "Tissue oxygen levels remain elevated for hours after a hyperbaric chamber session, and the infrared sauna heat increases circulation and stimulates the immune system. The two can be synergistic." Both therapies also boost parasympathetic nervous activity, which increases relaxation and healing.

HBOT and sauna therapy are just part of the treatment program that White uses to maintain his health and to help patients regain theirs. Diet, detoxification measures, and herbs are other components. Most importantly, patients with chronic illness need to pay attention to their bodies. White says, "It is important to temper treatments like hyperbaric oxygen, ozone and sauna with your body's ability to handle the treatment."

Huang C-Y, Chen Y-W, Kao T-H et al. Hyperbaric oxygen therapy as an effective adjunctive treatment for chronic Lyme disease. *J Chin Med Assoc.* 2014;77:269–271. Available at [www.jcma-online.com/article/S1726-4901\(14\)00042-2/pdf](http://www.jcma-online.com/article/S1726-4901(14)00042-2/pdf). Accessed April 24, 2015.

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Sexual Transmission of Lyme?

On January 25, 2014, a press release announced that *Borrelia* spirochetes had been cultured from semen and vaginal secretions from people who tested positive for Lyme. The study, presented at the annual Western Regional Meeting of the American Federation for Medical Research, proved that viable *Borrelia burgdorferi* (Bb) was present in human genital secretions. It did not prove that the infection could be transmitted during intercourse. Yet, a storm of headlines proclaimed that Lyme could be sexually transmitted. The study's authors were criticized for not being clear that their research was preliminary. The sensationalized headlines and subsequent outcry

nearly made me discount the study's importance. Then, I found version three (April 27, 2015) of the research article, written by Marianne J. Middelveen and a team of US and Australian researchers. The article, published with comments from peer reviewers/referees, discussion from scientists, and rebuttals from the authors, makes interesting reading.

Middelveen and colleagues recruited 17 participants for their study. Four of them acted as controls; they had no history of Lyme and tested negative for *Borrelia*. All 13 in the patient group had a history of Lyme disease. Ten of them were serologically positive, 2 tested negative, and 1 had equivocal results. The patient group included 4 pairs of heterosexual partners. Genital secretions from all 17 volunteers were cultured for 4 weeks. The researchers used several methods for examining the cultures for spirochetes including light and darkfield microscopy, silver staining, immunohistochemical staining, molecular hybridization, and PCR analysis. "Immunohistochemical and molecular testing was performed in three independent laboratories in a blinded fashion. Positive and negative controls were included in all experiments," say the authors.

No spirochetes were found in cultures from the control participants, but moving spirochetes were observed in genital culture concentrates from 12 of 13 patients. Molecular hybridization and PCR testing confirmed that the spirochetes were strains of *Borrelia*. The authors report, "PCR sequencing of cultured spirochetes from three couples having unprotected sex indicated that two couples had identical strains of *Bb sensu stricto* in their semen and vaginal secretions, while the third couple had identical strains of *B. hermsii* detected in their genital secretions." This finding supports the possibility that Lyme can be sexually transmitted. It is not proof. Some clinicians have noticed a higher failure rate of antibiotic treatment in their sexually active Lyme patients. It is possible that these patients are being reinfected by untreated partners, but more research is needed.

Controlled studies investigating sexual transmission of *Borrelia* in humans are unethical. (Remember the Tuskegee syphilis studies?) Consequently, researchers will have to rely on less direct means of studying this possibility.

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Skin Microbiota

Recent studies refute the longstanding belief that intact skin prevents microbes from crossing the epidermis to living cells below.

These studies found bacterial genes and DNA located in the dermis and subcutaneous layer of normal, intact skin. Over the past decade, researchers have learned that some commensal skin bacteria secrete compounds that inhibit excess inflammation during injury. Other secreted compounds directly affect the body's immune responses. Richard L. Gallo of University of California-San Diego School of Medicine and colleagues questioned how commensal skin bacteria influenced the immune system if their only contact were dead skin cells of the epidermis. The researchers examined sequential horizontal sections of normal human skin with qPCR and found bacterial 16S rRNA genes below the maximum depth of hair follicles in facial skin and below sweat glands in palm skin. "This means that the skin acts as a filter (rather than a barrier), and controls the balance of the dermal microbial communities," says Gallo.

In addition to their effect on the body's immune response, commensal bacteria enhance skin health by inhibiting pathogen growth. Pathogenic microbes have a harder time acquiring nutrients and space for growth when skin has an abundant population of commensals. Also, many commensals, including *Staphylococcus epidermidis*, secrete antimicrobial compounds that deter pathogens. Given the right conditions, however, many protective commensals can cause infection.

"In healthy adults, the amount of diversity seen in skin commensal bacteria is staggering," say James A. Sanford and Gallo. They cite a study, led by N. Fierer, that found 4742 species-level bacterial phylotypes on the hands of 51 healthy young adults with an average of 150-plus phylotypes on each palm. The phylotypes widely differed from person to person and even from a person's right hand and left: "... hands from the same individual shared only 17% of their phylotypes, with different individuals sharing only 13%." Other studies have shown that inhabitants of the same home (people and pets) have more similarity in commensal skin bacteria than strangers do.

Not surprisingly, microbiota composition on diseased skin differs from that found on healthy skin. Researchers have not yet determined "whether alterations in the microbiome

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lead to disease, or whether underlying conditions result in an imbalance in microbial communities," say Sanford and Gallo. However, a May 2012 investigation of skin microbiota and atopic dermatitis (AD) noted a decrease in microbial diversity during AD flare-up (up to 90% of detected microbes were *Staphylococcus* species) and increased diversity when the flares resolved with treatment. The association between dysbiosis and psoriasis is less clear cut.

Gallo writes, "Given the abundance and distribution of bacteria both at the surface and within us, it is most logical now to consider the human body as a heterogeneous collection of organisms hoping to work towards a common good."

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Sun Exposure and Skin Health

Public health campaigns that advocate total protection from UV rays overlook the crucial role that solar-derived vitamin D3 plays in overall health and in protecting skin from sun damage. Chronic sun exposure is a risk factor for both squamous cell carcinoma (SCC) and basal cell carcinoma (BCC). Although short-term intense UV exposure (sunburn), particularly during childhood, has been linked to malignant melanoma (MM) in epidemiology studies, chronic sun exposure has not. In fact, several studies indicate that chronic exposure that does not produce sunburn may protect against MM, according to Jörg Reichrath and Sandra Reichrath. They suggest that skin-produced D3 "may represent an evolutionary highly conserved feedback mechanism that protects the skin from the hazardous effects of solar UV-radiation." In vitro and in vivo studies support vitamin D's protective effect.

Vitamin D has been primarily associated with bone formation, but it also regulates numerous cellular functions, including cell growth and differentiation. Vitamin D receptors and the main enzyme needed to convert 25(OH)D to biologically active 1,25(OH)2D are found in tissues throughout the body. Without sufficient sun exposure, vitamin D deficiency results. Vitamin D deficiency is associated with increased risk or poor prognosis for several diseases including skin, colon, prostate and breast cancers; autoimmune disease; infectious disease; and cardiovascular disease.

"The important take home message for dermatologists and other clinicians is that health campaigns promoting strict sun protection procedures to prevent skin cancer may increase the severe health risk of vitamin D-deficiency," state the authors.

The Vitamin D Foundation recommends moderate, frequent sun exposure: "Large amounts of vitamin D3 (cholecalciferol) are made in your skin when you expose all of your body to summer sun. This happens very quickly: around half the time it takes for your skin to turn pink and begin to burn." People with very light skin need only 15 minutes of exposure, while those with darker skin may need a few hours to make a good supply of the vitamin. Dark skin contains more melanin that blocks UVB needed for D3 production.

Public health agencies usually advise people to avoid midday summer sun. The Vitamin D Foundation, however, points out that the earth's atmosphere blocks UVB rays early and late in the day and during most of the winter. Exposing as much skin as possible to sun for short periods near midday is the most effective way to increase D3. "A good rule of thumb is if your shadow is longer than you are tall, you're not making much vitamin D," according to the group's website. To avoid overexposure, the Vitamin D Foundation recommends covering up with clothing or staying in the shade. Studies investigating sunscreen's ability to prevent skin cancers have had mixed results.

Reichrath J, Reichrath S. Hope and challenge: the importance of ultraviolet (UV) radiation for cutaneous Vitamin D synthesis and skin cancer. *Scand J Clin Lab Invest*. 2012;72(Suppl 243):112–119. Available at EBSCO. Accessed April 24, 2015.

How do I get the vitamin D my body needs? [online article]. Vitamin D Foundation. www.vitamindcouncil.org/about-vitamin-d/how-do-i-get-the-vitamin-d-my-body-needs/#. Accessed April 24, 2015.

Tea Tree Oil in Dermatology

Topical application of tea tree oil (TTO), the steam-distilled essential oil derived from the Australian shrub *Melaleuca alternifolia*, is an effective treatment for many skin disorders. The oil contains terpinen-4-ol, a powerful antimicrobial and anti-inflammatory agent. TTO also has antioxidant and antitumor effects. A 2013 literature review, conducted by Nader Pazyar, MD, and colleagues, discusses in vitro, in vivo, and clinical research that supports TTO's use for dermatologic conditions.

The review is the first to summarize TTO's possible applications for dermatology. TTO has antiviral, antifungal, antiprotzoal, and antibacterial activity, according to the review. A 10% concentration of TTO showed results against *S. aureus* comparable to topical mupirocin. TTO is an "efficient" treatment for hand warts caused by human papillomavirus. Fungal infections of the nails, athlete's foot, and seborrheic dermatitis improve with topical TTO. A combination of TTO and lavender oil was highly effective for killing head lice, according to a 2010 study by Barker and Altman. In addition to having antimicrobial activity, a topical 10% TTO-dimethyl sulfoxide (DMSO) formula significantly slowed subcutaneous melanoma growth in mice. The authors say, "In conditions for which

continued on page 27 ➤



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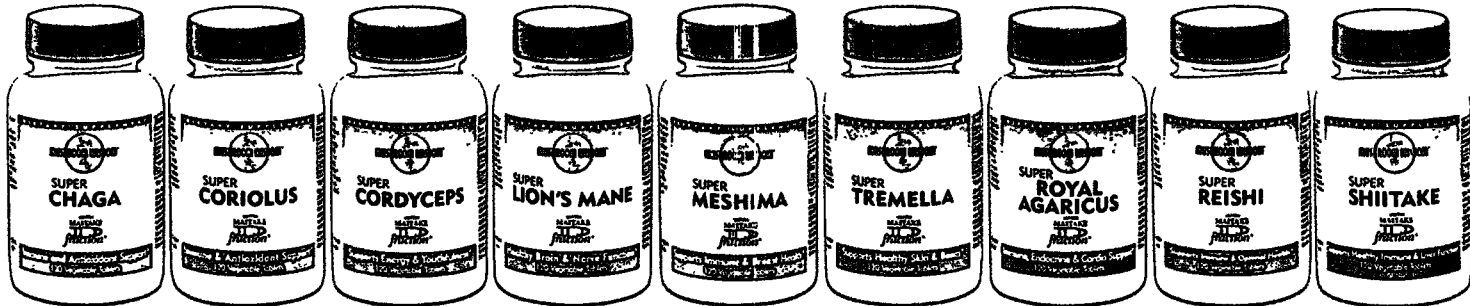
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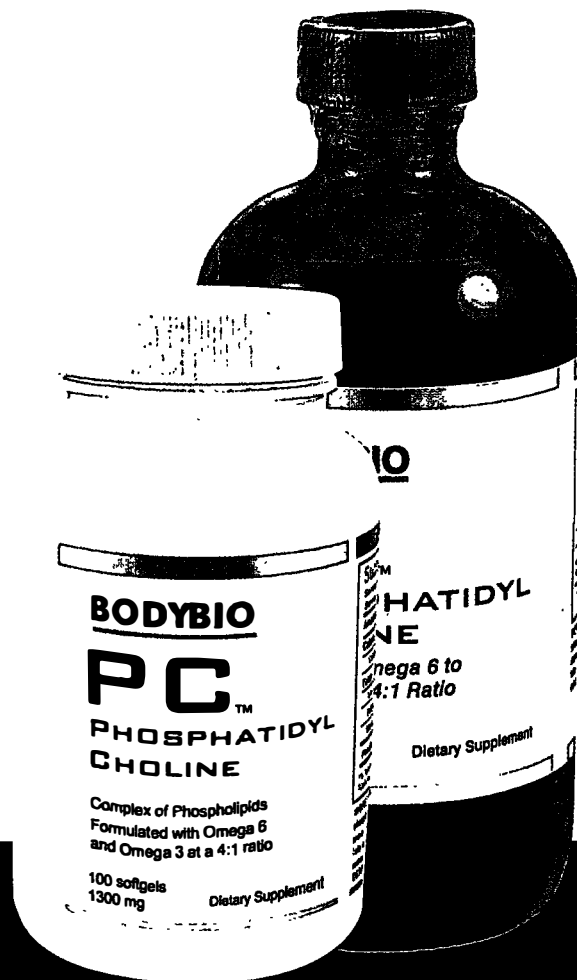
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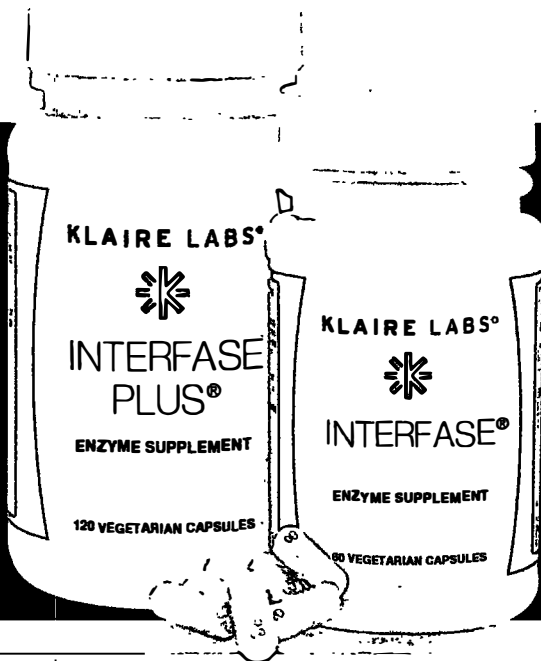
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► continued from page 20

TTO treatment is of benefit, further research is necessary to establish guidelines for its application, preparations, and therapeutic indices."

Topical application may not be the only method for using TTO. Karen B. Chin, RN, and Barbara Cordell, PhD, RN, reported clinical evidence that exposure to TTO fumes increased healing rate of infected wounds. Their small 2013 study involved 10 patients with abscessed wounds infected with *Staphylococcus aureus*. Two pairs of participants ("matched as closely as possible by age, gender, infectious vector, and number of days with infected wound") provided a mini-controlled experiment; 1 person in each matched pair received a standard dressing. The other 2 along with the 6 unmatched participants were treated with TTO dressings (changed every three days). The dressings consisted of 6 pipette drops (about 2 drops from a regular dropper) of full-strength TTO centered on the abdominal pad, Telfa pad, or gauze. Some participants also received antibiotics. Nurses recorded the wound size and description when dressings were changed.

Both matched pairs showed a marked difference between the conventional treatment and the use of TTO dressing. In pair A, the control's thigh abscess took over a week to begin healing, but the TTO patient's buttocks abscess showed major improvement after 1 day. In pair B, the control's calf abscess showed little improvement at day 19, whereas the TTO patient's scalp abscess was healing well at day 3. When the woman with the calf abscess (pair B) was given a TTO dressing, she reported that the wound closed after 1 day and was no longer painful. Five of the 6 unmatched participants also recovered quickly (an average of 4.4 days) with TTO treatment. One patient did not respond to TTO: a 70-year-old man with diabetes, hypertension, and respiratory illness.

"Further study is warranted to observe the effects of tea tree oil with and without conventional antimicrobial pharmaceutical treatment for both *S. aureus* and MRSA," say Chin and Cordell.

Barker SC, Altman PM. A randomized, assessor blind, parallel group comparative efficacy trial of three products for the treatment of head lice in children. *BMC Dermatol.* 2010;10:6. Available at www.ncbi.nlm.nih.gov/pubmed/20727129. Accessed May 15, 2015.

Chin KB, Cordell B. The effect of tea tree oil (*Melaleuca alternifolia*) on wound healing using a dressing model. *J Altern Comp Med.* 2013;19(12):942-945. Available at EBSCO. Accessed April 28, 2015.

Greay SJ, Ireland DJ, Kissick HT, et al. Inhibition of established subcutaneous murine tumor growth with topical *Melaleuca alternifolia* (tea tree) oil [abstract]. *Cancer Chemother Pharmacol.* 2010;66:1095-1102. Available at www.ncbi.nlm.nih.gov/pubmed/20577741. Accessed May 15, 2015.

Pazyar N, Yaghoobi R, Bagherani N, Kazerouni A. A review of applications of tea tree oil in dermatology. *Int J Dermatol.* 2013;52:784-790. Available at EBSCO Accessed April 28, 2015.

Ticks and Biodiversity

Although Lyme disease first gained attention in the 1970s, the causative spirochete *Borrelia burgdorferi* (Bb) is no newcomer. "Distinctive Bb genes have been identified in museum collections of ticks from the 1940s and of white-footed mice from the turn of the twentieth century," says science writer Sharon Levy. Decreased biodiversity in the environment may be one reason that Lyme incidence has risen to an estimated 20,000-30,000 cases annually in the US (CDC figures).

Deer ticks (*Ixodes scapularis*) are not born with Bb infection. The larvae, which hatch on the ground, become infected when they feed on blood from small infected animals. White-footed mice are the primary carriers of Bb. Other small animals, such as raccoons and skunks, are less likely to transmit Bb. If a tick larva has the misfortune of hopping onto an opossum for its first meal, it may not survive to see the next (nymph) stage of its life cycle. In 1990, epidemiologist Durland Fish found that 40% of the nymphs that had fed on white-footed mice acquired Bb (the highest infection rate for any tested host animal), but those that fed on an opossum died before researchers could test them for infection. Tiny nymphs need a second blood meal in order to become adults that reproduce. Both nymphs and adult ticks reside on vegetation instead of the ground, so their prospective hosts are larger mammals such as deer and humans.

The increased incidence of Lyme has been linked to at least two changes in biodiversity, according to Miriam Pfäffle, Nina Littwin, and Trevor N. Petney. First, the numbers of white-footed mice and other small mammals have increased because their major predator, the red fox, is less prevalent. Second, deer populations have also expanded with the absence of their primary natural predators. "Although deer are not competent vectors of *B. burgdorferi* s.s., and seem to have little or no role in the maintenance of infection in ticks, deer are the most important hosts for [adult] female ticks," explain Pfäffle and colleagues. More deer lead to more tick larvae, larvae that are available to become infected.

Levy S. The Lyme disease debate. *Environ Health Perspect.* April 2013;121(4):A120-129. Available at <http://ehp.niehs.nih.gov/121-a120>. Accessed April 24, 2015.

Pfäffle M, Littwin N, Petney TN. The relationship between biodiversity and disease transmission risk. *Res Rep Biodivers Studies* 2015;4. Available at www.researchgate.net. Accessed April 24, 2015.

The August/September 2015 issue will focus on cancer treatment and prevention. Look for it in the mail on August 20th.



Optimizing Metabolism

by Ingrid Kohlstadt MD, MPH
www.INGRIDients.com

Shifting Plates: Personalized Health Amidst Global Climate Change

Introduction

Remember the public discussions about whether global climate change is accelerated by human activity, and whether it even exists? Those have finally been booted off the public stage by ample evidence of its impact – even on human health.

The human health impact includes asthma, allergies, and cardiovascular diseases due to air pollution and lingering allergens. Diarrheal disease is increasing due to changes in water temperature and reduction in water quality and availability. Infections such as malaria and dengue fever are spreading to new areas and affecting more people, due largely to climate change that favors the spread of disease vectors such as mosquitoes, ticks, and lice. Degradation of the environment takes a direct punch at well-being and causes mental illness indirectly by forcing migration and sparking conflict over scarce resources. Extreme heat induces and worsens most chronic diseases, and severe weather takes a high toll on life itself.

When confronted with data as expansive and negative as health impact from global climate change, a very natural reaction is to feel overwhelmed or resigned that the only action one can take is to “do one’s part” at keeping the earth green and hope for the best.

This column takes a different approach, equipping readers with ways to proactively defend their health in the face of climate change.

Shifting Plates of a Different Kind

Allow me to start by presenting a new theory put forth by earth scientists: A warmer climate leads to melting of the earth’s fresh water supply. As polar ice caps shrink they exert less pressure on the earth’s tectonic plates. As the vise grip from polar ice loosens, even slightly, the plates are more able to shift leading to seismic activity at higher rates and frequencies such as those being observed today.

I found the theory fascinating and would like to make an analogy to shifting plates of a different kind, plates over which we do have some control: our food plates. With that I would like to introduce you, by way of an online video at <http://www.nutribee.org/beequest>, to a Maryland high school student named Sydney Miller.

Less packing in the landfill means more nourishing food for me, reasoned Miller. With that she led a shift in what she and her friends put on their plates. She created a scavenger hunt for middle school students to shop for the ingredients in three recipes. Scavenger hunt winners not only found the ingredients, they learned how to dispose of the trash. Those with fewest items in the landfill and the most in compost were considered the winners of the scavenger hunt because not only were they eco-friendly, they were adopting a diet in which processed food is replaced with local fresh foods – fresh corn instead of canned, for example.

I’ve personally used Miller’s scavenger hunt approach. It added pizzazz to my shopping especially because it was something that my children could really connect with, too. I noticed that it gradually influenced where I shop, more at the local Amish market and the Latino grocer that has the most real-looking, real-tasting fresh fruit.

Diversify Protein Sources

Many Americans have digestive problems of which they aren’t aware. The poor digestion gets in the way of the body’s natural ability to digest protein. Even though people may eat enough protein according to dietary intake charts, they aren’t meeting their bodies’ needs. But flipping an extra burger from the grill to your plate is not the answer. I encourage patients to address their digestive issues and diversify their protein sources. Since diversifying protein usually translates to eating less animal protein, I have found a clinical ally with the going-green movement.

Food Security, Revealing Answers for Current Health

Living from meal to meal is a daily reality for more Americans than we like to think. Food security is an important issue to which we need to attune ourselves as clinicians, if for no other reason than that it may prevent our patients from implementing our recommendations of any kind, not only those pertaining to nutrition.

While clinically relevant, asking patients if they have food in the house is awkward. Talking about the weather, a nonthreatening topic, is a way that I am able to indirectly ask

patients if they have food in the house. I'll mention something along the lines that global climate change brings more reasons for the power to go out. As part of this conversation, I ask them to describe their emergency food supply. That way, I screen for food insecurity and at the same time I can address the many people who have a 1-week supply of candy bars and soda. Yikes, from some of the replies, one might think chocolate candies, chips, cookies, and sweetened juices are staple foods!

Stopping a PET Peeve

Every doctor should discourage patients from routine use of water from plastic bottles. One reason is that bottled water is not screened for parasites and asbestos and is therefore held to a lesser standard than is municipal water. "Resealable" doesn't mean "reusable." Reusing plastic bottles is associated with high rates of germs, because plastic is like an agar plate for the growth of germs. As if those aren't reasons enough, most plastic bottles are made of polyethylene terephthalate (PET), a carcinogen. Sure, it's considered safe when kept at room temperature, but how do you know that the bottle didn't sit in a hot delivery truck? Or that it wasn't frozen and thawed to keep it cool for the sports event where you are purchasing it? Since PET plastics are usually destined for landfills or, worse yet, litter, the green movement may provide sufficient pressure for patients to use stainless steel water bottles or download an app for locating functioning water fountains.

Minimizing the Downsides

Fueling Existing Stigmas

Most unfortunately, the green movement has rekindled prejudice. Some people leverage the environment to promote dislike toward people who are obese. Obesity may prevent an individual from using the stairs or driving some smaller cars, and obesity predictably requires more food to meet metabolic demand. While these needs should not reflect on the people suffering from obesity, in reality it does. Because of the flare in overt prejudice, I am more proactive with my patients, getting them to support services and coming alongside their personally initiated fitness and diet efforts.

Tips for Avoiding Electrosmog

Virtual meetings and online books save fossilized trees (oil) and recently felled ones. However, prolonged exposure to electronic forms of communication can take a toll on health. The repetitive motion and postural strains are well known but

sometimes go unrecognized for extended periods of time. Even more, patients often don't realize that repeated or prolonged exposure to electrosmog can strain thyroid function, trigger headaches, or underlie their symptoms such as burning, cramping hands or a persistent rash. Helping people minimize exposure to electrosmog is an emerging clinical intervention.

Keep Up the Hygiene

Global climate change is leading to a rise in infections, even if hand-washing rates stay the same. Skimping on paper towels may save some trees in the short run, but it is a sufficient deterrent to hand-washing that in the long term it probably costs the environment.

Phosphates are removed from dish and laundry detergents out of appropriate environmental considerations. But there's a downside. Phosphates bind heavy metal toxins such as lead. For those who seek to preserve the old, such as historic homes and antique furniture, lead isn't exactly in public health's rear-view mirror. Clinicians should be vigilant about lead screening.

In theory, when you recycle something, you reuse both its benefits and potential toxins. I experienced such a situation firsthand. An eco-friendly building where a NutriBee program was hosted had a beautiful granite-like countertop. Oddly, as I was preparing food, the countertop splintered and a piece lodged in my hand. The wound took 2 to 3 times longer to heal than I would have expected. I later learned that the surface was made from recycled computer parts and was likely to contain several toxic metals.

Summary

As a physician and public health specialist, I have dual roles of helping my patients achieve the best health possible and promoting the public good. The two roles are usually synergistic. It is usually possible to simultaneously support ecology, which is vital to public health and survival of the planet. When the two topics are at cross purposes, physicians can screen for these risks and inform their patients, using the opportunity to affirm the doctor-patient relationship.

Ingrid Kohlstadt, MD, MPH, FACPM, FACN
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Literature Review & Commentary

by Alan R. Gaby, MD
drgaby@earthlink.net

Intravenous Vitamin C for Severe Sepsis

Twenty-four patients hospitalized in a medical intensive care unit with severe sepsis were randomly assigned to receive, in double-blind fashion, intravenous vitamin C infusions every 6 hours for 4 days or placebo (5% dextrose). Eight patients received high-dose vitamin C (200 mg per kg of body weight per day) and 8 received low-dose vitamin C (50 mg per kg of body weight per day). The mean plasma vitamin C concentration prior to treatment was 17.9 μM (normal range, 50–70 μM). Patients receiving vitamin C exhibited prompt reductions (improvements) in Sequential Organ Failure Assessment (SOFA) scores, whereas placebo patients exhibited no such reduction. Vitamin C significantly reduced the pro-inflammatory biomarkers C-reactive protein and procalcitonin. Unlike in placebo patients, thrombomodulin levels in patients receiving vitamin C did not increase significantly, suggesting that vascular endothelial injury had been attenuated. No adverse effects of vitamin C infusions were observed.

Comment: Vitamin C has multiple actions that might be of value for patients with sepsis, including anti-inflammatory and antimicrobial activity. The results of the present pilot study suggest that intravenous vitamin C infusions were safe and well tolerated in patients with severe sepsis, and may decrease inflammation, endothelial injury, and the extent of multiple organ failure. While some of the benefit may have been due to correcting vitamin C deficiency, it is likely that many of the effects of vitamin C were pharmacological in nature. Higher vitamin C doses than those used in this study might be even more effective, with respect to both its antimicrobial and anti-inflammatory effects. However, in patients with acute renal failure (which may occur in association with sepsis), large intravenous doses of vitamin C can cause soft-tissue deposition of oxalate, with potentially serious consequences.

Fowler AA III et al. Phase I safety trial of intravenous ascorbic acid in patients with severe sepsis. *J Transl Med.* 2014;12:32.

Low-Nickel Diet Helps Eradicate *Helicobacter*

Fifty-two patients (mean age, 42 years) with newly diagnosed *Helicobacter pylori* infection were randomly assigned to consume a standard diet or a low-nickel diet for 30 days. On the low-nickel diet, all foods high in nickel content were prohibited. Starting on day 15 of the diet, all patients were treated with 15 mg of lansoprazole, 500 mg of clarithromycin, and 1000 mg of amoxicillin, each twice a day for 7 days. *H. pylori* eradication was assessed by the urea breath test 4 weeks after the end of the treatment period. The *H. pylori* eradication rate was significantly higher among patients on the low-nickel diet than among patients on the standard diet (84.6% vs. 46.2%; $p < 0.01$). The incidence of treatment-related side effects did not differ between groups.

Comment: *H. pylori* contains a nickel-dependent urease enzyme. This enzyme catalyzes the hydrolysis of urea in gastric juice to form ammonia, an alkaline compound that protects the organism against gastric acidity. NiFe-hydrogenase is another nickel-dependent enzyme produced by *H. pylori* that helps the organism survive the acid environment of the stomach. The results of the present study demonstrate that the addition of a low-nickel diet to standard triple therapy significantly increased the *H. pylori* eradication rate. The decrease in *H. pylori* urease activity due to the low-nickel diet could expose the bacterium to gastric acid and increase its susceptibility to amoxicillin.

Campanale M et al. Nickel free-diet enhances the *Helicobacter pylori* eradication rate: a pilot study. *Dig Dis Sci.* 2014;59:1851–1855.

Arachidonic Acid for Parasitic Infection

Sixty-six Egyptian schoolchildren who were infected with *Schistosoma mansoni* were randomly assigned to receive a single dose of praziquantel (40 mg per kg of body weight), arachidonic acid (10 mg per kg per day, 5 days a week for 3 weeks; 15 doses total), or both treatments. Among children with light infection intensity (<100 eggs

per gram of stool), cure rates were 85% (12 of 14) with praziquantel, 78% (11 of 14) with arachidonic acid, and 87% (14 of 16) with the combination. Among children with moderate infection intensity (100 to 400 eggs per gram of stool), cure rates were 83% (5 of 6) with praziquantel, 44% (4 of 9) with arachidonic acid, and 100% (7 of 7) with the combination.

Comment: Schistosomiasis is a severe parasitic disease that is endemic in 78 countries and affects at least 243 million people worldwide. The treatment of choice is praziquantel, which produces cure rates of 60% to 90%. Side effects of praziquantel are relatively mild, and include headache, dizziness, rash, nausea, abdominal pain, and diarrhea. Arachidonic acid has been reported to kill juvenile and adult schistosomes in vitro and to reduce worm burdens by 50% to 78% in mice and hamsters infected with *S. mansoni* or *S. haematobium*. In the present study, arachidonic acid was nearly as effective as praziquantel in children with low infection intensity. Although arachidonic acid was less effective than praziquantel in children with moderate infection intensity, treatment with arachidonic acid may increase the efficacy of praziquantel in those children. Arachidonic acid is believed to work by disrupting the parasite's surface membrane.

Selim S et al. Efficacy and safety of arachidonic acid for treatment of *Schistosoma mansoni*-infected children in Menoufiya, Egypt. *Am J Trop Med Hyg.* 2014;91:973-981.

Vitamin D and Influenza Prevention: Examining Conflicting Evidence

Two hundred forty-seven Japanese high school students were randomly assigned to receive, in double-blind fashion, 2000 IU per day of vitamin D3 or placebo for 2 months during the 2009 pandemic of the H1N1 subtype of influenza A. The primary outcome was incidence of influenza A diagnosed by a rapid influenza diagnostic test. During the entire trial, the incidence of influenza A was nonsignificantly higher by 11% in the vitamin D group than in the placebo group (13.5% vs. 12.1%; $p = 0.75$): In post hoc analysis, during the first month, the incidence of influenza A was significantly lower in the vitamin D group than in the placebo group (1.4% vs. 8.1%; $p < 0.01$). However, during the second month, the vitamin

D group experienced more events and effectively caught up with or overtook the placebo group.

Comment: Previous research on the effect of vitamin D supplementation on influenza incidence has produced conflicting results. In a randomized controlled trial published in 2010 (Urashima M et al. Randomized trial of vitamin D supplementation to prevent seasonal influenza A in schoolchildren. *Am J Clin Nutr.* 2010;91:1255-1260), vitamin D supplementation (1200 IU per day) significantly decreased the incidence of influenza A among Japanese children (mean age, 10 years). However, vitamin D also

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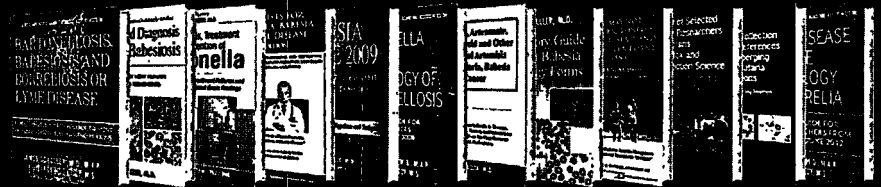
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Gaby's Literature Review

► nonsignificantly increased the incidence of influenza B compared with placebo (23.3% vs. 16.8%), and total influenza incidence (A plus B) did not differ between groups. In Japan, influenza A is usually prevalent in the early period of the influenza season (January), whereas the incidence of influenza B increases in March. In post hoc analysis of this trial, it was found that the total incidence of influenza (A plus B) was less in the vitamin D group than in the placebo group only during the first half of the study (and was apparently higher in the vitamin D group than in the placebo group during the second half).

Those findings, when combined with the results of the present study, suggest one of two possibilities. One is that vitamin D supplementation delays, but does not prevent, influenza. The other possibility is that an initial beneficial effect of vitamin D supplementation was negated or reversed by a progressive accumulation of this fat-soluble vitamin, which resulted in some type of deleterious effect on immune function due to subtle vitamin D toxicity. That possibility is supported by a subgroup analysis of the 2010 study cited above. In the group as a whole, children assigned to receive 1200 IU per day of vitamin D had a 42% reduction in incidence of influenza A, when compared with those assigned to placebo. However, the effect of vitamin D differed according to whether the children were taking other vitamin D supplements on their own. Among children who were not taking other vitamin D supplements, 1200 IU per day of vitamin D reduced the incidence of influenza A by 64% compared with placebo. However, among children who were taking other vitamin D supplements (average dose, approximately 150 IU per day), supplementation with 1200 IU per day of vitamin D nonsignificantly increased the incidence of influenza A by 11% compared with placebo.

Although the use of megadoses of vitamin D has become popular in recent years, the results of randomized controlled trials support the interim conclusion that moderate doses (such as 800 to 1200 IU per day for adults) are more effective and safer than larger doses, with respect to infection prevention, osteoporosis prevention, and treatment of conditions such as multiple sclerosis and chronic obstructive pulmonary disease.

Urashima M et al. Effects of vitamin D supplements on influenza A illness during the 2009 H1N1 pandemic: a randomized controlled trial. *Food Funct.* 2014;5:2365-2370.

Zinc for Atopic Dermatitis (Eczema)

Of 58 Korean children (mean age, 6.2 years; range, 2-14 years) with atopic dermatitis, 41 (70.7%) had a low hair zinc concentration ($< 130 \mu\text{g/g}$). In contrast, 41.9% of 43 control children had a low hair zinc concentration ($p = 0.003$ for the difference between groups). The patients with low hair zinc levels were randomly assigned to receive 12 mg of oral zinc (as zinc oxide; presumably per day, although this was not stated) or no zinc supplement

(control group) for 8 weeks. All patients received oral antihistamines and topical moisturizers. The mean Eczema Assessment Severity Index (EASI) score improved in both groups. After 8 weeks, the mean EASI score ($p < 0.05$), transepidermal water loss ($p < 0.02$), and visual analogue scale score for pruritus ($p < 0.001$) improved more in the zinc group than in the control group. At 8 weeks, the mean improvement in the EASI score was 74% in the zinc group and 48% in the control group. The mean EASI score was also significantly lower (better) after 4 weeks in the zinc group than in the control group.

Comment: Zinc deficiency causes eczematous lesions in both humans and experimental animals. The present study suggests that zinc is beneficial for children with atopic dermatitis who have a low hair zinc concentration. However, the concentration of zinc in hair does not appear to be a reliable indicator of zinc nutritional status, and it is not clear whether patients with atopic dermatitis need to be zinc-deficient in order to benefit from supplementation. A previous double-blind trial found that treatment with a sustained-release zinc preparation was not effective, and may have exacerbated atopic dermatitis in some cases (Ewing CI et al. Failure of oral zinc supplementation in atopic eczema. *Eur J Clin Nutr.* 1991;45:507-510). However, that study was confounded by the possibility that some children may have been allergic to one or more of the "inert" ingredients used to manufacture the sustained-release zinc preparation.

In my clinical experience, zinc does appear to be helpful for patients with atopic dermatitis. The efficacy of zinc seems to be increased by using it in combination with a source of essential fatty acids (such as sunflower oil, safflower oil, or flaxseed oil). Long-term zinc supplementation should in most cases be accompanied by a copper supplement, in order to prevent zinc-induced copper deficiency. A reasonable daily dose of copper would be 1 mg with 15 mg of zinc, 1 to 2 mg with 30 mg of zinc, and 2 to 4 mg with daily zinc doses greater than 30 mg.

Kim JE et al. Hair zinc levels and the efficacy of oral zinc supplementation in patients with atopic dermatitis. *Acta Derm Venereol.* 2014;94:558-562.

Iron Supplementation for Chronic Urticaria

Of 122 patients (mean age, 42.5 years) with chronic urticaria (i.e., urticaria present for at least 6 consecutive weeks) who had had a poor response to conventional treatment with antihistamines and glucocorticoids, 81 (66%) were found to have moderately low serum iron. The patients with low serum iron received iron supplements (105 mg per day, as ferrous sulfate) for 30 to 45 days. One month after the first visit, improvement was greater than 80% in the patients treated with iron, as compared with an improvement of 20% to 30% in patients with normal serum iron who did not receive iron supplementation. Two months after the first visit, all 81 patients treated with iron had normal serum iron. Sixty-four (79%) of those 81 patients had complete remission of urticaria and the remaining 17 patients (21%) had greater than 80% improvement. Among

the patients who received iron therapy, those whose serum iron levels were close to the lower limit of normal after treatment had less improvement than did patients whose serum iron levels were higher.

Comment: Chronic urticaria is a common and difficult-to-treat problem. The results of the present study indicate that patients with chronic urticaria frequently have low serum iron levels, and that in those patients, iron supplementation results in considerable improvement in, or complete remission of, the urticaria. While a low serum iron level by itself is not definitive evidence of iron deficiency, a therapeutic trial of iron supplementation would seem reasonable for patients with chronic urticaria who have low serum iron.

The study did not investigate whether iron supplementation would be beneficial for patients with normal serum iron levels. Iron supplementation can cause acute liver failure or other serious adverse effects in people with hemochromatosis; therefore, iron overload should be ruled out before starting patients on iron therapy. The mechanism by which iron supplementation improves chronic urticaria is not known.

Guarneri F et al. Oral iron therapy and chronic idiopathic urticaria: sideropenic urticaria? *Dermatol Ther.* 2014;27:223-226.

Vitamin B12 for Chronic Hepatitis C

Ninety-four patients with chronic hepatitis C who had not been previously treated were given pegylated interferon alpha plus ribavirin, and were randomly assigned to receive or not to receive (control group) 5000 µg of vitamin B12 (cyanocobalamin) intramuscularly, every 4 weeks for the duration of antiviral therapy. The duration of antiviral treatment was 48 weeks for genotype 1, and 24 weeks for genotypes 2 and 3. A viral response was defined as undetectable serum HCV-RNA. The proportion of patients who had a complete early viral response (12 weeks after starting treatment) (85% vs. 64%; $p = 0.03$), end-of-treatment viral response (24 or 48 weeks after starting treatment) (83% vs. 63%; $p = 0.03$), and sustained viral response (24 weeks after completing treatment) (72% vs. 38%; $p = 0.001$) was significantly higher in the vitamin B12 group than in the control group.

Comment: These results indicate that vitamin B12 supplementation, as an adjunct to pegylated interferon alpha plus ribavirin, significantly improved sustained viral response rates in patients with hepatitis C. As I mentioned in last month's editorial in the *Townsend Letter*, interferon alpha plus ribavirin may no longer be the treatment of choice for chronic hepatitis C, because a newer class of drugs produces a higher cure rate. However, the absurdly high prices for these new drugs (up to \$160,000 for a course of treatment) preclude their use in many cases. The results of the present study should serve to remind us that natural treatments such as nutrients and herbs, when given either alone or as an adjunct to conventional therapy, can in many cases improve outcomes and lower the total cost of treatment.

Rocco A et al. Vitamin B12 supplementation improves rates of sustained viral response in patients chronically infected with hepatitis C virus. *Gut.* 2013;62:766-773.

Gaby's Literature Review

Alpha-Lipoic Acid Promotes Weight Loss

Ninety-seven healthy overweight or obese women were randomly assigned to receive, in double-blind fashion, 1.3 g per day of eicosapentaenoic acid (EPA), 300 mg per day of alpha-lipoic acid (ALA), both treatments, or placebo for 10 weeks. All women were advised to follow a low-calorie diet (30% less than total energy expenditure). Seventy-seven women completed the study. Mean weight loss was significantly greater by 1.5 kg in the groups that received ALA than in those that did not receive ALA ($p = 0.03$). EPA had no effect on weight loss.

Comment: ALA is a cofactor for mitochondrial respiratory enzymes, and improves mitochondrial function. In previous research, ALA prevented the development of obesity in rodents by reducing food intake and increasing energy expenditure. The results of the present study indicate that, among overweight and obese women consuming an energy-restricted diet, ALA produced modest additional weight loss compared with an energy-restricted diet alone.

Huerta AE et al. Effects of alpha-lipoic acid and eicosapentaenoic acid in overweight and obese women during weight loss. *Obesity.* 2015;23:313-321.

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Blood Goes Mainstream

by Karina Gordin

What do Tiger Woods, Troy Polamalu, Hines Ward, Jon Dorenbos, and countless other professional sports players have in common? They all received platelet-rich plasma (PRP) prolotherapy for sports-related musculoskeletal injuries. For a steadily growing roster of athletes, this minimally invasive autologous injection therapy is a welcome alternative to the operating table, facilitating swift recovery and prompt return to the game. Initially making headlines as an injection procedure tantamount to blood doping, PRP is now a first-line treatment amongst team physicians, involving reinjection of drawn blood that has been centrifuged to concentrate platelets and growth factors responsible for tissue repair. And yes, this safe and cost-effective regenerative medical injection technique ultimately enables pros to train longer, recover faster, and play harder – legally.

During the 2009 AFC Championship Game (just two weeks before Super Bowl XLIII), wide receiver Ward sustained a debilitating medial collateral ligament (MCL) sprain. Such a serious injury takes at least 4 to 6 weeks to heal and may have potentially compromised Ward's chances of competing, as has happened with Ty Warren and Leigh Bodden.¹ Obviously, Ward competed and caught the decisive 38-yard pass, ultimately earning the title of Super Bowl MVP. However, what's less obvious is the fact that Ward's swift return to the field is largely due to a PRP injection into his overstrained MCL. The series of injections accelerated the knee ligament's rate of healing and restored its original tensile strength. Strong safety Polamalu promptly returned to top form following PRP treatment for a strained calf, enabling the critical 40-yard pass interception for a touchdown. L.A. Dodgers pitcher Takashi Saito partially tore his ulnar collateral ligament (UCL) just 2 months before the impending pennant race, in 2009. A surgical treatment approach may have shelved Saito's career for 12 to 14 months, ending his season, but Dodgers team physician promptly administered PRP into Saito's injured throwing elbow, and sent him off to pitch pain free.

There are countless notable examples of PRP reversing potentially career-ending injuries, sustained from the fierce physical nature of professional sports: full-speed collisions; blindsiding; battering ram; body checks; heading; kicking; repetitive serving at 140 MPH; pitching at 100 MPH; 5-minute-mile runs for 26 miles; and subsequent sprains,

strains, dislocations, hernias, laxities, partial tears, and plantar fasciitis – all of which PRP can tackle. All that's required of the athlete for the repair of such conditions is a few tablespoons' worth of drawn blood (regularly shed anyway in the name of athleticism) and an hour of time, once per week for up to 8 weeks.² In that hour a licensed prolotherapist may draw approximately 30 to 60 ml of venous blood, after which it's placed in an FDA-approved centrifuge and rotated for 15 minutes to separate blood into platelet-poor plasma (PPP) and platelet-rich plasma (PRP).³ This is where it gets interesting. PPP gets discarded, while the PRP is reinjected directly at the site of injury. No sweat.

Curing sports injuries while enhancing athletic performance has never been faster, more cost-effective, and safer. In effect, PRP prolotherapy is built on the principle of harnessing the body's own capacity to heal, via platelets.⁴ These discoid blood cells ordinarily constitute 6% of blood composition, or about 200,000 platelets/ μ L. As points of reference, red blood cells (RBC) constitute 93%, 1% are white blood cells (WBC), and the remaining 55% of blood volume is plasma.⁵ The basis for PRP benefit lies in reversing such blood component ratios. Upon centrifuging, RBCs decrease to 5%, considering their minor role in the healing process, while platelets increase to 94% to stimulate recovery. That's 4x baseline, or 1 million platelets/ μ L.⁶ Studies have shown that this supraconcentrated solution facilitates wound healing and hemostasis (blood clotting) through secretion of growth factors, contained within alpha-granule storage units of platelet intercellular structures.⁷ Normally at resting state, platelets require a trigger to develop pseudopods, which spread over injured tissue and jumpstart the healing process. The trigger in this case is a PRP injection that delivers proliferative growth factor proteins, such as epithelial growth factor (EGF) and vascular transforming growth factor beta (TFG-beta), which stimulate cell replication, regulate cell migration and proliferation, promote type I collagen and protein synthesis, and ultimately regenerate connective tissue.⁸

Growth factor mechanism of action is pivotal for repair of connective tissue such as tendons and ligaments, which, on account of poor blood supply, are vulnerable to injury and quite stubborn to heal. In fact, PRP is increasingly applied to chronic nonhealing tendon conditions of the Achilles, elbow, patella, rotator cuff, and other anchoring fibers that are subject to repetitive overuse and absorb the brunt of

tremendous mechanical forces. In such cases, collagen fibers may develop microtears, leading to tendinopathy as characterized by pain, localized tenderness, and swelling. Moreover, because of inherently poor vascularization, tendons heal by scarring, which compromises strength and elasticity of the tissue, increasing risk of reinjury.⁹ So that's where PRP comes in – the supply of nutrient-rich blood nudges the body to repair injured sites when the normal stages of healing are stagnating. Following injection, the body takes care of the rest on its own (Figure 1). Namely, PRP improves circulation and initiates deposition of new collagen, which shrinks as it matures, tightening the tendon and restoring strength, flexibility, and thickness. But don't take my word for it – take the word of Philadelphia Eagles long snapper Jon Dorenbos, who gave PRP a shot, so to speak, for an "extreme case" of tendonitis.

KG: How did you sustain the injury for which you sought PRP?

JD: Being an active long snapper and having gone into my 9th season in the NFL, I'd snapped a lot. The repetitive motion caused chronic pain on the inside part of my left elbow. The injury was classified as an extreme case of tendonitis.

KG: What treatment options did you consider before turning to PRP?

JD: I considered everything under the sun. We even discussed the possibility of surgery. Taking the tendon and placing it above the muscle or below, I forgot. Either way, it was an option that I wasn't excited about. The surgery would've resulted in months of rehab and we weren't very optimistic with it having the results we were wanting. Because of that, that option was in one ear and out the other. PRP was the last option that was presented to me, and it worked.

KG: Did you turn to any other therapy to treat your injury before and/or after?

JD: I had a few cortisone shots over the years in my elbow. Those only resulted in short-term relief. Unfortunately I don't remember the name of the procedure, but I had a series of 5 shots in my elbow well before the PRP injection. It was a shot that consisted of glucose water and the minerals that our body produces to create scar tissue. The thought behind the shot was to heal the tendon that had a few minor tears by creating scar tissue in and around the tendon. The shots were painful and didn't help at all. Not even short-term relief. During the season before I had the PRP shot I "rehabbed" my elbow,

for a lack of better terms. I would have the stem machine hooked up to my elbow followed by ice massages and I was constantly putting my elbow in the cold tub. The team also had a massage therapist come in often to do massage therapy on my left elbow, and of the treatments I tried, that was by far the most painful. All these treatments were done before the PRP treatment was really known. When I had the PRP shot two seasons ago it was still a fairly new procedure.

KG: How did you find out about PRP in the first place? Were you at first skeptical?

JD: The PRP shot was presented to me by our team's head trainer, Rick Burkholder, and team physician, Dr. Peter DeLuca. The risks were low, but the optimism was high. At that point, I had tried many alternatives and was on the verge of getting desperate to find an answer. The pain got worse as time went by and it started to affect my quality of life.

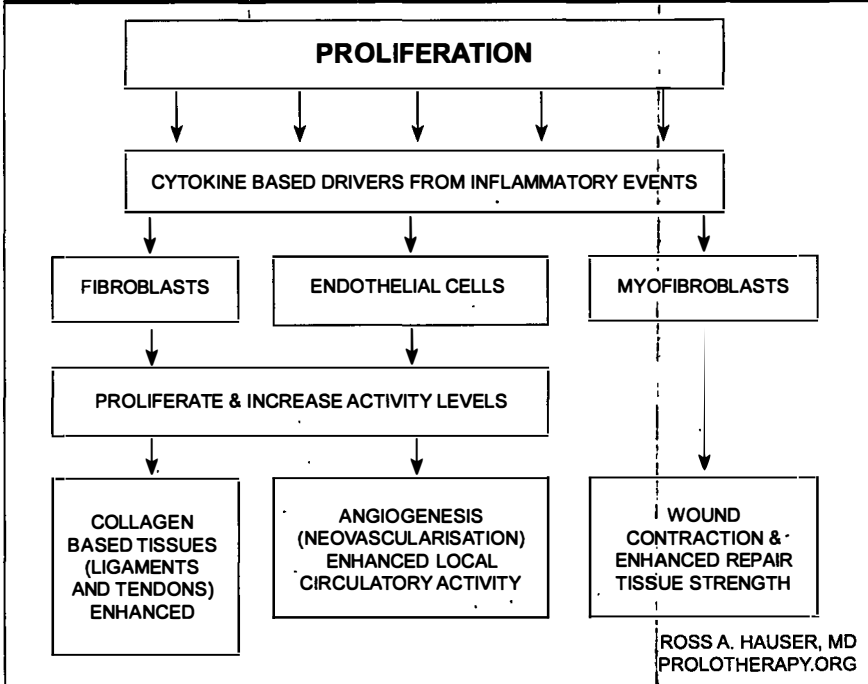
For instance, I'm left handed ... I write, eat, and brush left-handed. I started to eat and brush my teeth right-handed and started washing my hair with only my right hand.

KG: As expected, PRP relieved you of these inconveniences. What was your overall experience with it?

Figure 1

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Proliferation Cascade. Prolotherapy stimulates healing via inflammation. After prolotherapy solutions are injected into the injury site, a cellular reaction takes place in which various cells, including fibroblasts, endothelial cells, and myofibroblasts, form new blood vessels and ultimately lay down collagen, which enhances tissue repair and strength.



Blood Goes Mainstream

►

JD: Dr. DeLuca informed me that after the shot the pain would get worse before it got better. He estimated that if the shot worked, it would take about 8 weeks to heal. I got the shot right after the season ended. The pain definitely got worse. I couldn't bend my left arm for weeks. During this time I was wondering if I had done more bad to my body than good. Sure enough, week 8 came around and it seemed like it happened overnight, the pain was gone... and I mean gone. After dealing with this for years, after the PRP cycle, the pain was gone and I haven't had a problem since. I've changed nothing in my workout routine and still snap about the same amount.

KG: And how many injections did you receive to reach this point?

JD: 1 PRP shot

KG: Was the shot painful?

JD: Yes. Taking blood isn't bad. Then they spin it and separate the plasma and cells. The reinjection of the plasma isn't fun. They basically put the needle to the bone and injected the plasma as they pull the needle out. It isn't as bad as it sounds, but not fun. It's worth it.

KG: Did you experience any side effects?

JD: No, just the symptoms associated with the healing process.

KG: Do you recommend this therapy to others?

JD: Definitely. It all depends on the injury, but if your doctors are optimistic that the PRP shot would help, I recommend it.

KG: Would PRP be your first choice of treatment in the event you sustain another musculoskeletal injury?

JD: Yes, if it was an option. I would definitely try this before even considering any type of surgical procedure.

KG: Do you know of anyone else who received PRP?

JD: I have a few friends who have had the PRP shot. One had it 2 weeks before he played in the Super Bowl ... and they won.

I would like to revisit a few points that Dorenbos made, particularly his comment concerning surgery resulting in months of rehab. Except for a complete tear of a ligament or tendon, surgery may indeed be a less favorable option, considering the prolonged rehabilitation time, risk of complications due to removal of tissue, placement of foreign objects, infection and trauma, and of course differential costs. About the latter, one study determined an average cost of \$50,000 for an inpatient operative procedure and follow-up rehabilitation, with an average of 7 months before returning to full work duty.¹⁰ PRP is about 200 times less expensive, and there's no downtime. In fact, following treatment, the prolotherapist may likely recommend resuming exercise and daily routines as a form of rehab.

As with the surgical approach, Dorenbos was uncertain about cortisone shots, which he remarked only offered him short-term relief. PRP, on the other hand, is like spot-welding, reinforcing the impaired connection between muscles to bone (tendons), or bone to bone (ligaments), resulting in normal underlying function and long-term pain relief. Short-term pain associated with the injection is a slightly different story, considering the most sensitive fibrous layer, the periosteum, involved in the welding of already tender tissue. The temporary soreness may be likely due to stimulation of the body's inherent response to vital mediators, but the soreness is good, like the kind after a decent workout.

As Jon noted, "pain got worse," but then, "Sure enough, week 8 came around and it seemed like it happened overnight, the pain was gone ... and I mean gone."

But for the 260-pound tackler, who is quite sensitive to a needle pinch, perhaps some pampering may be in order. In such instances, the prolotherapist may include 1 cc of 1% of the local anesthetic lidocaine and 1 cc of 0.25 Marcaine in the injection, and offer a stress ball to squeeze. But as they say, *no pain, no gain*, and in this case there is much gained, with minimal to no risks and side effects. After all, PRP is minimally invasive, and uses one's own (autologous) blood, thus eliminating any risk of allergic reactions. In 1993, a survey was published examining prolotherapy injections performed by 95 physicians on a total of 494,845 patients.¹¹ Of these, 343,897 (70%) received the treatment for low-back pain, 98,430 (20%) for other spinal areas, and 27% for non-spine peripheral joint conditions. Only 66 secondary complications were reported, including 24 reports of minor reactions, and 29 cases of pneumothorax, in which cases the physicians inadvertently inserted the needle into the lung cavity; even so, these cases are isolated and resolved without serious setbacks. There were 14 reports of serious side

Figure 2



Resting platelets are smooth and disc shaped (left). Activated platelets have an irregular shape with many protruding pseudopodia.

(<http://www.perfusion.com/perfusion/articles/general9905-platelet-anatomy/>)

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effects, totaling the number of patients who experienced complications to 80 of 494,845, or 0.00016% risk probability.

For unresolved sports-related orthopedic injuries, PRP prolotherapy is steadily emerging as a cutting-edge, first-line treatment in regenerative medicine. The quick and simple preparation protocol, cost-effectiveness, clinical efficacy, and overall safety offer the type of reliable and innate care that both professional and recreational athletes may depend on. But whether treating an injury sustained in the name of a pastime or paycheck, PRP is an equal-opportunity injection that restores strength, enhances performance, and decreases recovery time, ultimately enabling prompt return to injury-prone games, in which the athletes may swing, jump, run, pitch, and bravely batter their way back to PRP.

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Notes

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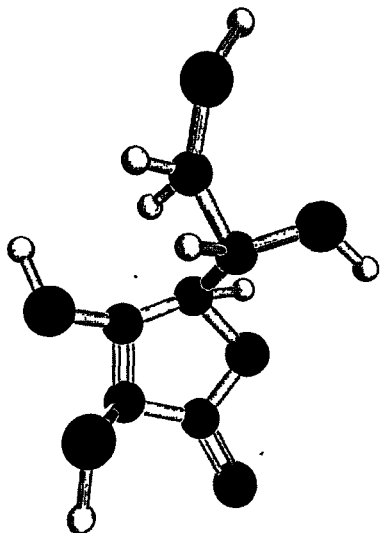
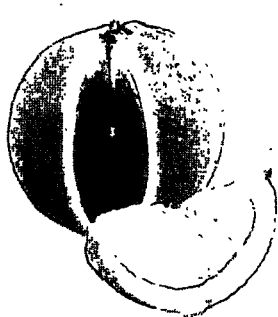
Blood Goes Mainstream

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Protein Intake and Mortality: New Research Says to Eat a Low-Protein Diet Until Age 65 Then Switch to High-Protein Diet

by Jacob Schor, ND, FABNO

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A recent paper is making a few waves and going to upset some people, such as advocates of certain high-protein diets. The research is also going to bother those companies selling supplements that promise to raise IGF-1 levels. Here's the quick story.

In early 2014, the journal *Cell Metabolism* published a paper on how high-protein diets affect lifespan.

Researchers compared dietary protein consumption in a US population cohort and looked for associations to overall and disease-specific mortality. The cohort studied consisted of 6381 adults aged 50 and over from the NHANES III, a nationally representative, cross-sectional study. Mean age was 65 years and was representative of the US population in ethnicity, education, and health characteristics.

The participants consumed 1823 calories on average per day, of which the majority came from carbohydrates (51%), followed by fat (33%) and protein (16%), with most of it (11%) derived from animal protein. The percent of calorie intake from protein was used to categorize participants into a high-protein (20% or more of calories from protein), moderate-protein (10%–19% of calories from protein), and low-protein group (less than 10% of calories from protein).

Mortality was followed via the National Death Index until 2006, which provide timing and cause of death. The 18 year follow-up period covered 83,308 total person-years

with 40% overall mortality, 10% due to cancer, 19% cardiovascular disease, and 1% diabetes.

Members of the study cohort aged 50 to 65 reporting high protein intake had a 75% increase in overall mortality and a 4-fold increase in cancer death risk during the following 18 years. These associations were either abolished or attenuated if the proteins were plant derived. Conversely, high protein intake was associated with reduced cancer and overall mortality in respondents over 65, but a 5-fold increase in diabetes mortality across all ages.

These results suggest that low protein intake during middle age followed by moderate to high protein consumption in old adults may optimize health span and longevity.¹

These data suggest that people should shift their dietary patterns in two key ways. First, patients younger than 65 should be discouraged from eating high-protein diets, especially diets high in animal protein. They should be encouraged to shift toward vegetable protein. Second, patients over 65 should be encouraged to consume more protein, as it reduces overall and cancer mortality unless the person is at high risk for diabetes.

The increased risk for diabetes seen in the middle-aged population on high-protein diets was striking: subjects with no diabetes at baseline had a 73-fold increase, while those in the moderate protein group had a 23-fold increase in risk of diabetes mortality. These increased hazard ratios may

be somewhat inaccurate due to the small sample size; there were only 21 diabetes deaths among persons who did not have diabetes at baseline, only 1 from the low-protein group.

Insulin-like growth factor-1 (IGF-1) was significantly lower among those 50 to 65 years old with low protein intake, while for those 65-plus the difference between the effects that high- and low-protein diets had on IGF-1 were insignificant.

The differences in mortality reported in this study are not small. The researchers found that eating a diet rich in animal proteins during middle age makes you four times more likely to die of cancer than someone with a low-protein diet; this is a an increased risk comparable to smoking.

The study was actually more complex than reported in the summary above. It also involved separate cell studies using yeast and animal studies using mice to examine these same questions. The impact that high-protein diets have on cancer progression was confirmed in mice implanted with melanoma cells. The tumors in the low-protein diet mice grew significantly slower and remained significantly smaller as the experiment progressed.

To test the hypothesis that older subjects on a low-protein diet become malnourished because they have difficulty absorbing amino acids, an experiment was run in which both young and old mice were fed either high-protein or low-protein diets. Old mice fed a high-protein diet for 30 days gained weight. Old mice but not young

mice fed a low-protein diet lost 10% of their weight by day 15, confirming the conclusion that the impact of high- or low-protein diets may vary with age.

Mice were also used to test the hypothesis that the growth hormone receptor and insulin-like growth factor-1 (IGF-1) together promoted cancer progression, melanoma cells were implanted into GHR and IGF-1 deficient mice along with normal controls. Tumor growth was far slower in the mice that did not have GHR or were lacking in IGF-1.

Protein intake was also tested on mice implanted with breast cancer. By day 18 after implantation, differences were already seen. Tumor incidence was 100% in the mice on the high-protein diet while only 70% in the low-protein-diet mice. By the end of the experiment at day 53, tumors in the low-protein-diet mice were 45% smaller.

Yeast were used to test the hypothesis that began to form after reviewing the human data, which suggests that the level of amino acids is linked to lifespan. Yeast were grown in media with differing amino acid concentrations. By day 5 of the experiment, the yeast exposed to high amino acids had a 3- to 4-fold higher mutation rate. By day 8, yeast grown in high concentrations of amino acids had a 10-fold decrease in surviving cells.

The strong association between protein consumption, IGF-1, disease, and mortality seen in this study has not been seen in some earlier reports; perhaps the age effect was not considered. Saydah for example reported no increase in all-cause, heart, or cancer mortality when comparing lower quartiles with highest quartiles of protein consumption in the NHANES III data.²

That the amount of animal proteins accounted for a significant proportion of the association between overall protein intake and all-cause and cancer mortality is in agreement with other recent reports on the association between red meat consumption and death from all-cause and cancer. Fung et al. reported in 2010 that low-carbohydrate diets are associated with increased all-cause mortality.³

In 2009, Sinha et al. used data from the National Institutes of Health Diet and Health Study cohort of half a million people aged 50 to 71 and reported that red and processed meat intake was associated with increased total mortality, cancer mortality, and cardiovascular disease mortality.⁴

Pan et al. reached this same conclusion in 2012 after analyzing data from 37,698 men in the Health Professionals Follow-up Study and 83,644 women in the Nurses' Health Study. They reported that red meat consumption was associated with an increased risk of total, CVD, and cancer mortality. After documenting 23,926 deaths during 2.96 million person-years of follow-up, Pan et al. calculated that for every 1 serving per day of unprocessed red meat eaten, the risk of total mortality increased by 13%. For processed red meat, 1 serving per day increased total risk by 20%. These researchers estimated that substitutions of 1 serving per day of other foods (including fish, poultry, nuts, legumes, low-fat dairy, and whole grains) for 1 serving per day of red meat were associated with a 7% to 19% lower mortality risk.⁵

The idea that a high-red-meat diet is associated with mortality is not new.

Although these new dietary suggestions are straightforward, it will not surprise us if proponents of various popular dietary regimes offer up resistance. A number of common dietary strategies, including Atkins, the paleo diet, and the ketogenic diet, can increase a follower's protein consumption to the degree that, if this study's conclusions are true, they may

adversely effect the person's health, increasing risk of diabetes, cancer, and early mortality.

Anyone advocating high-protein diets for people less than 65 years old should be ethically bound to either find some fault in this paper or find an overriding justification for such a diet that outweighs the impact that high protein may have on long-term survival. The same caution should apply to using dietary supplements for the purpose of increasing IGF-1 levels. Any benefit of increasing IGF-1 needs to be justified in light of these data that suggest an increased risk of cancer, diabetes, and bottom line, dying when IGF-1 levels are high.

This idea that protein consumption should vary with age is a new concept that the public is still unaware of. It will take effort to explain this idea. It might be a good idea to print off a copy and read the full paper yourself before attempting to speak to your patients. The full text is available online for free: [http://www.cell.com/cell-metabolism/pdf/S1550-4131\(14\)00062-X.pdf](http://www.cell.com/cell-metabolism/pdf/S1550-4131(14)00062-X.pdf).

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Insulin Resistance as a Symptom

by William Ferril, MD

If you acquire insulin resistance, it not only bolsters disease processes, it's also a symptom of a deeper underlying root. As I recall well-written articles that often mention insulin resistance, its root cause remains beyond the meat of the discussion. I believe that an intellectual dead end occurs whenever the vernacular, insulin resistance, is implied as the root abnormality.

I believe that insulin resistance is secondary to increased insulin need most of the time. But before I can attempt to convince you of this mostly overlooked fact, I need to perhaps widen your view of why you may find yourself needing excess insulin. Insulin on the rise is not the root of the problem most of the time, but rather just one of the symptoms of your body becoming imbalanced.

During my over 5-year sabbatical studying for clues to how bodies heal, I came across several important situations for why insulin need elevates (insulin resistance). Like other authors, I concur that until the liver begins to purge itself of excessive fat; increased insulin need (resistance) continues its stranglehold. However, some really pertinent science exists that bolsters the ability to heal from pathologies such as fatty liver, metabolic syndrome, and type 2 diabetes, but they require inquiring beyond insulin resistance.

Four root causes explain increased insulin need (resistance). Some of them are apparently secret. I find it very curious that although they are well documented by mainstream textbooks of yesteryear and credible literature of today, the significance of

these factors remains largely ignored. The big four factors promoting increased insulin need (resistance) are: falling nonsuppressible insulin-like activity of the blood stream, reversed mineral intake, heightened adrenal activity (not as extreme as Cushing's syndrome/disease but heightened), and excessive motivation through stress (type A personality disorder).

In other articles and books, I delve deeply into the evidence, endocrinology, physiology, and oxidative stress that these four aberrations promote. Here, I provide a brief introductory explanation for the sake of clarity. But first, a brief overview of what insulin is and is not.

Insulin Toxicity Symptoms

The majority of diabetic victims (90%) suffer from excessive body exposure to insulin (explained below). Mainstream medicine calls prediabetes, metabolic syndrome, and eventual blood sugar rise type 2 diabetes (90% of all diabetics). Unfortunately it is not commonly appreciated that both disease processes involve acquiring excessive insulin need (explained below).

Insulin levels determine much about how much fat collects in your arteries and how fat you become. You see, the insulin message is all about energy storage. Most body energy is stored as fat (cholesterol and triglyceride). Cholesterol and triglyceride are the body's main pH neutral type fat choices. Without being rendered pH neutral, excessive fatty acids cause metabolic havoc. For this reason, the amount of insulin arriving within the liver determines

how much cholesterol and triglyceride (fat) it manufactures. It also elucidates why high carbohydrate intake deleteriously affects your cholesterol profile.

Note that during the years a patient is in the prediabetic state (metabolic syndrome), the arteries are subjected to excessive insulin-directed LDL (cholesterol and triglyceride attached to specific carrier proteins) synthesis secondary to elevated insulin body exposure. Making matters worse, certain immune scavenger cells (macrophages) lining the arteries ingest LDL particles and grow and grow, eventually producing a fatty streak. The fatty streak is the earliest recognized lesion for developing heart disease. After many years, more and more macrophage cells are recruited and they stack on top of each other. In addition, other factors besides excessive sugar (a rusting agent) accelerate ongoing inflammatory injury to arteries (more on these factors are discussed in other articles). It is important for me to emphasize that the common terms plaque, CRP, and calcium accumulation superficially describe this ongoing injury resulting from oxidation (rust). For example, injured cells accumulate calcium. Somewhere down the road of this insidious process, an additional destabilizing event occurs (plaque rupture), promoting a clot to form. A heart attack or stroke follows. This series of events describes the first and third most common causes of death in the Western world.

Whether or not you suffer from elevated blood sugar or require excessive insulin to normalize your

blood sugar does not explain the complete mechanism of arterial insult. Both type 2 diabetics and prediabetics (metabolic syndrome) suffer from excessive insulin body exposure that fosters the above sequence, promoting heart disease. Also briefly introduced above, other inflammatory agents oxidize (rust) this accumulating debris, causing plaque to form and calcium to abnormally sequester. Suffering from these inflammatory promoters is commonly measured by CRP, homocysteine, hemoglobin A1c, and serum iron levels, to name just a few. Making matters even more alarming, mainstream medical adherents still promote elevating insulin secretion rates with medication! I ask how one hopes to enjoy wellness again if insulin levels rise.

It is important to understand that half of all heart attack victims enjoyed a "normal" cholesterol level. How could this be? Well, visualize with me that your liver is like a faucet that spews out newly manufactured LDL cholesterol. Your bloodstream is like the sink but your macrophage and abdominal fat cells are like the drain. So the bigger the drain is, the more potentially misleading cholesterol levels can be about your true risk of dropping dead. This relationship also elucidates why if you suffer from abdominal obesity, you are also likely silently suffering from fat growing in your arteries (whatever your cholesterol level), because insulin exposure determines abdominal visceral fat.

The initial take-home point is that you will never reverse this initially silent killer until you find a way to lower your body exposure to insulin. In order to do this, you need to know about the first perpetrated scientific secret. Insulin does not work alone!

Secret #1: Insulin Doesn't Work Alone

I find it more than curious that there is not even a cursory discussion in the popularized mainstream medical media and literature about the fact that insulin doesn't work alone. In

fact, normally insulin provides only 7% of the insulinlike activity within the body! However, when something happens to the other 93% of insulinlike activity, excessive insulin need arises to normalize a blood sugar elevating event (carbohydrate consumption). Many of my colleagues continue to call this *insulin resistance* because they stop looking beyond the fact that insulin has risen and hence fail to ponder why.

I suspect that hiding this fact from doctors helps sell more symptom-control prescriptions, but how cruel that patients may needlessly suffer only because their doctors remain in the dark about what science truly knows about sugar exiting the bloodstream. The good news is that the ignored 93% of insulinlike activity is amenable to being improved and hence healing is possible from the fatty liver, diabetes, or prediabetes state. Suffice it to say for now that the lack of this second component of blood sugar lowering ability promotes one mechanism (out of the seven) for being behind many *hungry cell syndrome*-originated diseases as well (explained in other articles). Here I will initially focus on how the disease processes of fatty liver, metabolic syndrome, and diabetes, in many ways, are all examples of varying degrees of lacking a secret hormone.

If you want to stay healthy or regain health, you must see beyond the insulin half-truth story perpetrated ad nauseam. It helps if you visualize the total insulinlike activity of your bloodstream as consisting of two types of "fuel nozzles." Just as your car requires a fuel nozzle before its tank can be filled, so too do body cells require a molecular nozzle to suck fuel from the bloodstream. The first is insulin and the other (called in the older literature) the nonsuppressible insulinlike activity of the bloodstream. It is important that I emphasize that this second, apparently secret, fuel nozzle ideally makes up 93% of blood-sugar lowering ability, while insulin makes up only the remaining 7%!

It turns out that the 93% of secret insulinlike activity (the nonsuppressible insulinlike activity of the bloodstream) provides the nutritional advantage to your bones, most organs, joints, and muscles. This is important because I have found that all healthy people enjoy ample amounts of this other secret fuel nozzle coursing within the bloodstream! I have further observed that lean body mass cannot be maintained/restored unless this secret fuel nozzle is optimized. It is important that I emphasize that restoring lean body mass only becomes possible when this secret hormone's levels improve (explained below).

In contrast, the other part of the secret concerns the fact that ideally insulin mostly shunts nutrition into your liver and fat (explained below). You see, because of your body anatomy, insulin always secretes from the pancreas into the portal vein, which heads straight into the liver, where it confronts 200,000 pure insulin receptors per liver cell. If you understand this mostly overlooked fact, you will now be in a place to see that your liver functions as a very effective insulin trap. This means that normally very little insulin traverses the liver to reach general circulation.

In addition, the message of insulin within the liver is all about energy storage. There is an upper limit for how much sugar the liver can store (100 grams in the best of circumstances) but no upper limit for how much it turns carbohydrate into fat (cholesterol and triglyceride). For reasons not entirely understood, some of this fat accumulates within the liver (the origins of fatty liver). However, most of it attaches to specific proteins and secretes as LDL cholesterol. Recall with me that LDL cholesterol has a particular affinity for abdominal fat cells and macrophage cells that line the arteries.

Visualize the ongoing "tug of war" being played out between these two fuel nozzles as to which cells in your body receive adequate nourishment!

Insulin Resistance

► Only if the nonsuppressible insulinlike activity of your bloodstream remains adequate can your cells outside your liver and fat cells ever hope to receive adequate nutrition. Beware of the secret; only when 93% of insulinlike activity remains sufficient will insulin need to be minimized when you encounter a blood-sugar elevating event (eat carbohydrates). Furthermore, only here will cells beyond the liver and fat such as bone, muscle, joints, and most other organs remain well nourished. Only in this situation will lean body mass, its functional component, continue to be adequately nourished!

However, if something starts inhibiting the nonsuppressible insulinlike activity of bloodstream levels, fuel nozzles beyond the liver will become scarce. Here insulin need increases, but because of body anatomy, liver and fat cells will become the nutritional hogs of the body. Slowly but surely these facts describe how both you and your liver become fatter!

So what promotes the nonsuppressible insulinlike activity of the bloodstream levels? The answer is multifaceted, but its comprehension and implementation allow healing from numerous *hungry cell syndrome*-derived diseases. In pursuit of this understanding, you must always remember that in most cases of diabetes the pancreas is perfectly healthy.

Secret #2: In Most Cases of Diabetes, the Pancreas Is Perfectly Healthy

In most cases of diabetes, the pancreas is perfectly healthy. If I can convince you of this, then you will be more amenable to pondering the evidence for what really causes the majority of fatty liver, metabolic syndrome, and diabetes: a lifetime of unhealthful living. More accurately, it is really just the first prerequisite. Our bodies were designed to eat real foods (explained in the potassium subsection) and move around. If you

practice a lifetime of forgetting these basic facts, around middle age, the stage is set for some very important hormone imbalances. Making matters worse, it is these same hormones that powerfully determine how much of the all-important, nonsuppressible insulinlike activity of the bloodstream you enjoy.

You cannot heal from fatty liver, metabolic syndrome, or type 2 diabetes until you raise nonsuppressible insulinlike activity of the blood stream. Mainstream medicine obstructs understanding this fact by using several different names for the nonsuppressible insulinlike activity of the bloodstream. Its other names include IGF-1 (insulinlike growth factor type 1), somatomedin, and sulfation factor. Perpetrating this disconnection keeps doctors practicing in the dark about the truth of this very important substance circulating within the bloodstream. From here on out I will discuss it as IGF-1, but remember its other three names used throughout the mainstream literature.

So often the real bedrock setback with fatty liver disease regards your impaired ability to manufacture IGF-1 again until enough fat purges from the liver. Adequate anabolic hormones arriving within the non-fatty liver instruct IGF-1's manufacture. Growth hormone release determines how much of it releases with the stored liver sugar and fat during a falling blood sugar (fasting, exercising, a good night's sleep). Notice how a fatty liver promotes diabetes because growth hormone is diabetogenic when your liver fails to make sufficient IGF-1. Thyroid hormone determines how much growth hormone your pituitary manufactures. Suffice it to say that it can become really complicated, but an overall appreciation for why fatty liver condemns your best efforts at healing until it is purged is warranted.

It is generally agreed that nonalcoholic fatty liver disease (NAFLD) risk is best predicted by the presence of insulin resistance. Other authors have noted lesser causes, but I note that most of these contribute to

your needing more insulin as well. For example, drugs such as tamoxifen, corticosteroids, and estrogens all increase your insulin requirements. Estrogen increases growth hormone release, but inhibits IGF-1 release, a diabetogenic effect. Tamoxifen shares estrogen's diabetogenic effects even though it is an estrogen antagonist in other regards, while corticosteroids increase gluconeogenesis, which also increases insulin need.

Most of these same papers point out that if you acquire metabolic syndrome, it increases your risks of suffering from NAFLD by threefold. Consistent with these observations regarding the primary predictor of risk being insulin resistance, by definition metabolic syndrome itself is thought to result from the consequences of insulin resistance.

For these and other reasons (read on), I suspect that NAFLD will turn out to be yet another common aberration inflicting bodily harm from the increasingly growing list of metabolic syndrome's presenting features such as visceral fat, increased thrombogenic potential, skin tags, hypertension, prediabetes, and abnormal lipid profiles.

Connect the dots of increased insulin need. A lifetime of unhealthful living leads to very little fasting, exercising, and proper sleep habits. These happen to be the main stimulants for your being able to release IGF-1, which lowers your insulin need. Then middle age happens, when you begin to suffer from tired-out endocrine glands and hence substances such as growth hormone and anabolic hormones wane. These facts alone contribute to your increased insulin need, which upregulates your fatty liver problem. But metabolic syndrome is even worse than this because another overlooked common aberration intensifies your suffering.

Secret #3: Cortisol Excess Promotes Fatty Liver Because It Is the True Cause of Metabolic Syndrome

Cortisol's message content to your body's cells is opposite to that

of the anabolic steroids. Cortisol represents the catabolic steroids that consume body structure (proteins) for fuel generation causing negative nitrogen balance. Adrenal glands secrete cortisol. Cortisol-endowed secreting machines were the warriors of primitive times, because in a pinch, ransacking proteins (cell "furniture") for fuel enhanced the physical strength and hence the odds for survival.

Unfortunately, today stress is largely both chronic and mental in nature. As a consequence, if you inherited this trait, you end up needing excessive insulin to bring down the inappropriate blood sugar surges because no physical threat ever comes. Compounding this trait's weight-gaining dilemma concerns the fact that once you experience elevated blood sugars caused by this mechanism, it also suppresses IGF-1 (the nonsuppressible insulinlike activity of the bloodstream) release.

If you experience excessive cortisol secreting out of the adrenals while awake, you will tend to endure slightly elevated blood sugars throughout the day (the true cause of metabolic syndrome). Unfortunately, it is a slightly falling blood sugar (about 70) that promotes the release of growth hormone, and subsequently this causes your liver to release IGF-1. Recall from above that IGF-1 keeps lean body mass well nourished and diminishes the need for excessive insulin. In contrast, as also mentioned above, insulin gives the nutritional advantage to liver and fat. Hence it promotes the liver as nutritional hog situation (excessive insulin need to normalize a sugar load described above).

So day after day, slowly but surely, what was metabolically active and structural enhancing tissue, proteins, and then sugar eventually becomes fat and deposits in the arteries and abdomen (metabolic syndrome). Without some doctors' realizing it, this heightened adrenal gland ability to secrete cortisol for a given stressor explains much about what scientists call metabolic syndrome (not all the way to frank Cushing's disease/

syndrome levels but heightened). It also explains the mechanism by which you become afflicted with this syndrome: you age so quickly (insufficient anabolism with heightened catabolism promoting excessive carbohydrate creation without even eating sugar). It also explains your relative carbohydrate intolerance (weight and cholesterol worsening) at its root if you suffer from metabolic syndrome.

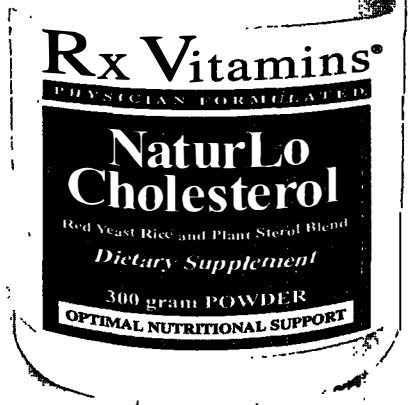
Insulin Resistance

I believe that some doctors fail to realize these basic scientific facts because they do not test for the important metabolites of cortisol exiting in urine. After reviewing thousands of 24-urine test results, a pattern for this association became clear to me. In fact, over 50 years ago, scientists documented the association

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OPTIMAL NUTRITIONAL SUPPORT

Insulin Resistance

of many obese (metabolic syndrome) patients and heightened cortisol metabolites exiting in their urine.

Secret #4: Potassium Deficiency Promotes Fatty Liver Disease

So far I have concentrated on the important role that hormones play in combating, if not defeating, fatty liver, metabolic syndrome, and diabetes. But these are multifactorial diseases, with other important causes. Potassium deficiency is one of them. I call this the reversed mineral intake state (explained below).

As a life-sustaining mineral, potassium is essential for proper kidney, nerve, heart, and muscle function. As an electrolyte, potassium helps to balance fluids in the body and enhance nerve cell signal transmission and muscle contraction because it is essential for creating cell voltage (explained in fatigue articles). Another way that potassium contributes to cellular function is by being centrally involved in delivering carbohydrates to cells. In fact, without sufficient potassium available, insulin cannot function. This fact describes yet another cause of insulin resistance (increased insulin need), unfortunately not appreciated by some doctors (see fatigue articles).

The sad truth about many of my patients', modern-day eating habits is that they are largely based on processed foods. Enticing as they may be, processed foods are woefully lacking in minerals and nutrients essential for fighting off diseases (such as fatty liver, metabolic syndrome, and diabetes) and aging. Processed food manufacturers today dump loads of harmful additives (e.g., sodium) into their products to increase shelf life and enhance taste. But in the process, they suck out the majority of the natural minerals, nutrients, and fibers to create what I call a reverse mineral content (consult a food mineral table). Potassium in particular takes a big hit (note the marked differences between unprocessed foods and natural foods'

mineral content in the mineral table consulted). Natural foods tend to be high in potassium and low in sodium. Processed foods tend to be just the opposite.

For this reason, it's nearly impossible to establish a proper ratio of nutrients and minerals in your body with a diet of too many processed foods.

Although this fact is not commonly appreciated, your body needs to acquire about four times as much potassium as sodium. But as you arrive at middle age with less efficient kidney function, the effects of a processed food diet begin to catch up; the ratio becomes unbalanced. Sometimes you can really suffer from them, becoming extremely unbalanced.

Visualize with me: after many years of reversed mineral intake, excessive sodium takes over, which leads to more fat buildup. Less potassium on hand means a diminished ability to deal with the carbohydrates that you eat, secondary to insulin malfunction, but also leads to more fat buildup because sugar storage ability requires sufficient potassium being available. In contrast, turning sugar into fat pathways does not require potassium within the liver. For this reason, that unbalanced ratio alone can put you at risk for fatty liver, metabolic syndrome, diabetes, and worsening cholesterol profiles. As more evidence accumulates, it will probably also explain yet another major contributor to fatty liver disease: insufficient dietary potassium with carbohydrate intake!

High-sodium, low-potassium diets are unhealthy for you, but it's an especially bad combination if you suffer from fatty liver, metabolic syndrome, or diabetes – because if you suffer from potassium deficiency, it alone creates insulin resistance (increased insulin need). Ingesting prescription medication that increases your pancreas insulin output only compounds the problem of upregulating fat synthesis (cholesterol and triglyceride). Remember, this abnormally triggers fat-making

messages in your liver from the carbohydrates ingested. With less available potassium, your liver is forced to upregulate cholesterol and triglyceride synthesis to normalize a sugar load. Some of this slowly accumulates inside the liver, causing it to eventually become fatty.

Because of these relationships, when your insulin, glucose, sodium, and potassium ratios become imbalanced, it puts you at a higher risk of developing any number of diseases, and it further complicates an already precarious situation for diabetics.

My goal is to balance levels of potassium sufficient to ward off the need for increased insulin. Proper potassium levels provide one pillar for keeping both carbohydrate and consequent insulin levels in line. It also provides you with the means for less fat (triglyceride and cholesterol) buildup. This step allows you a higher chance of ridding yourself of fatty liver, metabolic syndrome, and diabetes, if not preventing them altogether.

You can start the journey to better potassium intake today. In fact, the path is an easy one. Simply drop your dependence on processed foods and commit to a diet of real (natural) foods. Load up on low-carbohydrate but high-potassium fruits such as avocados and berries. Fresh vegetables are also a great source of potassium, as are unprocessed meats such as chicken, fish, turkey, lamb, and beef.

Simple as it sounds, the role that potassium plays in acquiring metabolic syndrome and diabetes is largely understated in the traditional medical community. Again, my strictly traditional colleagues are too focused on symptom control. But you don't have to be that narrow-minded. Take your newfound knowledge and put potassium to work for you the way it should.

Finally, note that I have removed many high carbohydrate-containing foods in the above short list even though they prove rich in potassium. There is a very good

reason for this precaution. Although not fully comprehended by some doctors, diabetes is a disease of sugar (carbohydrate) intolerance. The work of Richard K. Bernstein, MD, in *Diabetes Solution* (Little, Brown) convinced me of this truth, and I have been using his protocols for over 15 years with remarkable success. Beyond Bernstein's valuable contribution are the facts about fatty liver, type 2 diabetes, and metabolic syndrome being almost always caused at their root by an excessive cortisol secretion, reversed mineral intake, and consequent diminished IGF-1 levels (explained above).

The next time you read about insulin resistance, I hope you begin to suspect that it is only a sign that something deeper is at play. Its persistent implied explanation as a root jolted me to begin uniting other pertinent facts with this all too common scourge, such as the root of why insulin resistance (increased insulin need) occurs in the first place. After testing thousands of patients in the above-described manner, scouring the medical literature, and implementing countermeasures to heal my patients from fatty liver, metabolic syndrome, and diabetes, four commonly overlooked factors glare at me. Yes, I have found that increasing IGF-1 levels, identifying excessive cortisol metabolite exposure, restoring mineral balance, and stress reduction measures (a good night's sleep!) all profoundly contribute to my patients' healing.

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Text in 24 hrs June 1.
7/10/2011

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Dr. Ferril cofounded the Bio-identical Hormone society with Jonathan Wright, MD, in January 2005. In this capacity, he lectured hundreds of doctors on the biochemistry of the steroids and the best testing methods. Since childhood he has remained fascinated with answering the mystery of why some people age gracefully while others do not. The biochemistry of the steroids provides part of the answer.

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7/10/2015

Parasites: An Ancient Diagnosis for Modern Times

by Allison A. Hofmann, ND, and Dietrich K. Klinghardt, MD

Introduction

Practitioners of biological medicine should thoughtfully and routinely consider parasitic infection in their differential diagnosis. A differential diagnosis that fails to include parasitic infection, especially among the chronically ill patient population, is dangerously incomplete. Embracing an old and simple tenet common to all traditional medical systems may indeed serve us well: parasites infect us, often, and their presence can be life threatening.

Background

Protozoa and helminths are the broad classes of parasites that this article considers, as these are responsible for substantial morbidity and mortality worldwide.¹⁻³

Admittedly, our close interaction with these organisms elicits both positive and negative sequelae, and the definition of a parasite itself – an organism that lives at the expense of its host – fails markedly short of describing the intricate relationship that we have with parasitic beings. The emerging understanding of the relationship between our immune system, human biome, and all environmental influences is astounding. These complex relationships are none other than completely pervasive to who we are as human beings.

Within the past 25 years, the germ theory of illness has fully met its match in the hygiene hypothesis. A notable example is the use of *Trichuris suis* ova (pig whipworm eggs) in treating inflammatory bowel disorders.

However, it should be clearly understood that the vast majority of parasites, particularly those adapted

to infecting their human hosts, are not conveniently immunosuppressive in the authors' experience. Infestations may also not be localized in the gut; more commonly in chronic illness, patients become systemic with larval stages straying far from the intestines, setting up home in distant places such as connective tissue, liver, heart, and brain. By and large, parasites increase systemic inflammation and further disease processes.

Influenced by our ever-changing environment, ever-increasing toxic burden, and subsequent epigenetic mutations (epimutations), our coevolution with these creatures is an ever-changing phenomenon which is most certainly contributing to our human fate.

Research supporting the fascinating interaction between toxic burden and parasitic illness has been presented at several of Dr. Klinghardt's most recent and groundbreaking workshops. Parasites are not only more likely to infect hosts toxic with heavy metals, but many parasites also bioaccumulate metals and thus reduce the tissue levels of their hosts.⁴⁻¹⁰ Indeed, in modern times we may be in great debt to our parasitic infections. We should be mindful and respectful of this relationship, even when our end goal is eradication.

Beyond the Western Consensus

Physicians trained in Western medicine simply fail to consider that many parasitic infections exist in developed countries and thus clinicians lack the appreciation for the true threat that these diseases represent.¹¹ A telling example of modern susceptibility is *Taenia solium*

(pork tapeworm) infection, resulting in neurocysticercosis and seizures, occurring amongst the Orthodox Jewish population in New York City: patients who do not eat pork nor have traveled to an endemic country are still at risk.¹²

The most well-known and accepted parasitic infections in the US are pinworm (*Enterobius vermicularis*), *Trichomonas vaginalis*, *Toxoplasma gondii*, *Giardia intestinalis* (lamblia), and *Cryptosporidium* spp.¹ Otherwise, parasitology often falls under the jurisdiction of "tropical medicine," an area pertaining solely to developing countries and their travelers. A recent (2014) publication from *American Family Physician* titled "Neglected Parasitic Infections: What Every Family Physician Needs to Know" was a long overdue acknowledgment of some additional parasitic illnesses in the US, including Chagas disease (*Trypanosoma cruzi*), toxocariasis (*Toxocara* spp. of roundworms), and cysticercosis (*Taenia solium*), and their pervasiveness.

In actuality, the extensive medical literature concerning tropical medicine at large should not be considered "other." Our existence as a global community and the likelihood of parasitic exposure affects everyone despite modern hygiene, sanitation, and medical advances. Indeed, several parasites can infect humans without regard to hygiene and sanitation, via skin penetration: hookworm, *Necator americanus* and *Ancylostoma duodenale*, and the *Schistosoma* spp.

Emotional connotations surrounding parasitosis burdens its acceptance in general practice. Nothing is simultaneously more fascinating and

Willing organisms release biotoxins they have accumulated in HH's.

disturbing to the human experience than visualizing a macroscopic parasite. Passing a macroscopic parasite is a humbling experience indeed, and the mere possibility of doing so frightens most. Many patients seeking treatment of Lyme disease, autoimmune disease, heavy metal toxicity, and so on, are reluctant to embrace an additional or even more accurate assessment of their condition when parasitic infection is suggested. Much education and support must accompany parasitic illness, to gently desensitize patients to the diagnoses. Many patients become enthralled by their parasitic experiences, and we are privy to a plethora of visual documentation of their parasite "journeys": the macroscopic parasites, the unusual stools, dermatological manifestations, and what is understood to be the biofilm communities of parasites being expelled on a regular basis when the proper treatment is employed.

Lungworm, or a new species of roundworm identified by Lawrence A. Klapow as *Varestrongylus klapowi* (previously *Cryptostrongylus pulmonis*), is frequently encountered in the authors' experience. Many species falling under the "taxonomy unknown" category, including such phenomena as the rope parasite, are yet to be elucidated, but they are daily phenomena for our chronically ill population.¹³ It is speculated that the rope parasite is an intelligent biofilm community composed of different parasite DNA, previously referred to as simply "mucus" by the alternative health community.

Assessment and Diagnosis

Traditional Chinese Medicine, Ayurvedic medicine, and traditional naturopathic medicine have all classically considered parasitic illness in their teachings. It would serve our respective professions well to embrace these roots. The modern obscurity surrounding the existence and classification of parasites is upheld by diagnostic shortcomings. The rate of parasite detection is less than 12%, according to recent communication between Dr. Klinghardt and a leading parasitology lab. The gold standard of

many parasite diagnoses, stool ova and parasite testing, is notorious for its low predictive value; expertise in species-specific preparations and procedures is lacking.^{14,15} The eagerness to improve these bleak statistics is evident in a hopeful array of diagnostic advancements.^{16,17}

Nevertheless, performing a detailed physical examination, obtaining a thorough history of present illness, evaluating routine laboratory work, and utilizing the diagnostic prowess of established methods of biofeedback (e.g., Autonomic Response Testing, Matrix Reflex Testing, etc.) enable a practitioner to become perceptive to parasitic illness.

All aspects of the physical exam may pertain to this broad class of infectious disease. Organ-specific effects can be a consequence of a parasite's natural life cycle as well as accidental habitation. Abdominal palpation often reveals the "rope consistency" of chronic inflammation as most parasitic life cycles compromise bowel health. Palpable, nonfixated abdominal masses, gall bladder tenderness, and liver enlargements are frequent. Overt and subtle signs of nutritional deficiency (e.g., angular cheilitis, macroglossia, etc.), rashes and skin abnormalities (e.g., circumoral pallor), respiratory and cardiovascular abnormalities, fundoscopic findings, and so on may all support parasite involvement.

Review of systems is likewise all encompassing. Cyclical febrile illness, particularly correlating to moon cycles, and fatigue and various pain presentations including fibromyalgia should alert the clinician to infectious and toxic etiologies, including parasitosis. Any quality of bowel function may be present: diarrhea, constipation, and everything in between. Most patients are chronically constipated and/or experience irritable bowel symptoms. Patients may present with disturbed urinary function, unusual bleeding, shortness of breath, cognitive impairments, neurological symptoms (notably seizures), and/or a wide range of behavior (food cravings, sleep disturbance, etc.) Psychiatric symptoms may indeed be

pathognomonic for parasitic illness.

General laboratory investigation of the patient routinely reveals elevated eosinophil counts, especially under the duress of helminth infection.^{1,18} Protozoa are believed to reflect T cell, B cell, and macrophage dependent immune reactions, and thus a wide variety of white blood cell abnormalities are common.¹⁸

Absence of eosinophil elevation should not rule out parasitic infection. Note that the degree of eosinophil elevation may or may not correlate to severity of infection, both quantitatively and qualitatively; rather, correlation with present-time symptom severity is more likely.¹⁸

The authors routinely see eosinophils in high normal range, monocytes in high normal range in combination with a general low white blood count (5000 or below). Strong correlations exist between both microcytic and macrocytic anemia.¹⁹ Nutrient deficiencies are rampant, including decreases in fat-soluble vitamins due to sluggish bile. Marginally elevated liver enzymes are common with increased oxidative stress markers (e.g., elevated GGT). Lowered detoxification capacity and biotransformation can increase TGF beta 1, calcium protection, beta-glucuronidase, and so on, and these measurements should be considered dysbiotic consequences at large.

Indeed, the current concept of dysbiosis with its bacterial, viral, and fungal basis should be considered the "tip of the iceberg": deeper and more systemic parasitic issues are usually present. As mentioned earlier, heavy-metal toxicity and toxic burden findings at large should prompt a physician to consider parasitic illness.

In sum, many patients with negative parasitic labs will pass worms when empirically treated. Treatment often results in rapid improvements, although improvement can be delayed 4 to 6 months. Certainly the benefits of nutritional improvement, immune modulation, and so on take time, and the course of treatment needed for lasting improvement is quite variable. Many late-stage pathologies and

Parasites

► diagnoses, including any cancer under the diaphragm (liver, bowel, pancreas), should lead to parasitology workup and/or empirical treatment.

Treatment Options

Treatment of parasitic illness is an ever-evolving practice. Treatment options can be general and/or species specific but should always be tailored to the full range of functional diagnoses that each patient has. Those familiar with Autonomic Response Testing will observe that parasite illness often creates the phenomenon of "neurological switching." In the authors' experience, parasites are quite adept at controlling nervous function to their advantage. Therapies that amend any neurological switching in a patient will vastly increase the success of their antiparasitic regime. Those unfamiliar with Autonomic Response Testing can simply counsel their patients to pursue the full range of Emotional Freedom Technique tapping points every time they take their antiparasitic medicines. Acupressure with points concentrating on appropriate meridians is another effective tool that can augment any treatment program.

Rectal approaches to treatment are often paramount, allowing bioactive compounds to more effectively reach the liver and bowel. Chlorine dioxide enemas, eucalyptus enemas, milk enemas, and other herbal enemas and colon hydrotherapy are indispensable to most patients. We have seen the most success with chlorine dioxide enemas. Careful instruction and experience with chlorine dioxide ensures patient safety and success.

Nexus Suppository by BioPure (a garlic and artemisinin combination) is a powerful therapy for parasites burdening the liver. Rectal ozone insufflation should also be considered. Not all clinics are equipped for this application, but patients can buy home units from Longevity and other manufacturers – a fantastic investment.

Practitioners and patients should be aware that treatment length is substantial, and it is not unusual after

2 years for patients to still be passing parasites daily to weekly. Biological remedies should naturally be emphasized throughout this expected course, although pharmaceuticals certainly have a place in overall management, especially for specific phases of treatment. These include praziquantel, ivermectin, pyrantel pamoate, albendazole, nitazoxanide, and ivermectin. Simon Yu, MD, a pioneer in antiparasitic treatment, and his book *Accidental Cure* should be mentioned here for their substantial contribution to this field.²⁰ While injectable forms of these medications have been available in the veterinary and livestock arena for years, human use is only beginning to surface as a viable option for oral-resistant cases.²¹ Dr. Hofmann has worked with Key Pharmacy (Federal Way, Washington) to compound injectable ivermectin, and the authors have utilized it with great success.

Appreciation for the vast array of herbal approaches continues to grow amongst our practitioners and patients. Classic naturopathic herbals include *Artemisia annua* (sweet wormwood), *Artemisia tridentata* (sagebrush), *Allium sativum* (garlic), *Gentiana lutea* (gentian), *Mahonia aquifolium* (Oregon grape), *Zingiber officinalis* (ginger), *Tanacetum vulgare* (tansy), *Azadirachta indica* (neem), *Hydrastis canadensis* (goldenseal), *Thuja plicata* (western red cedar), *Syzygium aromaticum* (clove), *Juglans nigra* (black walnut), *Dysphania ambrosioides* (formerly *Chenopodium ambrosioides*; epazote), *Ailanthus altissima* (Chinese tree of heaven), *Quassia amara* (quassia), *Picrasma excelsa* (Jamaican quassia), *Uncaria tomentosa* (cat's claw), *Tabebuia impetiginosa* (pau d'arco), *Citrus paradisi* (grapefruit seed), *Cucurbita moschata* (pumpkin seed), and *Carica papaya* (papaya seed). A lesser-known antiparasitic herb, but a popular prescription of the authors', is *Mimosa pudica*. Traditional Chinese Medicinals include preparations from *Areca catechu* (betel nut) and *Omphalia lapidescens* (thunderball fungus). Herbs should be rotated, pulsed, and combined appropriately for the patient.

General treatment strategies should always involve mental/emotional support and electromagnetic frequency (EMF) remediation. Because of the psychospiritual/emotional connection between parasites and their hosts, active psychotherapy should be considered. We routinely see the resolution of psychological conflicts result in the spontaneous passage of parasites. Most modern-day EMFs create an immune suppression and parasite advantage. Therefore, mitigation of deleterious EMF at the home and workplace renders antiparasitic treatment more effective.

On the other hand, energetic approaches should be used to the patient's advantage. The "Sputnik," now discontinued, was one such approach. It was a small metallic capsule that when swallowed pulsed a parasite-disabling field along its course through the intestines. Currently, use of pulsed electromagnetic field therapy (PEMF) over the abdomen is a wonderful tool. Parasites become aggravated and weakened by these frequencies, and some may even die. Homeopaths (e.g., Pekana Helmin), particularly the nosodes, have classically been utilized with great success.

Note: Practitioners interested in learning more detailed treatment protocols can reference the Klinghardt Academy protocol manuals.

Treatment Considerations

The safety and efficacy of the many antiparasitic treatments available may certainly justify a therapeutic trial of treatment in many cases. However, physician direction and expertise in treating the chronically ill patient population are indispensable. A well-rounded protocol that addresses the patient holistically is required: parasitic infection never exists outside the context of other diagnoses and functional considerations. Both the direct and indirect immunological consequences of parasite-killing itself must be addressed. This may include anti-inflammatory support (even steroids when neurological manifestations such as seizures are a risk), detoxification support (particularly

heavy metal chelation and colon cleansing), and additional antimicrobial support for comorbid infections (viral and bacterial populations may increase during biofilm disruption and immune changes). Ketotifen, useful in decreasing histamine's effects, and rebamipide, helpful for mucosal protection and healing, may be employed.

In the authors' experience, individual parasite burden is most certainly proportional to a person's toxic burden. Minimally, the antiparasite treatment should be accompanied by aluminum, lead, and glyphosate detoxification protocols. Glyphosate toxicity likely creates a larval growth advantage in parasitic illness, and heavy metals are well-known components of biofilm communities in general. A dying parasite will release the stored toxins back into the host if proper support is lacking.

Therefore it is imperative to never give treatment without toxin binders to catch the biological "fallout" of antiparasitic treatment. The authors' favorites include ZeoBind (BioPure), G.I. Detox (Bio-Botanical Research), chlorella (BioPure), clays such as green clay (BioPure), and silica-rich products such as diatomaceous earth and microsilica (BioPure). The addition of weekly, biweekly, or even daily colon hydrotherapy sessions cannot be overemphasized. Replanting the gut with therapeutic amounts of probiotics is often necessary, and the immunological advances of fecal implants warrants their consideration in many cases.

As discussed, the mental and emotional manifestations of parasitic illnesses themselves must also be appreciated and addressed. Mental, emotional, and spiritual manifestations of parasitic illness have been given due consideration for millennia. Modern science and clinical observations uphold these associations. Our patients routinely present themselves clinically in predictable ways and also describe their experiences as harborers of parasitic infection in replicable ways.

Conclusion

Parasitic illness is so often a major contributing factor in a patient's ill health. We urge fellow practitioners of biological, functional, and holistic medicine to not overlook this possibility. Lack of diagnostic accuracy and the limited consensus reality at large should not prevent our patients from receiving this significant healing opportunity.

The legitimacy of observation and outcome should generally be honored. What comes out in the toilet and how the patient feels afterwards are invaluable data points. Indeed, the overall progression of a patient's health and well-being is the most important indication of great medicine, and certainly the most fulfilling for both practitioner and patient. We should continue to improve upon and refine the diagnosis and treatment of parasitic illness in the context of underlying toxic and genetic/epigenetic burden.

A great amount of clinical success can be found with antiparasitic treatment. Witnessing the success of our antiparasitic regimes continues to lend credence to our approach and bring joy to our practice of medicine.

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A lifelong learner, Dr. Hofmann finds that her greatest teachers have undoubtedly been her patients. The resilience, healing potential, and capabilities of the human body and spirit continue to inspire Dr. Hofmann.



Dietrich Klinghardt, MD, PhD, was born, raised and educated in West Germany, where he graduated from Freiburg Medical School/Albert Ludwigs University in 1975. He also studied psychology and completed a 3-year research project/PhD in angiology. He has been practicing medicine in the US since 1984 and specializes in the treatment of chronic illness and autism. He is widely recognized for his system of the "5 levels of Healing" and treatment of chronic Lyme disease without the use of antibiotics. His practice is in Woodinville, Washington.

Lyme Disease: A Microscopist's Search for an Antibiotic-Free Solution

by Bjørn Johan Øverbye, MD

We all know the official stories of how Dr. Allen Steere identified tick-transmitted arthritis in Lyme County in 1972.¹ We all rejoice in how Steere sent ticks from the county to Swiss-born entomologist Willy Burgdorfer in 1985 and asked him to search for parasites, and how Burgdorfer identified *Borrelia* spirochetes in the gut of the tick and then the link was established: Lyme disease caused by tick-borne *Borrelia*. Both became "immortalized"!

I was bitten by a tick a few years prior to these glorious events which changed the way that we have come to regard a number of previously unexplained diseases of organs, muscles, joints, and the nervous system. The year was 1978, and practically no one in my country, Norway, had ever heard about *Borrelia*, although German microscopist Otto Obermeier had identified the spirochete as cause of disease as early as 1868.² By 1908 it was baptized *Borrelia*.³ By 1920 it was widespread knowledge in German-speaking countries that bloodsucking ticks transmitted the spirochete and spread them from furry animals to humans and between humans. By 1950 Germans identified *Borrelia* in blood form by darkfield microscopy and gave *Penicillium* to the sick and they recovered, if not always forever.⁴

Due to the ignorance of the times I suffered untreated neuroborreliosis and went through living hell for 1 year with no help whatsoever. Later I came to learn that most Norwegian patients still do today, due to reasons called the "Lyme-borreliosis war" between antibiotic minimalists and more realistic doctors.⁵

In the following decades, I suffered badly and tried every treatment in the book, mostly alternative therapies, since the doors to hospitals were closed. In my search for help, I studied under German surgeon/microscopist/immunologist and natural therapist Wolfgang Gröger, MD. I learned about blood symbiotic, pleomorphic microbes, blood pH, and syphilis.⁶ No *Borrelia* was discussed!

I later studied with Professor Henk Oswald in the Netherlands in 1994; the same story as in Germany. No *Borrelia* was discussed! Swimming spirochetes were dismissed as "something else."

In 1999 I had a relapse of neurological symptoms and passed some years rebuilding myself with immunotherapy and electromagnetic therapy, to which I was introduced in Germany.

In 2010 while taking a course at the Monroe Institute in Virginia, now 100% symptom free due to natural therapies, I "stumbled over" inventor, microscopist, and electronic genius Jim Meisner, to whom I am still in debt. From him I learned the real thing: *Borrelia* when looking at live blood through his Olympus 50x darkfield microscope.⁷

I also learned how he was able to kill *Borrelia* using a replica of a machine made by French inventor George Lakhovsky and later adapted by American microscopist genius Royal Rife.^{8,9} I had the honor to meet his friend whom he had cured from severe crippling Lyme disease in 3 months using this electromagnetic wave equipment.

Inspired by such stories, a local businessman lent me a high-quality darkfield/phase contrast/

immunofluorescence microscope and I set out to do research.

Over the years I have collected a photo library of 1000 pictures taken from blood voluntarily donated by people who wanted to help me find an explanation for the nature of things.

By 2012, I had acquired a 15,000-page library on topics such as ticks, vectors, ecology, microscope building, optics, microbiology, *Borrelia* and spirochete sciences, therapies, politics of medicine, and not to forget electromagnetic science of curing the sick and killing microbes! By 2014 it all materialized into the biggest book on the topic ever written in a Scandinavian language, with the title *Sick from Tick Bite* (*Syk av Flåttbitt*; Veiviseren Forlag).

So what did I learn from the ill fate of suffering from the worst disease ever and bouncing back twice? The biggest evil on this planet is lack of true knowledge of the nature of things and the consequences thereof! By curing my own evils, I came out as a humbler and more knowledgeable doctor.

First I came to learn the nature of the enemy: a small spirochete of the magnitude of 1 to 2 RBC length (ca. 10–20 μm). Under a darkfield microscope at 1000 diameters of magnification, it looks like a small, thin, white thread with a characteristic wiggling movement.^{10,11} This is the "classical appearance" common to all spirochetes. I photographed them live and with still photos from the most unexpected volunteers, even healthy ones.

Thus I came to see with my own eyes the phenomenon so often not talked about: the *healthy* carriers! I followed the people's histories and came to see

for myself how a tick bite had infected them with spirochetes many years earlier, and how they stayed healthy. Yet later, when they were neglecting their health or exposed to traumas or immunosuppressing drugs, the spirochetes appeared from "nowhere" and caused symptoms related to Lyme disease or neuroborreliosis.

I also came to see a host of other phenomena known as *Borrelia* pleomorphism: spirochetes roll up into small discoid forms, change to smaller antigen and wall-free L-forms more apt to escape the immune system.¹² And I could see the phenomenon called shedding, in which spirochetes wiggle intensely and throw off immune-proteins meant to kill them.

In 2014 I went to Budapest to visit world-famous darkfield microscopy guru Dr. Bela Bozsik, who had photographed and filmed the blood of more than 100,000 Lyme-borreliosis patients.^{13,14} His records were impeccable: He could match the best serum tests and sometimes discovered *Borrelia* in previously seronegative sick patients.¹⁵ His Hungarian team had a 95% cure when steering their therapies with microscopy and serum testing in parallel; 3 to 4 months of antibiotics plus immune-supporting supplements were the secret.

During my second visit he offered me a job at a hospital in Hungary as a *Borrelia* physician to take care of foreign patients. I often wonder if I should have taken this job to learn more and increase my expertise and, above all, help people who are sick with no given answers as I had experienced.

From Bozsik I learned how *Borrelia* "blebs."¹⁶ I saw photographs of how spirochetes under attack would make small blebs, some empty as decoys for immune cells to chase in vain when a few other blebs containing valuable DNA meant to grow into new spirochetes could escape. I saw cysts with rolled-up spirochetes inside other cells, "nests of spirochetes" covered with biofilms, and how spirochetes cover themselves with slime (biofilm) to become "slippery" so that immune proteins could not stick to their surface antigens. I saw film of spirochetes breaking in two to create two offspring from one mother.

When I asked Bozsik, he admitted somewhat unwillingly, "Well, there is a chance that many therapies on the market that make people clinically healthy do not really kill off all the spirochetes; they just lower the number of spirochetes to a level where the immune system can keep them down; turning previously sick into healthy carriers."¹⁷

As a consequence of the mass of knowledge that I gathered from two continents and years of travel, I came to see the enemy as far smarter than the doctors chasing them. It is not a simple "bug" to be killed off by 2 to 4 weeks of penicillin or doxycycline. It is the perfect parasite honed by billions of years of evolution in the ecosystem, meant to survive at all cost and serving the highest purpose of James Lovelock's Gaia scenario: invade as many carbon-based warm-blooded organisms; stay when organisms get old or weak due to lack of proper food in the biotope; see to their premature weakening and eventual death, thereby promoting the younger, better fed, and more apt.¹⁸

It is not a parasite designed to succumb to a politically correct medical minimalism or even by antibiotic maximalists. I saw the spirochetes come back in patient after patient post treatment. And with awe and a curious admiration, I saw how they evaded the immune system and how serum tests came out negative in more cases than our official health system would like us to believe.

Burdened by the mass of knowledge loaded upon my shoulders after years of research, I ended up with a threefold strategy that I tested out on volunteers. The first part is immune therapy activating the natural inborn defense that we all have. The second part is sound wave therapy that I learned about in the US which activates the nervous system and our cells. The third part is based on my premedical studies in university – physics and electronics: the use of tuned focused pulsating electromagnetic fields enhancing immunity and the lymphatic system and balancing the nervous system by tapping into cellular communication pathways.¹⁹

Does it work? Well, it worked for me and a great number of volunteers. These days, I have had the honor to be

supported in my work by the people from Ondamed in Germany by doing a pilot project to use its electromagnetic generators to help people for a better outcome when suffering from Lyme wherein antibiotics have failed. Based on years of experience and research, I have a feeling that this may be the future.

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Dr. Bjørn Johan Øverbye has been practicing holistic medicine at his private clinic in Arendal in Southern Norway since 1977. His patient population is drawn to his clinic from all over Scandinavia and represents those who have been discarded by the official health system as incurable. Since 2001 he has worked mainly with hypothyroidism and metabolic disease, and since 2010 he has also been working with Lyme patients. He is the author of 8 books on topics such as acupuncture (3 books), Taoist self-healing (1 book), electromagnetic therapy (1 book), metabolic diseases (1 book), and tick-borne diseases (1 book). Dr. Øverbye has done extensive research on biophysics of electromagnetism on the human energy system (published in Canada) and completed a study on fibromyalgia under the tutelage of Dr. John C. Lowe in the US, published on www.thyroidscience.com. During the last three years, he toured Scandinavia and other parts of Europe lecturing on nutrition, anti-aging, the circulatory system, and hypothyroidism.

www.dr-overbye.no.

Lyme Survivor & US Track Athlete Perry Fields interviews with

Silvia Binder, ND, PhD, CEO of the Ondamed companies & Founder of The Binder Institute for Personalized Medicine

Q: Silvia, as you know I was using ONDAMED back in 2006 to get healthy. Being an ND, what are your thoughts on healing Lyme disease?

A: I believe healing is very possible with almost all disease processes including Lyme disease. I consider Lyme disease an immune deficiency disorder. The question that interests me, as a practitioner, is WHY the immune system of my patient is suppressed or dysfunctional. I am a firm believer in the theory that our physiology follows our minds and emotions. In other words, our body manifests unresolved emotional issues such as the trauma or shock we experienced at some point in our lives. The memory of such unresolved trauma or shock resides on a cellular level somewhere in our body hindering our body to be as strong, vibrant, and healthy as it could possibly be. We won't notice it right away. By nature the person doesn't remember these events since natural protective mechanisms may block out memories. But what I believe happens is that the shock or trauma disconnects us from the powerful self we have come to be and live in this lifetime. Most of us continue to stray away from "our path" and further disconnect from our truth. This, in a much broader sense and from a wider perspective on immune deficiency disorders, may be the causal factor for our disease journeys. It is our own disconnect from the power within which invites inflammation and infection.

Q: How does ONDAMED help those who are suffering from Lyme disease?

A: ONDAMED, with its unique biofeedback methodology, allows practitioners to find and treat dysfunctional tissue. The practitioner stimulates the patient with specifically chosen focused fields emitted from a handheld applicator that is moved along the body, from head to toe, front and back. Each time an area of the patient's body reacts to the stimulus (as felt through the biofeedback loop) the practitioner notes this reaction

area, concluding that there may be inflammation, infection, scar tissue, or memories of unresolved shock or trauma residing on a cellular level.

It is the patient who sits in the driver's seat, directing the practitioner to WHERE the weakest areas are located and WHICH focused fields are needed to stimulate these areas. Instead of using systemically pharmaceutical or nutraceutical approaches, the ONDAMED provides a focused "electroceutical" approach targeted to the areas in most need.

If you've ever seen Dr. Beverly Crusher in Star Trek: The Next Generation, she uses a Tricorder Technology to scan over the body to evaluate imbalances and once imbalances are found, she uses the Tricorder to send electromagnetic pulses toward the body area to stimulate healing processes. ONDAMED is quite similar, but may be the 1st generation of an advanced Tricorder since Star Trek plays in the 24th Century.

Now back to our time and your question, Perry, the ONDAMED stimulation attracts an immune response to the specific tissue area where applicators are placed. ONDAMED's focused fields cause an induction from within cells and tissue; cells and tissue are being vibrated or moved, which causes not only the immune system to respond, but also detoxifies the cells from toxins and waste product. While ONDAMED's stimulation impacts the targeted tissue areas, ONDAMED also has a systemic effect on the lymphatic system, the circulatory and the nervous system.

Lymphatic flow is being stimulated to help carry toxins and waste product from the cell environment to the respective organs to excrete the unwanted debris through stool, urine, or sweat to name a few.

While the ONDAMED is causing so many different benefits to happen all at the same time, its focused field stimulation also significantly impacts the nervous

system. Depending on the fields used, they either have a relaxing or exciting effect on the nervous system.

Q: Tell us more about using ONDAMED to address all the symptoms that people with Lyme suffer from?

A: I typically suggest that our practitioners use all four operating Modules the ONDAMED System offers. This will allow the addressing of patients' symptoms from different perspectives with a wide range of frequencies and programs.

First we work with frequencies which are specific to organs and organ systems, offering very precise frequencies to the areas in most distress. Then in Module 2, 170 different therapeutic programs offer a more generic approach while providing important information as to the patient's health journey. You may compare these programs, which are combinations of frequencies (but never more than 2 at one time) to a whole piece of music, or in other words, a symphony.

It is critical, especially for immune deficiency disorders including Lyme disease, to also use Module 3, frequencies that correlate to microorganism related information. This Module is the most specific of all, using only one frequency to primarily stimulate an immune response.

Module 4 is also essential since it deals with unique frequencies to stimulate assimilation of enzymes, vitamins, minerals, and more.

With use of these four Modules, Lyme sufferers have been shown to:

- relax deeply
- improve their sleep patterns
- detoxify by sweating more and with noticeable frequency and consistency changes in stool and urine
- improve facial complexion (patients' skin color changes from a grey tone to a healthy radiant tone)

- have more energy
- think clearer
- improve memory
- increase range of motion
- have less or no pain
- reduce or stop medication (with guidance of their physician)
- balance weight
- reduce or stop addictive patterns
- feel better about themselves and find it easier to manage their life's challenges
- improve quality of life significantly
- to undertake big life transformations, including a new career path, seeking additional or brand new education, changing or allowing a partnership, move, etc.

Q: What should one expect to feel after an ONDAMED session? When can people start to notice improvement with the use of ONDAMED? Is there a rough estimate of the number of sessions that someone may need before they start feeling symptoms get better?

A: The first thing people feel is that they relax and have less pain. Often patients report a better and deeper sleep. Pain levels drop and people can move better and easier and they have more energy! They feel better about themselves and start taking ownership of their lives again. Generally speaking, people should feel a change in the first 5 sessions and after 15 – 20 sessions (either weekly or bi-weekly) there should be a substantial improvement with their symptoms. I always tell my patients that the first step to getting them well is to stabilize them, which may take anywhere from 2-10 sessions. After they have achieved a stable level, we then work on building their health. So, even if the symptoms have been reduced it is important to work on building strength and health.

Q: If symptoms get better, is it safe to say that healing is actually taking place. Most people with Lyme are very

fearful. They fear that they aren't ever going to get well and if they do get well, they can expect to have another "attack" or "episode" of symptoms.

A: Yes, we can safely say that patients are healing. Of course, our belief in Naturopathic Medicine is to stimulate the patient's own healing abilities, which is always present in the body. I want to mention here that while our healing abilities are present, the patient may not yet be ready for healing. We must respect the patient's need and abilities and the right time for undertaking their healing journey. Another belief we hold in Naturopathic Medicine is to set the patient free and not bind them to yet another "thing". Therefore, while ONDAMED is a significant tool to stimulate wholesome healing, we must never forget or stop to educate our patients in terms of life style, nutrition, stress factors in their environment, etc. This way the patient is equipped with their own tools to better understand how to live a more aware, healthy, and joyful life. I can't stress enough how critical the work is with unresolved emotional shock or trauma. By stepping into a deeper level with patients, we are able to help them reconnect to their inner strength, and to follow this connection and strength. Once people "get a hold" of their power within and they start nurturing and growing their "inner seed", they have a fundamental base from which to direct their bodies and avoid ever having to experience another "attack" or "episode".

Q: How does energetic medicine fit into someone's recovery? How should it be used?

A: Good question, Perry. Since all matter including all beings is founded on electro-magnetic forces, it is a no brainer to understand the importance of using energy medicine. The field of Western medicine has long been using "energetic medicine" to diagnose – just look at MRI,

X-Ray, Ultrasound, EEG and EKG. These devices are an integral part of medicine.

Acupuncture, pulse reading and herbs have been used for thousands of years as part of many cultures.

The field of "energetic medicine" offers many different approaches. I am a skeptic and need to see evidence of safety and efficacy. All of you should ask questions when offered any form of treatment. I always encourage my patients to do their own research, since it puts them in a position of power.

There are fine & cutting edge energetic approaches, yet there also are many questionable methods, some even dangerous. If you wish to heal quickly and lastingly you must be treated in a focused and specific way. The body needs specificity for proper healing (the yogis already stated this a long time ago). A generic approach can be limited, may cost you time and money, and more importantly you may give up hope. We don't want that. Therefore, do your own research and use common-sense and practical thinking. It goes a long way and sets you free.

All in all, people need different elements of healing stimulation. Several pizza slices make a whole "pizza pie" and we need different slices of therapeutic interaction to reduce or even better to end people's suffering from their Lyme symptoms. It is absolutely necessary to include energetic medicine in order to tap into the cellular communication system of a person.

The return to Personalized Medicine, in a safe and common-sense kind of way, is the key for helping people to not only overcome their disease, but to learn from their disease journey and to become a more aware, healthy, strong, and joyful person.

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Unraveling the Mystery of Bartonellosis

by Scott Forsgren

Portions from an interview with
B. Robert Mozayeni, MD

As someone who struggled with *Bartonella* for many years, I know all too well the devastation that this infection can have on one's physical and mental health. While *Bartonella* can manifest in many different ways within the body, the challenges it presents extend far beyond the physical.

It is increasingly obvious that most people may not get through life without being exposed to one or more *Bartonella* species. Historically, we thought just the opposite was true.

— Edward B. Breitschwerdt, DVM

Bartonellosis is a poorly understood condition that is routinely overlooked by mainstream medicine. As a result, many cases go undiagnosed, leading to significant and unnecessary human suffering and substantial costs to society. While available testing options for *Bartonella* have improved greatly in recent years, there is still no perfect *Bartonella* assay available. Even when bartonellosis is confirmed through testing, the success of available treatment options is variable, and *Bartonella* may establish itself as a chronic infection that requires long-term management.

Fortunately for many of us, humans are not the only species affected by the genus *Bartonella*. In fact, much of the available research comes from the veterinary community, where

Bartonella is much more widely acknowledged and better understood than in human medicine.

Veterinary Medicine Leads to Help for Suffering Humans

Edward Breitschwerdt, DVM, is an infectious disease specialist at North Carolina State University College of Veterinary Medicine, an adjunct professor of medicine at Duke University, and chief scientific officer

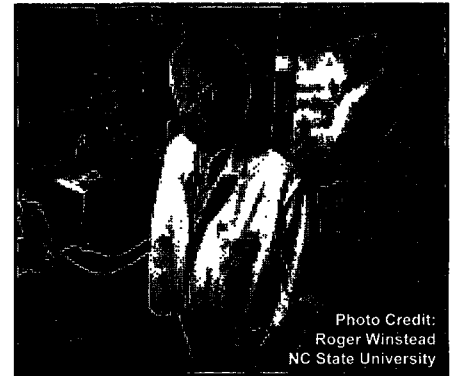


Photo Credit:
Roger Winstead
NC State University

Ed Breitschwerdt, an expert on infectious diseases and a doctor of veterinary medicine specializing in *Bartonella*.

cat and showed that cats can become chronically infected. This work was the catalyst that led Breitschwerdt down the path of unraveling the mysteries of *Bartonella*.

As Breitschwerdt lectured at veterinary conferences about *Bartonella* illnesses in dogs, numerous veterinarians approached him to discuss their own health challenges, such as multiple sclerosis-like conditions and rheumatoid-like diseases. Many had been sick for several years with no clearly defined diagnosis. When he started testing these veterinarians for *Bartonella*, his research team found that many of them tested positive for *Bartonella* DNA in their blood. If it were not for the translational research initially done with animals, the genus *Bartonella* and the disease bartonellosis would likely be even lesser known than they are today. This may be another example in which dogs truly are man's best friend. Over

at Galaxy Diagnostics. Early in his career, he focused on vector-borne, intracellular pathogens, including Rocky Mountain spotted fever (caused by a *Rickettsial* bacterium) and *Ehrlichia*. *Ehrlichia* was discovered in animals decades before it was identified in humans. His attention later shifted to *Bartonella* due to the historical association of one *Bartonella* species, *Bartonella henselae*, with cat-scratch disease (CSD).

The connection between the newly discovered bacterium and CSD was initially made by a rickettsiologist, Dr. Russ Regnery at the CDC, who recognized similarities between a newly isolated bacterium from an AIDS patient in Texas and bacteria visualized in lymph nodes of patients with CSD. Regnery made the first isolate of *Bartonella henselae* from a

the next two decades, research would demonstrate that dogs often develop the same pathologies as those of their human counterparts.

Bartonella Basics

Bartonella was named after A. L. Barton, who in 1909 identified organisms that adhered to red blood cells. *Bartonella* is a genus of gram-negative, aerobic bacteria belonging to the Bartonellaceae family and Rhizobiales order. It is in the Alphaproteobacteria class, which is part of the Proteobacteria phylum. *Bartonella* organisms are considered facultative intracellular parasites, meaning that they may resort to parasitic activity although they do not rely on a host to complete their life cycle. Other well-known pathogenic Alphaproteobacteria include *Rickettsia*, *Anaplasma*, *Ehrlichia*, and *Brucella* species.

While *Bartonella* can infect healthy people, it has generally been believed by mainstream medicine that the infection is cleared by the immune system in the majority of cases. Many view these bacteria as opportunistic pathogens in conditions wherein the immune response may be suppressed, such as in AIDS and chronic Lyme disease. As the ability to identify *Bartonella* has improved, patients with numerous long-term illnesses have been found to harbor the bacteria in their blood and tissues. Additional research is needed to determine what role *Bartonella* plays in these illnesses. Persistent bloodstream infection with numerous *Bartonella* species is increasingly being linked to a host of chronic illnesses.

Bartonella targets erythrocytes (red blood cells), endothelial cells, microglial cells, macrophages, and CD34 progenitor cells.¹ Once the infection is in the body, it commonly resides in red blood cells and in the endothelial and pericyte cells lining the blood vessels throughout the body. *Bartonella* may use these cells and various tissues in the body to hide from the immune system and

to establish a chronic, persistent infection.

Bartonella and Human Infection

According to Breitschwerdt, there are nearly 40 different species of *Bartonella* known today, and 15 of these have been found to infect human beings (see Table 1). The more commonly known *Bartonella* species include *Bartonella henselae*, *Bartonella quintana*, and *Bartonella bacilliformis*.

Bartonella henselae is the causative agent in CSD. In

addition, this *Bartonella* species can cause numerous symptoms in both immunocompetent and immunocompromised individuals. Infection may result in bacteremia (presence of bacteria in the blood), myocarditis (inflammation of the middle layer of the heart wall), peliosis hepatis (a vascular condition which results in blood-filled sacs in the liver), neuroretinitis (inflammation of the neural retina and optic nerve), bacillary angiomatosis (a proliferation of blood vessels leading to tumorlike masses), enlarged lymph nodes, and

Table 1: List of *Bartonella* species, excluding *Candidatus*. Those that have been identified as pathogenic in humans are indicated with a check mark. The table also identifies the pathogens (exposure only) that are detectable using IFA serology. It is important to note that IFA serology can only detect previous exposure to *Bartonella*, not current infection. Because the preparation of materials for IFA serology is complex, you can see that this test is only available for a limited number of *Bartonella* species. Conversely, Galaxy's ePCR test uses genus level primers that are designed to detect all pathogenic species of *Bartonella* and an enrichment process to confirm infection.

No.	<i>Bartonella</i> Species	Pathogenic in Humans?	Detectable with current IFA?	Detectable with ePCR?
1.	<i>B. alsatica</i>	✓		✓
2.	<i>B. bacilliformis</i>	✓		✓
3.	<i>B. clarridgeiae</i>	✓		✓
4.	<i>B. elizabethae</i>	✓		✓
5.	<i>B. grahamii</i>	✓		✓
6.	<i>B. henselae</i>	✓	✓	✓
7.	<i>B. koehlerae</i>	✓		✓
8.	<i>B. quintana</i>	✓	✓	✓
9.	<i>B. rochalimae</i>	✓		✓
10.	<i>B. tamiae</i>	✓		✓
11.	<i>B. vinsonii arupensis*</i>	✓		✓
12.	<i>B. vinsonii berkhoffii*</i>	✓		✓
13.	<i>B. washoensis</i>	✓		✓
14.	<i>B. rattimassiliensis</i>	✓		✓
15.	<i>B. tribocorum</i>	✓		✓
16.	<i>B. bovis</i>			✓
17.	<i>B. doshiae</i>			✓
18.	<i>B. talpae</i>			✓
19.	<i>B. schoenbuchensis</i>			✓
20.	<i>B. senegalensis</i>			✓
21.	<i>B. silvatica</i>			✓
22.	<i>B. silvicola</i>			✓
23.	<i>B. rattaaustraliani</i>			✓
24.	<i>B. pachyuromydis</i>			✓
25.	<i>B. peromysci</i>			✓
26.	<i>B. phoceensis</i>			✓
27.	<i>B. queenslandensis</i>			✓
28.	<i>B. jaculi</i>			✓
29.	<i>B. japonica</i>			✓
30.	<i>B. coopersplainsensis</i>			✓
31.	<i>B. callosciuri</i>			✓
32.	<i>B. capreoli</i>			✓
33.	<i>B. chomelii</i>			✓
34.	<i>B. birtlesii</i>			✓
35.	<i>B. acomydis</i>			✓
36.	<i>B. australis</i>			✓
37.	<i>B. vinsonii vinsonii*</i>			✓
38.	<i>B. taylori</i>			✓

*Subspecies of the same *B. vinsonii* species^{46,47}

Source: Galaxy Diagnostics

Bartonellosis

►
fevers.

Bartonella quintana is known as the causative agent in trench fever. It was first described during World War I as over a million soldiers in Europe became infected after exposure to the human body louse. Urban trench fever has reemerged among homeless populations around the world. It may present with fever, severe headaches, back and leg pain, and skin rashes. It can be a factor in endocarditis and may result in bacillary angiomatosis in those coinfecting with HIV. Evidence of *Bartonella quintana* has been found in the dental pulp of soldiers from Napoleon's Grande Armée, and many of his soldiers died from infectious diseases, including trench fever and epidemic typhus (*Rickettsia prowazekii*).²

Bartonella bacilliformis leads to a condition known as Carrion's disease, or Oroya fever. Carrion's disease was named after Daniel Alcides Carrion, a Peruvian medical student who died after an experiment where he inoculated himself with the bacterium. *Bartonella bacilliformis* is found primarily in Peru, Columbia, and Ecuador and is transmitted by sand flies. It can have a mortality rate of 40% to 90% in untreated patients.

More recently, other *Bartonella* species have been associated with human infection. *Bartonella clarridgeiae* may lead to CSD. *Bartonella elizabethae*, *Bartonella vinsonii* subsp. *berkhoffii*, and *Bartonella vinsonii* subsp. *arupensis* may be factors in endocarditis. *Bartonella grahamii* has been found in the ocular fluids of a human with neuroretinitis.³ *Bartonella washoensis* has been implicated in myocarditis.⁴

Candidatus (an interim status for a yet-to-be officially named organism) *Bartonella melophagi* was discovered in 2009 by Dr. Ricardo Maggi, a research microbiologist working with Breitschwerdt, while testing human blood specimens.⁵ *Candidatus Bartonella mayotimonensis* has been

identified in a case of endocarditis, and recent evidence suggests that bats may be the reservoir for this *Bartonella* species.^{6,7} In that research publication, a compelling statement was made: "This case reinforces the hypothesis that any *Bartonella* species can cause human infection."

Human infection with *Bartonella* may be the result of arthropod vectors, including fleas (and flea feces), biting flies such as sand flies and horn flies, the human body louse, mosquitoes, and ticks; through bites or scratches of reservoir hosts; and potentially from needles and syringes in those who are drug addicted. Needle stick transmission to veterinarians has been reported. *Bartonella henselae* and *Bartonella clarridgeiae* have been transmitted to cats through blood transfusion. Recently, 3.2% of healthy blood donors in Brazil were found to carry at least one of these two cat flea-associated *Bartonella* species in their blood.⁸ *Bartonella* DNA has even been found in dust mites.⁹ Fleas have been shown to be infected with *Bartonella henselae*, *Bartonella clarridgeiae*, *Bartonella koehlerae*, and *Bartonella quintana*; body lice with *Bartonella quintana*; and ticks with several species of *Bartonella*.¹⁰ Vector biologists and others with extensive arthropod exposures are at increased risk for acquiring *Bartonella* infections.

Working or living with flea-infested pets or other animals is a notable risk factor for *Bartonella*, so much so that up to 28% of veterinarians tested positive in one study compared with 0% of controls.¹¹ Cats and dogs exposed to arthropod vectors and that live in close proximity with people put humans at increased risk for acquiring *Bartonella* infections. The concentration of *Bartonella* in the blood of an infected cat may be as high as a million times that of an infected human. Recent evidence indicates that many of the *Bartonella henselae* strains that commonly infect cats are not found in humans, suggesting that virulence factors play an important role in determining pathogenicity and whether or not a

cat poses a risk for transmission of a pathogenic strain to a human.

Animals that are exposed to fleas and ticks have a high likelihood of being infected with *Bartonella*. About half of all cats may be infected with *Bartonella*, as high as 80% of feral cats and near 40% of domestic cats.^{12,13} In one study that tested 108 domestic dogs in Peru serologically, 67 of the dogs were seropositive for *Bartonella rochalimae*, while 43 dogs were seropositive for *B. vinsonii berkhoffii*.¹⁴ In a study of dogs in Iraq, the seroprevalence of *Bartonella* infection was 47.4%.¹⁵ An incidence of 10% has been reported in healthy dogs in the eastern United States, and 35% of coyotes were seropositive for *B. vinsonii* subsp. *berkhoffii* in California, with 28% being bacteremic.¹⁶

Additionally, although presumably an infrequent mode of transmission, evidence now suggests that *Bartonella* may be transmitted from mother to child in utero, potentially leading to birth defects.¹⁷

Bartonella Symptoms

The symptoms of *Bartonella* may affect numerous body systems and can range from mild to severe, and may even be fatal.¹⁸ Breitschwerdt's own father passed away from complications associated with *Bartonella* infection.¹⁹ One may be asymptomatic or may never become symptomatic enough to pursue medical evaluation. Presenting symptoms may wax and wane over time.

The more common symptoms of *Bartonella* include swollen lymph nodes, gastritis, sore soles of the feet most noticeable in the morning, fasciculations (muscle twitching), headaches, abdominal pain, striae (irregular areas of skin that look like stretch marks), skin rashes, tender subcutaneous nodules in the extremities, fevers, anxiety, depression, anger, and obsessive-compulsive thoughts or behaviors.

B. Robert Mozayeni, MD, has a private rheumatology practice in Rockville, Maryland, and is a

leading expert in the treatment of *Bartonella*. He serves as chief medical officer for Galaxy Diagnostics and works closely with Breitschwerdt. In a recent interview for this article, Mozayeni explained that he sees a pattern of symptoms in his patients with *Bartonella* infection. He noted that bartonellosis is primarily an infection of the blood vessels, the blood components, and the bone marrow. While *Borrelia burgdorferi*, the causative agent in Lyme disease, can be found in the blood and as well as outside the vascular system, *Bartonella* species primarily congregate within red blood cells, endothelial cells, and bone marrow cells. It may also be found in cysts, having been isolated from an otherwise "benign" breast cyst in one patient.

As bartonellosis is principally an infection of the vascular system, it leads to inflammation and endothelial proliferation, disrupting blood flow at the small vessel level, such as in the capillaries and arterioles. The end result is compromised microcirculation throughout the body which can lead to the appearance of fluctuating and migrating symptoms. The manifestation of symptoms is largely associated with where in the body the blood flow compromise happens to be located.

Pain in the soles of the feet upon waking, for example, is likely due to inflammation of the blood vessels in an area that endures ongoing microvascular trauma as a result of regular weight-bearing activity; the pain is then exacerbated by the presence of *Bartonella* and small vessel inflammatory disease.

Patients may present with POTS (postural orthostatic tachycardia syndrome) or other forms of dysautonomia wherein the autonomic nervous system is affected; this is another manifestation of small vessel disease. The nerves of both the sympathetic and parasympathetic nervous system are compromised in their function due to changes in microcirculation and interruption in blood flow.

In every patient, *Bartonella* is infecting the vascular system throughout the body. Endothelial infection with *Bartonella* can damage veins and the valves of veins. Endothelial cells also line the heart valves. However, such infection can progress to infiltrate the deeper connective tissue of the heart in rare cases. This type of deeper heart valve infection with *Bartonella* is usually detected too late and almost always leads to heart valve replacement surgery. In a recent study of healthy coyotes in California, *Bartonella* DNA was found in the heart valves of 17%

It appears that chronic intravascular infection with a *Bartonella* spp. may induce a degree of immunological anergy, resulting in undetectable levels of organism-specific antibodies in some chronically infected patients.

— Edward B. Breitschwerdt, DVM

of the coyotes studied, and DNA of the bacterium was preferentially amplified from the aortic valve, the valve most commonly involved in both dog and human endocarditis.²⁰ Fortunately, as a rheumatologist, Mozayeni has not seen anyone in his patient population who has developed serious heart valve complications as a result of *Bartonella* infection.

Some believe that devitalized teeth and jawbone cavitations may be associated with *Bartonella* infection. Mozayeni collaborates with an endodontist who indicated that *Bartonella* "is big in the endodontal scientific literature," as it is known to cause cysts around dental roots that may lead to chronic, hard-to-diagnose head and face pain. Additionally, Mozayeni has observed a very high incidence of root canals in his practice which may be related to a compromise of small blood vessels that feed the dental pulp, another manifestation of small vessel disease. Although a single case report, a veterinarian infected with *Bartonella henselae* and *Bartonella vinsonii* subsp. *berkhoffii* developed neurological symptoms and periodontal disease concurrently.²¹

***Bartonella* and Psychological Manifestations**

Bartonella patients often describe a number of psychoemotional manifestations of the infection. These may include anxiety, depression, anger, obsessive-compulsive thoughts or behaviors, rage, and even suicidal thoughts.

Mozayeni shared that small vessel disease manifests in the central nervous system and the brain and affects executive function, often leading to mild or moderate cognitive impairment. As people become increasingly unable to process information, anxiety may develop. While there is generally no dementia or long-term memory impairment, short-term working memory and reaction time are often affected. "People become unable to think their way out of a negative thought, which leads to anxiety and fear," shared Mozayeni.

Mozayeni has said, "Many neuropsychiatric conditions can often be traced back to an infectious cause." A common pattern may be that a patient initially presents with anxiety, which goes hand in hand



Bartonellosis

with depression if not treated. They are two sides of the same coin. Over time, as a result of overstimulation, neurotransmitter depletion may lead to depression.

In a recent post on his blog, "LymeMD," Daniel A. Jaller, MD, stated, "Bartonella, as I know it, is frequently associated with specific neuropsychiatric symptoms, which may include: irritability, anxiety, rage and many others."³¹

More and more clinicians are beginning to recognize the impact of *Bartonella* on psychological well-being. Further research is needed to validate what these experienced clinicians have observed and to highlight that *Bartonella's* impact extends far beyond its physical manifestations. Many of those that have personally experienced *Bartonella* can attest to having

experienced a number of these distressing neuropsychiatric symptoms.

Bartonella Testing

Bartonella presents a diagnostic challenge for clinicians. A number of different laboratory tests exist to support the confirmation of *Bartonella* infection. However, cases of *Bartonella* infection may exist even when available tests are negative. Although current diagnostic tests have limitations, it is of critical importance to attempt to confirm a diagnosis of bartonellosis before embarking on a long, costly, and complex treatment regimen. As has been stated in the medical literature for many decades, "The kindest form of therapy is an accurate diagnosis."

The first option is an antibody test looking for IgM and IgG antibodies in serum using an indirect fluorescent antibody (IFA). Many commercially available antibody tests focus primarily on *Bartonella henselae* and

Bartonella quintana, a small subset of the *Bartonella* species that can cause human disease. *Bartonella* testing that relies on an antibody response is generally not very sensitive, as *Bartonella* has many properties that allow it to evade the immune system. Diagnostically relevant production of antibodies is not generally observed with *Bartonella* infection. As many as 85% of those chronically infected with *Bartonella*, confirmed through DNA amplification of blood or tissue, may have negative test results with IFA assays (for antibody detection).³² Further, there is a potential for cross-reactivity between *Bartonella* and other organisms such as *Coxiella burnetii* and *Chlamydia*, which further complicates interpretation of the results.³³ Furthermore, antibodies indicate evidence of prior exposure and do not confirm active infection.

Traditional PCR testing for *Bartonella* is available, though these assays were designed to only detect *Bartonella henselae* and *Bartonella*

Table 2: Bartonella symptoms experienced in humans²²⁻³⁰

Body System	Symptom
General	Fatigue, malaise, chills, sweats, fevers, chronic fatigue, somnolence, restlessness, poor sleep
Cardiovascular/ Circulatory	Endocarditis, myocarditis, pericarditis, hemolytic anemia (through invasion and destruction of RBCs), hypertension, pulmonary embolism, systemic vasculitis, arrhythmias, ischemic stroke, cardiomegaly (enlargement of the heart), heart valve problems, palpitations
Gastrointestinal	Poor appetite, weight loss, mild sore throat or feeling that one is about to get a sore throat, gastritis, muscle weakness leading to changes in speed/strength/coordination of digestive organs, difficulty swallowing, elevated liver function tests (LFT), hepatomegaly (enlargement of the liver), GERD (acid reflux), jaundice, gallbladder dysfunction, granulomatous hepatitis, peliosis hepatis, abdominal pain
Head and Neck	Headaches (can be severe or ice pick-like), migraines, vision changes or deficits, blurred vision, double vision, dry eyes, red eyes, conjunctivitis, neuroretinitis (ocular inflammation), ocular neovascularization, uveitis, retinal vasculitis, eye pain, floaters, photophobia, Parinaud's oculoglandular syndrome, papilledema, tinnitus, hyperacusis, nonhealing infections of the jawbone, cavitations, devitalized teeth
Hematological	Elevated VEGF, elevated LFTs, elevated alkaline phosphatase, pancytopenia, thrombocytopenia
Immune	Immune suppression; weakened immune response
Skin	Skin rashes, papules or papulovesicular rash, striae (stretch-mark appearance), acne, crusty scalp, Henoch-Schönlein purpura (purple spots on the skin), bacillary angiomatosis, petechiae, spider veins, varicose veins
Lymphatic	Splenomegaly (enlargement of the spleen), lymphadenopathy (enlargement of the lymph nodes)
Musculoskeletal	Fibromyalgia pain, pain in the soles of the feet (especially in the morning), ankle pain, rheumatoid arthritis, myositis (inflammation of the muscles), myalgia, systemic lupus erythematosus, osteomyelitis, joint pain, knee pain, pain in the chest or sternum, twitching or fasciculations, subcutaneous nodules, osteolytic lesions, bone destruction, stiff legs, shin bone pain, granulomatous inflammation
Neurological	Hallucinations, peripheral neuropathy, polyneuropathy, multiple sclerosis-like symptoms, transverse myelitis, encephalitis, meningitis, hyporeflexia or areflexia (below normal or absent reflexes), seizures, crawling sensations, burning sensations in the skin, vibrating or shooting sensations, tremors, cognitive deficit, memory loss, radiculitis, transverse myelitis, chronic demyelinating polyneuropathy, brain fog, temperature dysregulation, hyperesthesia (increase in sensitivity to stimuli)
Psychological	Irritability, panic disorder, agitation, impulsivity, anxiety, depression, obsessive-compulsive thoughts or behaviors, anger, rage (often termed " <i>Bartonella</i> rage"), combative behavior, suicidal feelings, bipolar disorder, hallucinations, confusion, disorientation, mood swings, antisocial behavior

quintana DNA in the patient's specimen, thus potentially missing many cases of *Bartonella* infection. Another challenge with traditional PCR testing is that *Bartonella* often infects humans at very low levels, making it difficult to detect with standard PCR techniques.

Three of the top specialty laboratories for *Bartonella* testing include Fry Laboratories, Galaxy Diagnostics, and IGeneX.

Fry Laboratories (Scottsdale, Arizona) offers IgM and IgG IFA testing for *Bartonella* as well as *Bartonella* PCR testing using molecular diagnostics. The *Bartonella* spp. by PCR testing screens for numerous clinically relevant species of *Bartonella* using a genuswide PCR method. Additionally, they offer assays using a modified May-Grünwald and other stains; however, these stains are not specific for *Bartonella*.

Galaxy Diagnostics (Research Triangle Park, North Carolina) has emerged as one of the more recent options for *Bartonella* testing. Its ePCR panel uses the BAPGM (*Bartonella* alpha-Proteobacteria growth medium) platform to optimize results. *Bartonella* are usually present in very low numbers. Therefore, an initial step to culture the blood and "enrich" the number of "copies" of *Bartonella* is usually necessary to enable PCR detection. Three of four positive tests are usually enabled by the enrichment culture step. All PCR-positive results are confirmed by DNA sequencing to confirm *Bartonella* and to identify the species. Galaxy specializes in *Bartonella* testing and has emerged as a leader in the field.

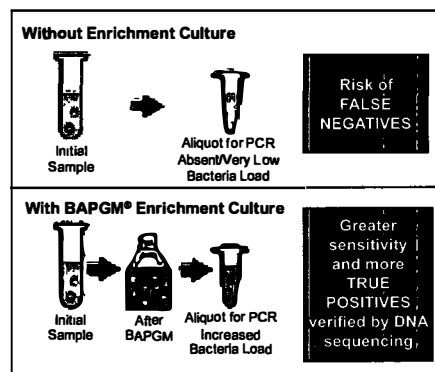
IGeneX Inc. (Palo Alto, California), a respected laboratory in vector-borne infections, offers IgM and IgG IFA testing, PCR testing, and a *Bartonella* FISH (fluorescent in situ hybridization) assay. The advantage of the FISH assay is that it looks for *Bartonella* RNA and thus does not rely on the response of the host immune system in order to identify the presence of the organism.

It has been suggested that vascular endothelial growth factor (VEGF) may be elevated in a subset of those infected with *Bartonella*.³⁴ It is believed that *Bartonella* may produce VEGF to stimulate its growth in the endothelium or VEGF may result from endothelial inflammation and infection. There may be an association between *Bartonella*-induced VEGF and various skin lesions such as bacillary angiomatosis or even striae. Some have suggested that monitoring VEGF throughout treatment may serve as an indicator of treatment progress, but Mozayeni has not found this to be a consistently reliable marker.

Mozayeni has found that patients solely infected with *Bartonella* can have elevated C4a levels, an inflammatory marker commonly high in patients with Lyme disease or those with biotoxin illnesses such as mold illness.

Galaxy Diagnostics ePCR

With the Galaxy ePCR Panel, there has been a confirmed positive rate of 9.2% as compared with only 0.98% for traditional PCR testing. Conventional IFA testing identifies exposure but cannot confirm presence of active infection. In terms of sensitivity, the ePCR is reported to be 10 times more sensitive than PCR and is 100% specific; each positive ePCR test result is confirmed by DNA sequencing. Conventional IFA testing is reportedly only 66% to 76% specific, which means that even when the result is positive, it may not be the result of *Bartonella* infection.



Galaxy Diagnostics *Bartonella* ePCR is optimized to detect low-level infection and minimize false negative results.

There is a cyclical nature of *Bartonella* bacteremia that may influence test results. *Bartonella* generally resides in the tissues that line the blood vessels and is not consistently found in the bloodstream. This makes testing more difficult and potentially leads to higher false negative test results. This drawback is minimized by Galaxy's True Triple Draw which collects three blood samples over a 7 to 8-day period to maximize test performance. The ePCR is believed to have a 90% reduction in false negatives as compared with conventional PCR testing.

In order to optimize the sensitivity of the testing, it is recommended that patients be tested prior to starting antibiotics or be off all antimicrobial therapies, including antimicrobial herbs, for 2 to 4 weeks prior to the blood draws. *Bartonella* exists in human blood in very low amounts and maximizing the numbers in the blood optimizes test results.

For those with chronic Bartonellosis, the ePCR test panel is an ideal tool as compared with conventional IFA testing which generally has high false negative rates for chronically infected individuals. IFA testing may be a better option for classical CSD.³

Bartonella is a stealth infection. The bacteria have a long division time of around 22 to 24 hours which makes diagnostic testing much more difficult as compared with testing of more rapidly dividing bacteria. A special growth environment was needed for *Bartonella* species, and it was an innovation from Breitschwerdt's research team at North Carolina State University College of Veterinary Medicine that led to a new culture medium. Considering that *Bartonella* is often found in sand flies, fleas, lice, and other insect vectors, an insect biochemical composition was evaluated as opposed to a conventional mammalian growth medium. This became the "secret

Bartonellosis

➤ sauce" of the Galaxy Diagnostics ePCR platform and is known as BAPGM (*Bartonella* alpha-Proteobacteria growth medium) With this enrichment medium, *Bartonella* is exponentially grown to levels that can

"Knowledge of *Bartonella* is virtually nonexistent in those who treat human patients. Veterinarians know much more about *Bartonella* than most medical doctors. With all the research that has been done on *Bartonella*, it is still a struggle to get people to notice it. When it infects heart valves, the damage is done before it is noticed."

- B. Robert Mozayeni, MD

be detected with a PCR assay, making false negative test results much less common, even in those with low levels of *Bartonella*

The Galaxy *Bartonella* ePCR is designed to detect all known pathogenic *Bartonella* species Although blood is the most readily available diagnostic specimen, BAPGM enrichment prior to PCR has facilitated the detection of various *Bartonella* species in cerebrospinal fluid, joint fluid, aqueous fluid, and pathological effusions such as pleural, pericardial, and abdominal effusions.

***Bartonella* Prevalence in Humans**

Mozayeni began working with patients with small vessel disease (SVD), relying on a careful subcortical neurological examination to find evidence of the disease in the nervous system Once he had a group of patients who met the criteria for SVD, he started testing this group for evidence of *Bartonella* infection.

Studies were done comparing 296 of Mozayeni's patients (some with a prior diagnosis of Lyme disease); 192 high-risk patients with animal exposures or veterinarians with fatigue, joint pain, and arthritis; and 32 healthy medical school volunteers serving as controls.

Of the 32 controls, only 1 had positive *Bartonella* antibodies and none of the 32 had a positive test using the BAPGM platform The high risk patients had a seropositivity rate of 49%; whereas Mozayeni's patients had a positive rate of 63% Using ePCR testing, 24% of the high risk group were positive; whereas 41%

of Mozayeni's patients had a positive test result which means that one or more *Bartonella* species were isolated or DNA of the bacteria was PCR amplified.³

Based on serological testing, blood donors have a *Bartonella* positivity rate of around 3.6%, veterinarians between 6% and 9%, and forestry workers between 10% and 40%. The Mozayeni patient population was higher than any of these groups. Importantly, serology may underestimate active infection with *Bartonella* in 50% and 75% of bacteremic individuals; therefore, serology has diagnostic limitations and the true prevalence of infection may be even higher than what is noted in these studies.

***Bartonella* and Lyme Disease**

Bartonella has been described as a common c-infection found in people with Lyme disease Mozayeni hypothesizes that the reason some patients with Lyme disease do not improve with treatment is because the emphasis may often be put on the wrong infections. Practitioners may focus on *Borrelia burgdorferi*, the causative agent of Lyme disease, rather than *Bartonella*, *Babesia*, and various protozoa such as *Protomyxzoa rheumatica*.³ It may be

these other microbial burdens that create the majority of the symptoms in what many refer to as "Lyme disease" rather than *Borrelia* itself.

"The paradigm in Lyme disease has been that one of the reasons the disease persists is because *Borrelia* has a complicated genome, is a smart organism that is very stealthy, has different forms, and evades the immune system," said Mozayeni. He continued, "We are now entering a second way of thinking about it, a 'Lyme 2.0.' We are trying to understand the ecosystem and the microbiome of the different organisms that are involved."

As common as *Bartonella* may be in those with Lyme disease, it can certainly exist on its own Many people with *Bartonella* alone may not express symptoms severe enough to be recognized and may be asymptomatic carriers. Over the long term, Mozayeni hypothesizes that carrying *Bartonella* chronically may cause a variety of common human diseases including arthritis, arteriosclerosis, and a host of other conditions. *Bartonella* is known to cause immune suppression in dogs and is immunosuppressive in humans, which may make people more prone to harbor other opportunistic microbial burdens and may fail to make antibodies to germs which might enable their serological detection.

Conventional Treatment Approaches

The most frequently used antimicrobial drugs for *Bartonella* are those that can enter the cell as the microbe is most commonly found intracellularly. While treatment is often successful in reducing symptoms, there are those where persistent infection has been identified "There is increasing evidence of treatment failures in people with normal immune systems, and it is not uncommon to see relapses in immunocompromised patients who were treated for six weeks or longer."³

According to the Infectious Disease Society of America, erythromycin and doxycycline are

drugs of choice with clarithromycin or azithromycin as alternatives. For those with central nervous system disease, they suggest that the combination of doxycycline and rifampin may be the preferred treatment. They note that in immunocompromised patients with repeated relapses such as in those with HIV, treatment may need to be indefinite.³

Bartonella is susceptible to numerous antibiotics in vitro, but many of these have only bacteriostatic activity; they inhibit reproduction but are not bactericidal; they do not kill the bacteria. Gentamicin and, to a lesser extent, Rifampin have been found to be bactericidal.⁴ However, Gentamicin may only be bactericidal when the bacteria emerge from the red blood cells and are extracellular.

Based on a 2004 study, antibiotics which may be helpful in the treatment of *Bartonella* include doxycycline, erythromycin, rifampin, doxycycline with rifampin, doxycycline with gentamicin, gentamicin and ceftriaxone with or without doxycycline, chloramphenicol, ciprofloxacin, or streptomycin.⁴¹

The Lyme and Tick-Borne Diseases Research Center at Columbia University has recommended that azithromycin or doxycycline combined with rifampin, clarithromycin, or a fluoroquinolone may be useful regimens.⁴²

Some practitioners have found drugs such as ciprofloxacin (Cipro), levofloxacin (Levaquin), gemifloxacin (Factive), and moxifloxacin (Avelox) to be helpful in those with *Bartonella*, though these drugs may lead to long-term tendon damage and ruptures, retinal detachment, and a host of other symptoms that may persist long after these medications have been stopped. They may result in what is known as fluoroquinolone toxicity syndrome, or what some term as having been "floxed." Mozayeni has suggested that the risks of these medications may outweigh the benefits, and he rarely uses quinolone antibiotics for the treatment of *Bartonella*.

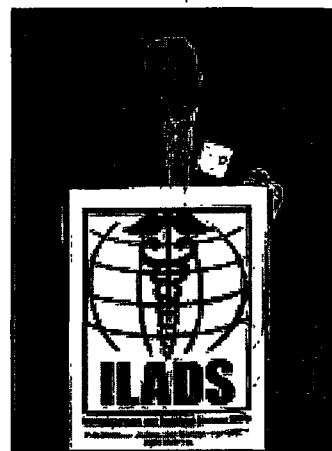
Mozayeni generally uses clarithromycin or azithromycin,

though there is a higher resistance to azithromycin in animals. He later adds rifampin with clarithromycin. If a patient is on other medications whose metabolism may be impacted by rifampin, he may then use Mycobutin. He has not found a need to treat any of his *Bartonella* patients with IV therapies except in rare cases where patients cannot take pills. He has noted that antibiotics used for the treatment of *Borrelia burgdorferi*, the causative agent in Lyme disease, may push *Bartonella* organisms further into the cells making treatment more challenging.

Both adrenal health and thyroid health should be considered before treating with these antibiotics, especially rifampin, in order to allow the patient to better tolerate the treatment. Mozayeni has found that one pitfall in *Bartonella* treatment is related to adrenal fatigue. Adrenal issues must be identified and managed to optimize therapy tolerance. Rifampin, a cytochrome P450 inducer, causes a more rapid metabolism of sterol hormones and can lead to severe symptoms where a Herxheimer-like reaction is juxtaposed with a condition of depleted adrenals and low cortisol. This can put the patient into adrenal crisis with greatly amplified symptoms including severe pain and hemodynamic instability. This reaction may be mistaken for a Rifampin allergy and therapy stopped. Thus, withdrawal of a useful drug may result in a lost opportunity to treat and lead to treatment failure.

The pharmaceutical protocol that Mozayeni utilizes in his practice has been shown to work well for many of his patients. If someone is extremely ill and may not be strong enough to tolerate treatment with antibiotics, he may start with herbal antimicrobial and supportive interventions. It will take time and additional data to determine whether or not the herbal approaches prove to perform as well, but they are useful considerations. After an antibiotic regimen is complete, Mozayeni may then move a patient to more natural *Bartonella* treatment options. These may be

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B. Robert Mozayeni, MD, chief medical officer at Galaxy Diagnostics, speaking on *Bartonella* at the 2014 International Lyme and Associated Diseases Society in Washington, DC.

more sustainable and can be used for longer-term maintenance therapy if needed.

Once someone is infected with *Bartonella*, it is possible that they will never fully clear the infection; it may become a matter of how the infection manifests within the body. Response to treatment of any regimen may vary based on the immune status of the host, the response of the immune system, and the infecting *Bartonella* species.

Alternative Treatment Approaches

While pharmaceutical options for *Bartonella* treatment are often very helpful, those with chronic *Bartonella* infection may benefit from looking at natural solutions. These may be combined with pharmaceutical options or used alone.

Mozayeni has an interest in allicin, an extract from garlic, and sulforaphane, a compound derived from cruciferous vegetables. Sulforaphane has broad spectrum antimicrobial properties against both gram-negative and gram-positive bacteria while also being anti-inflammatory, supporting detoxification, and serving as a powerful antioxidant.

Bartonellosis

Stephen Harrod Buhner is a one of America's preeminent herbalists and wrote *Healing Lyme Disease Coinfections: Complementary and Holistic Treatments for Bartonella and Mycoplasma* in 2013.⁴³ It contains

Bartonellosis, caused by the diverse members of the genus *Bartonella*, may prove to be the most important emerging infectious disease of the next decade.

- Edward B. Breitschwerdt, DVM

some of the most current information on herbal and holistic treatment of *Bartonella*. In the book, Buhner goes into extensive detail on *Bartonella* characteristics, symptom presentation, cytokine shifts that may occur, and natural treatment options based on his own clinical experience and literature reviews.

Buhner has created a protocol that is outlined in his book and consists of therapeutic options such as *Sida acuta*, *Isatis*, *Houttuynia*, *Alchornea cordifolia*, Japanese knotweed, EGCG, hawthorn, cordyceps, L-arginine, milk thistle, and others. He further outlines interventions that may be helpful based on specific symptom presentations. The book is a very detailed resource on *Bartonella* and is highly recommended for anyone who wants to learn more.

Many other natural products or formulations are available that some practitioners have found helpful. These include Beyond Balance MC-BAR-1 and MC-BAR-2; BioPure Quintessence, O3 Oil Gamma, Lyme and Co-Infection Nosode Drops, Cryptolepis, and Czaga (chaga); Byron White Formulas A-BART; Maypa Herbals Formula Bart; Jernigan Nutraceuticals Lymogen, NutraMedix Samento, Banderol, Quina, Cumanda, and Houttuynia; Researched Nutritionals BLT Microbial Balancer #1, CryptoPlus Microbial Balancer #2, and LymPlus Transfer

Factor; Deseret Biologicals *Bartonella* Series Therapy; Woodland Essence C.S.A. Formula; Mountain States Health Products *Bartonella* Nosode; Professional Formulas Tick Pathogen Nosode Drops; Dr. Zhang's HH and HH-2; freeze-dried garlic, and others. Injectable artesunate administered by a doctor has been found to be of clinical benefit. Some practitioners

have found essential oils of clove, thyme, marjoram, melaleuca, cypress, rosemary, and cinnamon to be helpful.

Recently, at the American Academy of Environmental Medicine workshop, low-dose antigen (LDA) therapy for Lyme disease was introduced by Ty R. Vincent, MD, and has reportedly been helpful for those dealing with *Bartonella* and other Lyme-related issues.

Synergistic Treatment Options

While the underlying microbial burden itself must be addressed, there are a number of synergistic interventions that may improve patient outcomes. Mozayani has noted that the most proximal cause of symptoms of *Bartonella* is the small vessel disease, and addressing this aspect of the condition is an important part of the treatment program. Two primary areas of focus include evaluating and treating coagulopathies and reducing inflammation.

Small vessel disease results in a form of brain injury, though the injury does not have to be permanent. Treatment must be approached in a manner very similar to how a brain injury would be treated. The nerves may be stunned or hibernating as a result of trauma; these may be resuscitated with glutathione, hyperbaric oxygen, or other interventions.

Bartonella often causes low grade inflammation in the body. This can manifest in more significant ways, such as inflammatory arthritis or a neurovascular problem, in people with specific MHC (major histocompatibility complex) or HLA (human leukocyte antigen) genetic predispositions. The infection may result in chronic, low-grade, smoldering symptoms even in those that considered themselves to be asymptomatic. How the disease manifests is determined by the biological terrain and how the host immune system responds to the infection and less by the infection itself.

For hypercoagulation, agents that dissolve clots or help to reduce clot formation may be helpful. The more the blood pH is acidic, the more coagulation is likely to be a concern. Substances that alkalize the body help to reduce microscopic clot formation. High dose vitamin C, apple cider vinegar, and an alkaline diet are useful tools. Incorporating fresh vegetables into the diet both helps alkalize the system and introduces enzymes that help to break down clots and biofilms (a polysaccharide layer produced by a community of organisms that serves to protect them from antimicrobial therapies).

Boluoke, lumbrokinase, or serrapeptase are commonly considered, as are Wobenzym and Researched Nutritionals InflaQuell. When patients are not improving with or tolerating treatment, Mozayani's first question is often whether they are keeping up with their enzyme intake; he finds that 80% to 90% of the time they are not. During a Herxheimer reaction, enzymes and alkalization can often provide relief. If neurological symptoms are severe, low-dose Lovenox or heparin may be considered and often leads to dramatic improvement.

Reducing inflammation using natural options such as curcumin, quercetin, and astaxanthin may be very helpful.

Treatment may include a focus on the health of the lining of the

endothelium where *Bartonella* congregate.

Nitric oxide is produced in the endothelium and may be impaired when the endothelium is unhealthy. Thus, therapies such as Xymogen AngiNOX, Thorne Perfusia Plus, or L-arginine, which increase nitric oxide production, may support blood vessel health. Increasing nitric oxide production may counteract some of the detrimental effects of *Bartonella*.⁴⁴ Low-dose baby aspirin may reduce the stickiness of the platelets.

In Chinese medicine, the herb *Dan Shen* (*Salvia miltiorrhiza*) is a "blood-invigorating" herb that is thought to make the blood flow more freely and has angiotensin-blocking properties. Vinpocetine is derived from the periwinkle plant and leads to dilation of blood vessels and improved blood flow. Hawthorn berry, in a form called Cratoxy, may support cardiovascular health and dilate the blood vessels. As a blood vessel dilator and platelet inhibitor, *Ginkgo biloba* may be beneficial.

Prevention and Management of Pet Exposure

In *Bartonella* patients with pets in the home, consideration should be given to the potential for reexposure from the pet or from fleas or ticks that these animals may bring into the home. Cats represent a higher risk for human exposure, though dogs may present with more symptoms when infected. Anyone infected with *Bartonella* and living with household animals should consider having their animals evaluated and treated for the infection in order to minimize the potential for reinfection. Veterinarians are generally well versed in testing and treatment of animals with *Bartonella*. Most importantly, keeping fleas and other vectors from infesting pets will reduce and potentially eliminate the possibility of transmission from a pet to a family member.⁴⁵

Conclusion

While more and more is being learned about *Bartonella* and its impact on human health, there are

still many unknowns that require further exploration. We are learning and will continue to learn. Very few medical doctors are familiar with *Bartonella* and people suffer needlessly, as bartonellosis is rarely on the list of differential diagnoses for the conditions that it may cause. Most infectious-disease doctors have very limited or no experience with identifying or treating *Bartonella* and believe that it is generally a benign condition that resolves without treatment.

Thanks to our animal friends and those who care for them, there is an ever-increasing focus on *Bartonella* and human health implications. The work of Drs. Ed Breitschwerdt and Robert Mozayeni has enlightened many about this previously underestimated microbe and continues to lead to improved testing and treatment options for both animals and humans. While getting people to recognize *Bartonella* has been a struggle, the tide is shifting.

Available tests have notably improved over the past several years, and treatment options are available that generally lead patients to higher ground. While there is more work to be done, the mysteries of *Bartonella* are beginning to unravel. Here's to your health!

Scott Forsgren is a health writer, advocate, and coach. He is the editor and founder of BetterHealthGuy.com, where he shares his now 18-year journey through the world of Lyme disease and the myriad of factors that it often entails. He has been fortunate to have written for publications such as the *Public Health Alert*, *Explore!*, *Bolen Report*, and *Townsend Letter*. Scott was personally affected by *Bartonella* for many years and today enjoys a state of good health. More information on his work is available at <http://www.BetterHealthGuy.com>.

Dr. B. Robert Mozayeni is an expert in translational medicine, the science and art of advancing medical science safely and efficiently. He is the chief medical officer of Galaxy Diagnostics. He specializes in autoimmune diseases and the effects of chronic infection and inflammation on vascular physiology and neurovascular conditions commonly seen with autoimmune and neurovascular diseases. He has a clinical practice in Rockville, Maryland. I thank him for his time in participating in this article and sharing his experience with the treatment of *Bartonella*. For more information, visit <http://tmgmd.com>.

Bartonellosis

In Memoriam

Linda "Angel" Heming worked closely with me for many years on a number of the articles that I have written. She was a warrior in the Lyme community and gave so much of herself to help other people. Given the time commitment required for each article, I was unable to do as many as I once did. A year ago, Linda asked me whom she could get who would interest me enough to do another article. At that time, I responded that an article on *Bartonella* with Drs. Ed Breitschwerdt and Robert Mozayeni would be compelling. Linda unfortunately became ill with another battle with cancer and passed away in October 2014. It was a great loss to the Lyme community. About a week after she passed, I was connected with a colleague of Drs. Breitschwerdt and Mozayeni, and an opportunity to do this article presented itself. Of course, I couldn't say no, as it was clear to me that Linda was still running the show. You will be missed and truly are an angel!

Bartonellosis

Upcoming Conference

On July 24, 2015, from 1 to 4 p.m. at the Hyatt Regency in Cambridge, Maryland, there will be a conference on diagnosing and treating *Bartonella*. Speakers will include Drs. Edward Breitschwerdt and Robert Mozayani. Conference is open to medical, counseling, and veterinary professionals. Marilyn Williams from the Lyme Disease Association of the Eastern Shore of Maryland is organizing this exciting event. For registration, information contact the LDAESM at easternshorem Lyme@yahoo.com.

Disclaimer

Information is not intended to treat, diagnose, cure, or prevent any disease. Nothing in this text is intended to serve as personal medical advice. All medical decisions should be made only with the guidance of your own medical authority.

Portions of the information presented in this article come from various conferences as well as an interview with Dr. Robert Mozayani for the purposes of this article.

Additional Resources

Breitschwerdt EB. Bartonellosis: a one health approach to an emerging infectious disease. Video of lecture available at <http://norvect.no>.

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Diagnostic Laboratories

Fry Laboratories: <http://www.frylabs.com>
Galaxy Diagnostics: <http://www.galaxydx.com>
IGeneX: <http://www.igenex.com>

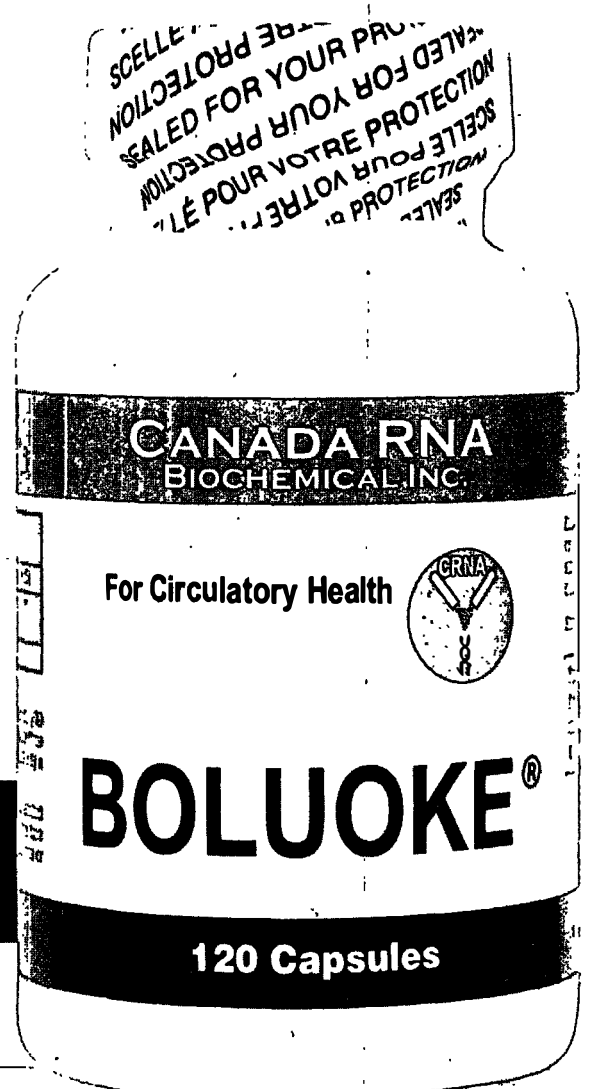
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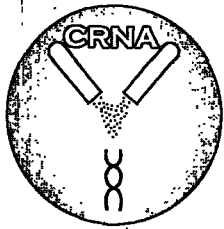
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Advanced 2015 *Babesia* Care: Profound Testing Defects and Preventing Disability and Death

by James Schaller, MD, MAR, and Kimberly Mountjoy, MS

Babesiosis is one of the most deadly tick infections on earth. It is not rare, a mere "coinfection," or "occasionally" present. It should always be checked in anyone with a history of a tick bite, possible Lyme disease, high suburban or rural exposure, or a transfusion, using specialty direct and indirect labs. Almost all *direct* testing is 95% inaccurate, with the exception of IGeneX, which detects *Babesia* approximately 25% of the time if you perform full testing of *Babesia microti* and *Babesia duncani* antibodies, PCRs, and a FISH test. This 25% is approximate and comes from 11

years of our own patients and our consult patients, who were tracked very closely on *Babesia* infections. Initial testing by over 100 physicians and our repeat testing yielded this percent in the absence of meaningful new exposures. Also, *Babesia* is routinely missed in slides by specialty laboratories.¹

Babesia can cause crippling fatigue and migraines. Weight loss or gain is routine, but so is disability from an additional 50 possible symptoms which were carefully derived from a decade of our research and presented earlier in our *Checklist* book.²⁻⁴ (See

Figures 1-3 and note the *Babesia* inside the red blood cells.) Despite these intimidating images, some patients have no symptoms. For others, their first or most serious symptom can be death by stroke, heart attack, blood clots in the legs or the lungs, or cancer.

Babesiosis, according to the CDC, can cause low and unstable blood pressure, severe hemolytic anemia (hemolysis), a very low platelet count (thrombocytopenia), disseminated intravascular coagulation (DIC, or consumptive coagulopathy), which can lead to the above-mentioned

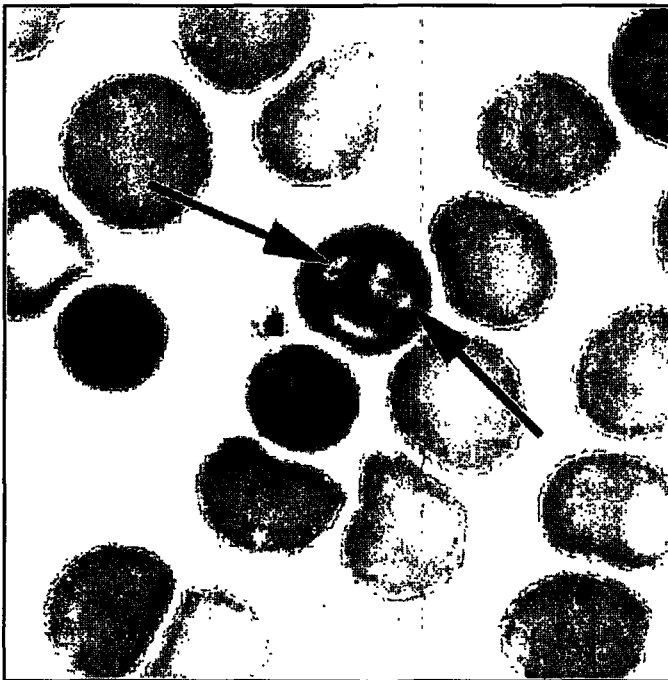


Figure 1: *Babesia* lives inside red blood cells. This routine Giemsa-stained blood smear is MO-1, which means that the first patient lived in Missouri. It was fatal.

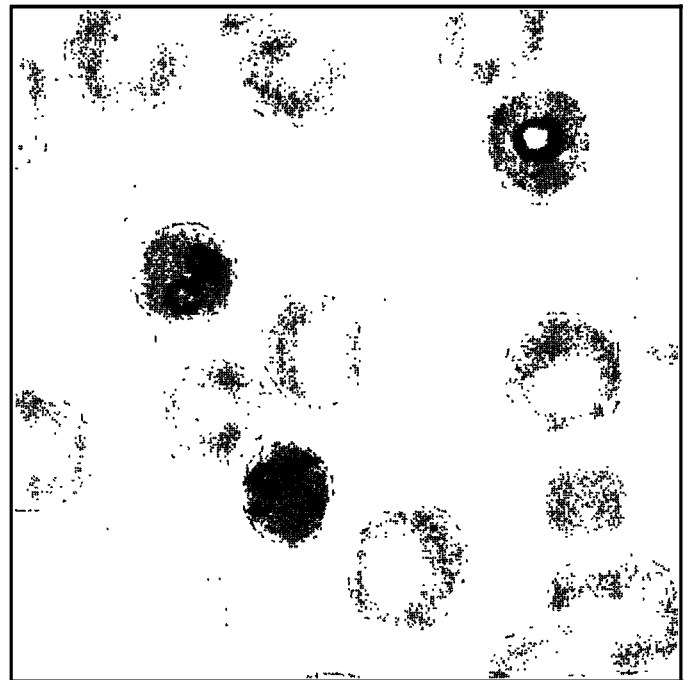


Figure 2: *Babesia* lives inside red blood cells. This routine Giemsa-stained blood smear is WA-1, renamed *duncani*.

blood clots and bleeding, malfunction of vital organs (such as the kidneys, lungs, and liver), and death.⁵ In 2001, Mylonakis was already warning of major risk of heart attacks, renal failure, and other catastrophic disease states from babesiosis.⁶

We first became concerned with *Babesia*, as an infection that affects clotting, when we noticed patients clotting very fast when cut by shaving accidents or blood draws. Very high D-Dimer and thrombin-antithrombin complex formation (TAT) blood levels were found in our *Babesia* patients, as well as research in humans and dogs.⁷⁻⁹ Our worry about death from clots in humans was further supported when some patients required prescription blood thinners to prevent a clotting death.

Further, another issue emerged also related to clotting. We were consulted on patients who had strokes. The trouble was that they would take Coumadin or other medicines to keep their blood at a constant level of "thinness" to prevent another stroke, but their clot measurement labs were too variable for optimal clot prevention. All of these patients were eventually found to have *Babesia* and other tick infections, and their unstable blood thinning or clot risk labs, the PT, aPTT, and INR, changed due to a *Babesia* infection.

Heart and brain infarcts are the leading causes of death in adults. *Babesia* increases the incidence of these infarcts by an unknown percentage. The chemical changes that *Babesia* causes in the body may add to this problem. This is partly discussed in our book, *Babesia 2009 Update*.¹⁰ *Babesia* occasionally increases red blood cell size (MCV) so that red blood cells that normally measure 8 microns often can barely pass through 10 to 20 miles of human capillaries.

Babesia's "sibling," malaria, another single-celled red blood cell parasite, routinely causes brain infarcts, causes white blood cell adhesion, and impairs venous blood flow causing pressure, edema, and

other coagulation disasters.^{11,12} No single test should ever be used to rule out a clotting risk from *Babesia*.

Another top cause of death for adults is cancer. We believe that *Babesia* increases the rate of some cancers. The amount of natural killer cells with an outer CD57 marker can decrease with the presence of Lyme disease, but *Babesia*, when it is killed, can decrease the levels of this type of cancer killer cell even more.¹³ For example, in proprietary research and in our physician patients who were only given *Babesia* medications, the levels of cancer killer cells with CD57 decrease very rapidly with effective *Babesia* killing. Antimalaria medications used alone may also acutely decrease the levels of CD8 markers by *Babesia* death and presence of Lyme in the body.

Another common cancer issue is the lack of testing for *Babesia* in most hemolytic anemia workups – the red blood cells are being destroyed, and hematologists seem to have no awareness that this may not be cancer but *Babesia*, even when told that it is *Babesia* related.

Let me use a routine article to show this error. Nackos reports on a man who was admitted to a hospital weak and disoriented.¹⁴ His complete blood count (CBC) revealed mild anemia (hemoglobin 11 g/dL) and low platelets. His level of platelets had been normal 3 weeks earlier. My first appeal is that *Babesia* does not replicate very quickly. Nackos et al. seem to assume that *Babesia* replicates like weeds because he seems

to think all of the patient's *Babesia* load came from a single walk in the "country grass." We have serial *Babesia* smears examined every 2 to 4 months which show otherwise, proving that *Babesia* is very slow growing.

However, he is correct to consider a new additional bite is a trigger to acute illness and decreased function. The signs and symptoms were new in this poor man, but he already likely had *Babesia* present in his blood for years.

Sometimes someone has a tick-infection trigger event such as a car accident, flu, another tick bite, a surgery, an airplane trip, a divorce, or the death of a close family member, which may lead to the sudden emergence of *Babesia* symptoms. However, the patient may have had *Babesia* for 20 years. Occasionally, there is no particular trigger.

In this case, the patient was possibly bitten again "in country grass a few weeks before his symptoms started." The author, Nackos, might

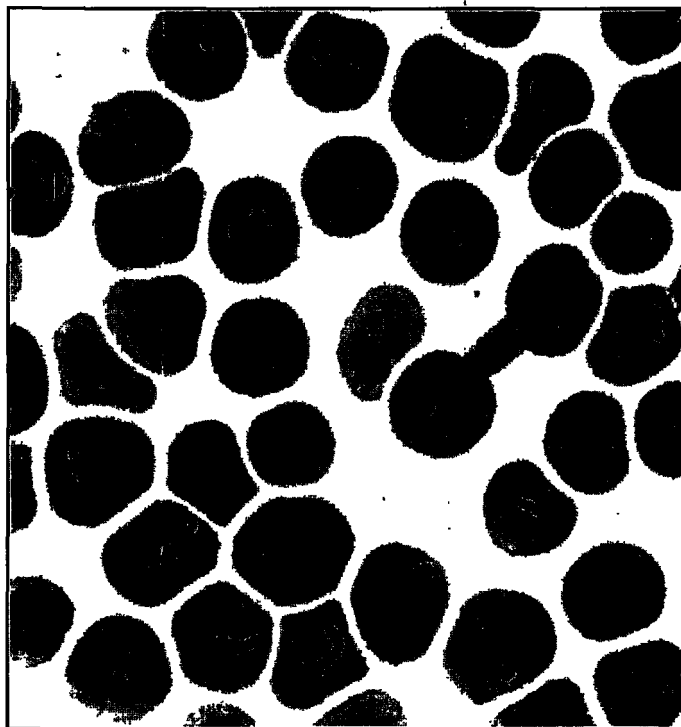


Figure 3: A wide range of *Babesia* single-celled infections inside the critical red blood cell – the cell carrying oxygen to all of the body's cells. The density of infected cells shown here is not routine. What is routine is to examine a blood sample slide for 3 minutes as the CDC suggests, and see no infected cells.

Babesia Care

► be confusing a trigger event with the onset of infection. I suspect that the patient had walked in exposed areas hundreds of days. My position is – and the evidence indicates – that this was not his first tick bite. One final point: it is theoretically possible that *Babesia* may create symptoms like a high mysterious fever on a first bite, but we believe that this is not the norm. We believe that the norm is that initial tick bites are experienced as trivial or merely a brief cold or flu, and after years of reproduction, serious symptoms start.

Further, Nackos's patient showed evidence of shredding red blood cells. In response to the destruction of mature red blood cells, young red blood cells come out of the bone marrow quickly. These are called reticulocytes, and his count was 5.56% (0.5% to 2.17% normal range). Lactate dehydrogenase was also very high at 540 IU/L (98 to 192 IU/L normal range), consistent with the deadly excess destruction of red blood cells called *hemolysis*. His peripheral blood smear showed many parasites that looked like *Babesia*.

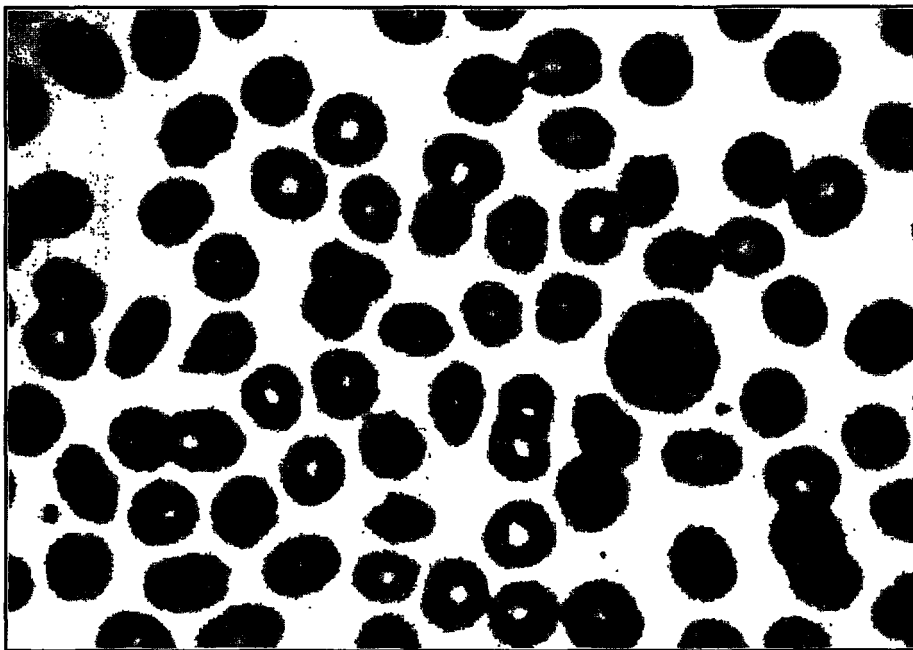


Figure 4: This European woman had been treated with routine *Babesia* medication by infection specialists. Do you think that the treatments and duration worked? This is a deadly number of parasite-filled red blood cells.

It is highly unusual for hospital clinicians to consider *Babesia*. Most clinicians do not consider *Babesia* when a patient presents with these symptoms – they are often more cancer oriented in their thinking when hemoglobin or white blood cell levels decrease. They can actually initiate chemotherapy for a supposed cancer.

It takes a rare and exceptional lab staff to visualize *Babesia* on a slide. As the author of six *Babesia* books and other tick infection writings, I have sent many *Babesia* and tick infection cases for blood smear testing. I have had antibody-positive and PCR (DNA)-positive *Babesia* patients, yet the most respected routine labs never see the *Babesia* organisms on a smear. Nackos had a good laboratory team. (Perhaps the blood *Babesia* volume was showing infection in a high percent of cells, as seen in Figure 4).

Babesia microti can cause deadly hemolytic anemia and/or dangerously low platelet levels (thrombocytopenia) per Nackos. I would add that it can also alter other labs such as decreasing white blood cell levels to 1200 to 5000. Altered red blood cell (RBC) numbers can also hint at the presence of *Babesia*.

A Cancer Caused by *Babesia*

We published a functional cure for hypereosinophilic syndrome (HES), a blood cancer, from patient care provided in 1999–2000.¹⁵ Over 250 physicians missed the primer of this blood eosinophil cancer – a *Babesia* infection.¹⁶

Diagnosis

Direct testing for *Babesia* at the major world labs fails both patients and the doctors who treat them. Both antibody testing and DNA (PCR) testing at the largest world laboratories have little sensitivity. No laboratory tests are perfectly sensitive; however, the best and most advanced laboratory for *Babesia* antibody testing, PCR, and smear visualization is IGeneX. I am often perplexed when someone tests for "Lyme" at IGeneX and no other infection, such as babesiosis, is explored. I have had patients who were positive for *Babesia* and *Bartonella* with no Lyme disease.

Further, another trouble in diagnosis is from the impact of *Bartonella* on *Babesia* antibody testing. *Bartonella* is more common than either Lyme or *Babesia*, and it is immunosuppressive, so at times in diverse ways it alters direct and indirect testing results for *Babesia*.^{17–22}

One simple tool used to diagnose *Babesia* is to start treatment with *Babesia* medications. The more treatment given the patient, the more likely you are to get a positive direct – or indirect – *Babesia* test. Physicians are trained not to treat without good evidence of a positive diagnosis. Unfortunately, that means using routine local labs that will almost always miss the *Babesia*. However, once the physician cancer patient in our HES study was exposed to two strong *Babesia* medication options and the semisynthetic herb artesunate – not the weaker artemisinin – his *Babesia* antibody test converted from negative to positive, and some other indirect chemical markers of the presence of *Babesia* changed as *Babesia* organisms were killed.

Zhao provides a useful example of the defect of PCRs in a *Babesia* transfusion death.²³ A patient given *Babesia*-infected blood died from the *Babesia*. Although tragic, we should not be surprised since only last year Goodell reported that no *Babesia* test exists or is utilized to screen blood donations in the US.²⁴

In investigating this man's death, 13 blood donors were tested for *B. microti*. All donors were negative by PCR. However, one donor living in New Jersey had a profoundly elevated *B. microti* antibody titer (1:1024).

FISH Testing

iGeneX has a test that helps visualize *Babesia duncani* and *microti*. It uses a probe that has one end that attaches to the organism and another end that is vividly visualized to help a microscope expert see the *Babesia*. However, as you recall, seeing *Babesia* is an activity that takes more than an hour unless you are infected with a dangerously high level of *Babesia* organisms. Our position is that this is a good test to determine quantity. If you are positive, you likely have a high level of *Babesia*.

This does enhance examination of a blood smear, but roughly 85% of the positives will be missed. *Babesia* is missed by LLMDs and infection physicians because of their reliance on laboratory results rather than the art of diagnosis which uses a good patient history and examination.²⁵ This overreliance on lab results is one of many reasons for the failure of LLMDs and infection physicians in tick infection care. Lyme is seen as the "core infection," and *Babesia* is a coinfection. This is an error. Typically, people who have a positive FISH become negative with effective care. This does not mean that they are cured; it means that they are losing body load, making *Babesia* harder to see, even with this smart enhancement tool (FISH). I do believe that the test is worth ordering if the patient can afford it. (Figure 5 is a black-and-white picture of the FISH test showing various types of *Babesia* inside red blood cells.)

FL1953 or *Protomyxzoa Rheumatica*: The *Babesia*-Like New Protozoan

This protozoan was sent to the CDC, which reported that it was a protozoan and not malaria or *Babesia*. The agency could

not identify it. Dr. Stephen Fry reports that many studies are being conducted with the goal of publication in a close time frame. We believe that the DNA sequencing done by the Fry Laboratory genetics experts shows a new form of protozoan that is a biofilm engine. For more information, see my summary article at <http://www.personalconsult.com/posts/FL1953.html>. We also offer options for treating FL1953 biofilm issues in the best-selling biofilm textbook in the world.²⁶ (Figure 6 shows hundreds of black tiny red blood cells with a large white collection of complex biofilm jelly generated by FL1953.)

Variation in Diagnosis and Treatments

100 years ago, before antibiotics, Rocky Mountain spotted fever (RMSF) was death to between 20% and 80% of the people who contracted it – the difference in death rate depended on a small difference in location. The next town could have a radically different outcome. My appeal is that we know that *Babesia* species have many strains. The variation of strains

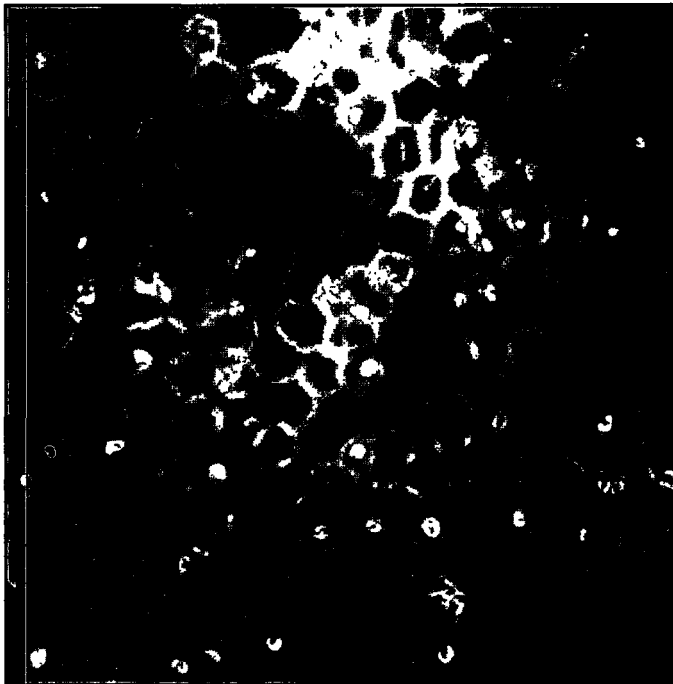


Figure 5: The FISH image, in black and white, enhances the visibility of *Babesia microti* and *duncani*. About 15 different *Babesia* forms are shown. This was supplied by Dr. J. Shah of iGeneX.

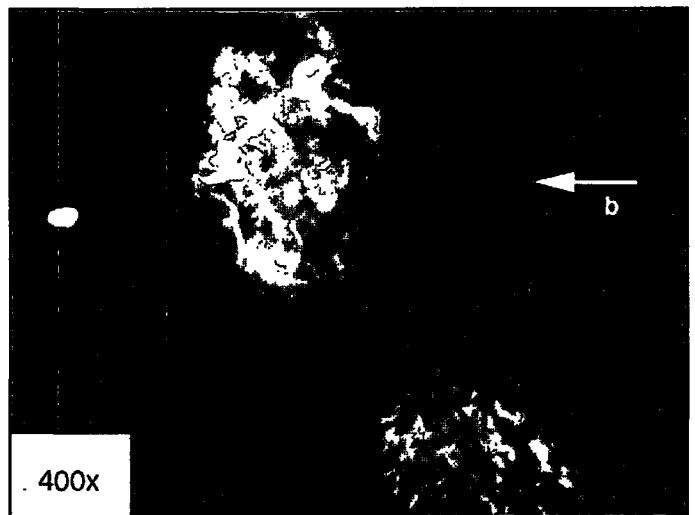


Figure 6: FL1953 or *Protomyxzoa rheumatica* is shown creating biofilm. The CDC reports that it is a genetically unique protozoan per Dr. Stephen Fry of Fry Laboratory. The small black circles marked by a "b" arrow are hundreds of red blood cells. Notice the two massive white biofilm balls that are approximately 100 times the size of a red blood cell. Source of image: Fry Laboratory.

Babesia Care

► from state to state determines some of the variation in symptoms of patients from different areas of the country.

Treatment

One of the disasters in treatment is based on the variation we mentioned with RMSF. Baseline pre-2015 treatments were weak and showed ignorance of the power of *Babesia* – it is vastly harder to kill than malaria. Therefore, simply plugging in malaria herbs or drugs can fail. Modern treatments might be founded on a famous paper that seemed to define perfect *Babesia* care rigidly for 15 years in infection care both by infection physicians and LLMDs.²⁷

In 2000, Krause, who had published concerns of suboptimal results with clindamycin and quinine therapy, reported low side effect and high success with a short course of atovaquone (Mepron) liquid at a dose of 1500 mg/day used in the presence of azithromycin (Zithromax) 500 mg/day.^{28,29} While the addition of another tool in the treatment of *Babesia* should be welcomed, many aspects of this approach are found to be flawed in 2015.

1. A review of *Babesia* species that infect humans shows immense variation, and the idea of one dosage for everyone is limited medical science. The infinite complexity and uniqueness of your body as it changes each month in hundreds of systems confuses *Babesia* treatment with the purity of basic arithmetic.
2. Human immune systems are complex and never clones in which everyone in a city could be given the same exact treatment for the same duration.
3. The determination of the presence of infections such as *Babesia* in ticks and human bodies is markedly limited. (Figure 4 [p. 68] is a picture of a patient after this treatment who suffered severely due to its ineffectiveness.)
4. Some patients are extremely sensitive to the atovaquone and they do not have an allergy. They experience a dose flaw. It is too effective. Not everyone can handle 1500 mg per day.
5. The issue of resistance to atovaquone is a concern in 2015.^{30,31}
6. Ignoring the resistance question, another option is that some people need a higher dose, as correctly advocated to me by a kind physician, Richard Horowitz, over 10 years ago.^{32,33}
7. The role of azithromycin and the dose used seems arbitrary. We can easily measure blood levels and on 500 mg/day the range of levels are highly variable.

Proposed 2015 Treatment Options

Lesson one is that all treatments should be started at a low dose and increased to make sure that no one is made to feel discomfort by the dose of the medication or herb. Treatments tend to make people feel better and then make them slightly uncomfortable. Most can tolerate an increase after days or a month. No single recipe exists. The patient and physician need to talk.

Malaria medications should be examined but not blindly

added. I do not find any evidence that doxycycline, minocycline, clarithromycin, azithromycin, or sulfamethoxazole/trimethoprim (Bactrim) kills *Babesia*. I think that the side effects of clindamycin and quinine usually would not make them a first-line treatment.

Of the treatments that do work, we believe that *Babesia* is so dangerous and disabling in the long term that at least three direct *Babesia* killers should be used before someone is considered ready for the end of treatment.

Artemisia or sweet wormwood (*Artemisia annua*) may prevent a malaria infection by just working in the harvesting and tending of this herb. (See Figure 7 of *Artemisia annua*.) But to kill *Babesia*, semisynthetic derivatives are required. Qing Cai Zhang, the expert herbal physician sent to the US at the request of our nation, has discussed *Babesia* treatments.³⁴ In his artemisia and double-potency Artemisiae-2 products, he uses artesunate, a synthetic modification of sweet wormwood. Please note: this is not wormwood (*Artemisia absinthum*).

One routine *Babesia* treatment used by Lyme-literate MDs (LLMDs) and functional medicine healers is artemisinin. I wrote in three texts that this derivative is dated, weak, and not even potent enough to kill malaria.^{35,36} The inability of artemisinin to kill malaria due to resistance is one of many reasons that top malaria agencies are looking for and using other artemisia derivatives.

Artemether/lumefantrine (Coartem or Riamet) is one medication that is partly an Artemisia derivative. This effective malaria medication is also often an effective *Babesia* medication, and does not have the "yellow paint" taste of atovaquone. In the US it only comes as a tablet: 20 mg of artemether with 120 mg of the synthetic lumefantrine. In many other nations, a generic is 4 times as potent. It is an appealing, small tablet that is dosed 8 tablets daily for 3 days for the treatment of malaria. That dose is profoundly too high for *Babesia*.



Figure 7: Some *Artemisia annua* or sweet wormwood plants, which are grown all over the world.

Patients may detect benefit at a dose as low as ¼ to 1 tablet a day. You can determine the upper dose with your physician. I was concerned about the risks of artemether 10 years ago, but after seeing and reading about its safe use in millions, I no longer fear this option.

Atovaquone/Proguanil (Malarone) is a combination of the atovaquone mentioned above (Mepron) in a tablet form combined with proguanil.³⁷ This is primarily the only way for most physicians to prescribe proguanil. One initial issue is that it can cause nausea far in excess of what is tolerable. Therefore, we suggest taking the medication in ¼ or ½ tablet units inside empty gelatin capsules, first inside a 0 and then inside a 00, so that the tablet has a double coating. This will end nausea based on stomach contact. If nausea is still present due to direct stomach contact, the addition of marshmallow root is effective at slowing and protecting an irritated stomach mucosa. Dosing should be started at ¼ to ½ tablets per day on the first two days, and then adjusted based on patient experience and insurance coverage. We believe that this is a good, modest *Babesia* treatment option.

Synthetic garlic derivatives are not your average garlic extract. If one reads Professor K. Huang and Dr. Zhang, it is clear that synthetic drugs have a massive foundation of research in China and Asia, and these do kill babesia.³⁸ But they also kill everything that a tick or flea might carry. You can carry a garlic smell for 36 hours, and dosing needs to start with a pinch or ⅓ to ½ of an allicin capsule from hepapro.com. It may say "allicin" but it is not mere natural garlic.

Mefloquine (Lariam) does kill *Babesia*.³⁹ However, in our very precise examination of insomnia, mood, irritability, restlessness, and concentration, I believe that the medication tends to create side effects unrelated to the killing of *Babesia*. I do not think that in most situations these common problems, minimized by some, warrant using it for *Babesia*.

Nitazoxanide (Alinia) is a newer medication in the parasite community and in the US; it is approved for diarrhea caused by *Giardia* and *Cryptosporidium parvum* in children. The French report effective use against *Fasciola*, a human parasitic disease.⁴⁰ Dupouy-Camet usefully makes this statement about the uniqueness of nitazoxanide: "[It is] a well-tolerated anti-parasitic agent with a broad

Babesia Care

spectrum because it is active on many intestinal protozoa and helminths. It acts in the same metabolic way as the 5-nitro-imidazoles inhibition of the ferredoxine reductase) but without synthesis of free radicals and DNA deterioration of the target cell. It is thus neither teratogenic nor

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▶ mutagenic."⁴¹ However, this drug does exit the intestinal track and has other actions that we believe include effective *Babesia* killing in some individuals and with some strains.

Essential oils have many compounds that can kill *Babesia* and also human cells. If anyone is truly interested in using this useful and dangerous class of treatments, please start by reading Tisserand and Young's excellent textbook, *Essential Oil Safety: A Guide for Health Care Professionals*, 2nd edition.⁴² If your healer has not read this book, s/he has possibly become out of date. If the person proposing essential oils is not familiar with the book, propose it *for your safety*. Further, we currently have the best-selling biofilm book in the world, and it discusses many of the most effective antibacterial, antifungal, and antiprotozoal essential oils.⁴³

Herbal options for *Babesia* were initiated above in our discussion of some of the most common medicines in the world – artemisia or sweet wormwood, artesunate, artemether, and artemisinin. But many other herbs exist that have antimalarial and anti-*Babesia* properties.

As a starting place, after Huang and Zhang, Buhner's herbs are ones that we also have researched most heavily. We have reviewed all of his textbooks published since 2000. We think that both Buhner's and Zhang's herbs are useful and have credible research and experience. We think that most should never be started at a full dose or tablet on the first day, and that they all tend to have a weaker action than some synthetics – which is actually a very good feature. Why? Almost all practitioners overdo treatments on inflamed tick-infected people. This long-term inflammation caused by tick infections causes autoimmunity, allergies to foods and medicines, and less coping ability and makes some patients "overly sensitive" to any treatment. I am not going to list and

comment on each one of the herbs that Buhner publishes in his books. Buy his books or ask your librarian to order them through the interlibrary loan program. If you are interested in artemisia in its natural and synthetic forms, consider our text, found inside both our large 2006 *Babesia* book or as a smaller textbook only discussing the major artemisia derivatives.^{44,45}

Hyperbaric oxygen therapy (HBOT) has many exceptional benefits, but it does not kill *Babesia* or any major tick infection. For example, we financed a study for over \$100,000 to determine the ability of HBOT to kill *Babesia*, Lyme, and *Bartonella*. Participants received 110 to 120 treatments at 2.4 atmospheres for 90 minutes. There was no change in their indirect or direct lab results, or their clinical function.

Infrared sauna treatment is based on both high temperature and infrared waves to kill infections. *Babesia* is very hard to kill, and most of your 100 miles of blood vessels are out of the range of the rays, and high external temperature is not curative of *Babesia*. A high internal temperature, such as a fever, is a body response to a pathogen.

Ozone is gaining resurgence in interest due to biofilm resistant infections. In terms of ozone, I would suggest as starting points the research and books of Bocci, Shallenberger, and other healers looking for treatments outside of synthetic pharmaceuticals.^{46,47} Certification and training societies now exist all over the world. We think that direct injection of ozone into veins is malpractice, but that ozone is a useful option that has extensive research and utility in careful wise hands. It has to be dosed carefully, since it is common that individuals kill more of an infection than intended, which causes profound inflammation and discomfort. Ozone is used many ways in industry and health.⁴⁸⁻⁵³ It is also an effective killer of microorganisms in pools. One common route to use ozone as a therapeutic agent is to remove blood, add an anticlotting agent, and return the treated blood

to the body. This is not a cure, but our observations over 20 years show that it delays *Babesia* relapse and is a useful tool to augment treatment: Again, the dose of ozone needs to be low on initial trials.

A Sample of Other Treatment Options

Other common *Babesia* treatments on the Internet and in functional medicine are Rife machines, very low-dose herbal tinctures, acupuncture, intravenous immunoglobulin (IVIG), homeopathy, and energy medicine. Due to space limitations, we will only mention that we are very aware of these modalities, and that some patients and healers report that they work for *Babesia*. We have inherited patients who were using these options, and their reports and laboratory findings are worthy of further examination. We appreciate that some people report that they believe these treatments are reliable and even curative.

In conclusion, *Babesia* is a very serious infection with poor to fair sensitivity of laboratory testing, and effective treatments need to be tailored to each patient – mill medicine and simplistic "protocols" lead to poor 5- to 10-year outcomes. Finally, all the comments made in this article are only to be considered with the licensed counsel of your local healing professional. Nothing expressed is to be considered the standard of medical care in the US.

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James Schaller, MD, MAR, is an top-rated physician with awards from physicians and national and international patients. He has written 6 books on *Babesia* and 13 books on tick infections. His newest book on biofilms is the best-selling book on the topic in the world, and the second best-seller under cystic fibrosis. He has published respected journal articles and books on cancer cures; parasites; nutrition; hormones; mystery illnesses; depression; inflammation; flea, louse-, and tick-borne infections; and a respected NIH-endorsed set of entries on *Babesia*, *Bartonella*, and Lyme disease. He is the author of over 30 books and 27 top peer-reviewed journal articles.

Kimberly Mountjoy, MS, is a chemist, researcher, and coauthor of seven books and eight peer-reviewed articles in chemistry and biochemistry. She has worked in research in industry and in respected science centers for many years. Most recently, she has been involved in hands-on patient care before joining Dr. Schaller.

Multifactorial Approaches to Lyme Infection

by Dan Kenner, PhD, LAc

The term *Lyme-literate* is used to describe physicians and health-care providers who regard Lyme disease as a complex multifactorial and hydra-headed clinical syndrome. There are numerous challenges to understanding the complex nature of its clinical manifestations and developing an effective approach to treatment. The 19th-century reductionist model of a pathogenic microbe as the sole cause of a disease and the corollary notion of curing the disease by killing the "bad germ" is obsolete for treating persistent chronic infections.

This reductionist viewpoint limits thinking. A treatment goal of killing the pathogen microbe is the traditional formula for failure. The clinical strategy of a systems biology viewpoint is not to attempt to kill the microbe by direct contact with a poisoning drug, but to change its habitat. A healthy condition of the gut and tissues will not support the overgrowth of toxic microbes. This is the traditional view of German biological medicine and the French concept of the terrain.

In German biological medicine, the model proposed by Dr. Hans-Heinrich Reckeweg in his seminal work *Homotoxicology* classifies all pathological phenomena in a system of nosological units based on each tissue of the body according to its stage of deterioration. Reckeweg developed a flow chart to be used as a fundamental model. His chart had a vertical line listing all of the body's tissues, grouped according to their embryonic origin. In the horizontal aspect, he listed 6 stages of pathological development, three

"Humoral" phases and three "Cellular" phases. Phase One is the "Excretion Phase," followed by the "Reaction Phase," which is acute inflammation; and the Deposition, Impregnation, Degeneration, and Neoplasm Phases represent a nosology of disease vectors. Movement from Phase One toward Phase Six maps the decline of the body's structural and functional integrity. This model is called "The Health-Disease Continuum." In the early "humoral" phases of pathology, the principle of regulation is excretion of pathogens and all harmful elements. In the cellular phases, the principle is precipitation of nocive substances and a resulting congestion of the extracellular matrix, which is referred to by its older term, the *mesenchyme*. This congestion results in a loss of functionality and a chronic inflammatory condition or degeneration of proximate tissues as cells are deprived of nutrients and bathed in their waste products. One of the main characteristics of chronic disease in this model is the congestion and subsequent erosion of the matrix tissues and the ground substance, the intercellular material in which the cells and fibers of the connective tissue are embedded.

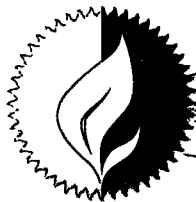
One of the important treatment strategies to relieve congestion is *drainage*, which is the process of removing obstruction and restoring what Reckeweg called "the dynamic flow" of the tissue fluids at this level of anatomical structure.

The term used to describe a model of the condition of the whole system is the *terrain*, a term in use since the late 19th century in France in the era of Louis Pasteur and Claude

Bernard. The terrain concept was originally a description of the body's fluids and the system of metabolism in its entirety. The most prominent proponent of the terrain philosophy was the famous physiologist Claude Bernard. Bernard proposed that the factors regulating the cellular environment play a primary role in the functioning of the organism as opposed to external factors. Bernard was famously at odds with the equally prominent and internationally famous Louis Pasteur when he said, "The germ is nothing; the terrain is everything." Pasteur's discoveries led to the "germ theory" of disease, in which toxic microbes are considered to be responsible for their related infectious diseases. In the terrain theory, treating an infection requires changing the microbial habitat; that is, the body's global condition, rather than simply attempting to kill a single type of microbe. Bernard also referred to the terrain as the *internal medium* (*milieu intérieur*).

Of course external factors such as toxic microbes can also play an important role in pathogenesis, but Bernard and the terrain-model advocates claimed that disease germs were a trigger and a marker for infectious disease, but the actual cause was the internal predisposition or weakness in the body's internal environment. This emphasis on a person's biological individuality means that a terrain model is much more complex than a simple cataloging of diseases and the germs that supposedly cause them. In chronic infections, the terrain concept may be superior "software" for dealing with its inherent complexity.

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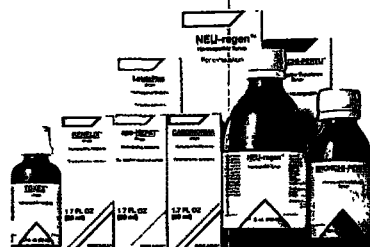
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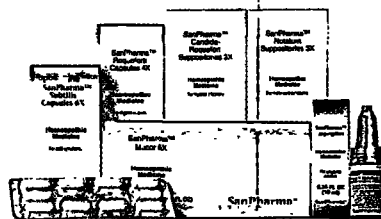
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Lyme Infection

► continued from page 74

Lyme disease in its chronic form is sometimes called “tick-borne illness” because of coinfections by other microbes including *Babesia*, *Bartonella*, *Ehrlichia*, *Mycoplasma*, and others. In addition, opportunistic infections with the yeast *Candida albicans*, the bacterium *Brucella*, and a host of viruses including herpes 1 and 2, HHV-6, Epstein-Barr, Coxsackie, and cytomegalovirus have been reported.

Kristine Gedroic, MD

An increasing number of doctors are developing new strategies to accommodate the needs of patients with the range of symptoms attributed to chronic Lyme disease. Kristine Gedroic, MD, is the founder of the Gedroic Center for Integrative Medicine in Morristown, New Jersey. Her approach to Lyme disease reflects this view of a complex pathological syndrome, not as a specific entity with a microbial etiology (i.e., Koch’s postulates), but an intricate set of interactions between the host and microbe populations. She uses various botanical, biological, and homeopathic medications in an integrative approach that involves detoxification of environmental toxins and heavy metals, correcting gut dysbiosis, draining and unburdening the extracellular matrix, and assisting the patient in cultivating lifestyle habits that support positive treatment outcomes.

Relieving congestion in the extracellular matrix is of primary importance in her treatment approach. For cellular detoxification to occur and to facilitate the clearance of microbes, the matrix must be detoxified in order to accommodate the flow of microbial debris. The body possesses an innate flow system that represents the natural inertia of healing. In the ideal scenario, the practitioner is able to harness this mechanism to facilitate the healing process naturally. Gedroic also believes that the cell membrane

disruption is at the root of chronic *Borrelia* infection. She uses several diagnostic tests from Stony Brook University, Fry Labs, IgeneX Labs, Advanced Labs, Doctor’s Data and Vitamin Diagnostics, BodyBio, as well as national labs – Quest, LabCorp, and BioReference, in order to run a comprehensive panel that assesses global balance of the immune system, as well as nutritional deficiencies that may be confounding the treatment paradigm. Testing can identify the presence of various toxins, as well as the presence of coinfections from *Bartonella*, *Babesia*, *Ehrlichia*, *Mycoplasmas*, *Chlamydias*, viruses, and *Protomyxzoa*.

For a qualitative assessment of the patient’s condition, she emphasizes the importance of using the Stained Blood Film Test (epierythrocytic bacteria) from Fry Labs to evaluate the presence of biofilms. Gedroic says, “If I could only do one test, it would be this. It gives a visual read on the degree of polymicrobial infection and congestion of the matrix.”

Biofilms are multicellular communities held together by a self-produced extracellular matrix. The CDC estimates that 65% of infection today involves biofilms. Estimates suggest that biofilms are up to 1000 times more resistant to antibiotics than planktonic forms.

Constitutional assessment is another important factor to determine if the patient can withstand stimulation or a challenge to the immune resources; that is, if a patient is “stable” or “depleted.” The depleted patient may require additional nutrient and herbal support.

Borrelia does not necessarily make someone symptomatic. It is opportunistic and takes advantage of a compromised host. Sometimes the Lyme microbes are simply there and don’t play as important a role in the chronic nature of the problem. These are cases that need to be approached differently. This is described as membrane instability: when a patient’s cellular membranes are impaired to the extent that they are not capable of maintaining immune

regulation. In these cases, *Borrelia* is often only a small part of the problem. Once cellular membrane integrity is restored, symptoms abate. Antibiotics are rarely needed for longer than 2 or 3 months. If the patient continues to be symptomatic, it means that a different pathogen is dominant or cellular integrity needs to be restored.

The PEKANA medications play an important role for regulation and drainage of the matrix. Biological medicines from Syntion and SanPharma are considered immunomodulatory or “cellular reprogramming therapy” and are vital to immune support for treatment of infections. SylImmune is important for latent infections and to nudge the immune system out of anergy, SyCircue for inflammation and matrix congestion, SyResp and SyGest for gut dysbiosis, SyRegule for fungal infections, and SyInfect as a broad-spectrum agent for infections. SanPharma medications have similar effects for infections, inflammation, and dysbiosis. Herbal tinctures from Beyond Balance are also used for disinfection, detoxification, and inflammation. Gedroic likes the traditional Ayurvedic herb *guggul* (*Commiphora wightii*) for treatment of biofilms.

Treatment of gut dysbiosis often requires a combination of the paleo diet with probiotics and GI Repair Nutrients (from Vital Nutrients).

Her favorite protocol is:

SyGest or SyFungin (Syntion): ½ tab to 1 tab t.i.d.
Apo-STOM +/- SyDetox (PEKANA/ Syntion): 5 drops b.i.d. to ½ tsp b.i.d.
Opsonat (PEKANA): 3 drops daily to 15 drops b.i.d.
Core Pau D’Arco (Energetix): 5 drops daily to 30 drops b.i.d.
Core Berberine Blend (Energetix): 5 drops daily to 30 drops b.i.d.

Stabilization of the nervous system:
PsyStabil (PEKANA):
Viscum (PEKANA)
SyDetox (Syntion)
SyCircue (Syntion)

Dalektro N (PEKANA), E-Lyte

(BodyBio)

SR3 Balance Oil

Phosphatidylcholine (BodyBio or Nutrasal)

The triad of apo-STOM, SyDetox, and Viscum is particularly effective as a starting point.

Intravenous treatment can be a decisive element in intractable cases. Gedroic has discovered that intravenous phosphatidyl choline can break up the congestive membranes of biofilms. This is sometimes followed up with IV glutathione. Additional detoxification measures may also be taken intravenously.

Countermeasures for Herxheimer reactions:

Increase drainage of the extracellular matrix

Parsley/burbur/Pinella from NutraMedix

Options for alkalization:

Alka-Seltzer Gold

Alkabase

Magnesium carbonate (BodyBio or BioTech)

Potassium bicarb (BioTech)

Lemon water

Gedroic notes that since the introduction of matrix drainage and German biological medicine therapies, there has been a 75% reduction in Herxheimer reactions in her practice.

Gedroic's research projects are under the umbrella of the Gedroic Research Initiative (GRI), associated with the Institute for Infectious and Inflammatory Disease (I3D) at Rutgers University, a project with more than 2000 contributing scientists. One proposed study is designed to assess the impact of helminthic infections in the gut microbiome and host susceptibility, another is to evaluate the influence of biofilms and molds on the intractability of chronic infection. A third research project involves examination of the effect of occult viral infection on ADHD.

Ann Corson, MD

Another physician who specializes in treatment of chronic infections in

all their complexity is Ann Corson, MD, of Cochranville, Pennsylvania. To explain the complexities of how she treats a chronic infection, Corson uses a very vivid depiction of the terrain: "I'm not just going into battle. I need to get the lay of the land and get the situation to my advantage. I have to destroy the enemy and carry the dead bodies away and then break down the debris. Then, the battlefield needs to be cleaned up and restored. This can take time." This metaphor also describes the strategy of drainage of the extracellular matrix. This is how Corson has adapted the principles of German biological medicine to serve the needs of her patients.

Corson explains that German biological medicine provides a framework for restoration of overall vitality, gastrointestinal function, and endothelial integrity; for drainage of the extracellular matrix, restoration of GALT, MALT, spleen and bone marrow function, and assistance in the regulation of neuroimmune, neuroendocrine, and neurovascular control mechanisms. It is necessary to have a complex model because there are multiple challenges in treating patients with tick-borne disease: polymicrobial infections with all classes of organisms, gut dysbiosis, liver/gallbladder dysfunction, hormonal and metabolic dysfunction, systemic inflammation – unregulated and stuck in the "on" position, vasculitis and hypercoagulability, a toxin-encumbered extracellular matrix, metabolic faults in detoxification, mitochondrial dysfunction, and immune-system/bone marrow dysfunction.

For stable improvement, it's necessary to address all of these issues and take countermeasures as required. These include the control or elimination of dominant pathogenic microbes, elimination of environmental toxins, reduction of inflammation and neuroexcitotoxins, restoration of gut and respiratory mucosal integrity, the buffering and elimination of hypercoagulation, the support of genetic and epigenetic weaknesses and restoration of

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neuroendocrine system function. It requires identifying allergies to food or environmental factors, lifestyle changes, stress reduction to address emotional and spiritual needs, and, often, structural work. Throughout treatment it is helpful to mitigate the effects of treatment such as Herxheimer reactions as progress is monitored.

Evaluation of each patient's needs requires a thorough history, physical examination, and strategic laboratory testing. A full history includes details from conception to the present, trauma history, both physical and emotional, surgical and dental history, and family history. A social history includes details of home, work, and school as well as environmental issues such as mold exposure, EMFs in home or work environment, and any arthropod insect exposure risk factors. History and physical exam include a history of symptoms and their time of onset, a complete review of systems, vital signs, and physical and neurological examination.

Laboratory evaluation necessarily includes tests for many classes of infection, a full medical workup with CBC with differential, CMP, UA, HLA typing for biotoxin illness, methylation genetic profiling, hypercoagulability evaluation, inflammatory cytokines, and autoimmune makers. Other important tests include hormonal evaluation, stool testing, food allergy assessment, urinary kryptopyrroles (KPU), heavy metal challenge test, and nagalase testing.

Treatment

Dietary compliance is of utmost importance. Diet should be fully organic, grass-fed meats, free-range poultry, wild fish, no processed foods, no GMOs, sugar limited to fresh fruit, no dried fruit, and often limited grains, gluten, and dairy. In this process, it is often necessary to support special nutritional needs. There are several homeopathic products for this purpose along with

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➤ warming Chinese herbs and warming foods. Nutritional supplements such as EFAs, trace minerals, B vitamins, vitamin C, vitamin D, vitamin E, R-lipoic acid, and transfer factors are sometimes needed. Some nutrients are necessary to target specific problems such as zinc, biotin, manganese, magnesium, B-6, P5P, arachidonic acid, L-methionine, lithium, and special B vitamin preparation. Mitochondrial resuscitation support can be invaluable.

Detoxification should be initiated very gently at first. It is also important to reduce inflammation at this time with homeopathic immune modulators. Proteolytic enzymes and cytokine modulators are also important in reducing inflammation.

Corson uses both allopathic and nonallopathic antimicrobials. Nonallopathic antimicrobials can be single botanicals or herbal mixtures and play a large role in reducing pathogenic microbe populations.

Spagyric combination homeopathics regulate organ function, restore the regulatory set points of metabolism, hormones, the immune system, and the extracellular matrix. Immunobiological medications are useful for specific targeted activities for disinfection and other vital functions such as detoxification support and tissue drainage.

The combination of nourishing and strengthening the patient, detoxification, disinfection, and drainage are essential features of any of Corson's treatment plans. But another necessary component is troubleshooting. There are two main categories of troubleshooting: nonresponsiveness and Herxheimer reactions. Both situations are discouraging for the patient and can affect compliance with the treatment plan. The first thing to do when patients are not getting better, however, is check for compliance. For recalcitrant patients, look again and again for unrecognized infections, a hypercoagulation state,

biotoxin illness from mold exposure, methylation faults, heavy metal toxicity, and occult dental infections.

Hypercoagulation can be a problem for anyone, but a genetic abnormality found in 1 out of 5 people makes their blood more "sticky." This is exacerbated with acute and chronically upregulated systemic inflammation, exacerbated with Herxheimer reactions, with the vasculitis caused by infections and with any febrile illness. Treat with fibrinolytic enzymes or heparin. Note: According to Corson, heavy metals and mold toxins worsen hypercoagulable symptoms exponentially. Herxheimer reactions almost always require an increase in enzymes.

Biotoxin illness is another reason patients may not be getting better. To quote Corson, "Mold! Mold! Mold! Mold is everywhere!"

It is necessary to ask about mold once, twice, 3 ... 10 times, because it is an ubiquitous yet often hidden problem; ask about water intrusion, leaky pipes, musty smells, and so on. One in four people have a genetic fault that predisposes them to the chronic inflammatory state caused by exposure to molds and their toxins. The innate immune system doesn't recognize and "present" these biotoxins to the cellular immune system for antibody production and subsequent elimination.

Methylation is required in the synthesis and repair of RNA, DNA, and proteins; in immune system function; neurotransmitter production; intestinal barrier integrity; and removal of wastes. Methylation plays a key role in the ability of our immune system to recognize foreign bodies or antigens to which it needs to respond. Whenever there is an assault on the immune system, the body must synthesize new T cells (which fight viruses, parasites, and control functioning of B cells). Unless methylation is operative, the immune system may react when it's not needed, creating autoimmune disorders, or fail to respond to actual threats when it is needed. Methylation

is also directly related to substances in your body that affect your mood and neurotransmitter levels of both serotonin and dopamine. In addition to its direct role as a neurotransmitter, dopamine is involved in assuring your cell membranes are fluid and have mobility. This methylation of phospholipids in the cell membranes has been related to ADD/ADHD. Membrane fluidity is also important for a variety of functions including proper signaling of the immune system as well as protecting nerves from damage. A number of serious neurological conditions cite reduced membrane fluidity as part of the disease process involved in modulation of NMDA (glutamate) receptors, acting to control excitotoxin damage.

Herxheimer reactions are always an emotional challenge for patients. They require reassurance and patients suffering from them may need help with motivation to continue compliance with the treatment regimen. Drainage remedies can be useful for symptomatic relief. Proteolytic enzymes can relieve the intensity of the symptoms by reducing inflammation. Fibrinolytic enzymes that dissolve excess soluble fibrin are an essential part of the treatment of all Herxheimer reactions. All of Corson's patients are treated with fibrinolytic enzymes at some point during their care. Never forget the chelate heavy metals when patients are having Herxheimer reactions, as killing off of infection often causes the release of toxic heavy metals from biofilm communities. Sequestration of toxic bile can relieve many symptoms of the reaction. Restorative fats, trace minerals, and correcting methylation problems can help relieve the "healing crisis." Corson warns other practitioners to be prepared to handle very toxic downloads when breaking up fibrotic debris by having proper drainage medicines, enzymes, and bile binders on board.

Patient protocols may include support for the hypothalamic pituitary axis, thyroid, adrenal, and gonadal systems that can be botanical or

homeopathic. Methylation reactions and liver detoxification is supported as needed. Additional etiologies for patient symptoms are structural problems, which are often relieved by cranial osteopathy or other bodywork. Emotional therapy may be required. Spiritual concerns may benefit from participation in orthodox religions or personal cultivation. Corson recommends Falun Dafa, a life cultivation practice of both mind and body, as the best path to spiritual perfection.

Essential Oils for Disinfection

Another group of doctors who treat infections without antibiotics are practitioners of French phytotherapy, based on terrain models such as the Five Elements of acupuncture and the neuroendocrine system. In addition to tisanes and tinctures, pharmaceutical-grade essential oils are integrated into the treatment plan.

Jean Valnet, MD, was impressed with the effect of essential oils in treating infections from battle wounds and even gangrene when he was chief physician at the Gulf of Tonkin Hospital in Vietnam in the early 1950s. When he returned to France, he was determined to develop a clinical science of phytotherapy (botanical medicine) and incorporate the power of essential oils in primary care. The research he and his colleagues initiated uncovered several unique phenomena:

- A microbicidal effect is produced despite the fact that the concentration in vivo is far too low to kill microorganisms by direct contact.
- The most active essential oils are not always those reputed to have antiseptic properties or those that contain antiseptic active principles.
- A single type of microbe possesses no specific sensitivity to a particular essential oil and vice versa. There is no phenomenon of acquired resistance of microorganisms to essential oils. For a given microbe isolated at a given moment, the essence will retain all its efficacy.
- One of the proposed mechanisms of the disinfection properties of essential oils is the ability to dissolve biofilms.

- The same type of microbe appearing in different tests in contact with essential oils is affected differently in each aromagram. The sensitivity of the microbe to essential oils is thus a function of the host organism; that is, a function of the terrain. In other words, essential oils that were effective against a pathogenic microbe in a petri dish were not necessarily effective in patients infected with the same microbe.

They realized that it was necessary to match the properties of an essential oil with the global physiological activity. They made clinical observations about the effects of essential oils, as well as other plant extracts to determine how to treat the terrain. Doctors who practiced acupuncture classified plant medicines according to the Five Elements of Chinese Medicine and other groups of doctors used the neuroendocrine model.

For example:

Oil of thyme (*Thymus vulgaris*) has these various properties:

Immune: anti-infectious (ENT, pulmonary, intestinal, pharyngeal, urinary, genital, cutaneous), antifungal, antibacterial (gram +, gram -), antiviral, antihelminthic, wide spectrum antihelminthic, vermifuge; immune stimulant; antioxidant, anti-inflammatory; febrifuge;

Pulmonary: mucolytic, expectorant, antitussive; thyme oil is the great mucous membrane cleanser, whether they are respiratory or digestive membranes;

Digestive: neurotropic digestive carminative, eupeptic, choleric, antigastric; ANS: parasympatholytic (strong vagolytic)

Endocrine: adrenal cortex stimulant;

Gonad: binds to estrogen, progesterone receptors;

Neuro: analgesic;

Neuromuscular: spasmolytic;

Renal: volumetric diuretic;

Use: dysmenorrhea, paralytic fear, spasmophilia, hypotension, infections, digestive disorders, rheumatic disorders;

Contraindications: Pregnancy, glaucoma, hyposecretory states.

Oil of clove (*Syzygium aromaticum*):

GI: eupeptic (stimulates all secretions), digestive, carminative, antiulcerative, choleric, balances intestinal flora;

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Metabolic: hypoglycemic;

Dental: all infectious maladies of teeth, pulp, gingiva;

ID: (tisane, too) anti-infectious (genital, urinary, gastric [*H. pylori*], intestinal, ENT, respiratory, mouth), antibacterial, antiviral (esp. herpes), antifungal, antiparasitic (protozoa, trypanosomes);

GU: antigonorrheal;

Neuro: antispasmodic (Ca-channel blocker), increases alpha-brain waves; antialgic, antineuralgic, local anesthetic

Immune: anti-inflammatory (inhibits synthesis of prostaglandins and chemotaxis of leukocytes), antiallergic (reduces histamine release from mast cells); ANS: sympatholytic, parasympathomimetic;

Endocrine: central: oxytocic;

Use: dysbiosis, digestive disorders, chronic cough (dry), inflammatory disorders, dental disorders, osteoarticular and rheumatic disorders, emotional overactivity, spasmophilia, learning disorders, autistic children, headaches, sexual frigidity, impotence;

Contraindications: pregnancy (oxytocinlike). Note: Avoid in bath; never use undiluted on skin or mucosa.

Endocrine phytoaromatherapy eventually evolved into a system of medicine called *endobiogeny*. Endobiogeny is the study of the internal networks and pathways within the body and in relationship with its environment, stressful influences, and so on, from the standpoint of the neuroendocrine system as the manager of human life. It combines an integrative understanding of the structures and functions of the body, a rational approach to understanding physiology, an empirical assessment of history and symptoms, and utilization of a dynamic, integrative metabolic assessment of network management of the organism called the *biology of functions*, which is based on calculations of several blood test values. (See www.endobiogeny.com.)



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Essential Oils and Lyme

A pharmacist named Bernard Christophe in Strasbourg developed a natural antimicrobial compound for Lyme treatment containing essential oils from aromatic plants and concentrated extract of propolis. Even in France, treatment with essential oils is still too little known to doctors. Christophe's product contained more than 170 molecules. It was very successful as a treatment for Lyme, sometimes even as a stand-alone treatment. Although it was labeled for topical use, many Lyme patients used it internally. It was eventually taken off the market in France, supposedly because it contained oil of sage, which contains thujone. Even though the quantity of thujone was below the allowance made by the health authorities it was still removed from the market. There was a public outcry but the authorities refused to relent.

In 15 years of the marketing of this product, there had never been a case of a toxic reaction, even in subjects who took the product for years without interruption and sometimes at doses higher than recommended. Nonetheless AFSSAPS (Agence française de sécurité sanitaire des produits de santé; French Department of Health Product Safety) took it off the market.

According to Christophe: "In the case of aromatherapy, known as

essential oils, they also have several special properties: they easily cross the membranes of various tissues and organs and contain several active bactericidal or bacteriostatic substances. In the case of Lyme it appears that *Borrelia* is unable to adapt to the multiple substances in a single essential oil. This is the explanation of the fundamental superiority of essential oils over antibiotics."

In addition, they always recommended the product to pregnant women and young children at doses of 1 to 2 drops three times daily. No cases of poisoning occurred in 40 years of work and 15 years of marketing the product.

Phytomicrospheres

Another type of medication from France is phytomicrospheres. Phytomicrospheres are made using a special plant fiber that acts as a carrier to bring herbal constituents into the bloodstream unattenuated. Nothing is lost from the extraction process. The microspheres contain no excipients and they transport the phytochemicals to the serum without degradation from the digestive tract.

Iderne Pharmaceuticals developed the microspheres process, which has been designated an ethical medical modality that will replace the use of tinctures in phytotherapy practice by the European Union. Iderne Pharmaceuticals has produced a number of compounds, including one that is designated as a treatment for diabetes by the European Union.

Stiruba, a microsphere extraction of the herb *Simarouba amara*, is a powerful antiparasitic and has proved helpful for some cases of *Babesia*. Several phytomicrospheres and essential oils products will be available in the US through Ormed and BioResource later in 2015.

Conclusion

Does it work? is the question that most of us ask about any supposed treatment or remedy, but I think that's the wrong question. Practically every nutrient or remedy that you have seen or heard about anecdotally has worked for someone. The real question is, what works for whom, and why? Obviously there is no single cure for everyone, but the complexity of Lyme infection requires a diverse approach and individual investigation and experimentation. Hopefully new scientific models will emerge and develop from traditional sources of whole-system thought. Meanwhile we can learn from these traditional European models of whole-system therapeutics.

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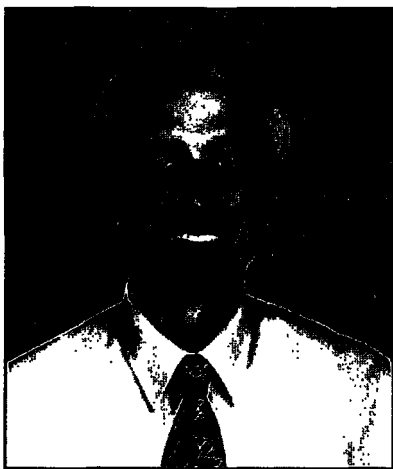
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Dan Kenner, PhD, LAc, is a writer and consultant in integrative medicine with over 35 years of clinical experience in both Oriental and naturopathic medicine. In addition to authoring numerous articles, he is author of *The Whole-Body Workbook for Cancer* (New Harbinger; 2009), *Acupuncture Core Therapy* (Paradigm; 2008), *AHCC - The Japanese Medicinal Mushroom Immune Enhancer* (Woodland; 2001), *The Science of AHCC* (Basic Health Publications; 2009), and *Botanical Medicine: A European Professional Perspective* (Paradigm; 1996). He is author of *The Science of Natural Cancer Therapy* (Éditions Trédaniel; 2015 [in French]). He is licensed to practice Oriental medicine in both Japan and the US. Dr. Kenner has constructed a website with alternative health-care research and information at www.dankenresearch.com. He is also on the board of governors of the National Health Federation (www.thenhf.org).

Iowa Congressman Declared his Lyme Disease Cured by Bovine Colostrum and Helped Create the Law Guaranteeing Availability of Natural Remedies (DSHEA)

Not many dietary supplements can claim that they were instrumental in the passage of the Dietary Supplement Health and Education Act (DSHEA). Yet, that's precisely what happened.

Former Iowa Congressman Berkley Bedell (D-Iowa) suffered from Lyme disease and did not recover with multiple, long-term antibiotic use. Due to severe debilitation,



COURTESY OF DEB MONIER REGISTER

Congressman Bedell eventually had to retire from Congress, but first, he paved the way to DSHEA. On the way to personal healing, Congressman Bedell discovered a farmer who was using cows to develop specific ("hyperimmunized") colostrum for specific diseases. Fortunately, the congressman experienced a full recovery from Lyme disease. Unfortunately, the farmer was arrested for practicing medicine without a license.

It didn't seem right that an individual could be punished for supplying a safe, all-natural substance which promotes healing simply because it was overlooked by mainstream medicine, so Congressman Bedell joined forces with like-minded senators and in 1994, the Dietary Supplement Health and Education Act was signed into law by President Clinton. DSHEA was very important to the development of bovine colostrum as a health-enhancing supplement.

In the last 20 years, colostrum manufacturing for human consumption has undergone a tremendous transformation. We now know that hyperimmunization of cows is not a requirement for colostrum's broad spectrum capabilities and that fresh, raw colostrum can be made into sup-

plements with an extended shelf-life. Better processing methods also allow for colostrum to contain a higher percentage of bioactives. Colostrum-LD® has been perfected to be effective against thousands of infections, something not possible in Congressman Bedell's day. Colostrum-LD®'s **unique broad spectrum profile** sets it apart from ordinary colostrum, and treating bacterial infections with Colostrum-LD® can be effective where antibiotics have failed.

Colostrum-LD® does not contain specific antibodies for Lyme. It doesn't need to. It helps the immune system successfully fight the pathogens within the body. Among the more than 280 bioactives known to affect the immune system by killing pathogens or modulating T-cell and macrophage production, the Proline-Rich Polypeptides (PRPs) are the most significant. In fact, PRPs can increase natural killer cell activity by up to forty times. Lyme disease is just one of thousands of bacterial and viral infections that Colostrum-LD® can effectively treat, including HIV, E-coli, C-difficile, Rotavirus and Cryptosporidium.

A major downside to conventional Lyme disease treatment with antibiotics is that it actually worsens a patient's prognosis. Antibiotics are never 100% effective; they leave resistant bacteria behind; and when taken for extended periods of time, cause destruction of the gut lining and increased gut permeability, or Leaky Gut Syndrome (LGS) which in turn, contributes to autoimmune conditions. Acknowledging the causal effect of antibiotics on LGS will help practitioners more fully understand why colostrum is so critical to a successful treatment program. Douglas Wyatt (Founder and Medical Director of the Center for Nutritional Research)

recommends Colostrum-LD®, the only colostrum laboratory-certified to contain all of the growth factors clinically proven to heal LGS. Put your patients on the path to recovery, even after years of antibiotics have already taken their toll. If you have a patient with Lyme disease who is not recovering and may have subsequently developed another autoimmune condition, Mr. Wyatt is available for consultation. *To learn more about the bioactives in Colostrum-LD® responsible for immune health, please visit www.ColostrumTherapy.com/lyme.html.*

Colostrum-LD® Broad Spectrum Immune Modulator and Natural Killer of Pathogens



Medical professionals may receive professional pricing by registering on www.ColostrumTherapy.com or call Sovereign Laboratories at 928.202.4036.

Consumers may purchase at www.SovereignLaboratories.com

 **Sovereign Laboratories**

Lyme Disease and Detection in 2015

by Stephen E. Fry, MS, MD

Introduction

Lyme disease is a bacterial infection caused by the spirochete *Borrelia*, usually *B. burgdorferi*. *B. garinii*, *B. afzelii*, and *B. miyamotoi* are *Borrelia* species that may also cause disease. Wilhelm Burgdorfer, a Swiss zoologist and microbiologist researcher who spent much of his later career at the Rocky Mountain Laboratory in Hamilton, Montana, is typically credited with the discovery of Lyme disease. By using chemical staining and light microscopy techniques, Burgdorfer was able to detect spirochetes that were found in deer ticks. Now, we know of at least 36 distinct species of *Borrelia* with at least 12 of them known to cause borreliosis or Lyme disease. The genomic sequence of *B. burgdorferi* strain B31 has revealed a 910,725 base pair core genome with at least 17 additional genomic plasmids totaling a combined 533,000 base pairs. There are approximately 853 genes to carry out the basic survival and reproduction of the organism. At the time of its sequencing in 1997, the majority of the 430 genes on plasmids did not have any known function.¹ More recently, it has been reported that much of the ability of the organism to cause disease is encoded by these plasmids.^{2,3}

Lyme disease is commonly transmitted by an insect bite, most commonly by ticks, but other insects have been implicated in disease transmission. The prime environmental reservoirs for the *Borrelia* sp. include deer, mice, squirrels, and lizards. In the endemic areas the primary vector for *B. burgdorferi* is the *Ixodes* tick. *Borrelia*

sp. is primarily found in both Europe and the US, but pathogenic strains have been observed on all continents with the exception of Antarctica. In the US, *Borrelia* is commonly found on the East Coast, and in the Midwest and Northwestern states, with a majority of cases concentrated in the Northeast and upper Midwest. Multiple studies have demonstrated low prevalence of the potential reservoirs and insect vectors in the Southwest US.⁴ Most human cases that occur in the Southwest are those with recent exposure in endemic regions. A statewide study of Texas and northern Mexico revealed that the farther west ticks were collected, the less *B. burgdorferi* was present as determined by molecular analysis (PCR).⁴ It is believed that the hot, arid conditions associated with the Southwest are inhospitable for known tick vectors.^{5,6}

Symptoms of Lyme disease may include the classic erythema migrans "bull's-eye" rash, fever, malaise, meningitis, headache, photophobia, and joint and muscle pain. Treatment of the acute phase of infection is effective using common antibiotics such as doxycycline, azithromycin, amoxicillin, or cefuroxime axetil. Neurologic, cardiac, and resistant cases may be treated with intravenous ceftriaxone or penicillin. In a subset of patients, chronic symptoms may occur. Treatment of these "chronic Lyme" cases is difficult and controversial in the mainstream infectious disease community.

There are several additional infections that can "hitchhike" along with *Borrelia* sp., as they share the same vectors and mode

of transmission (e.g. a tick bite).⁷ These infections include *Anaplasma*, *Ehrlichia*, and *Babesia* species. Frequently these coinfections are overlooked when Lyme disease is suspected, but can cause significant illness as well.

A number of tests have been traditionally used to diagnose Lyme disease, while more tests are in development. Could the issue in "chronic Lyme" simply be that these patients really have another Lyme coinfection that causes similar symptoms? Are there testing methods which document that the "chronic Lyme" patients are suffering from spirochetal disease? Could there be undiscovered microbes that cause a "chronic Lyme"-like disease state?

Technologies

The detection of Lyme disease and the spirochete that is responsible is performed by a clinical microbiologist using several testing techniques. These testing technologies include microscopy, culture, specific proteins, serology, metabolite detection, and molecular diagnostic technologies. It is the general consensus among microbiology and infectious disease specialists that molecular detection technology, whether protein-based assays such as electrospray mass spectrometry (ESI-MS), MALDI-TOF (matrix assisted laser desorption ionization time of flight), or DNA-based technologies, is the future of microbiology.

Currently available tests can be divided into two major categories, direct and indirect. Direct tests assay for direct evidence of the microbe. Direct tests include observation by

microscopy, culture (growing the organisms from patient samples), and molecular methods that detect an organism's unique molecular signature. Often direct testing methods are considered the "gold standard" or most convincing test method in microbiology. Indirect test methods seek to measure how the patient's body responds to a pathogen and assumes that a response is evidence of exposure. These tests include serological tests (such as immunofluorescence, western blots, or ELISA-based assays) or lymphocyte response assays. Indirect tests are less specific than direct tests; however, they may be the best diagnostic tool available for a given disease. It is widely recognized by immunologists and microbiologists that a patient's exposure to other organisms can produce similar immune responses, where the antibodies produced by the patient can cross-react with other organisms. This can cause false positive results in certain serological tests.

The CDC suggests that Lyme disease testing be performed in two steps. First a screening ELISA for *B. burgdorferi* should be performed. If the screening test is positive, then a

confirmatory western blot should be performed.

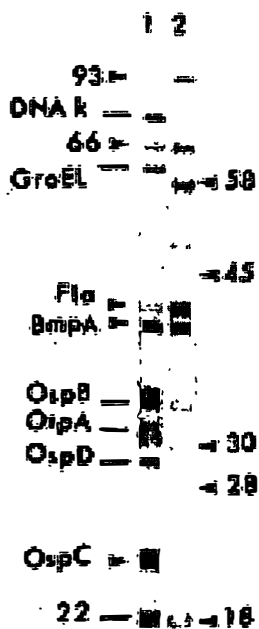
Although not all practitioners agree with them, the following are the official guidelines from the CDC website for Lyme disease testing:

1. The immunoblot should not be run without first performing an EIA or IFA.
2. The immunoblot should not be run if the EIA or IFA tests are negative.
3. A positive IgM immunoblot is only meaningful during the first 4 weeks of illness.
4. If you've been ill for longer than 4 to 6 weeks and the IgG immunoblot test is negative, it is unlikely that you have Lyme disease, even if the IgM immunoblot is positive.

Most serologic tests aim to detect antibodies produced from the body's response to an antigen or foreign protein of an infectious organism. *Borrelia* proteins, such as p41 or p23, are expressed by the organism and commonly recognized by the body's immune system as foreign. Therefore, the body will produce antibodies that recognize those specific foreign proteins. In infectious diseases, there are two relevant classes of antibodies: the IgG and IgM antibodies. The CDC

criteria for Lyme disease require 5 out of 10 bands for IgG or 2 out of 3 bands representing *Borrelia* proteins for IgM to be considered a positive result. The potential of serology cross-reactivity with other microorganisms is one of the main reasons for the band count criteria. Simply, the antibodies produced to fight a different organism that a patient was exposed to, such as *Treponema pallidum*, the spirochete that causes syphilis, can cross-react with similar proteins of *Borrelia sp.* and produce a "false positive" result.⁸

It has been documented that some individuals can have a diminished response to antigens, also known as anergy. These individuals, when tested for antibodies in any serology test would yield a negative result. Recently, the FDA has approved two new Lyme disease assays, called the C6 Lyme peptide ELISA and the Prevue B tests. These tests are more sensitive and specific than previous serology-based assays. Another available type of indirect test includes lymphocyte-based assays (e.g., ELISpot assay [Enzyme-Linked ImmunoSpot]). Although sensitive, this assay can still have nontrivial rates of cross-reactivity.



Lyme western blot



Borrelia burgdorferi spirochetes, CDC

Lyme Disease and Detection

Interestingly, the emerging species *Borrelia miyamotoi*, is not detectable by standard western blot and requires either a special serology, PCR (targeted or genuswide), or DNA sequencing for conclusive detection. This newly recognized species is thought to represent over 10% of the Lyme cases in the US and may be significantly underdiagnosed.⁹⁻¹²

In a recent study by John T. Belisle at the University of Colorado, a series of metabolic markers have been discovered in acute Lyme infections and may prove to be the foundation of a new Lyme disease assay. This experimental assay, using metabolomics, holds promise for detecting Lyme disease.¹³

An additional indirect/direct methodology is the Nanotrap Lyme Antigen test under development by Ceres Nanosciences with cooperation from George Mason University. This test measures a specific highly conserved C terminal domain of the OspA protein excreted into the urine and uses a Nanotrap particle, a polymer meshwork containing a chemical bait, to capture and concentrate the antigen.¹⁴

Testing for the molecular signature of organisms is the “gold standard” in microbiology. This direct approach includes methodologies such as polymerase chain reaction (PCR) and

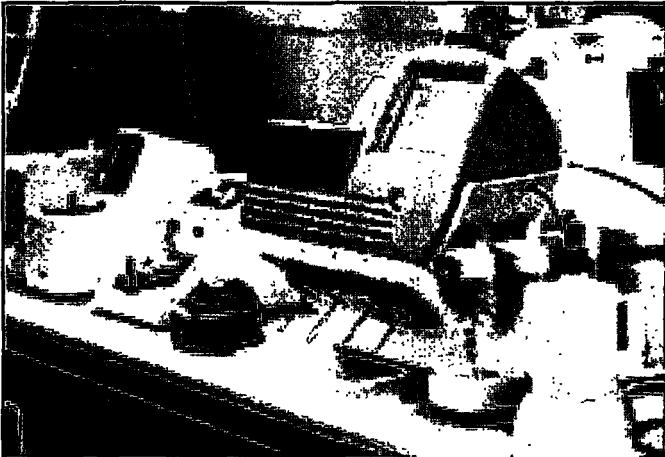
Next-Generation DNA sequencing (NGS), and is very accurate. The use of these direct testing methods will increase sensitivity and specificity and can be used to test patient samples including human blood.

PCR is a technique that amplifies specific DNA fragments. This process can be accurate and incredibly rapid. Multiplex PCR is a technique wherein DNA can be detected from multiple organisms simultaneously. The multiplex PCR approach, which is currently used for microbial screening, may be replaced by NGS technologies in the future.

There are two newly developed systems for broad characterization of microbial populations. The Abbot IRIDICA system uses a preliminary PCR step to amplify the DNA of the organisms. It then uses mass spectroscopy electron spray ionization for identification.¹⁵⁻¹⁷ This methodology is similar to the recently approved MALDI-TOF systems, but identifies a PCR product instead of protein markers.^{15,18} These systems, while rapid and having a significantly broader range of detection than currently available tests, are limited by the organisms included in the electrospray ionization database.

The use of DNA sequencing technologies is becoming more common in laboratories. A number

of laboratories are developing these systems by adapting technologies that are currently used to sequence human genes. These platforms allow for a “metagenomics-based” approach. Metagenomics is the ability to characterize a population of organisms in a nonexclusive way. This approach involves amplification of DNA directly from samples that can be sequenced using a variety of sequencing instruments. A sequencer of note is the IonTorrent PGM, which relies upon a microchip for DNA sequencing.¹⁹⁻²¹ These sequencing systems must successfully combine biochemistry, microchip or laser imaging, and computational power to assist in detection and identification of organisms. This type of technology may address many of the outstanding questions in infectious disease research. The final figure shows the mapping of a reference strain of *Borrelia burgdorferi* from our laboratory. Sequencing has the ability to detect and characterize previously unknown organisms because of its nonexclusionary approach. In addition, these platforms allow for the characterization of mixed populations of organisms in a sample. Our laboratory is currently involved in five independent review board (IRB)-approved studies, some of which are completed and pending publication.



IonTorrent PGM Sequencer

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B. Burgdorferi sequence results, Fry Laboratories

Lyme Disease and Detection

Additionally, we have recently published a report outlining our diagnostic approach utilizing NGS technologies.²²

Summary

Borrelia species that cause Lyme disease and other illnesses are an important and increasingly significant health care concern in the US and worldwide. There are a variety of tests each with its own unique pros and cons that may be used to diagnose disease. The detection of Lyme disease in the mainstream medical community currently relies on the two-tiered approach as defined by the CDC, a positive serologic ELISA test followed by a western blot. Improved use of advanced serologic assays, microscopy, and culture methods may assist in the timely and accurate diagnosis in suspect cases. However, molecular methods hold great promise, including NGS, which may elucidate the causal microbial population of the chronically ill.

Acknowledgements

I would like to thank Drs. Jeremy Ellis, Dara Missan, and Lance Liotta for their kind editorial assistance.

Disclosures

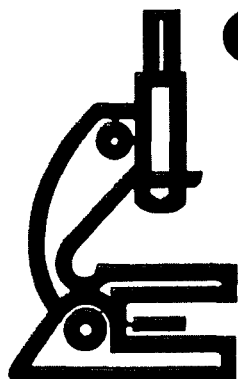
Dr. Fry is the founder of Fry Laboratories LLC and coinventor of a Next Gen sequencing system RIDI for the detection of existing and novel virus, bacteria, fungi, and protozoa. He is a primary care physician and has a BS in microbiology and MS in molecular biology. He is a member of the American Society of Microbiology and the International Lyme and Associated Diseases Society.

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Dialogue: An Overview of Lyme Testing and Treatment

Steven Harris, MD, and Mischa Grieder, ND

an interview with Nancy Faass, MSW, MPH

Context

There have been scarcely two generations of medical practitioners and researchers in the scientific effort to decipher Lyme disease. At present, the CDC estimates 300,000 new cases are infected in the United States alone, every year. For the past twenty years the number had been listed at 30,000 cases with admitted underestimates by at least a factor of ten.¹ In the United States, this may amount to six million people infected in the last twenty years. In other parts of the world, prevalence is in unknown numbers, but Lyme is documented to now be endemic in Scandinavia, Russia, Nepal, China, Japan, Australia, New Zealand, South America, Mexico, Canada, Scotland, England, France, Germany, and Eastern Europe.

A recent study by Johns Hopkins showed 65% of patients still have Lyme symptoms after treatment with standard therapies.² This leaves millions of patients diagnosed with Lyme disease still with symptoms. Many of the patients with Lyme disease or post-treatment Lyme syndrome (PTLS) are remarkably ill. Most are quite proficient in the state of their knowledge regarding Lyme treatments and are desperate for any progress, evidence-based or not. How do we approach these patients? What about the patients who have Lyme disease, but have not been diagnosed?

Diagnostic Considerations

Dr. Harris: In the diagnosis of Lyme, we want as much hard evidence as possible in order to have statistical probability that we will be able to help the patient – to have a reasonable assurance that the clinical diagnosis is

supported by high-quality scientific evidence. There are also medical and legal issues, and we want to maximize the possibility that our patients will be reimbursed by insurance.

Dr. Mischa Grieder: With the instant availability of content online, we now have access to an enormous amount of information on emerging treatments. Everyone learns very quickly of new developments, right down to specific herbs, herbalists, and protocol. We know those. What I believe is of interest for us as practitioners is to get a better grasp on how we organize and use those resources – how we integrate that information into a meaningful treatment plan for each specific patient.

Ms. Nancy Faass: If you think of this as a multiple matrix like a hologram, it's a chessboard in three or four dimensions. You have the individual genetics, their phenotype, all these infectious agents (and all the associated commensals and pathogens in the human microbiome). Then there are treatment successes and treatment failures, plus the patients' economics and what they bring to the treatment. How do you organize all that in an individual patient?

Dr. Harris: We're hopefully thinking about all of that all the time: we're thinking about the bugs, we're thinking about the patients' nutritional status, their phenotype or epigenetics, their detox pathways, and environmental factors (toxins, metals, etc.). We're thinking about their travel history and the risk of parasites and infections, including viruses. We're thinking about their psychological and emotional state, their history of interpersonal

Table 1. Differential Diagnosis: Selected Evaluations*

*This list will be determined by the patient's history, health status, and presenting symptoms.

<ul style="list-style-type: none"> • Thyroid disease • Parathyroid disease • Autoimmune disorders • Rheumatic fever • West Nile virus • Histoplasmosis • Invasive fungal species and potential fungal or mold sensitivity or reactivity • Other bacterial infections 	<ul style="list-style-type: none"> • Amyloidosis • Sarcoidosis • Syphilis • Guillain-Barre • Glycogen storage disorders • Neurofibromatosis • Mitochondrial disorders • Bone, blood, carcinoid tumors • Heavy metal toxicity 	<p>The following can coexist with Lyme as coinfections:</p> <ul style="list-style-type: none"> • Human herpes virus 6 • Epstein-Barr virus • Cytomegalovirus • Mycoplasma pneumoniae • Chlamydia pneumoniae • Candida spp. • Coxsackie virus • Relapsing fever borreliosis
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Table 1. Differential Diagnosis © Steven Harris, MD 2015.

relationships, and any history of abuse, as well as their health history, birth history, and past experience with medications, trauma, and surgeries. As practitioners we consider all these factors, and we internalize them, regarding them as a gestalt in relationship to that patient to create a meaningful plan.

Differential Diagnosis

Dr. Harris: I think we should include differential diagnoses in the work-ups for Lyme patients. Standard tests need to be done. Lyme testing needs to be done, and it should not all be done based on non-validated tests. There still needs to be a strong scientific approach.

We can employ a three-tiered paradigm of testing and treatment for Lyme disease and other infectious disorders (such as Dr. Horowitz' MSIDS, multiple systemic infectious disease syndrome): this could be described as

a hard-evidence model, a soft-evidence model, and an alternative model. The hard evidence includes prospective, randomized trials, clear causality, and a well-defined endpoint. Whenever we can use this approach, I feel that this should be a goal. There are many patients with Lyme who do not utilize or adhere to integrative medicine, who are not going to be reached if hard scientific evidence is not the basis of their diagnostic and treatment approach. In addition, whenever we can develop cogent research studies and treatment approaches to raise the state of the science, more universal acceptance of the disease is probable.

Dr. Grieder: Although we're going to use all the hard evidence we have, we also need to use data points that are less well-established. This is a multifaceted approach. The solution is going to require deepening our investigation and our understanding of the multiple layers of the disease process – and then integrating that into clinical practice.

Table 2. Approaches to Diagnosis and Treatment

Mainstream Diagnosis	Lyme-Literate Diagnosis
MDs, DOs	MDs, DOs, Integrative doctors, naturopathic doctors, functional medicine doctors
Two-tiered approach: strict testing criteria – a negative test defines the absence of disease; IFA or ELISA and WB; clinical diagnosis only if bulls-eye rash present after a tick bite or 2 of 3 bands on IgM or 5 of 10 bands on IgG	Testing criteria open to broader but evidence-based criteria. Clinical diagnosis is paramount—a negative test does not rule out disease. Screening ELISA not very useful; IFA or ELISA not necessary; bulls-eye rash not necessary. WB 2+ bands on IgM (inc. 23, 34, 39, 31 +/- 31, 83-93); or 2+ bands on IgG or PCR in blood or tissue, or signs and symptoms consistent with Lyme with likely exposure to ticks, even in the absence of a positive test after other disorders have been ruled out
Primarily relies on validated laboratory data, history, physical evidence, statistics, differential diagnoses, and interpretation	Primarily relies on validated laboratory data, history, physical evidence, statistics, differential diagnoses, and interpretation
	Integrative Approaches
	Use laboratory data, history, physical evidence, statistics, differential diagnoses, environmental factors, subtle nutritional status, detoxification capacity, and interpretation May utilize tests that do not have the rigor to meet certain standards of care. Certain PCR tests that use less than 10 BP for primers; some Lyme culture tests; lymphocyte transformation tests; over-sensitized ELISA/IFA tests; quantitative PCR test; dark field microscopy; some other direct visualization tests Some ancillary tests to address health status include: salivary hormone testing, nutritional profiles, provoked metal testing, stool health tests, organic acids testing, methylation testing, kryptopyrrole testing, MELISA testing, CD57 testing, IgG allergy testing, autoimmune testing, urine amino acids testing, hair testing, saliva/stool antigen testing, and mycotoxin testing
Mainstream Treatment	Lyme-Literate Treatment
Treat for 10-28 days with doxycycline 100 mg bid or amoxicillin 500 mg tid. For severe cases (cardiac or neurologic), penicillin G or ceftriaxone 2 g qd x 30 days. Consider co-infections and treat as necessary: human granulocytic anaplasmosis, human monocytic ehrlichiosis, babesiosis Retreatment is not necessary. Scant mention of adjunctive testing and other complex management	Treatment is based on clinical response. Treat with one or more antibiotic combination as long as clinical picture is consistent with active infections. Consider co-infections and treat as necessary for anaplasmosis, ehrlichiosis, or infections due to babesia, mycoplasma, bartonella spp., relapsing fever borrelia, rickettsia spp. Retreatment may be necessary; some adjunctive testing such as co-infections testing is important; management of other aspects of health may be related and necessary.
	Integrative Therapies
	Use of non-pharmaceutical regimens; herbal combinations; herbs in concert with pharmaceuticals; probiotics; timing of herbs, meds, probiotics, minerals; suppositories, enemas, transdermals, sublinguals; supplements to enhance detoxification; lifestyle choices to increase function of chemical treatments; saunas, heat, exercise; pulsing of medications and herbals. Use of some studied adjunctive methods such as oxidative therapies—H2O2, ozone, ultraviolet irradiation; use of intravenous herbal remedies; of hyperbaric oxygen in concert with antibiotics; of acupuncture, massage, colon hydrotherapy, meditation, or fasting

Table 2. Approaches to Diagnosis and Treatment. © Steven Harris, MD, 2015.

Lyme Testing and Treatment

► Jerry Stine: Guest commentary: Lyme initially has the appearance of a straightforward infectious disease, but in practice, it often does not respond in that way. In mainstream medicine, a standard approach is to render down the treatment into identifiable steps in diagnosis, treatment, and follow-up. Lyme does not consistently respond in that fashion. As a result, practitioners dealing with this on an ongoing basis are compelled to broaden their focus to see what other resources and issues need to be brought to bear that may have some relevance to the patient. This doesn't mean abandoning traditional, medical evaluative and treatment approaches, but for the sake of patients with persistent symptoms, practitioners have to be open to other kinds of analytical assessments to develop an effective treatment plan.

In the evaluation of possible Lyme infection, there are levels of diagnostic approach and those start with commonly recognized lab tests. Depending upon what's learned and how well people respond, a practitioner may be pushed into looking at other considerations beyond that. It is not that the data lacks rigor or specificity; rather it is a response to the demands of the case, based on the patient's health status and the outcome of treatment. Information the provider learns from the initial round of tests and interventions moves the clinical process to the next phase of therapy. If those work, that is cause for celebration. If they do not, then the investigation goes to the next level. The table describes considerations and steps at those various levels.

Genetic Testing

Ms. Faass: Do either of you use genetics and do you see any subsets within the genetics. Dr. Grieder, you have mentioned 23andMe.com.

Dr. Grieder: I use methylation panels to help support the process, but I haven't seen that approach work by itself alone – genetic information is a useful tool in fine tuning treatment approaches, but genetic data alone does not define the pathophysiological process. We should be reminded of the old adage "treat the patient, not the paper," which is also emphasized by methylation pioneers such as Ben Lynch and Paul Anderson.

DNA methylation patterns that act as a bridge between genotype and phenotype are highly responsive to environmental and extracellular data. In the future, when we understand the causal relationships between environment and DNA methylation better, we may be able to be more targeted with our therapies. Until then, we have the patient and their phenotypic expression right before us, so we can learn from those patterns, because they give us hints as to the underlying processes.

Dr. Harris: I agree. We're certainly looking at the MTHFR gene and some of the methylation panels, the more

complex ones such as those featured on the HolisticHeal.com website of Amy Yasko's work. They do help guide us as to how aggressively we can treat and how much we need to consider detox, and bypass certain enzyme mutations.

Treatment

Diagnostic Trials

Dr. Harris: Many times the diagnosis will trail the treatment. It's not very elegant, but the strategy is to do empirical treatment, and then see how the body responds. Ultimately it comes down to a risk-benefit analysis. What is the risk of using a certain treatment? What is the potential benefit? And what is the likelihood that the patient actually has that condition. Sometimes you don't know the full answer until you have initiated controlled empirical treatment and assessed response. For example, the tests for bartonella spp. miss more than half of the likely cases. When a patient presents with hallmark symptoms of bartonellosis, using a low dosage of a botanical formula such as A-BART, BLT, Bar 1, or Bar 2, or a medication such as Septra or Rifampin can really clarify the diagnosis.

Dr. Grieder: When I start seeing a patient, I have an initial set of information, and I begin initial treatment. The next time the patient comes back, there is a specific response that I'm going to compare with the patient's prior health status before the initial treatment. That response can give me distinct pieces of information. At that point, my goal is to determine which issue(s) are most on the surface. That way I can begin to move forward into increasingly complex processes, but always maintaining a sense of direction by looking at what issue is most on the surface and then focusing deeper as we go through the treatment process. We need to approach the Lyme by looking at the individual before us, using the information we have available. We also want to remember that herbs and antimicrobials and support therapies and detox therapies all have interactions. We can't just use individual data points – we also need to look at the bigger picture.

Dr. Harris: I agree. This is the way the most successful practitioners engage patients who have these very complex conditions – by gathering information, initiating treatment, keeping the patient safe, and delicately advancing treatment. The key is to make something happen, watch the response, and based on that response, make something else happen. It's a slow, steady process with periodic dips in the road.

At the same time, we're still gathering hard evidence to accumulate as much data as possible, because we need to know the underlying dynamics: How is liver function, how is kidney function? Which infections are testing positive, and is that in DNA testing or antibody testing? What's going on with the immune system? We need to accumulate all this data, but the data still don't tell us the treatment priorities at that particular moment. We have a multitude of treatments that might address the various problems, but how do we triage – in what order do we sequence them? There is no

single test that we can use. That's why some people shift to alternative approaches, such as bioenergetics, applied kinesiology, or some other intuitive approach.

Multifactorial Treatment

Dr. Harris: Many of us compare the process of treating Lyme to peeling an onion. From this perspective, the layer on the surface is different for each patient. Uncovering those layers and determining which one to treat is also a very personal process. Sometimes we might find for example, that helminths are the primary issue, and that parasites are at a deeper layer, but that *Borrelia burgdorferi* is at the core. There may be numerous ways to get into the so called core, but choosing the correct layer to address in sequence may hasten the recovery. Each practitioner, through his or her experience, openness, and pure intention to help is going to find ways to get into those layers.

Dr. Grieder: I begin by identifying what is most on the surface. There are many very subtle symptom pictures associated with various aspects of molds, the coinfections, environmental toxins, and even the psychospiritual/psychoemotional aspects of the disease. Depending on what comes up in the visit, that is what will be addressed in the coming weeks. That way we narrow down the issues to set priorities.

Dr. Harris: I essentially take a by-any-means-necessary/safe-approach. I have several patients on IV antibiotics, on multiple IV antibiotics, and on IV antibiotics combined with oral antibiotics. I pulse certain antibiotic regimens, or use antibiotics with herbs, I use numerous homeopathic blends, homeopathic nosodes, and also combination remedies, as well as immune supportive agents. For certain types of infections such as babesia, I'll stack one treatment on top of another on top of another. For other infections such as bartonella, I'll rotate drugs fairly rapidly. With Lyme, I might pulse the treatment at one point, then become more consistent, and then make a surgical strike with heavy antibiotics at another time. If it seems that there is a predominance of one symptom complex reflecting specific disease activity over another, that is the one I am usually going to treat the most assertively at that time. I do think it is difficult to treat Lyme and all the infections simultaneously, so there is often a rotation of treatments. I treat fairly aggressively to knock the bugs out; at the same time, I try to keep the body as safe as possible, and protect the gut. I also address other issues such as molds, toxins, heavy metals, and hormonal imbalances at the same time.

Ms. Faass: Are these patterns that you carry in your own intuitive knowledge base, that you've defined or codified? Has anyone codified them and made them available to other providers, for example as decision trees? Is there consensus on either genetics or symptom patterns in subsets of patients, and do other providers find them relevant?

Dr. Harris: I think there are definitely decision trees. If you look at the work of Klinghardt, he has a multilayered approach and often does things in a certain order. Some

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people will test for mold, and will try to address mold issues before they deal with some of the other infections. Many of us will try to detox patients before we kill more bugs and create more inflammation. Other providers will just go in and kill the bugs, straight away. Some will make sure that the patient is nutritionally replete before detox. Others will try to drain toxins before they detox more, rather than just move the toxins around, and all this before they kill organisms. We may determine that we want to treat coinfections first, or viruses first, or parasites first. Many of us create models that seem to work in a certain order. I do think the treatment plan needs to be individualized. In my experience over the last 15 years, I have not found a cookbook approach that works across the board. There has to be openness.

Ms. Faass: How often do you see people and how do you organize all this information so you can keep track of where you are with that individual and so that they don't get into trouble?

Dr. Harris: We try to see patients every four to eight weeks. There are obviously patients who are out of the country and out of town that we can't see that frequently, so we may have to talk on the phone or skype. All our patients are generally required to have a primary care doctor as well, to help them address issues that come up day-to-day, especially if they're out of town. We measure liver function and blood count on an ongoing basis and give patients information on the side effects and risks of taking certain medications. It takes time to sort out when someone is having a die-off or Herxheimer reaction or a side effect from a medication, or whether the disease itself is getting worse, or if another infection is becoming predominant, coming up as the "top layer of the onion." Essentially all Lyme-literate practitioners track those types of issues.

Inflammation and Pain

Dr. Grieder: Everyone working in this field tends to develop a particular focus, based on their insight and the patterns they see. My own recent interest has been a focus on the inflammatory processes that often underlie the manifestations Lyme patients experience, which can be a major factor in pain and cognitive dysfunction. One of my interests is how we strengthen and stimulate the immune system, and at the same time downregulate it to normalize immune function and reduce proinflammatory damage. We have some herbal tools: curcumin is well studied and can reduce cytokine expression. Fish oil has an effect on prostaglandins and can be used to attenuate the immune system without suppressing it as steroids tend to do.

When a patient comes in with pain, there is a tremendous amount of information we can learn from that pain. Is it neurologic or inflammatory pain, for example? Does it come from a specific location in the body or is it a

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➤ more generalized pattern? This information will lead to a broader range of choices – choices that are more interesting and potentially more successful than simply looking at biochemical or pharmaceutical aspects of pain.

Low-Dose Immunotherapy Therapy

Dr. Grieder: I've also been interested in LDI, based on an allergy treatment (LDA or EPD), that was originally brought to the U.S. by Dr. Butch Shrader. Over the past three years, Dr. Ty Vincent in Alaska has been looking at an approach using extremely low-doses of antigen groups in combination with beta glucuronidase – an enzyme abundant in the human gut that appears to act as an immune attenuate without suppressing normal immune responses. Lyme involves chronic infection but also over the years, the potential for developing immune dysfunction. Low-dose immunotherapy appears to address immune-dysregulatory aspects of the Lyme disease complex.

Ms. Faass: What do you see when you use this approach with your patients—do you see a subset of people who do better? Is there any pattern to the people who are more responsive with this approach?

Dr. Grieder: As with any treatment, often there will be a subset of people who respond better. In this case it tends to be those who are experiencing true immune regulatory dysfunction. When I find the correct dose, the optimal concentration of the antigen with the enzyme, within a matter of hours or days patients have reduction in pain or inflammation, and they begin to sleep well.

Some patients say they awaken the next morning feeling as if a switch had been flipped. They wake up and feel normal. I'm still exploring how to develop this as a lasting treatment. Currently there are certain rules associated with its use: until recently we believed that we could not use it more often than every seven weeks because of the eight-week life cycle of white blood cells; the logic was that giving shots too frequent could trigger a symptom flare, so we had to wait for a new generation of cells. However, we are now finding that there is a way to speed up that process dramatically as long as we don't exceed a certain total antigenic load. This is exciting, because as we all know, it

has always been difficult to find a way to sustainably calm inflammatory processes.

Ms. Faass: In terms of linking these outcomes to a subset of patients, is there any hard data on markers that might correlate with that improvement, such as elevated CD-57 or CRP or others?

Dr. Grieder: With that treatment, we don't yet have additional specific datasets available, but I would expect elevated C4a complements to normalize. In terms of chronic fatigue, for example, a study just published by Lipkin and Montoya³ showed that certain cytokines are elevated and others are depressed in the presence of chronic fatigue, but these patterns change over time, and it does not occur equally in all patients. One set of studies doesn't fit all properties. There are Lyme patients, for example, who have immune dysregulation, but others who do not.

Psychoneuroimmunology

Dr. Grieder: There is also the psychoemotional aspect of the condition. It is well established that the inflammation associated with Lyme disease can affect the CNS profoundly and cause neuro-psychiatric symptoms such as depression, anxiety, and cognitive impairment. There is another related aspect that tends to be underestimated and that is the psychological trauma that develops from having to live with an often debilitating and poorly understood disease. Many of the patients that I see have been sick for many, many years and therefore have seen a number of doctors. Shuffling from one specialist to the next, getting frequent dismissals, and then landing in the psychiatrist's office is traumatizing in itself. That creates a dynamic that makes the treatment process more complicated. Yet we can't simply recommend a psychotherapist in the first visit, because the patient may feel we are suggesting that the disease is "all in their head."

Dr. Harris: We can't just kill the bugs. We can't just offload the toxins. We also have to address all the psychoneuroimmunological factors that have been at play. You can use whatever methodology you want. Engaging with deep psychological processes is one of the key tenets in achieving wellness. It doesn't have to be our Western model of psychotherapy. There are a thousand other ways to engage the mental, emotional, psychological, and spiritual aspects of the patient.

Dr. Grieder: We want to think carefully about how and when we introduce this idea to the patient, in a delicate dance to avoid another layer of trauma. Psychotherapy, meditation, yoga, weekend retreats, EFT, or shamanism from various cultures can all be effective, depending on the patient. The beauty of this is that as human beings, we are multifactorial organisms, so there are many ways to treat. Finding an underlying process that fits that one particular patient is the key to treatment.

Dr. Harris: As the second tier of Lyme practitioners, we are standing on the shoulders of giants – the people

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who did the early work – physicians such as Burrascano, Drulle, Bach, Jones, Horowitz, and Liegner, to name a few of the integrative and naturopathic doctors who were early adopters. There is a great deal of communication between the naturopathic and integrative physicians and some of the scientists and researchers. Were it not for that foundation, we would not be able to do what we do.

Steven Harris, MD

Dr. Harris has been in private practice since 2001 and established Pacific Frontier Medical, Inc., a California medical corporation, in 2006. His work focuses on the diagnosis and treatment of chronic and persistent Lyme disease and other tick-borne co-infections, incorporating strategies from conventional, functional, and complementary medicine. Dr. Harris has taken a leadership role in lymedisease.org (formerly CALDA – the California Lyme Disease Association), a group focused on research and patient advocacy that has been largely responsible for spearheading favorable legislation, protecting patient rights, expanding Lyme disease awareness, and fostering continued public health education. He is also an active member of ILADS (the International Lyme and Associated Diseases Society), a professional medical society of physicians and scientists that has become the authority on effective treatment for chronic Lyme disease. Dr. Harris is a consulting associate professor at Stanford University School of Medicine.

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Dr. Grieder received his medical training at Bastyr University in Seattle and practices at the San Francisco Preventive Medical Center in collaboration with Paul Lynn, MD. Dr. Grieder is a member of the American Association of Naturopathic Physicians and the California Naturopathic Doctor Association and serves as an adjunct professor at the American College for Traditional Chinese Medicine in San Francisco. Lyme disease is a primary interest and specialty of Dr. Grieder, and he has worked collaboratively with Lyme-literate physicians that include Steven Harris, MD, Christina Green, MD, and Raphael Stricker, MD. Born and raised near Zurich, Switzerland, Dr. Grieder experienced a holistic lifestyle and the benefits of natural medicine early on. Frequent trips to Switzerland enable him to draw from a wealth of European research and leading-edge expertise in health care. He also regularly participates in conferences on the rapidly emerging findings in Lyme disease and associated complex chronic diseases.

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Editorial

This article is based on an interview and the writings of Steven Harris, MD, and Mischa Grieder, ND. Editorial services were provided by Nancy Faass, MSW, MPH, a writer and editor in San Francisco who has worked on more than 40 books for publishers that include Elsevier, Harper, McGraw-Hill, New World Library, and others. Director of the Writers' Group, she also provides articles, white papers, and writing for the Web and can be reached at info@HealthWritersGroup.com.

Thanks to Jerry Stine, NC, for insightful commentary. He can be reached at Lifespan Institute, 415-883-9033.

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Ozone Therapy Used Instead of Antibiotics for Microbiome Restorative Therapy Yields Successful Outcomes for Dogs and Cats with Fecal Transplants

by Margo Roman, DVM, CVA, COT, CPT

Abstract

Microbiome restorative therapy (MBRT) can improve a range of gastrointestinal-related health issues such as irritable bowel disease, diarrhea, GI infections such as *Giardia* and *Clostridium perfringens*, skin disorders, and aggressive disposition in cats and dogs. MBRT can be administered by oral or enema fecal transplant from a healthy donor. Using ozone therapy as a rectal insufflation along with subcutaneous saline with O₃ is important in reducing the colon biofilm. Prebiotics, probiotics, and gastrointestinal gut support have always been a part of our clinic's care, but the addition of MBRT speeded the recovery remarkably and kept the balance by adding hundreds of species to the gut flora. Animals who had already tried probiotics and diet change for months without complete improvement reverted from positive *Giardia* and *Clostridium perfringens* to negative fecal tests within 5 days after the MBRT. Including ozone is crucial for overall healing and success of MBRT treatments.

Introduction

The importance of the microbiome endogenous to the gastrointestinal tracts of all mammals is becoming increasingly apparent. It is now known that the human body relies on over 100 trillion microbes of over 500 species of microorganisms.^{1,2} Based on this abundance and variety of microbial life, it has been estimated that 75% to 85% of the human immune system depends upon the gut microbiome, and imbalance in the normal symbioses of these species could lead to immune system failure resulting in disease, cancer, or mental health disruption.² Currently, probiotics are used to support gut health, but these provide only between 1 and 20 species of microorganisms – a small subset of the actual microbial diversity.

Microbiome restorative therapy (MBRT) offers a natural and effective way to improve a variety of gastrointestinal-related health problems that are difficult or impossible to treat using standard methods. The author coined the term *microbiome restorative therapy* in 2012 to highlight the need for restorative support for the microbiome. The previous term, *fecal microbiota transplant*, lacked a focus on the restorative nature of the treatment and focused on the word fecal, which is a distasteful term to use when talking to clients.

In recent years, MBRT has been increasingly used as a successful treatment option in human mainstream medicine, including at hospitals such as Massachusetts General in Boston, the Mayo Clinic, Cedar Sinai in LA, and Sinai Hospital at Johns Hopkins.³ Research on this promising therapy in humans is not yet abundant, but it is growing along with research initiated by the Human Microbiome Project (HMP), a NIH program, with millions of dollars being spent to identify the human microbiome.

In the natural world, many species of wild animals and even domesticated farm animals will eat the feces of other individuals. Among canines and some felines in the wild, this behavior, coprophagia, is normal. After a dog or cat kills its prey, it first eats the visceral organs of the abdomen, thus ingesting the prey's microbial flora along with fibrous digested plant matter. Many pet dogs will eagerly eat the feces of other animals when the opportunity arises. However, coprophagia is generally frowned upon and disallowed by pet owners in developed areas of the world.

Ozone has been used as a rectal insufflation for years, and many think that this procedure alone can correct irritable bowel and other GI issues. But how does one reestablish a microbiome that could have been destroyed by overuse of antibiotics, chemicals, toxins, preservatives, chlorine, fluoride,

GMOs/glyphosate, and other insults? Standard fecal transplants require several courses of antibiotics to reduce bacteria in the gut prior to transplant, but would ozone treatments be a better way to prepare the gut than adding the effects of even more antibiotics? We have found this to be the case.

It is logical to assume that the most efficient way to restore the microbial load and therefore gastrointestinal function to a human or animal patient with deficient gastrointestinal flora would be transferring fresh feces from a human or animal donor with a healthy gut microbiome. In fact, veterinarians and veterinary students have been doing rumen transfers and pig feces transfers for at least 4 decades.⁴ The immune system uses the body's innate mechanisms to utilize newly introduced microbes to restore symbioses that enable systems and tissues to heal.

In humans, studies have shown that frequent use of antibiotics disrupts the natural gut flora.⁵ One study, for example, finds that the rate of irritable bowel disease among children has doubled over the last decade as a result of frequent antibiotic usage.⁶ As cases of gastrointestinal problems, including irritable bowel syndrome (IBS) and inflammatory bowel disease (IBD), have skyrocketed at the same time that the quality of diets and environmental exposures have become more compromised, one can infer that individuals with healthy biomes would be relatively free from serious health issues, have had low exposure to chemicals (such as pesticides, pharmaceuticals, or water with added chlorine or fluoride), eat a raw diet, and spend ample time outdoors in a natural environment.

Studies have shown that both oral and rectal transfer of fecal material in humans has reintroduced a balanced gastrointestinal microbiome and decreased *Clostridium difficile* overgrowth.^{7,8} Early evidence points to microbiome-restorative therapies as key to future treatments for gastrointestinal and immune system problems in mammals.⁹

If the gut microbiome, which has hundreds of species of microbes

in balance, creates 75% to 85% of the immune system, can nutritional approaches alone replenish and reestablish the balance of this biome in an animal? The microbiome can be supported by good nutrition, but without all the symbiotic species we may never be able to balance the health of an individual.

No one has a definitive answer yet, but the knowledge of the numerous possibilities that exist to improve overall health that begin with a healthy gut should challenge all of us.

At my Massachusetts clinic, Main Street Animal Services of Hopkinton (MASH), we have been successfully using progressive nutritional approaches for 30 years. We had always told clients that it would probably take about six weeks for a significant turnaround in the health of their pets. Now, having performed more than 800 MBRTs on dogs and cats, we are consistently seeing significant reversals in health conditions in as few as two to three days. The results have been so positive that we want to share our methods and cases and encourage other veterinarians to consider MBRT in combination with ozone therapy.

Methodology

Appropriate fecal donors have minimal exposure to chemicals, ranging from fluoride and chlorine in water supplies to chemical fertilizers or disinfectants used around the home. The suggestions that we have are ideal and we believe that any practitioner should strive to attain a naturally healthy donor that comes as close as possible to that ideal. We only use donors that are minimally vaccinated with vaccines that do not contain mercury preservations and aluminum hydroxide. We use titers for protection, as well as natural pesticides instead of chemical flea and tick spot-on or oral compounds. Before donating, we give donors fecal parasite exams and screen them with Plechner's Endocrine Immune Evaluation to check for normal immunoglobulin A, G, and M levels.⁹ Periodic PCR evaluations of the donors' feces also provide information about the gut composition. We

believe that a raw meat diet free from antibiotics and also full of organic pureed vegetables and organic non-genetically modified grains and seeds such as oats, millet, and quinoa is important. We think that it is essential that donors not have taken antibiotics, nonsteroidal anti-inflammatory drugs, and other gastrointestinal suppressive medication, at least within the past several years, but optimally never during their lifetimes. There is a real concern that once the microbiome has been exposed to a course of antibiotics or strong chemicals, many species can be eliminated from the balance forever, since they don't spontaneously create themselves. Additionally, we look for donors that are physically active and raised with extensive time in outdoor natural environments. Optimal donors will be third-generation animals raised naturally according to these criteria.

The fecal sample for donation is simply collected from the yard or litter box soon after defecation, making sure that we know from which donor the sample is produced. It can also be manually extracted directly from the donor, but for humane reasons we prefer the first technique. We use a litter that does not have any odor-neutralizing antimicrobials which could affect the sample. Samples from multiple generations of dogs can be mixed to capture the experienced microbiome of an older dog and that of a young animal with all the natural growth hormones and youthful biome vigor.

We use four methods to administer MBRT: (1) a piece of stool given directly, orally; (2) stool mixed with saline and placed in capsules and given orally; (3) stool mixed with saline in plastic cups, filtered via a sieve and given rectally; and (4) stool mixed in saline in a small personal blender and given rectally. To thoroughly implant the biome throughout the entire gastrointestinal system, oral administration is the most effective, as it is more likely to repopulate the upper part of the GI system. However, going through gastric secretions may compromise some of the biome. Therefore,

➤

Ozone Therapy

➤ additionally doing a rectal infusion may ensure that more biome is transferred. Also, it is usually easier to administer the treatment rectally to animals that resist oral administration. The ozone therapy is done before the MBRT is started. Subcutaneous ozonated saline is given first and then rectal insufflation with O₃ gas (see Table 1 for amounts and gamma.) We retain the ozone within the colon for about 5 minutes by tucking the tail. We then allow the dog or cat to defecate. At that point we introduce the MBRT.

1. The first method of administering MBRT is to give it orally in the form of a small feces ball pushed to the back of the throat of the recipient animal. We give the patient a freeze-dried raw treat before and after the feces. We sometimes conceal the fecal material in raw ground meat to encourage the patient to take the donor feces.
2. We sometimes mix the fecal material with a small amount of normal saline which we place into capsules to administer orally. We do not prefer this method, because the capsules dissolve very rapidly.
3. We also administer fecal material in an enema. A "three-cup method" can be used where no blender is available. When using the three-cup method, one cup is used to mix the saline and feces, one cup is pierced through the bottom in multiple places to make a sieve, and one is used to collect the mixture filtered through the sieve cup. The filtered slurry is administered through a syringe.
4. We liquefy the fecal material with saline in a small blender for maximizing utilization of the stool biome. To blend, 15+ grams or 1+ tablespoons of the fecal sample are combined with the saline enough to make a liquid slurry, and then strained. The mixture is blended as little as possible to avoid heating. Then we administer in an enema. Some of that liquid can also be given as an oral slurry with a syringe.

We coined the term *microbiome mixology* (MBM) as a way of combining feces from multiple dogs

to enhance the possible responses. These combinations are done to give the natural hormones of an intact male to a male, and that of a female to a female. For dogs with cancer, we use the feces of Geneva, who has survived breast cancer for more than 5 years, and her grandson or granddaughter who are puppies, and can offer youthful vigor. These puppies have been exposed to Geneva's biome, as well as their mother's, so they hopefully share a similar group of species.

Once the fecal mixture is created, it is loaded into a syringe. Before the fecal enema is given, a syringe of ozone/oxygen gas at 80 gamma is administered to the colon through a catheter. We use a natural lubricant with natural aloe and no antimicrobial properties in order to remove or impact the biofilm. Removing the colon biofilm with ozone gas and introducing more oxygen into the digestive tract increases the success of the fecal transfer. The catheter is removed and the patient's tail is tucked and held for 5 minutes to prevent leaking. The patient is taken outside to see if it is able to defecate to remove any of its own feces after the rectal ozone insufflations. To give the enema, a catheter lubricated with natural aloe is inserted into the colon. After administration, the tail of the patient is again tucked for 5 minutes and the abdomen of the pet is massaged to help the enema reach as much of the descending colon as possible. For at least 4 hours posttreatment – the longer the better – patients are prevented from defecating.

Discussion and Conclusion

Our patients have been treated with MBRT for a variety of diseases and clinical symptoms. The overwhelming majority of our patients show improvement in a short amount of time.

To increase the success of the fecal transfer, the patients' gut flora is primed with digestive enzymes, probiotics, whole food glandulars, and raw meat diets. This helps establish a hospitable environment for the new biome. The example that I give to my

clients is, "If I asked you to move into a concrete apartment with a concrete bed and no carpets or amenities of life, would you want to live there and want to stay? Adding GI-supportive nutrition gives the transplanted microbiome all the comforts of home, which will create an environment in which the microbiome will want to revivify and thrive." Some patients are also supported with additional antioxidants, organic spirulina, ultrafatty acids, and glandulars. Additionally, patients receiving enemas have their colons primed with ozone to degrade the biofilm that could reduce the uptake of the transferred microorganisms. Transferring the donor microbiomes into primed patient digestive systems seems to better allow the new microbes to survive and promote normal symbioses in the patients' systems.

Many patients are given MBRT in combination with other treatments, and the MBRT seems to accelerate positive outcomes. Some of these other treatments included acupuncture, homeopathy and chiropractic work. But ozone has been a big part of the initial implantation. (However, when owners have come in for just an oral piece of feces to give at home, ozone is not usually taken home.)

Patients are given individualized courses of MBRT according to their clinical symptoms being treated and to their recovery progress. Some patients show complete and sustained recovery after just one MBRT fecal transfer. Others need the implant to be repeated regularly after 1 or 2 weeks for continued improvement. For these patients, we hope to fine-tune dosages and timing so that caretakers might be able to take home chilled MBRT material to administer to their pets multiple times per month to maintain the benefits of the treatment. More study is needed to determine how much fecal matter should be transferred and over what period of time to rebalance the gut microbiome optimally.

We believe that MBRT has been successful in replenishing the normal symbioses of endogenous

gut microbiota in vast majority of the cases we have treated. Currently, separate glandulars, prebiotics, and probiotics are given to patients to support different organs or systems. Because the gut microbiome plays such an important role in many body systems, MBRT could be a more complete, effective, and inexpensive treatment option. Drugs and antibiotics currently used as standard treatment for many clinical signs, are less broadly effective, more costly, and can also weaken the gut flora of patients. Ozone therapy has been a crucial component of MBRT. Conventionally in humans, the treatment protocol prior to administering MBRT includes a course of antibiotics to disinfect the gut. Vancomycin is the usual antibiotic used in humans.¹⁰ However, the overuse of antibiotics

in humans often accounts for cases of *Clostridium difficile* and other gut dysfunctions. Ozone therapy reduces the biofilm and supports the gut mucosa, so it is the sensible application of ozone that can foster success with MBRT.

With increased research in fecal bacteriotherapy, further knowledge about the hundreds of species endogenous to the digestive system and their relation to bodily health could be clarified. MBRT could then be optimized and used in place of other drug and antibiotic treatments.

I would like to describe several of our cases that highlight the considerable transformation that the introduction of MBRT effected, over using *only* nutritional supplements, ozone therapy, and other alternative therapies. The following seven cases show the efficacy of adding

microbiome therapy to the treatment protocol. The remarkable changes seen in over 800 treatments have been nothing short of inspirational. (For the delivery system, amount, and concentration of ozone, please refer to Table 1.)

Stovin, a white male 3-year-old standard poodle, had been sick since he was 9 weeks old with chronic diarrhea while on multiple antibiotics throughout his life. By the time he arrived at MASH as a patient, he had been diagnosed with Addison's disease and was severely anemic even after a blood transfusion, was having seizures, and had chronic cystitis and bloody diarrhea. He received ozone therapy and nutritional

Table 1

Animal weight lb/kg	O ₃ -subcutaneous saline/cc or ml	O ₃ -rectal gas/cc	O ₃ combination saline/cc/gas/cc	Vitamin C+B complex cc+cc
10/4	40	40	25/25	0.5 + 0.2
20/9	75	60	50/40	1.0 + 0.5
30/13	100	75	80/45	1.5 + 0.5
15/22	130	100	100/50	2.0 + 0.75
75/34	145	120	125/70	2.2 + 0.75
100/45	190	140	170/100	2.5 + 1.0
125/56	210	150	180/120	3.0 + 1.0
150/68	250	150	200/150	4.0 + 1.3

NOTES:

- a) Gas administered rectally
- b) If animal has low body temperature, decrease saline by 30%
- c) If animal has cardiac issues, decrease saline by 40%
- d) If animal is older and/or weaker, keep warm at all times and add more gas rectally, up to 25% more
- e) If animal is younger and more vital, can give more saline subQ, up to 25% more
- f) Wait at least 10 minutes post O₃ administration before giving vitamin injection subQ
- g) If animal has been on oral antioxidants, may not need vitamin injections

Ozone Dosages:

1 gamma = 1ug/ml = 1mg/L
 1LPM = 1000cc/min = 1000ml/min
 1/8 LPM = 125cc/min = 125ml/min

Saline is ozonated at 1/8 LPM and a setting at 8 = 88 gamma

Saline only saturates to 37 gamma

Unit is run for 30 minutes to saturate 1000cc of saline

Gas is given at 1/8 LPM and setting of 5 = 61 gamma

Cardiac issues and low body temperature will reduce the ozonated saline volume by 30%

Ozone Therapy

➤ support as well as acupuncture and homeopathics, after stopping all antibiotics and anti-inflammatories. After 5 days, he had the first normal bowel movement since he was 9 weeks of age. He was considerably recovered, but not completely. The fecal transplant given orally two weeks later gave him a total recovery and stabilized his Addison's disease. Rebalancing the gut flora allowed for normal absorption of the food and nutrition and gave Stovin a normal life.

Archie, a 7-year-old neutered wire-haired fox terrier, presented with aggressive behavior and digestive issues. Pharmaceutical medications for his behavior failed, and he was kept on a very strict diet with lots of nutritional support for years along with many other integrative approaches, but still was very aggressive with his sister. Less than 24 hours after the MBRT, he was grooming and kissing his sister and could eat foods that he hadn't been able to eat without experiencing massive diarrhea. This agreeable behavior change stopped when he was given the heartworm medication Interceptor (milbemycin oxime). Less than 24 hours later, his aggressive behavior returned when he attacked his sister. Monthly heartworm pills are antibiotics, and damaging the microbiome with an antibiotic can throw off the immune system and create behavior issues. Since subsequent MBRT treatments and being kept off the monthly heartworm preventative, Archie remains well behaved and has minimal diarrhea.

Dudley is a 12-year-old male neutered poodle cross who was diagnosed with T-cell lymphoma. The owners decided to use an integrative approach to his cancer care. With his first round of chemo he received an MBRT and ozone therapy with ultraviolet blood therapy (UVBI) after each round of chemo. His blood work stayed completely normal throughout his chemo and he did not develop the interstitial cystitis that is common

with this protocol. When he went out of remission, he was treated with an experimental lymphoma vaccine protocol, and received MBRT and ozone with UVBI after each vaccine in the series. When his lymphoma returned for a third time, he was treated with rounds of chemo and again after each round, MBRT, ozone, and UVBI. He has consistently had nutritional support with combinations of herbs and nutraceuticals. Now surviving 20 months, he bounces around like a puppy. He has received some of the useful biome from a young puppy in our donor group as well as MBRT from a cancer survivor. The youthful microbiome has given him the energy of a puppy. Throughout his conventional care, Dudley has been allowed to receive all of this alternative treatment, and we believe that this has enabled him to have more successful remissions.

Sadie, a 10-month-old female spayed English golden who had giardia for 6 months, was treated and referred by Angell Memorial Hospital for MBRT. After 2 days she had a normal bowel movement and at days 5, 14, and 30 had negative giardia tests and continues to be negative 4 months later. By receiving a more balanced microbiome that had not been subjected to multiple antibiotics, the balancing of the biome has helped to stabilize Sadie's gut.

Bode, a 4-year-old neutered male golden retriever, had chronic diarrhea for over 2 years and an unresolving *Clostridium perfringens* for the past two months. He had been on multiple antibiotics and nutritional supplements from four different veterinarians. With ozone therapy and after 2 days post-MBRT, the diarrhea stopped and 1 week later the *Clostridium perfringens* was negative. By repopulating the gut with a new balanced biome, the diarrhea was stopped and the balanced gut flora somehow pushed out the overgrowth of *Clostridium perfringens*.

Mojo, a 7-year-old neutered male domestic short-haired cat, lived years with atopic dermatitis and had been treated with multiple

courses of antibiotics, cyclosporin, antihistamines, and steroids. He was put on nutritional support and ozone and given a rectal MBRT from a 1-year-old Siamese male kitten. Within a week he stopped scratching his face and was able to remove the Elizabethan collar. His attitude was much happier than it was prior. Six weeks later, the owner requested a second MBRT. When he continued to improve, she requested a third treatment 6 weeks after the second. The owner called 2 days after the third fecal transplant and said that there was a problem with his anal glands as he was acting odd. When she came in for me to see the anal glands, they were normal. After discussion of his behavior, we realized that he was sexually humping her arm. Had we given him the hormones and youthful vigor of the 1-year-old Siamese cat who was the new fecal donor? My comment to the owner was "Forget Viagra; we may have found the fountain of youth." Mojo continues to get better and better.

Norman, an 8½-year-old neutered male ragdoll cat, had been vomiting for months and also exhibited inappropriate urination. He had lost over 3 pounds and seemed nauseated all the time. He had been on nutritional supplements and some were added, but 2 days after his ozone and MBRT, he started eating as if he had not eaten for days and gained back 2 pounds. With another MBRT 10 days later, he seemed really happy and has not vomited since that time. The microbiome was rebalanced and allowed digestion to occur normally.

Norton, a 2-year-old Labrador retriever pup, had been trained by New England Dog Assistance as a therapy dog. He did not advance into the program for several reasons, but coprophagia was one of them. We started him on some nutritional support (he had already been on a limited raw diet) and gave him ozone rectally to address the biofilm. Two days after the fecal transplant, his coprophagic behavior stopped. We can hypothesize that missing certain microbes in his gut made him seek out stool. Even though he was eating

his own stool, it was from the same microbiome. Allowing a new more complete biome to set up a terrain in his gut enabled him to process his food more efficiently and most likely extract more nutrients to his body, thereby eliminating the urge to eat feces.

The range of clinical symptoms that benefitted from MBRT in the cases presented here, and the even wider range that we have treated in our practice, gives hope that MBRT along with ozone therapy could be beneficial in treating even more medical conditions.

In the near future, we hope to establish a fecal donor directory that could match healthy dogs and cats with animals in need of fecal transplant to reestablish their biomes. Veterinarians will then have a source of healthy animals to provide flourishing, active microbiome samples.

More natural approaches to veterinary care, including herbs, homeopathy, acupuncture, and chiropractic work, reduce the need for drugs and antibiotics in patients. Raw meat diets, including green tripe with lots of pureed raw vegetables, give canines and felines more of the evolutionary diet of their ancestry. We are also interested in the development of bowel nosodes of particular fecal groups as another method for administering fecal material. Bowel nosodes involve performing a series of dilutions on a fecal sample to increase the potency of the material to create a homeopathic remedy, which could make MBRT even easier to administer. The original bowel nosodes in humans were developed in the 1930s by Edward Bach, MD, a bacteriologist, and John Patterson, a homeopath, at the London University College Hospital.

Bowel nosodes are prepared from cultures of non-lactose-fermenting flora of the intestinal tract. More studies are needed to determine the viability of microorganisms in homeopathic fecal capsules as compared with those in natural stool.

The possibility of premade fecal capsules could eliminate some of the

“grossness” factor that unfortunately may have been inhibiting the development of MBRT to this point.

Massachusetts General Hospital has done a study to show that frozen capsules of feces have cured *Clostridium difficile* in over 90% of cases.¹¹ Clearly, MBRT is a promising therapy that should be developed for widespread use in veterinary, as well as human, medicine. The use of ozone therapy to reduce the biofilm and numbers of unhealthful bacteria in the gut, as well as to heal it, seems to be the best way to introduce a new microbiome.

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Margo Roman's integrative veterinary practice, MASH Main Street Animal Services of Hopkinton in Massachusetts, has been a cutting-edge center since 1983. The addition of ozone therapy in the past 12 years and microbiome restorative therapy and biophotonics for over 3 years has enhanced her 37 years of practicing conventional and alternative modalities, giving her more chances to enhance the health and well-being of her patients. Dr. Roman graduated from Tuskegee University Veterinary School in 1978, held an internship at Angell Memorial Animal Hospital in 1979, and was on Tufts University School of Veterinary Medicine faculty for 8 years. As a national and international speaker on an array of integrative veterinary topics, she has a deep passionate to enlighten and educate both students and the profession as whole of the need for integrative health. With the production of the *Dr. DoMore* documentary preview and “Dr. ShowMore Calendar,” millions of people have been exposed to other ways to help their health and that of their beloved animals. Dr. Roman has stood up to Tufts University and the Massachusetts Veterinary Board to protect animals' health by including integrative medicine as a choice for health care. The standard of care is euthanasia instead of ever offering an integrative approach, and she has gone all the way to the Supreme Court of Massachusetts to try to make sure that owners of animals have choices. She has published articles and books on these topics. Dr. Roman's three grown children and husband support her need to educate as she lives with her four standard poodles, Siamese cat, and cockatoo in Hopkinton.

Ozone Therapy

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Acknowledgement

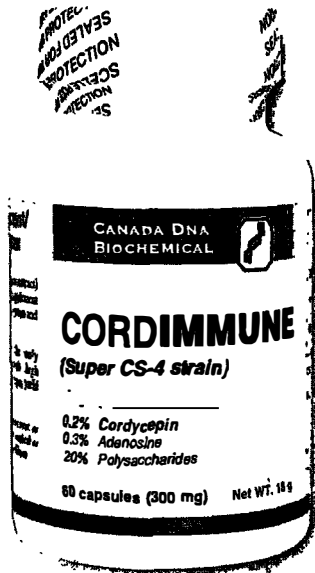
Much appreciation goes to Leona Chan for her statistical analysis for an earlier version of this article (and now subsequent paper), as well as for all her great work on our website, www.EatSh-tAndLive.com/org.

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Field Control Therapy: Successful Approach to Lyme Disease and Coinfections

Part 1

by Savely Yurkovsky, MD

I have been whole-heartedly referred to Dr. Yurkovsky by an acquaintance of mine. I looked around the website and found with amazement a lot of information and principles that are concurrent with my experience and philosophy. I'm not a medical professional but I did learn a lot while dealing with my own health issue. I went through many therapies, Lyme-literate doctors, healers, naturopaths, nutritionists, homeopaths. Even did a prolonged course of antibiotics for about 9 months, herbs, structured water, Rife machine, hyperbaric oxygen chamber, bioresonance (also Spinor/Metatron). The list goes on and on. I'm better now, but I still have lingering symptoms like brain fog, small fevers, migrating joint pains which come and go, excessive sweating, headaches, lack of energy, photophobia, etc. It comes and goes like with all Lyme there are good and bad days but even in the good days, I'm not 100% well and in the bad days I'm still incapacitated.

Ms. A.

I have been on antibiotics for 8 years now ... in many ways they have helped me to regain some sort of life back but there have been other issues that have gotten worse and new problems that have come up that medical doctors are having a very hard time figuring out. I am sick and homebound more than not and I remain on Lyme and Babesia treatment. ... I am looking for someone who can help me get to the core of what is going on. My biggest problem is my severe sensitivities to alternative treatments, as well as prescription meds. I have tried to do different alternative treatments through the years but they seem to make me much worse and set me back, causing me months to try and get back to baseline ... this includes everything from supplements, energy work, and types of detoxing.

Mrs. B

Dear Dr. Yurkovsky, I watched your DVD series on Lyme, and at this time I am leaning very much toward following your program. I've done oral antibiotics, antimalarial, and antifungals, in rotation for over a year. I've considered Rife machine and medicines (intramuscular injections, plus orals, plus supportive treatments) and your approach, and I'm still confused as to where to place my confidence, but my strongest feeling is right now to commit to your approach. I am honestly quite overwhelmed, and frightened, as I feel I am continuing to get worse.

Ms. C

Besides encountering countless e-mails like these and their authors, I have also encountered similar statements by J. Schaller, MD, published in this periodical underscoring the failures of the prevailing treatments for Lyme disease and coinfections.¹

Our average patient has been to anywhere from 10 to 50 physicians, but has not returned to his/her baseline level of functioning.

"Lyme literate doctors" really means that you have gone to a couple of conferences. ... These are a good starting place, but do not make one tick-infection literate in any serious manner. Finding someone who knows how to use a wide range of labs, has read thousands of articles, and checks for a direct and indirect presence of the infections in ticks is extremely rare in the world.

Routine speed IV treatment of most new patients is a mistake.

The most common treatments for Bartonella lead to a relapse, even when they appear to work for variable periods of time.

Following the guidelines of practitioners with famous names, university titles, or organizations leadership positions is an error in judgment.

No single organization or group of organizations can provide people with authoritative instructions in how to treat an individual patient.

The human body when infected with a cluster of tick-borne pathogens is a billion times more complex than any automobile.

Antibiotics rifampin, azithromycin, HBOT, Rife, special saunas, ozone, IV nutrients to "boost immunity," chelations, confused detox formulas, Artemisia derivatives, essential oil combinations, IV medications, various weak alcohol-based herbal programs, various energy machines, and 100 other options found in chat rooms and Lyme disease information sites are not meant to be the sole or primary style of all patient treatment.

Lyme Disease and Coinfections

And finally, the great philosopher of science, Thomas Kuhn, has shown that there are so many variables affecting all scientists, the notion that any group of physicians can give unbiased pure scientific recommendations is impossible. Obvious errors are present in all current tick and flea-borne infections guidelines.

Following this last quote, I must state that we had all better beware of statements that we choose as our support because these can also lead to just the opposite. This is particularly true when bringing the philosophy of science into the battle by quoting its Professor Thomas Kuhn, since one of the main objectives of the philosophy of science is to protect science from scientists, because the latter are often afflicted with "human weakness," to quote American Nobelist in Physics Professor M. Gel-Mann. Besides other misgivings, this element of "weakness" often prompts scientists and us health practitioners, according to Harvard biology Professor Eugene Wilson, to confuse sound scientific hypotheses with just plausible-sounding speculations, whether concerning any disease or any scientific field. Unfortunately, the thorough scientific criteria to distinguish between the two are not taught in medicine. As a result many spirited arguments for or against different methods within such inherently inexact science such as medicine, often resemble soft science of blowing one's own trumpet, rather than true science.

Even though most of the actual clinical outcomes of the prevailing Lyme and all chronic infections do concur with Dr. Schaller's statements, yet some of his assertions do conflict with the rules of science concerning the subject matter.

Starting with the "famous names," even though, yes, there is a general herd mentality in medicine to blindly follow some prevailing method or authority, a blanket statement concerning an authority being necessarily wrong should not be made, because strictly speaking, if the rules of science do not prevent even a village fool from being correct on a good day, why can an authority not be correct sometimes too?

Just because many of us have not studied Schaller's writings, who has "read thousands of articles," this does not make anyone medically inferior per se. For one, it is impossible for an average practitioner to read millions of pages of medical material published only yearly, and for two, the rules of science, paradoxically, tell us that the mere consumption of large volumes of data may end up with its consumers becoming more confused than enlightened. This is simply because no single datum can solve any problem per se, but only if properly connected with other relevant data by a *sound scientific theory*.

Without such a sound connecting theory, just reading or adding more findings into one's head may automatically

lead to the increased odds of connecting these incorrectly or ineffectively, whether in Lyme or 1300 other chronic diseases. As an example, if Thomas Edison faced 125 million wrong connections to choose only 5 correct elements out of 100 to construct a working lightbulb, we doctors face trillions of wrong connections between thousands of data for Lyme or any disease. Moreover, quoting my acquaintance, expert in decision science and MIT physics Professor George E. Pugh, "There are no exact formulas that even exist how to properly engineer or connect data into viable theories." Columbia University Medical School immunology Professor Stuart Firestein, PhD, among other medical scientists, underscored this issue of sheer piles of scientific information: "Neither I nor my colleagues can keep up with all the findings in our field, these confuse me and we are just trafficking in findings."

Next, gastroenterology professor and book author Michael Gerson, MD, from Columbia University Medical School: "Half of what I teach (e.g., scientific findings and theories) today will be obsolete tomorrow." That is why the foremost authority in the philosophy of science, whose work has been revered by many Nobel laureates in science, Austrian Professor Carl Popper said: "Accumulated knowledge, by and of itself, paradoxically, is not as important as people think. It is only a sound theory that determines and connects only the most important findings that counts."

So, when any theory yields poor outcomes in Lyme or other treatments, we ought to suspect that it is either has misconnected some formally correct medical findings, or that it missed more important ones, or both. The more important ones science views as being akin to the rank of a four-star general, versus sergeants and lieutenants. Popper also specified a sound theory as capable of offering a superior understanding or explanation of a problem, as well as sound reasons for occurred failures. Sound reasons do not imply a mere lack in more scientific activity or treatments, per se, because since there is no end to scientific findings in medicine, then, there logistically can be no end to just "more and better research and treatments," for better or worse.

What have I found in my practice as a superior or FCT theory to treat Lyme and coinfections? Presenting it in points:

Point 1

The infectious agents *Borrelia*, *Babesia*, *Bartonella*, *Ehrlichia*, *Mycoplasma*, and so on are neither the primary nor the most important causes of Lyme disease and coinfections, but are only their triggers, or the proverbial final straws that broke the camel's back. Besides other supporting scientific theories, one of the most significant of these dissipative structures in biological evolution by

Lyme Disease and Coinfections

Russian-born quantum chemist and Nobel Prize laureate Professor Ilya Prigogin states that it is a preceding history, as embedded in the current state of an organic or living structure, that determines how it will or will not change or evolve in response to any external influence. In our case, an external influence can be any infection, Lyme, herpes, parasitic, yeast, HIV, HPV, EBV, or thousands of others, where a change becomes a given disease.

Q: What usually determines the preceding history of our bodies in our modern environments before we may encounter Lyme or other microbes?

A: Good and bad genetics, higher or lower amount of toxic environmental pollutants, including mercury and other heavy metals, higher or lower

number of mercury fillings, higher or lower side effects from antibiotics and other treatments, higher or lower number of other infections present in the body, higher or lower consumption of sugar and other substance abuse, of junk food, higher or lower levels of stress, high and even higher levels of electromagnetic fields (EMF).

Q: How will such a preceding history and current state influence physiologic response of a corresponding living system to contact with a given microbe?

A: It will make its susceptibility to it, depending on an individual combination of the aforementioned factors, correspondingly, high, very high, moderate, low, or very low. Just between mercury and

EMF alone, with both being ubiquitous in populations, the immunosuppressive effects of these have been cited in hundreds of scientific reports.^{2,3} Likewise, if an infection develops it might become severe, very severe, moderate, or mild.

If it is mild, many treatments, even if imperfect, such as antibiotics, herbs, immune enhancers, oxygenative, and electrocuting, may succeed, at least in the short run. If the infections are severe or very severe, the aforementioned treatments, as a rule, fail. With moderate infections it can go either way, but the trend is toward recurrence.

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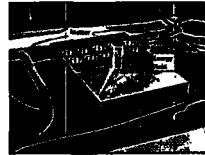
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Lyme Disease and Coinfections

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Point 2

Using effective diagnostic means to determine most of the factors which undermine resistance to current and future Lyme and other infections.

Since neither imaging nor lab tests can identify most of these factors and their resulting damage where it counts the most or inside the immune or endocrine systems, brain, or other organs, FCT uses noninvasive bioresonance testing to obtain this crucial information right from the internal organs themselves. Here, too, only a handful of the most important data, or "four-star generals," are sought and addressed, such as mercury and other heavy metals, certain organic environmental pollutants, electromagnetic radiation, residues of antibiotics, parasites, candidiasis, and some viruses. The rest usually is overcome by the body on its own, given also proper patient compliance.

Bioresonance testing also serves other crucial clinical goals. One is to diagnose Lyme bacteria and their coinfections, since the lab tests often issue false negative reports. Two is to determine the end point of the treatment, whether antibacterial, anticandidiasis, or detoxifying of mercury or other toxicants. This is regardless of the nature of a treatment;

for example, allopathic, homeopathic, herbal, antibiotic, chelator, electrical, or any other, since a simple, very important question – what has the treatment actually accomplished? – is not an idle one.

Point 3

The homeopathic treatment used is based on up-to-date conventional medical knowledge, not classical or complex homeopathy, along with a healthful well-balanced diet, EMF reduction guidance, and effective (vs. just presumed) EMF stress-reducing technology such as memon.

Point 4

Based on the aforementioned reasons and very successful outcomes, which confirm this agenda of treating the *total* patient with Lyme, rather than Lyme without a patient, one is to only approach Lyme and coinfections as strictly and always individual disease. It is because every combination of the many aforementioned factors plays a crucial role in the history and state of each individual patient's disease. That is why, strictly speaking, pure or "Lyme disease" by itself does not even exist. What does exist and matters the most is a unique mosaic of these factors in the body, including the severity of the

microbial invasion. Thus, if we have a million patients formally diagnosed with Lyme disease, we have a million different Lyme diseases. That is why many of these patients also present numerous problems that are not part of your medical textbook Lyme disease: different degrees of chemical or electromagnetic sensitivities, food and mold allergies, gastrointestinal disorders, cycle problems in women, sinus, skin, genitourinary, or other infections.

Numerous clinical examples attesting to the efficacy of this FCT approach even in the very severe and great variety of cases.

Notes

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◆



Saveli Yurkovsky, MD, graduated from II Moscow State Medical Institute in 1975 with a degree in pediatric medicine. He completed his training in internal medicine and cardiology at Coney Island Hospital of Downstate Medical School, and is board certified in internal medicine. He has been in private practice since 1984 with a special focus on identifying and successfully treating the main causes of chronic diseases via bioenergetic modalities – bioresonance testing and homeopathy, correspondingly, or FCT.

Dr. Yurkovsky has founded a teaching organization, SYY Integrated Health Systems Ltd., dedicated to training in FCT. It has been presented extensively in the US and Europe to medical practitioners since 1999 and demonstrated numerous documented reversals in a variety of chronic diseases.

His book, *Biological, Chemical, and Nuclear Warfare Protecting Yourself and Your Loved Ones: The Power of Digital Medicine*, was endorsed for scientific validity by two prominent physicists: MIT Professor George Pugh, PhD, and former chairman of materials science at Stanford University, Professor William Tiller, PhD, and also by Mehmet Oz, MD, from Columbia University Medical School. Its diagnostic and homeopathic aspects were also presented at the annual BTR (bioterrorism) conference in 2005: Unified Science & Technology for Reducing Biological Threats & Countering Terrorism, affiliated with the Department of Homeland Security and the US Army, as well as at the Department of Psychiatry of Massachusetts General Hospital, Harvard Medical School, and many other professional symposia.

In collaboration with the Department of Gastroenterology of Johns Hopkins University School of Medicine, he has contributed a chapter on homeopathy to the textbook *Integrative Gastroenterology* (Oxford University Press, 2011) and authored numerous articles on different medical topics.

Dr. Yurkovsky's seminars on DVDs, devoted to autism, other brain disorders, and Lyme disease, serve as a virtual step-by-step textbook classic explaining the fundamental nature of all chronic diseases (available at www.yurkovsky.com). His book in progress explains the inevitability of the current epidemics of autism and numerous other brain and somatic diseases and how to solve them.

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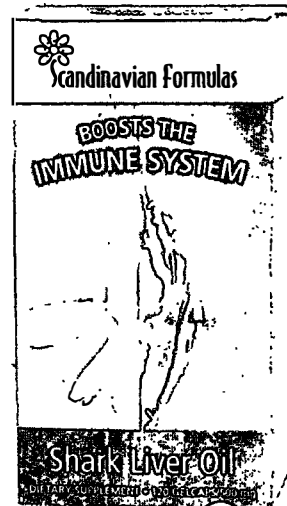
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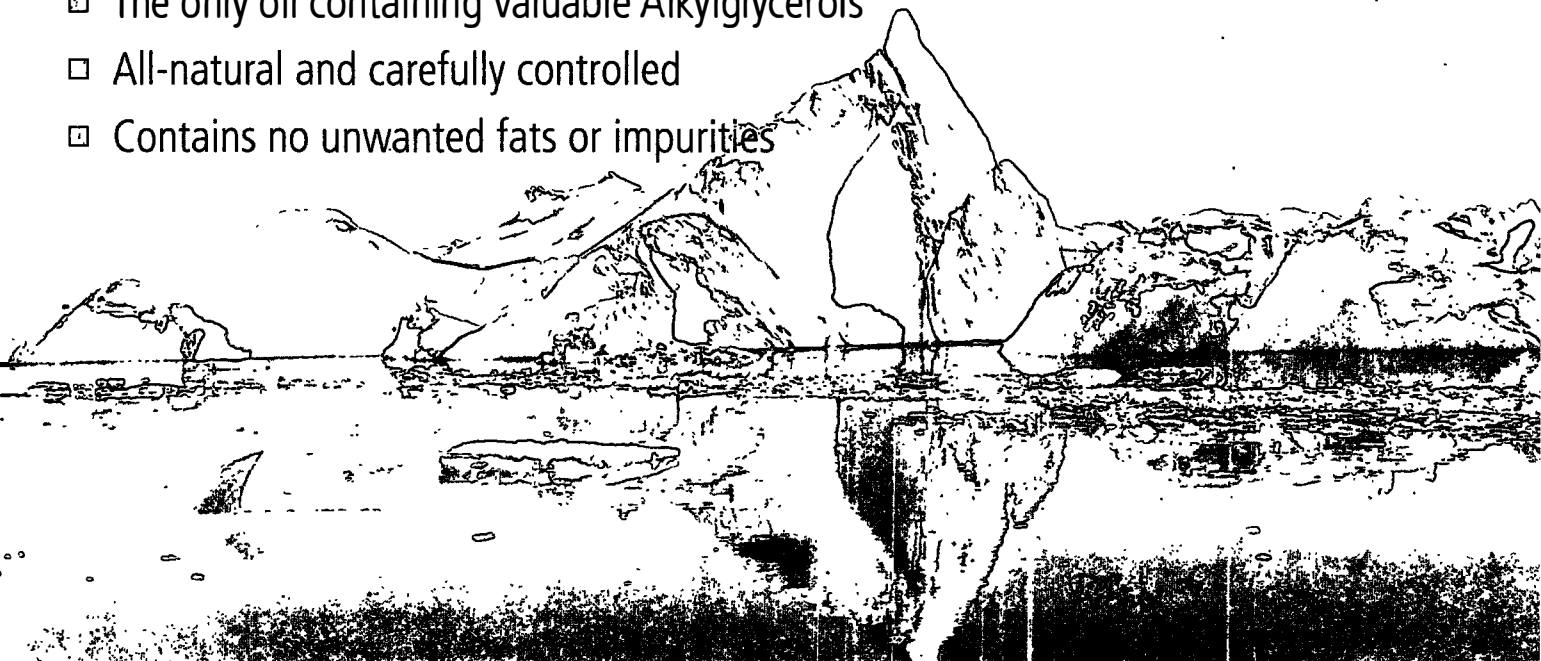
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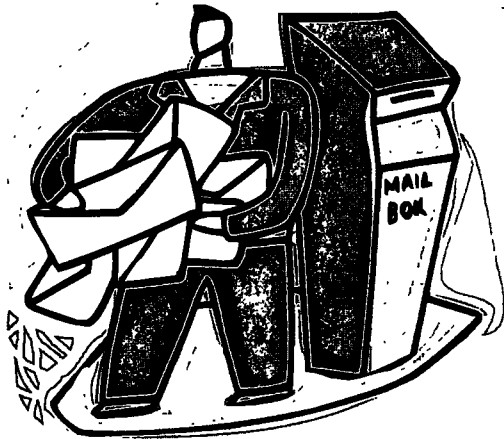


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Letters to the Editor

Mannitol as Effective as D-Mannose in Treating Urinary Tract Infections

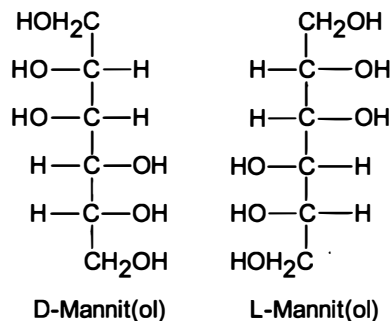
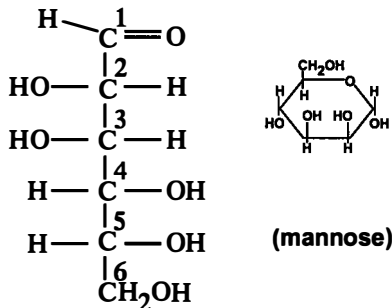
D-mannose has been touted in the alternative medical field as an effective treatment and prevention for urinary tract infections. Several studies show that d-mannose inhibits the adherence of *Escherichia coli* to urinary epithelial cells in humans. Many supplement companies sell d-mannose for treatment of urinary tract infections. One of the main prohibitive aspects of d-mannose is the cost. Many patients find that it is simply too expensive. A 50 to 85 gram bottle of d-mannose powder typically costs over \$40.

The etymology of mannose and mannitol comes from the root word *manna*. Manna is a sweet secretion of several trees and shrubs from the Middle East. In biblical times it was manna that helped to sustain the Israelites in their journey from Egypt to Israel. Mannose and mannitol are naturally occurring sugars found in some fruits and vegetables, including cranberry, blueberries, tomatoes, broccoli, and beans.

I have used mannitol instead of d-mannose in the treatment and prevention of urinary tract infections. I have found that mannitol works just as effectively as mannose. I decided to do some research to substantiate my theory and observations in clinical practice. While searching Medline, I came across an interesting scientific article that supports this. An article called "Effects of Carbohydrates

on Adherence of *Escherichia coli* to Human Urinary Tract Epithelial Cells" appeared in the *Journal Infection and Immunity* in November 1980. In the study, uroepithelial cells were obtained from freshly voided midstream urine samples from premenopausal women who had no history of urinary or vaginal infections and were not taking contraceptives or antibiotics. The harvested uroepithelial cells were mixed with *Escherichia coli* and then mixed with different carbohydrates including mannose and mannitol. The molar concentrations of the different sugars were determined to produce a 50% inhibition of bacterial adhesion on the uroepithelial cells. Mannose and mannitol were the best sugars to reduce adherence. Both sugars had almost identical concentrations required to produce a 50% reduction of bacterial adherence.

D-mannose and mannitol are 6 carbon hexose monosaccharides. The molecular formula of d-mannose is C₆H₁₂O₆ and C₆H₁₄O₆ for mannitol. Mannose is formed by oxidation of mannose. D-mannose exists as a ring structure or a straight chain. Mannitol exists as a straight chain. This study makes the point that the ring structure or straight chain doesn't matter for the efficacy of



sugar on bacterial adherence. Both structures are equally effective. It was further surmised that d-mannose and mannitol inhibit bacterial adherence by competing with mannose-like receptors on the uroepithelial cells. They prevent bacterial adherence by a competitive binding to receptors on the bladder lining. The piled bacteria are prevented from binding to the bladder wall and literally slip away.

Further investigation helped to elucidate the stereochemical specificity of the carbohydrate binding site to the bacterial ligand. The bacterial-carbohydrate binding site appears to be the carbon 2 position of d-mannose and its derivatives. This position is an unmodified hydroxyl group at the carbon 2 atom. The C2 binding site is not altered whether the sugar molecule is in a ring structure or straight chain. The study concluded that d-mannose and mannitol are both equally effective in inhibiting bacterial adherence to uroepithelial cells in humans.

Mannitol is just as effective in treating and preventing urinary tract infections as d-mannose. My clinical experience supports the conclusions reached by the researchers in this study. The obvious advantage of mannitol is that it costs substantially less than d-mannose. The cost of mannitol is about 25% the cost of mannose. This is an obvious advantage for patients who desire to use this sugar for urinary health. Other clinicians should start to recommend mannitol as well as d-mannose for prevention and treatment of urinary tract infections.

Reference

Schaeffer AJ, Amundsen SK and Jones JM. Effect of carbohydrates on adherence of *Escherichia coli* to human urinary tract epithelial cells. *Infect Immun*. November 1980;531-537.

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Reader Seeks Help with Neurofibromatosis-1

An 11-year-old family member of mine, let's call him "Joe," was born with the "café-au-lait" brown spots that are diagnostic of the genetic disease von Recklinghausen disease, a.k.a. neurofibromatosis 1, or NF1. It's related to "Elephant Man disease."

The symptoms of NF1 are usually marble-sized lumps under the skin surface anywhere or everywhere on the body. These bumps are nerve tumors, usually small and benign, embarrassing at the least and potentially deadly at worst.

When Joe's deciduous teeth erupted, they were blackened and eventually crumbling with severe dental fluorosis. When he was 6 years old, the family had to borrow \$8000 to have all his teeth temporarily capped. I believe now that there's a definite fluoride connection with NF1. Working temporarily as a waitress, his mother drank a good deal of tea during his gestation. Tea is naturally very high in fluoride (up to 8 ppm), and it was also made with fluoridated water. She drank fluoride-free water at home.

Fluoride can cause DNA damage resulting in genetic diseases and cancers. A British study compared the incidence of Down syndrome incidences between the fluoridated and unfluoridated areas in Britain. They found Down's syndrome to be 30% higher in fluoridated communities. A NJ Health Dept. study found osteosarcoma, a rare bone cancer, to be 3 to 7 times higher in fluoridated areas. This fluoride connection was confirmed in an EPA rat study. This cancer, osteosarcoma, usually hits young 8 to 10 year old boys during their early bone growth spurts and is often fatal. It is very rare in adults.

I believe NF1 and osteosarcoma are results of fluoride toxicity which causes a rather newly recognized disease, called hypothyroidism 2. This disease was named by Mark Starr, MD, author of *Hypothyroidism Type 2 the Epidemic*. I read an article by him and wrote to him about a fluoride connection. It wasn't mentioned in his book, but he added my suggestion on p. 199 in his next edition and kindly sent me a copy. Note: Dr. Starr tragically died a relatively young man this past October.

I believe that NF1 and osteosarcoma are both results of hypothyroidism caused by fluoride. Joe has very little evidence of the disease now. The symptoms don't usually manifest until after puberty. This disease, unlike traditional low thyroid, is at epidemic levels in this country. Some doctors who can recognize it have reported it in up to 80% of their patients from only about 40% reported in the 1970s.

Regular hypothyroidism can be diagnosed by a blood test for adequate thyroid hormones but hypothyroidism 2 is caused not by an absence of the thyroxin hormone but by the hormone's inability to enter the cells where it can raise body temperature. To test for hypothyroidism, the temperature is taken before arising in the morning. It should be at least 97.9 °F. Joe's temperature runs about 95 °F all the time.

When a person's temperature is low, the body partially shuts down the blood supply to the skin, to preserve body heat. Without a supply of antioxidants from the blood, in the case of the tumors of NF1, they don't manifest internally but right at the skin level because there's not a sufficient blood supply there to fight their creation or elimination.

I would greatly appreciate it if those professionals who treat NF1 patients or the patients themselves after taking a few morning temperature readings would contact me and relay the results of this readings. If thyroid medication is being taken, please relay that also.

Additionally, any professional who wants to try this hypothesis, after finding a low temperature reading in an NF1 patient, please keep in touch and share any results. An 11-year-old boy needs help before NF1 symptoms appear. Thanks.

Janet Reed Pettit, Palatka, Florida
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Connecting Childhood Nutrition and Ailments

review by Katherine Duff

Cure Your Child with Food: The Hidden Connection between Nutrition and Childhood Ailments

by Kelly Dorfman, MS, LND

Workman Publishing Co. Inc., 225 Varick Street, New York, NY 10014-4381

© 2013; softcover; \$13.95; 348 pp.

Kelly Dorfman, MS, LND, is a nutritionist who specializes in treating children's disorders. Often, her clients come to her after seeing several medical specialists who have not had success in resolving the child's issues. In her book *Cure Your Child with Food*, Dorfman describes the conditions that can be affected by diet and methods for parents to discover possible dietary and nutritional connections. To be clear, this book is not about miracle cures for everyone, as she points out that these interventions will work for some but not all children.

The subtitle of the book, *The Hidden Connection Between Nutrition and Childhood Ailments*, more accurately describes the efforts detailed in this book. The author hopes to teach parents to become detectives in their search for possible dietary and nutrient issues affecting their children. The puzzles to be solved are an array of problems that can include developmental delays, learning and behavior challenges, digestive troubles, and emotional issues. Starting with her Nutrition Detective Principles included throughout, we learn that the first is the Binary Law of Nutrition: "All nutritional problems fall into one of two categories: Either something is bothering the body or something is missing." In some cases, the solutions will be found in both circumstances.

There are many culprits when the child is irritated by certain foods. Milk, particularly the protein casein, can cause several symptoms, as demonstrated in a case study about a young boy. His difficulty sleeping, mood swings, and frequent illnesses were resolved when he was taken off dairy and given a multivitamin and calcium supplements. A young girl found relief from her constant stomachaches and headaches after gluten and lactose-heavy dairy products were removed from her diet.

Supplementation begins with a good multivitamin for all children. There are conditions that will need even more supplements to counteract a deficiency. An example of this is described in a boy who presented with symptoms of keratosis pilaris ("chicken skin"), constipation, and lack of thirst. His diet was low in fiber and high in white processed foods. The author suspected an essential fatty acid deficiency, and the child was successfully treated with fish oil supplements. Another example is of a 2-year-old who had been on prescription medicine for reflux. In addition to investigating possible irritating foods that could be causing the problem in the first place, Dorfman put the child on probiotics and zinc carnosine to aid in healing the digestive tract.

How the author gets to the solution is the most interesting part of this book, as it is superbly organized for the reader to follow. Each chapter addresses a different health issue using a

"All nutritional problems fall into one of two categories: Either something is bothering the body or something is missing."

case study to describe what can be called a journey of discovery. Dorfman readily acknowledges that this will be an effort of trial and error, which should relieve any anxiety that parents may have about addressing their child's health issues on their own. The interview, which includes the history of the ailment, the child's diet, and symptoms, is meant to get the issue down to the bare facts. Often, Dorfman has noted, the beginning of symptoms occur when the child changes to adult food that is heavy in what she calls the white diet – high in sugar, bread, and pasta, while being low in fresh fruits and vegetables.

There are a series of questions for readers to determine if the condition fits their child and where to start if it does. Treatment may call for a visit to a specialist, whereupon the author offers advice for the visit. If nutritional investigation is the answer, the author provides age-appropriate dietary and/or supplement changes. She explains how she arrived at her decision and why she is recommending the particular change.

Something that becomes very clear while reading this book is that diet-influenced conditions in a child can be a family affair. Sensitivities and intolerance to certain foods may run in the family. Though they may express it differently than a young child, a parent or older sibling may also be suffering from the same issues. Dorfman cites instances when other family members' health improved when the child's irritant food was removed for the whole family.

A picky eater can turn every meal into a stressful and combative situation that can test the patience of any parent. The author is sympathetic and offers a plan that will remove irritant foods and introduce healthful ones over a period of time. This plan allows the parent to take control and hold firm and, most importantly, relax, knowing that it is a process which takes time.

Dorfman has called her consultations a last resort for many parents, but one wonders why that has to be the case. She cites the increase in children with chronic health conditions and the fact that children are consuming 40% of their total calories in "empty calorie" foods. With cultural changes such as this, it seems wise to consider dietary influences on children's health much sooner than as a last resort.

This book is an entertaining read and is well resourced, which makes it very accessible. In the absence of having such a knowledgeable nutritionist as Kelly Dorfman available for consultations, this book could help parents and physicians alike.

Help Patients Harness Habits

review by Jacob Schor, ND, FABNO

The Power of Habit: Why We Do What We Do in Life and Business, by Charles Duhigg

Random House

© 2012*

All our life, so far as it has definite form, is but a mass of habits – practical, emotional, and intellectual – systematically organized for our weal or woe, and bearing us irresistibly toward our destiny, whatever the latter may be.

– William James, *Talks to Teachers* (1899)

I was late to work this morning, and I can't blame anyone but myself. I've driven the same route for 20 years. Driving to work is a habit, and I don't think twice about where I'm going once I click my seatbelt on until I unlock my office door. Denver is installing new light posts on Monaco Boulevard, necessitating lane closures, mergers, and traffic coming to a total standstill. Of course I should take another route to work; Quebec would certainly be faster, yet I never think of this while I'm driving. Habit takes over, I make the turn onto Monaco, and the next thing I know I'm stuck in traffic and late to work again. I am a creature of habit.

Habits and how much they control our lives have been on my mind this summer as I've slowly worked my way through a fascinating book called *The Power of Habit*. Written by Charles Duhigg, it is an important book to read, understand, and incorporate into our practices.

I make a list for each patient at every visit of things that I think they should do in regard to exercise, diet, and supplements, under the assumption that because I told them to do these things, they will. How ludicrous. What I should be doing instead is to reinforce my patients' good habits and help shift their bad habits to be less harmful. Duhigg's book explains how to do this.

Here's one example from the book of how habits work. Scientists implanted probes into the brains of rats to monitor brain activity and then placed these rats one at a time at one end of a maze with some chocolate at the far end. On its first time in the maze, a rat would slowly meander its way though until finding the chocolate. The brain probes revealed this seemingly lackadaisical behavior on the part of the rat was a false impression. The neurosensors recorded intense neural activity; the brain was furiously at work with every sniff. Each time the rat was placed back in the maze, it found the chocolate faster. As the rats learned their ways through the maze, something happened in their

brains; the faster they ran, the less brain activity occurred. As the path through the maze became automatic the brain worked less. As the habit was formed, the brain had less work to do and the rat thought less and less about what it was doing.

The process when the brain converts a sequence of actions into automatic behavior is called *chunking*, and we rely on chunking to get us through the day, from brushing our teeth, to making our coffee, and to driving the car; these complex sequences of activity and thought have become automatic. Chunking and habit forming occurs so naturally, we rarely notice that it is going on.

As Duhigg writes, "Left to its own devices, the brain will try to make almost any repeated behavior into a habit,

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▶ because habits allow our minds to conserve energy. It feels good to conserve brainpower; it feels good to be lost in the routine of a habit.

"We've devised a clever system to determine when to let a habit take over. It happens whenever a chunk of behavior starts or ends. Our brains look for cues that tell it when to turn on or off the automatic program."

Our brains create habits in a three-step loop. First, there is a cue, a trigger that tells the brain to go into automatic mode and which habit to use. Then there is the routine, which can be physical or mental or emotional. For the rat, it is running through the maze. For my morning commute, it's driving the car. Finally, there is a reward, the end gain that lets your brain know that this particular automatic behavioral loop is worth remembering for the future.

This loop of cue, routine, reward; cue, routine, reward, becomes automatic, so neurologically intertwined, that a sense of craving emerges. The rat wants its chocolate as soon as the door to the maze clicks open. Most cues and rewards are so quick and so subtle that we are rarely aware of them in day-to-day life. But our brains do notice them and build neural pathways that reduce effort in every way that they can. Once a habit is established, your brain stops participating in the decision making; it's riding on cruise control. No wonder it is so hard to change people's habits.

It's easy enough to make a list of lifestyle changes for patients that will make them healthier, but we should be focused on how to turn these lifestyle behaviors into habits. Simply understanding how habits are formed helps people

gain better control over their lives. Duhigg reviewed a study in which 256 participants took part in classes on the importance of exercise. Half the participants took a second class on the theories of habit formation. The study participants who took the habit class identified the cues and loops in their lives that affected their exercise and in the end spent twice as much time exercising.

If you want to start running each morning, choose a simple cue (like leaving your running clothes next to your bed) and a clear reward (like a midday treat or ritually recording your distance or times in a log book). Your brain will anticipate that reward, either the treat or the sense of accomplishment, and that's how the habit is formed.

Habits may be deeply rooted in the mind but they aren't destiny. People can choose their habits. They have to figure out what their cues and rewards are and then make the decision to change.

Duhigg writes in a style reminiscent of Malcolm Gladwell. Each has a way of weaving multiple stories together into a single narrative that slowly builds on an idea from a variety of angles, that creates a complex story that is pure pleasure to read, but in the end takes you to the conclusion he is hoping you will reach.

Duhigg provides a valuable resource, a body of knowledge that can help you do a better job with your patients. Having read this book, I see my job a bit differently; my task is to help my patients identify, preserve, and strengthen the positive habits in their lives and to alter their bad ones. ♦

A Film Concerned with Early-Onset Alzheimer's

review by Robin Sharan

Director of The Annapurna Center for Self-Healing

Still Alice, written by Lisa Genova and Richard Glatzer; directed by Richard Glatzer and Wash Westmoreland

Sony Pictures Classics

© 2014; 101 minutes

Alice is always running, living an overachieving, linear life as a professor of linguistics at Columbia University in upper Manhattan – seemingly the American Dream. There are three beautiful children and a loving husband. She goes to a neurologist when she feels that something is wrong with her. Diagnosing her as early onset Alzheimer's, the neurologist gives no support for recovery, no warmth, and no suggestions of out-of-the box thinking. He advised to continue her running routine; no suggestion of slowing down to practice a more nurturing activity such as yoga,

tai chi, or chi gong, instead of running in a very toxic urban environment, sucking up even more than her share of chemtrails and petrochemicals that of course worsened her condition.

Alice begs her husband to take a sabbatical to spend time together; but he instead escapes into a job that is a two-hour flight away from New York City. She does not accompany him, as she cannot cope with an unfamiliar environment as even the familiar is disintegrating before her very eyes. He doesn't have the fortitude to see his formerly energetic,

capable, brilliant, and beautiful wife disintegrate before his eyes, so the not-so-successful actress daughter comes to live with her mother.

There is no second opinion, no functional, Oriental, or Ayurvedic doctor consulted, no suggestion of changing Alice's standard American diet, and no water filter on the pretty little fashionable faucet in the kitchen.

When I visited my friend in New York whose mother was also a professor at Columbia, the water that came out of the bathtub spigot was rusty brown. I freaked out that I was bathing in such toxic water – and that was more than 50 years ago!

In a famous study of healing Alzheimer's, Dr. Mary Newport helped her stricken husband regain his brain function with coconut oil. Both are interviewed in January 2014 and can be viewed on YouTube ("How a Doctor Reversed Her Husband's Alzheimer's Disease in 37 Days").

Dr. Norman Walker, in his book *Colon Health*, reiterated that a short series of colonics twice a year prevents senility at any age. I personally met Dr. Walker when he was 100 years old; he had all his mental and physical faculties and died at age 109.

There are herbs and supplements such as *Bacopa monnieri*, vinpocetine, phosphatidylserine, acetylcholine, niacin, B6, *Gingko biloba*, and gotu kola, and so many more, all of which have positive effects on brain function. All one has to do is search the Internet on herbs for enhancing brain function. All the characters in the movie were all so intelligent and yet could not think outside the major paradigm.

Then there is the scene wherein Alice's neurologist encourages her to discuss the progression of her dysfunction in front of an Alzheimer's association. She received a standing ovation for her very poignant talk that seemed a bit all too positively reinforcing. Of all the people in that association, no one knew about Newport and how she helped heal her husband, or all the herbs and supplements that support brain function. It just seemed a bit preposterous to me.

Still Alice is a film with a name that seems to be a misnomer. Alice went

from success to senility due to environmental toxins and a society that doesn't think outside the box.

Thinking more peripherally in the production of this film could have been so much more satisfying and uplifting. When I left the theater, I could palpably feel the fear from the audience. I hope this review will shed light on a different approach.

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Melanoma on the Rise

Skin cancer (cutaneous melanoma) is the most common form of cancer in the US. The overall incidence of skin cancer increased nearly 8-fold during a 39-year period, among middle-aged men and women. Jerry Brewer and colleagues from Mayo Clinic (Minnesota, US) completed a population-based study using records from the Rochester Epidemiology Project, selecting participants aged 40 to 60 years with a first lifetime diagnosis of melanoma between January 1, 1970, and December 31, 2009. The researchers found that among white, non-Hispanic adults, the incidence of skin cancer increased 4.5-fold among men and 24-fold among women. In particular, women under age 50 showed a marked increase in melanoma. Overall chances of surviving melanoma increased by 7% each year of the study. Further, the researchers found the steepest increase in melanoma occurred in the last decade covered by



7 SKIN PROTECTING NUTRIENTS

A BOUQUET FOR BEAUTY

1 Green Tea

Exerts protective effects against sunburn inflammation & ultraviolet radiation damage.¹



2 Coffee



4 or more cups a day may lower a person's risk of malignant melanoma by as much as 20%.²

3 Omega-3 Fatty Acids

Modulate photoimmunosuppression in skin.³



4 Pine Bark

Contains leelamine, a compound that disrupts the cellular processes necessary for cancer cells to survive.⁴



5 Milk Thistle

Contains silybinin, an extract shown to kill skin cells mutated by UV-A radiation.⁵



6 Licorice

The root extract of *Glycyrrhiza inflata* (Chinese Licorice) is a source of Licochalcone A, shown to protect the skin from damage after UV irradiation.⁶



7 Aspirin

Large-scale study suggests that women who used aspirin for five or more years are at 30% lower melanoma risk.⁷



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7. Large-scale study suggests that women who used aspirin for five or more years are at 30% lower melanoma risk. *J Invest Dermatol*. 2004;123:1033-1038.

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To assemble a bouquet for beauty, consider these skin-protecting agents.

the study, 2000 to 2009. The uptick, researchers speculate, may be connected to the popularization of tanning beds in the 1980s and 1990s. The study authors conclude: "The incidence of cutaneous melanoma among middle-aged adults increased over the past 4 decades, especially in middle-aged women, whereas mortality decreased."

Lowe GC, Saavedra A, Reed KB, et al. Increasing incidence of melanoma among middle-aged adults: an epidemiologic study in Olmsted county, Minnesota. *Mayo Clin Proc.* 2014 Jan;89(1):52-59.

Early Sunburn Predicts Later Risk

Among women, frequent sunburns in your 20s may sharply raise your risks of future skin cancers. Abrar A. Qureshi and colleagues from Brown University (Rhode Island, US) assessed data collected on 108,916 women, aged 25 to 42 years at the study's start, enrolled in the Nurses' Health Study II. Following the participants for 20 years, the team observed that those who had at least five blistering sunburns when they were 15 to 20 years old were at a 68% increased risk for basal cell carcinoma and squamous cell carcinoma of the skin, and an 80% increased risk for melanoma. Those exposed to the highest amounts of cumulative ultraviolet radiation in adulthood had no increased risk for melanoma, but had a 2.35-fold and 2.53-fold increased risk for developing basal cell carcinoma and squamous cell carcinoma of the skin. The study authors submit: "In a cohort of U.S. women, we found that sun exposures in both early life and adulthood were predictive of [basal cell carcinoma] and [squamous cell carcinoma] risks, whereas melanoma risk was predominantly associated with sun exposure in early life."

Wu S, Han J, Laden F, Qureshi AA. Long-term ultraviolet flux, other potential risk factors, and skin cancer risk: a cohort study. *Cancer Epidemiol Biomarkers Prev.* June 2014 23:1080-1089.

Protect Skin with Sunscreen

Thus, it is imperative to wear sunscreen, the single most basic intervention for skin cancer. While it is generally accepted that sunscreen helps to minimize burning, whether sunscreen helps to prevent skin cancers has been the subject of some debate. Elke Hacker and colleagues from the Queensland University of Technology (Australia) have elucidated the molecular mechanism of sunscreen. The team confirmed previous findings that sunscreen protects against all three forms of skin cancer: BCC (basal cell carcinoma), SCC (squamous cell carcinoma), and malignant melanoma. Further, these researchers observed that sunscreen is effective at shielding the p53 gene, a gene that works to prevent cancer.

Hacker E, Boyce Z, Kimlin MG, et al. The effect of MC1R variants and sunscreen on the response of human melanocytes in vivo to ultraviolet radiation and implications for melanoma. *Pigment Cell Melanoma Res.* 2013 Aug 21.

Sun Lotion Also Slows Aging

And, the daily use of a broad-spectrum sunscreen slows, and may even prevent, sags and wrinkles – the hallmarks of aging skin. Maria Celia B. Hughes and colleagues from the University of Queensland (Australia) asked 903 Australian men and women, aged 55 years and younger, to use a broad-spectrum sunscreen daily and/or to consume a dietary supplement of beta-carotene (30 mg) daily. Subjects were followed for a 4-year period, with dermatological assessments conducted to analyze changes in skin appearance. The researchers found that the daily sunscreen group exhibited no detectable increases the aging at the end of the study term. Further, the subjects who used sunscreen daily showed 24% less skin aging, as compared with those who used sunscreen periodically. No effect was seen for beta-carotene supplementation.

Hughes MCB, Williams GM, Baker P, Green AC. Sunscreen and prevention of skin aging: a randomized trial. *Ann Intern Med.* June 4, 2013;58(11).

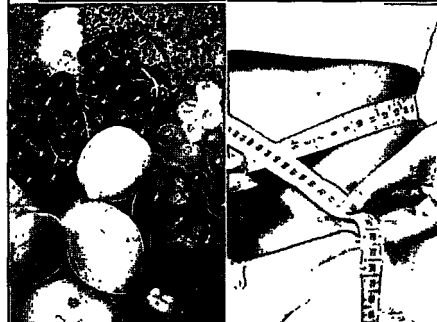
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JUNE 25-28: HEALTH FUSION- CANADIAN ASSOCIATION OF NATUROPATHIC DOCTORS NATIONAL CONFERENCE in Calgary, Alberta, Canada. CONTACT: https://www.cand.ca/Conference_Health_Fusion.healthfusion.0.html

JULY 8-12: VEGETARIAN SUMMERFEST 2015 in Johnstown, Pennsylvania. CONTACT: <http://vegetariansummerfest.org>

JULY 17-19: 21ST ANNUAL INTERNATIONAL INTEGRATIVE MEDICINE CONFERENCE in Melbourne, Australia. CONTACT: <https://www.aima.net.au/21st-annual-international-integrative-medicine-conference/>

JULY 31-AUGUST 2: HORMONE REPLACEMENT THERAPY SEMINAR (Session 1) with Dr. Neal Rouzier in Charlotte, North Carolina. CONTACT: <http://www.ducerecorp.com/Seminars.aspx>

AUGUST 5-8: AMERICAN ASSOCIATION OF NATUROPATHIC PHYSICIANS (AANP) 30TH ANNIVERSARY CONFERENCE in Oakland, California. CONTACT: <http://www.naturopathic.org/aanp2015>

AUGUST 19-22: 24TH ANNUAL IAACN SCIENTIFIC SYMPOSIUM – PREVENTIVE BIOCHEMICAL INTERVENTIONS & NOVEL THERAPEUTIC OVERTURES FOR THOSE WITH CANCER in Minneapolis, Minnesota. CONTACT: <http://www.iaacn.org/symposium/>

AUGUST 21-23: INTEGRATIVE ADDICTION 2015 in Myrtle Beach, South Carolina. CONTACT: 954-540-1896; Sharon@integrativeaddiction2015.com; <http://integrativeaddiction2015.com>

AUGUST 27-30: NORTHWEST HERB SYMPOSIUM – Botanicals at the Beach @ Camp Casey Conference Center, Whidbey Island, Washington. CONTACT: 425-868-0464 or 800-468-0464; info@treefarmtapes.com

AUGUST 29-30: WILLIAM REICH'S ORIGINAL ORGONOMIC DISCOVERIES FOUNDATIONS AND SCIENCE in Ashland, Oregon. CONTACT: <http://www.orgonelab.org/events.htm>

SEPTEMBER 5-7: 43RD ANNUAL ALTERNATIVE THERAPIES CANCER CONVENTION in Los Angeles, California. Doctor Symposium on **SEPTEMBER 8.** CONTACT: <http://www.cancercontrolsociety.com>

SEPTEMBER 9: TOUR OF MEXICAN CANCER CLINICS from Universal City, California. Also, **SEPTEMBER 12.** CONTACT: <http://www.cancercontrolsociety.com>

SEPTEMBER 11-13: CURING THE INCURABLES in St. Louis, Missouri. Fibromyalgia and chronic fatigue. CONTACT: <http://iamconf.com>

SEPTEMBER 14-15: 15th INTERNATIONAL CONFERENCE ON AYURVEDIC MEDICINE in Paris, France. CONTACT: <http://aapna.org/conferences/15th-conference-september-2015-paris-france>

SEPTEMBER 17-19: BIO-IDENTICAL HORMONE REPLACEMENT THERAPY SYMPOSIUM in New Orleans, Louisiana. Also, **NOVEMBER 19-21** in Vancouver, British Columbia, Canada. CONTACT: 561-893-8626; <http://www.A4M.com>

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SEPTEMBER 18-29: 16TH INTERNATIONAL CONFERENCE ON AYURVEDA & PSYCHIATRY in Vevay, Switzerland. CONTACT: <http://aapna.org/conferences/16th-conference-september-18-19-2015-switzerland>

SEPTEMBER 25-26: NEW FRONTIERS IN GI MEDICINE in Dallas, Texas. CONTACT: 561-997-0112; <http://www.a4m.com/2015-09-dallas-gi-symposium.html>

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OCTOBER 2-4: HORMONE REPLACEMENT THERAPY SEMINAR (Session 2) with Dr. Neal Rouzier in Chicago, Illinois. CONTACT: <http://www.ducerecorp.com/Seminars.aspx>

OCTOBER 9-11: 15th INTERNATIONAL CONFERENCE ON AYURVEDA & AUTOIMMUNE DISORDERS in San Jose, California. CONTACT: <http://aapna.org/conferences/15th-conference-october-9-11-2015-san-jose-ca-usa>

OCTOBER 14-17: MINDFUL PRACTICE ADVANCED WORKSHOP: Enhancing Quality of Care, Quality of Caring, and Resilience in Batavia, New York. For healthcare practitioners. CONTACT: <http://www.urmc.rochester.edu/family-medicine/mindful-practice/presentations-workshops.aspx>

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NOVEMBER 7-8: ARIZONA NATUROPATHIC MEDICAL ASSOCIATION 2015 FALL CONTINUING MEDICAL EDUCATION CONFERENCE in Scottsdale, Arizona. CONTACT: <http://www.aznma.org>

NOVEMBER 11-14: 56TH AMERICAN COLLEGE OF NUTRITION ANNUAL CONFERENCE in Orlando, Florida. CONTACT: <http://www.naturalhealthresearch.org/annual-conference/>

NOVEMBER 12-14: SOCIETY FOR ACUPUNCTURE RESEARCH 2015 CONFERENCE in Boston, Massachusetts. CONTACT: <http://www.acupunctureresearch.org/events>

NOVEMBER 12-15: AMERICAN FUNCTIONAL MEDICINE ASSOCIATION ANNUAL CONFERENCE in Atlanta, Georgia. CONTACT: 1-855-500-2362; <http://www.afmassociation.com/calendar/>

NOVEMBER 13-15: IGNITE CONFERENCE 2015 – The Business of Better Medicine in San Diego, California. CONTACT: <http://eeignite.com/coming-soon-the-business-of-better-medicine>

NOVEMBER 14-16: 12TH INTERNATIONAL CONFERENCE OF THE SOCIETY FOR INTEGRATIVE ONCOLOGY in Boston, Massachusetts. CONTACT: <http://www.integrativeonc.org/annual-international-conference>

NOVEMBER 19-22: 5TH ANNUAL PRO-AGING EUROPE CONFERENCE with Dr. Thierry Hertoghe in Brussels, Belgium. CONTACT: <https://www.weezevent.com/pro-aging-europe-2015>

DECEMBER 10-13: 23RD ANNUAL WORLD CONGRESS ON ANTI-AGING MEDICINE in Las Vegas, Nevada. CONTACT: 561-893-8626; <http://www.a4m.com/anti-aging-conference-lasvegas-2015-dec.html>

JANUARY 29-31: WORLD CONGRESS ON NATURAL MEDICINES in Tampa, Florida. CONTACT: http://www.smoch.org/world_congress_tampa.php

FEBRUARY 19-21: LDN 2016 CONFERENCE in Orlando, Florida. CONTACT: <http://www.ldn2016.com/townsend/>

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Women's Health Update

by Tori Hudson, ND
womanstime@aol.com

Omega-3 Fish Oils for Dry Eye and Comparing Inositols for PCOS

Oral Omega-3 for Dry Eye in Contact Lens Users

One of the most common but frequent consequences of hormonal changes associated with perimenopause and menopause is ocular dryness. In fact, the majority of those who suffer dry eye are midlife women and older. In one study, presented at the annual meeting of the North American Menopause Society, 96% of 582 women with dry eye symptoms were perimenopausal or menopausal. Unfortunately, only 27% of these women had actually been diagnosed with dry eye syndrome by a practitioner and most did not know that there was any relationship with menopause. In this same study, 95% of gynecologists and primary-care providers never asked about dry eyes during a medical history. Clearly, health-care providers need more information about this common and problematic health issue.

Contact lenses present a specific and unique problem for the eyes because prolonged exposure of a contact lens on the corneal surface reduces the thickness of the prelens lipid layer and increases the rate of tear evaporation. Contact lens wearers experience dry eyes quite frequently. Not only does this lead to discomfort and potential inability to wear the contact lenses, but prolonged use of contact lenses may lead to a decrease in conjunctival goblet cells and cell changes in conjunctival epithelial cells.

Omega-3 fatty acids from fish oils have been shown to be beneficial in several eye conditions, including age-related macular degeneration and dry eye syndrome. However, efficacy and safety of oral fish oils have not been documented for dry eyes related to contact lens use. The current study set out to see if omega-3 supplementation improved dry eye symptoms, contact lens comfort, goblet cell density, conjunctival epithelial cell changes, and clinical measures of tears.

A prospective, multicentric, randomized, double-blind interventional study was carried out at three eye centers in Northern India. Selected exams and tests were performed at the same time of day at each visit which occurred at baseline, 3 months, and 6 months. Contact lens wearers (n

= 496) were randomized to one of two groups: omega-3 capsules twice daily or placebo corn oil capsules twice daily for 6 months. The omega-3 fatty acid group received a dosage of two 300 mg capsules (1 cap = 180 mg EPA and 120 mg DHA) twice daily for 6 months.

The primary outcome was a decrease in subjective dry-eye symptoms and lens-wear discomfort after the 6 months of intervention. A symptom scoring system of 0 to 3 was used that included ocular fatigue, blurry vision, itching or burning, sandy or gritty sensation, and redness. A score of 0 meant no symptoms, a 1 meant sometimes present, a 2 meant frequently, and 3 meant always present. A total score of 0 to 6 represents mild, 6.1 to 12 moderate, and 12.1 to 18 is severe dry eye. The comfort related to lens wear was graded 0 to 6, with 6 being severe discomfort.

In the fish oil group, 22.3% of patients were mildly symptomatic, 72.7% moderately, and 5% severely symptomatic at baseline. After 6 months of treatment, 18% were asymptomatic, 76.5% mildly symptomatic, and 5.5% were moderately symptomatic. In the placebo group, 34.4% were mildly symptomatic, 61.7% moderately, and 3.8% severely symptomatic at baseline. After 6 months of placebo, 41.2% were mildly symptomatic, 56.3% moderately, and 2.5% severely symptomatic.

The mean improvement in symptom scores in the omega-3 fatty acid group was 4.7 compared with 0.5 in the placebo group. Lens wear comfort levels improved significantly in the fish oil group as well and was associated with a significant increase in tear film stability and increase in tear film production and a reduction in the number of blocked meibomian gland ducts in the fish oil group.

Comment: Previous studies have demonstrated that individuals who wear contact lenses have subclinical inflammation on the ocular surface, which may be correlated with dry eye severity and even corneal surface injury. With newer contact lens options, individuals are wearing their contact lenses for longer and longer periods of time, which can lead to a change in the lipid layer of the cornea and increase tear evaporation rates. The influence

of omega-3 fatty acids appears to work especially by decreasing inflammation and stimulating tear secretion. The dry eyes themselves are initiated by inflammation in some cases and low tear production in the rest. In addition, once the eyes become dry, inflammation sets in and the process progresses. Omega-3 fatty acids reduce inflammation by generating anti-inflammatory prostaglandins and by competitively inhibiting the elongation of omega-6 fatty acids, resulting in the inhibition of arachidonic acid. The increase in prostaglandin E1 stimulates tear production (and salivary gland secretion). Several studies have determined an association between the intake of omega-3 supplements and meibomian gland oils and the implication for dry eye disease. Flaxseed oil, sea buckthorn oil, and fish oil have all shown some efficacy in dry eye symptoms. (See my earlier column on dry eye syndrome published in the *Townsend Letter* October 2013 issue for more details.)

The results of the current study clearly demonstrate benefit with the use of omega-3 fatty acids in alleviating dry eye symptoms in contact lens wearers. I would encourage consumers, patients, and clinicians to use the current study as a guide regarding dosage, both in the total amount of omega-3 and in the specific amounts of EPA and DHA: two 300 mg capsules (1 cap = 180 mg EPA and 120 mg DHA) twice daily for 6 months for a total day's amount of 720 mg EPA and 480 mg DHA.

Bhargava R, Kumar P. Oral omega-3 fatty acid treatment for dry eye in contact lens wearers. *Cornea*. 2015;34:413-420.

Comparison of Myo-Inositol and D-Chiro-Inositol in PCOS Women

Both myo-inositol and D-chiro-inositol have been shown to affect ovarian function and metabolic factors in women with polycystic ovarian syndrome (PCOS). They have been shown to improve androgen levels, increase the action of insulin, reduce systolic blood pressure, and more.

The purpose of the current study was to compare the effects of myo-inositol and D-chiro-inositol in women with PCOS. Fifty women with a diagnosis of PCOS according to the Rotterdam criteria were enrolled. They were randomized into two groups; 25 were treated with 4 g of myo-inositol plus 400 mcg of folic acid daily for 6 months, and the other 25 were treated with 1 g of D-chiro-inositol plus 400 mcg folic acid per day.

In the myo-inositol group, there were statistically significant reductions of diastolic and systolic blood pressure; lowering of luteinizing hormone (LH); lowering of the LH/FSH (follicle stimulating hormone) ratio; and lowering of total testosterone and free testosterone, androstenedione, prolactin, and the HOMA index (homeostatic model assessment to check for insulin resistance). These same patients also had a statistically significant increase of sex hormone-binding globulin (SHBG) and the glycemia/immunoreactive insulin ratio.

In the D-chiro-inositol group, there was a statistically significant reduction of systolic but not diastolic blood pressure and a statistically significant reduction of

the Gallwey-Ferriman Score (a measure of hirsutism), LH, LH/FSH ratio, total testosterone, free testosterone, androstenedione, prolactin, and the HOMA.

Both inositols reduced systolic blood pressure, LH, LH/FSH ratio, circulating androgens, and prolactin, and increased insulin sensitivity and SHBG. Myo-inositol may decrease the LH/FSH ratio, total testosterone, and the HOMA in a more statistically significant way. D-chiro-inositol is likely to reduce mostly, but not statistically significantly, the LH and free testosterone levels and may increase, but not significantly, the glycemia/IRI ratio.

It could be concluded from this comparison that both the inositol isoforms are effective in improving the ovarian function and metabolism of women with PCOS, although myo-inositol showed the greater impact on the metabolic profile and D-chiro-inositol affected more positively the hyperandrogenism measurements. In comparing the two products pre- and posttreatment, there was a higher regularization of menstrual cycles in those treated with D-chiro-inositol compared with those with myo-inositol, although this was not statistically significant.

Comment: PCOS is one of the most common endocrine disorders in reproductive-aged women. The majority of women with PCOS (about 74%) do not ovulate, almost half (about 42%) have insulin resistance, and almost half (48%) have hyperandrogenism. It's important to remember that PCOS is a syndrome – and not all women with PCOS have any one sign or symptom. Not all actually have multiple cysts on the ovaries, not all have excess body hair, and not all have abnormal menstrual cycles. In women with PCOS, though, the insulin resistance is commonly associated with hyperinsulinemia, which then enhances the production of androgens by the ovarian theca cells, leading to a reduction in circulating levels of SHBG, which leads to increased levels of free testosterone. Nutritional, lifestyle, supplemental, and pharmaceutical strategies try to address the syndrome by targeting this core issue of improving insulin sensitivity, which thereby addresses the signs and symptoms of PCOS. Both myo-inositol and D-chiro-inositol, in the doses used in this study, improve ovarian function and metabolism in PCOS, but myo-inositol showed the most effect on the metabolic profile and D-chiro-inositol reduced the hyperandrogenism better.

Pizzo A, Lagana A, Barbara O. Comparison between effects of myo-inositol and D-chiro-inositol on ovarian function and metabolic factors in women with PCOS. *Gynecol Endocrinol*. 2014;30(3):205-208.

Dr. Tori Hudson graduated from the National College of Naturopathic Medicine (NCNM) in 1984 and has served the college in many capacities over the last 28 years. She is currently a clinical professor at NCNM and Bastyr University; has been in practice for over 30 years; and is the medical director of the clinic A Woman's Time in Portland, Oregon, and director of research and development for Vitanica, a supplement company for women. She is also a nationally recognized author, speaker, educator, researcher, and clinician.

Huge Vitamin C Price Increase May Be a Blessing in Disguise for Cancer Patients, Says Vitamin C Foundation

With the recent price jump in sterilized vitamin C for injection, the Vitamin C Foundation shares that the sharp increase may be better for patients in the long term. Doctors who utilize intravenous vitamin C therapy will be spurred to seek out lower-cost options that may work even better.

"Prices have more than quadrupled overnight," says Vitamin C Foundation founder and CEO Owen Fonorow. Factors that have contributed to the price increase include the cost of ingredients, new regulations and restrictions, cost of sterilization and insurance, loss of economy of scale by manufacturers, and increased demand. These changes make it unlikely that pharmacies will be able to offer vitamin C injections at a lower cost in the future.

Intravenous vitamin C (IV/C) is widely used in the treatment of cancer, but it is often an out-of-pocket expense for patients, meaning that even small price increases have serious ramifications. The Vitamin C Foundation believes that doctors will then be motivated to look for lower-cost options and may return to the ways of the early pioneers of vitamin C therapy, which could be a blessing in disguise.

Vitamin C pioneers, such as Fredrick Klenner, MD and Robert Cathcart III, MD, would mix the vitamin for infusion just prior to the intravenous administration, keeping the vitamin fresh. Their methods may be vastly more potent than today's commercial vials, where the vitamin C is in solution for long periods on the shelf. The results of the early pioneers using vitamin C intravenously were so profound that two-time Nobel Laureate Linus Pauling began championing the cause of vitamin C.

For strictly legal reasons, most doctors have preferred to purchase sterile solutions from national pharmaceutical companies rather

than mixing the vitamin C with stock solution shortly prior to use. According to the late Cathcart, the world expert, the sterility of the stock solution did not worry him "because sodium ascorbate in solution is very bactericidal." Therefore, there is nothing better than vitamin C itself to sterilize the mixture. More than 14,000 patients from around the world received their intravenous vitamin C from Cathcart.

"Safety has never been an issue with intravenous vitamin C, even at dosages as high as 200 grams," says Fonorow. "The new regulations must have more to do with protecting pharmaceutical interests than the public health."

By mixing prior to infusion, the resulting vitamin C solution is stronger, whereas the foundation has estimated that the premixed commercial vials of vitamin C for injection may lose 50% potency on the shelf. Even a compounding pharmacist's solution will experience a loss in potency.

Another problem is that the commercial firms offer the premixed products in vials for injection marked "ascorbic acid." This mislabeling can be confusing, especially for doctors new to IV/C therapy, because these labels imply that ascorbic acid can be used intravenously. If the company is responsible, these products labeled ascorbic acid for injection are buffered in accordance with the US Pharmacopeia, but they are still premixed, allowing the ascorbate to rapidly break down by at least 50% in 4 hours.

Experts such as Cathcart told the foundation that they recommend using only sodium ascorbate intravenously. Cathcart recommended using major brands of sodium ascorbate powder, such as Bronson or Wholesale Nutrition sodium ascorbate, but the foundation recommends using the China-free DSM QUALI-C sodium ascorbate powder.

The Vitamin C Foundation Approved sodium ascorbate (Cathcart's) formulation is amazing. Again, I was shocked at how superior it was compared to the other vitamin C powder I was using. Patients noticed it; I noticed it ... pretty incredible.

— Daniel CK, MD, Boston, Massachusetts

The DSM QUALI-C (Cathcart's) sodium ascorbate powder approved by the foundation is less than \$50 for 250 grams, versus the \$100 from a compounding pharmacist, and \$375 for the equivalent in standard vials.

For more information on the preparation and use of intravenous vitamin C, please view this tutorial video: <https://www.youtube.com/watch?v=Zgi-7xPrCAg> or read this document: <http://www.vitaminfoundation.org/pdfs/civprep.pdf>. Visit our forum conversation on this topic: <http://www.vitaminfoundation.org/forum/viewtopic.php?f=21&t=11811>.

About the Vitamin C Foundation

The Vitamin C Foundation is a nonprofit, charitable corporation devoted to preserving the vital knowledge about ascorbic acid and its role in life and use in medicine. Headquartered in Illinois, the foundation is dedicated to the memory of Linus C. Pauling. The organization has been assigned the IRS tax-exempt 501(c)(3) designation and its activities are funded by charitable contributions.

Vitamin C Foundation Approved is a registered trademark of the Vitamin C Foundation. QUALI-C is a registered trademark of DSM Nutritional Products.

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office: 630-983-6707

Integrative Therapeutics Introduces two ProThrivers Wellness Supplements with specialty ingredients from Kyowa Hakko USA

Integrative Therapeutics has introduced two new supplements focused on two key pathways: immunity and cognition.

A unique combination of herbs and nutrients, including Setria Glutathione, helps set ProThrivers Wellness Flavonoid Complex apart from other antioxidant supplements. This formula relies upon the combination of ingredients to support antioxidation versus the use of single or sequential compounds.* It unites curcumin, green tea extract, resveratrol, Alpha-Glycosyl Isoquercitrin, and coenzyme Q10 with Setria Glutathione.

Glutathione is the major low-molecular-mass thiol compound in plants and animals. Although some researchers have questioned whether oral glutathione is adequately absorbed, a randomized controlled trial demonstrated that oral glutathione (Setria brand) supplementation raised tissue and blood levels of glutathione in a dose-dependent manner in healthy adults.*

For more information about ProThrivers Wellness Flavonoid Complex, visit <http://www.integrativepro.com/Products/Vitamins-Minerals/ProThrivers-Wellness-Flavonoid>.

ProThrivers Wellness Brain contains Cognizin Citicoline plus Theracurmin, *Hericium erinaceus* (lion's mane mushroom), and acetyl-L-carnitine. All individually support brain health; combined in ProThrivers Wellness Brain, they are intended to support cognitive clarity.*

Citicoline is a compound that promotes the production of phosphatidylcholine, which is important for brain function. Clinical research has shown that citicoline has multiple applications and is able to improve various aspects of the brain's physiological activity.*

For more information about ProThrivers Wellness Brain, visit <http://www.integrativepro.com/Products/Vitamins-Minerals/ProThrivers-Wellness-Brain>.

About ProThrivers Wellness

The ProThrivers line of products has been specifically designed with the guidance of Lise Alschuler, ND, FABNO, a respected naturopathic doctor for 20 years and an expert in thriver care. Her present clinical focus is on addressing the needs of this specific population of thrivers using evidence-informed strategies. In addition, Dr. Alschuler is a thriver herself and uses her own experience to better understand their unique challenges.

About Integrative Therapeutics

Located in Green Bay, WI, Integrative Therapeutics LLC (integrativepro.com) is a leading manufacturer and distributor of science-based nutritional supplements committed exclusively to health-care professionals. Integrative develops unique formulations that can be safely and effectively integrated with diet and lifestyle recommendations for improved patient health. For over 35 years, the company's dedication to social responsibility and charitable giving has been evidenced by their commitment to supporting medical schools, community clinics, and professional associations. In partnership with its medical advisory board, Integrative has developed professional resources, education programs, and patient-centered therapeutic programs to help cultivate healthy practices and advance the field of integrative medicine.

About Setria Glutathione

Setria Glutathione, manufactured by Kyowa Hakko USA, is a clinically studied form of glutathione that, when taken orally, has been shown to replenish the body's reserves, which may be depleted as a result of poor lifestyle choices, stress, or natural aging.¹ Called the "master antioxidant," glutathione helps protect cells in the body from the damaging effects of oxidative stress and toxins. Setria Glutathione is manufactured through a patented fermentation and patent pending for increasing natural killer (NK) cell activity and is pure, vegetarian, and allergen free. For more information about Setria Glutathione, visit www.setriaglutathione.com.

Notes

1. Richie JP Jr, Nichenametta S, Neidig W, Calcagnotto A, Haley JS, Schell TD, Muscat JE. Randomized controlled trial of oral glutathione supplementation on body stores of glutathione. *Eur J Nutr*. May 2014. doi:10.1007/s00394-014-0706-z.

*These statements have not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure, or prevent any disease.

** Setria is a registered trademark of Kyowa Hakko Bio Co. Ltd.

*** ProThrivers is a trademark of Integrative Therapeutics LLC.

About Cognizin

Cognizin is a branded form of citicoline, a natural substance found in every cell of the body and especially vital to brain health.* Citicoline is broken down during intestinal absorption and, after passing through the blood-brain barrier, is reconstituted in the brain as citicoline.* Citicoline is a water-soluble compound that supplies precursors for the synthesis of phospholipids, including phosphatidylcholine, a major constituent of brain tissue*; helps maintain normal levels of acetylcholine, a chemical that regulates memory and cognitive function*; enhances communication between neurons*; supports visual function*; protects neural structures from free radical damage*; enhances metabolism and healthy brain activity*; and helps sustain healthy cellular mitochondria for sustained energy*. Cognizin is also highly stable, GRAS, ultrapure, and allergen free. For more information on Cognizin, visit <http://www.cognizin.com>.

*These statements have not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure, or prevent any disease.

About Kyowa Hakko USA

Kyowa Hakko USA is the North American sales office for Kyowa Hakko Bio Co. Ltd., an international health ingredients manufacturer and world leader in the development, manufacturing, and marketing of pharmaceuticals, nutraceuticals, and food products. Kyowa is the maker of branded ingredients including Cognizin Citicoline, Lumistor L-Hydroxyproline, Pantestin Pantethine, and Setria Glutathione, as well as Sustamine L-Alanyl-L-Glutamine. For more information, visit <http://www.kyowa-usa.com>.

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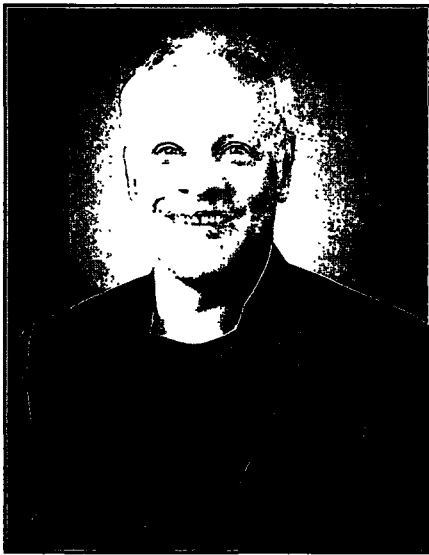
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How Your Food Is Cooked May Affect Your Health

Considering that we existed before we discovered fire, it would be reasonable to assume that humans were designed to subsist on raw foods. There is evidence that consuming more raw foods can have various health benefits, including improvements in hypertension, rheumatoid arthritis, and fibromyalgia; a reduction in inflammation; and an increase in antioxidant activity.

Of course, cooking foods has some beneficial effects. For example, cooking kills pathogenic microorganisms and inactivates undesirable substances in certain foods (such as the trypsin inhibitor in soybeans). However, cooking also has deleterious effects, particularly when foods are heated to high temperatures. Cooking can alter the structure of proteins, thereby decreasing their digestibility and potentially increasing their immunogenicity. Moreover, a number of toxic compounds are produced during cooking, including lipid peroxides, polymerized fats, cholesterol oxides, advanced glycation end products, heterocyclic amines, polycyclic aromatic hydrocarbons, and acrylamide. Each of these compounds may play a role in the pathogenesis of one or more chronic diseases (including

cardiovascular disease and cancer), and some may accelerate the aging process. In addition, cooking can lead to the destruction or leaching of essential nutrients, thereby lowering the nutritional value of food. Thus, the manner in which foods are cooked may be an important determinant of whether those foods are beneficial or harmful.

Advanced Glycation End Products (AGEs)

Perhaps the best studied of the molecules formed during harsh cooking are advanced glycation end products (AGEs). These compounds are produced by a reaction between a reducing sugar (glucose, fructose, or lactose) and a protein or amine-containing lipid. AGEs are absorbed from food intact and persist in tissues, where they can modify protein structures. AGEs appear to promote inflammation and insulin resistance, both of which play a role in the pathogenesis of many chronic illnesses. In addition, AGEs are thought to contribute to the development of cardiovascular disease, diabetes complications, and fibromyalgia, and to accelerate the aging process.^{1,2}

AGEs, Diabetes, and Inflammation

In an animal model of type 1 diabetes, feeding a low-AGE diet (as compared with a standard diet) significantly decreased the incidence and delayed the onset of diabetes, decreased the degree of inflammation of pancreatic islet cells, decreased the severity of nephropathy, and prolonged survival.^{3,4} Consumption of a low-AGE diet also improved insulin sensitivity, decreased islet cell destruction, reduced the severity of nephropathy, and promoted weight loss in an animal model of type 2 diabetes and obesity.⁵

The potential of AGEs to promote inflammation and insulin resistance has also been demonstrated in humans. In one study, 74 overweight women were randomly assigned to consume a diet high or low in AGEs. The difference in dietary AGE content was achieved primarily by differences in cooking methods. Compared with the high-AGE diet, the low-AGE diet significantly decreased insulin resistance, as determined by the homeostasis model assessment of insulin resistance (HOMA-IR).⁶ In another study, 13 patients with diabetes were randomly assigned to consume a diet for 6 weeks that was high or low in AGE content. The two diets had a similar amount of protein,

carbohydrate, and fat, but differed by approximately 5-fold in AGE content, which was achieved by varying the cooking time and temperature. After 6 weeks, the mean C-reactive protein concentration (an indicator of inflammation) increased by 35% relative to baseline on the high-AGE diet and decreased by 20% relative to baseline on the low-AGE diet ($p < 0.02$ for the difference between groups). Levels of other inflammatory mediators also increased on the high-AGE diet and decreased on the low-AGE diet.⁷

How to Reduce the Amount of Dietary AGEs

Raw foods are virtually devoid of AGEs, so eating more raw foods will decrease AGE intake. Cooking at lower temperatures and in the presence of water results in relatively little AGE formation, whereas more

AGEs are formed when foods are cooked at higher temperatures and in the absence of water. Temperature and cooking method seem to be more important factors for AGE formation than cooking time. Emphasizing boiling, poaching, and stewing over frying, broiling, and roasting may decrease daily AGE intake by up to 50%. Microwaving foods increases their AGE content only modestly, to about the same extent as boiling.⁸

High-fat and high-protein foods (such as meat and cheese) tend to have the highest AGE content. High-carbohydrate foods typically contain much less AGEs. However, within the carbohydrate group, commercially prepared breakfast foods and snacks have a high AGE content compared with oatmeal. This relatively high AGE content is presumably due to the fact that processing of some ready-to-eat cereals includes heating to

temperatures over 230°C. In addition, many cereals and snack foods undergo an extrusion process under high pressure, which can promote the formation of AGEs.⁸

Alan R. Gaby, MD

Notes

1. Hein G, Franke S. Are advanced glycation end-product-modified proteins of pathogenetic importance in fibromyalgia? *Rheumatology*. 2002;41:1163-1167.
2. Ruster M et al. Detection of elevated N epsilon-carboxymethyllysine levels in muscular tissue and in serum of patients with fibromyalgia. *Scand J Rheumatol*. 2005;34:460-463.
3. Peppas M et al. Fetal or neonatal low-glycotoxin environment prevents autoimmune diabetes in NOD mice. *Diabetes*. 2003;52:1441-1448.
4. Zheng F et al. Prevention of diabetic nephropathy in mice by a diet low in glycoxidation products. *Diabetes Metab Res Rev*. 2002;18:224-237.
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The Institute for Functional Medicine and the Personalized Lifestyle Medicine Institute Team Up to Present Free, Six-Part Series

The Institute for Functional Medicine (IFM) is pleased to announce an exciting, six-part series in partnership with Dr. Jeffrey Bland and his Personalized Lifestyle Medicine Institute (PLMI): "Functional Medicine and Genomics: Is the Future Here?" IFM's 2015 Annual International Conference, The Omics Revolution: Nature and Nurture, will serve as the launch site for the first presentation in the series, "Functional Medicine and Genomics: A Match Made in Heaven." The remaining presentations will run monthly through December 2015. All presentations will be available for free on IFM's website and will include links to outside resources for additional information.

In this series, Bland will take viewers on an extended journey into genomic applications in functional medicine, creating a vision of health care that includes a deep understanding of how to help patients achieve true wellness and resilience using real-time, personalized information. Through such fascinating and clinically relevant topics as epigenetics, genomic testing, and genomic applications in cancer, we will learn how genes get messages from interactions with our environment and our behaviors, translating those messages into cellular instructions that guide our trajectory toward health or disease. No one can deliver this inspiring message better than Jeffrey Bland: genetic inheritance is not fate – we have the opportunity and the power to shape our own patterns of health and longevity, and we can do it now.

"I am so pleased to be a part of the development of this six-part series on the application of the 'omics' revolution to functional medicine. This series will be an excellent follow-on to the topics covered at the Annual International Conference and will provide news to use as it relates to the creating of a personalized approach to the prevention and treatment of chronic disease," says Bland.

IFM Chief Executive Officer Laurie Hofmann, MPH, adds, "We are thrilled to collaborate with Dr. Jeffrey Bland, one of the principal thought leaders of the functional medicine movement, to offer this in-depth exploration of genomics and functional medicine."

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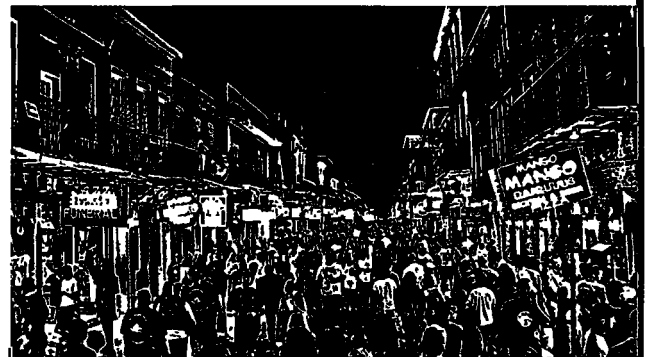
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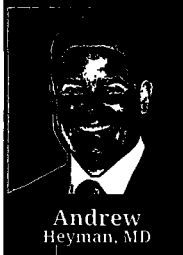
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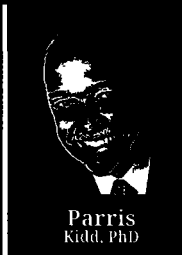
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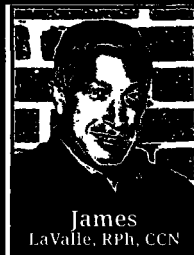
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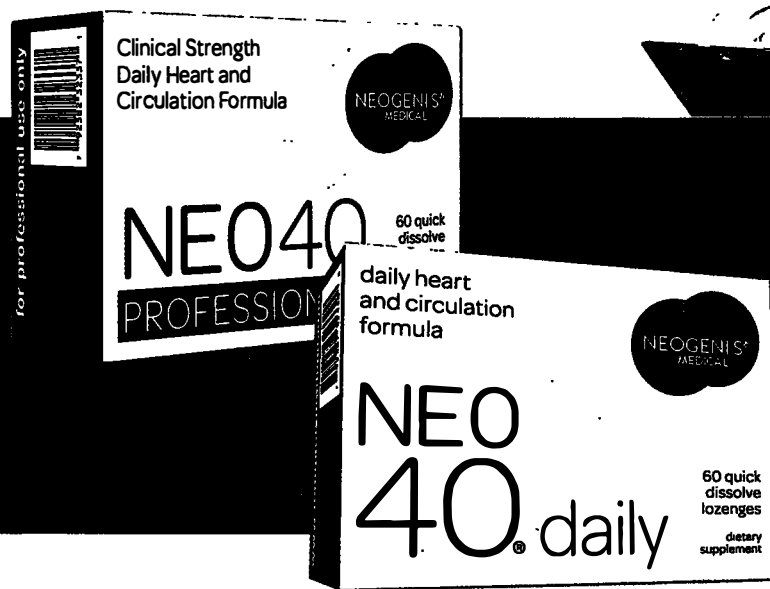
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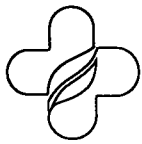


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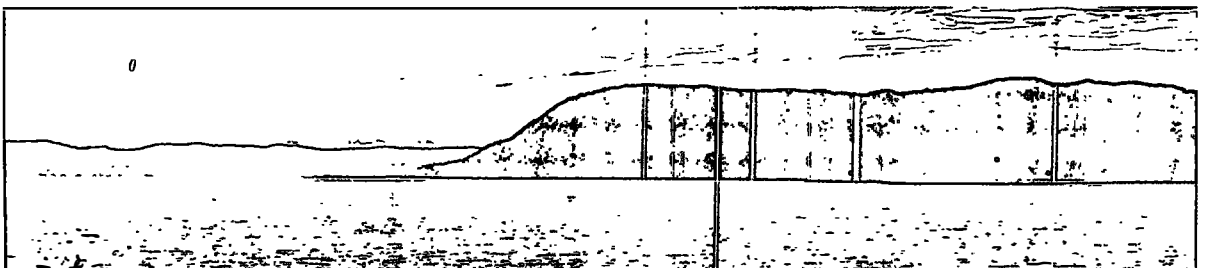
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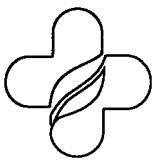
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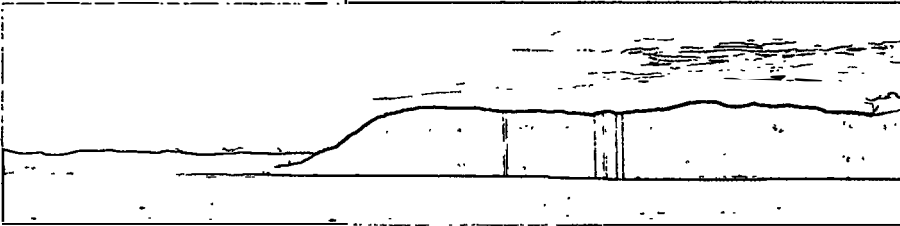
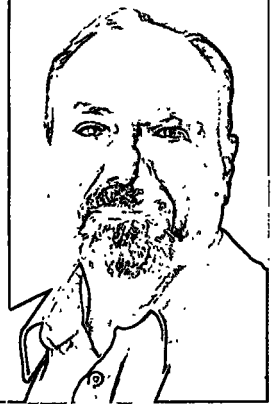


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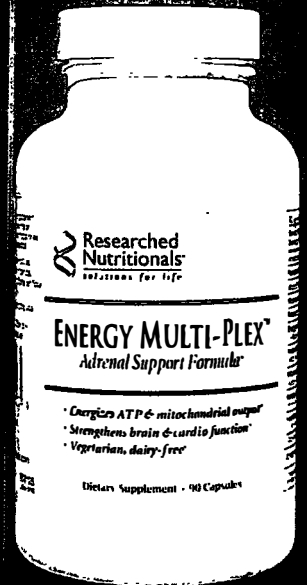
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Sugars	5g	**
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D-Ribose	5g	**
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L-Glutamine (free form), L-tyrosine, Phosphatidyl Serine, Huperzine A		
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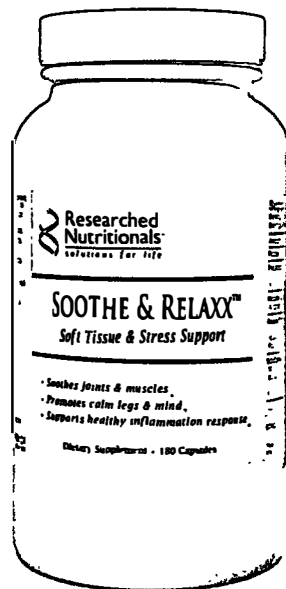
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Glucosamine Sulfate, MSM, Chondroitin Sulfate, Hyaluronic Acid		
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