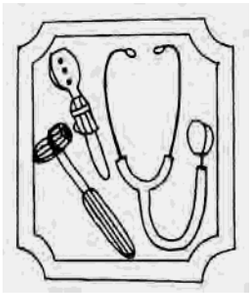

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From the Editor's Desk

by: Jack Kessinger, DC, ND, DABCI
jack@drkessinger.com

We all know that patients respond to a natural approach to most healthcare problems more successfully than forcing the body to react to a chemical or drug. I have always maintained that *medicine and drugs may save your life, but they will never make you healthy*. I tell my students that if I am in a car crash, I don't want to be given Vitamin C, I want to be taken to a hospital. However, if I am ill, I expect to be treated safely and naturally!

The discovery of specific dietary insufficiencies, causing, or acting as a determining factor to the development of common illnesses played a central role in the development of modern nutritional science[1]. It has long been recognized that nutritional deficiencies contributed to many diseases. One of the first recorded clinical trials was in 1773. That trial proved that consuming citrus fruit would prevent scurvy in sailors while on long ocean voyages. This is the first documented observation leading to the recognition that nutritional deficiencies would cause, or contribute to, any chronic and degenerative health issues.

However, it wasn't until 1941 that the first recommended dietary allowances (RDA's) were formulated, which established a scientific basis for modern dietary guidelines[2] and are followed to this day. These efforts set a precedent for the creation of guidelines by first considering nutrient targets and then translating these targets into food recommendations. The Dietary Guidelines for Americans have been published jointly every five years since 1980 by the Department of Health and Human Services (HHS) and the Department of Agriculture (USDA). The Guidelines provide authoritative advice for people two years and older about how healthy dietary habits can promote health and reduce risk for major chronic diseases. They serve as the basis for Federal food and nutrition education programs.

Much more recently, The Dietary Guidelines for America, 2005, reported that "Eating right and being physically active" are necessary for good health, including cardiovascular health. And breaking with a long tradition, the FDA recently ruled food companies can now make certain claims about the heart-healthy benefits of omega-3 fatty acids on their packages. The decision by federal regulators not only provides what is effectively an additional endorsement of these so-called "healthy fats," but will allow consumers to more easily identify brand-name products that contain them.

What a concept! I have been singing this song to my patients for nearly 50 years. EFA's are a part of holistic protocols for cardiovascular disease. As a matter of fact, many holistic health care providers recommend dosages daily as preventative health care.

However, the fact that optimum health demands adequate nutritional support from dietary and nutritional supplementation has been bullied into submission by pharmaceutical companies, and modern medicine by proxy.

The FDA has concluded that while these particular fatty acids are not essential to the diet they may be beneficial in reducing coronary heart disease," said FDA acting commissioner Lester Crawford.

While the agency limited the claims to two omega-3 acids, known as EPA and DHA, these two fatty acids are frequently and well documented, reported for their potential health benefits.

However, a third fatty acid, alpha linoleic (ALA), found in some leafy vegetables, vegetable based oils, and nuts was not included. Alpha linolenic acid is referred to as an essential fatty acid because it cannot be produced within the body and must be acquired through the diet. It is for preventing and treating diseases of the heart and blood vessels. WebMD reports it is also useful for treating rheumatoid arthritis (RA), multiple sclerosis (MS), lupus, diabetes, ulcerative colitis, and Crohn's disease.

The Health and Human Services (HHS) agency said it determined there was enough scientific evidence to allow companies to make what is known as qualified health claims, which can be included on food labels based on a preponderance of research results and usually refer to a specific heath benefit. In the case of the Omega-3s, the FDA will allow claims that discuss heart health.

In addition, several studies report that, according to a study in mice, "eating a diet rich in a certain type of omega-3 fatty acids may slow or prevent Alzheimer's disease." Researchers, reported in the Journal of Neuroscience, found that feeding mice that were specifically bred to develop Alzheimer's disease a diet rich in docosahexaenic acid (DHA) slowed the accumulation of two proteins associated with the brain-clogging plaques and tangles implicated in the disease.

It takes science and big pharma a long time to accept that natural therapies are often a superior method of health care.

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The Legacy Continues

Dr. Jay Kessinger

by: A. Jay Kessinger IV, DC, ND, DABCI
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These are just a few questions that health care providers ought to be asking. What are we selling? How are we selling it? Are we selling out? Contemplating these questions, here are my answers for us as a whole.

We are selling a service, our expertise, to provide people with the tools of knowledge to get better, stay better, and live healthier. There are so many people to provide for, that there is truly no need for us to rough-shod over one another; however, all too often it's a dog-eat-dog world even within our professional circle. It appears the line of acceptability is often blurred when you think, "How low can you go to assure your competitions' cut of the economic health care pie?" The best that we can do is, speak the truth and back it up with referenced research and a sound reasoning that our patients can understand.

To quote Glenn Beck, "I've never seen anyone go to Washington (DC) and come back a better person." Apparently, in order to get anything done in that town, you not only have to stay in line, but you have to allow the winds of power to erode your own values; potentially to the point of being unrecognizable to most by-standers, unless you were a snake to begin with, and then, what should people expect? Being a mid-term election year, I find this analogy to the predicament we find ourselves in on a daily basis, to be apropos. As a sign I saw at a 'Señor Frogs' says, "If you don't like our service please lower your standards;" we are, as a whole, lowering standards/principles in our treatment of the masses for the supposed 'greater good' of being able to sell more to more of the general public and at a bigger profit. Profit is good when merited, as it is the fruit of harvest for a job well done. Unmerited profit is not only short lived, but it also spoils the field for all workers; diligent and indigent alike.

A few examples of the aforementioned are the presently accepted insulin dependents' prescribed dietary intake of "eat whatever you want and adjust your pump accordingly," and the "if my insurance won't cover it, I can't afford it," mentality. The former example given is the most ludicrous and directly detrimental to the unknowing professional health care provider advice taker. Insulin is not an independent, docile, glucose-lowering element. It is a powerful insulin receptor stimulant, an energy storage enhancer and, according to many current studies, a longevity depriver. The second example of the lowering of our standards to contour to the patients is from the overall concept that health insurance covers all necessary health care. As an example, Medicare insurance, after the age of coverage has personally been reached, automatically deems itself the primary insurer; therefore, whatever Medicare says is not a coverable treatment procedure is also not covered by any other 'supplemental' policies.

An example I like to use to illustrate my point about health insurance is to compare it to car insurance. Car insurance is catastrophic insurance, period. If you are involved in an accident your insurance company will buy you a new car, etc. However, it will not change your oil, buy you tires or provide any other service or parts that are routinely necessary to maintain the proper running/driving condition of your car.

Health insurance, on the other hand, has become so expensive, because so much has been put on its plate of responsibility by the providers, truth be told, more so than the consumers, that the health care consumers truly believe that 'if their insurance won't cover it, then they can't afford it.'

Do we really think that patients' can ever reach a point that diet and exercise is not enough? What kind of line is, "whenever diet and exercise are not enough?" It is an advertising ploy to sell a pharmaceutical product; i.e., statin drugs et.al. These treatments relieve the burden of care from the primary culprit, which is the patient, but all the while using misinformation, and deceit to direct them away from taking personal responsibility for the destruction, as well as, toward the rebuilding of health that lies ahead.

We are selling a necessary, yet all too often watered down service to bolster the quality of life by providing those we have the privilege to serve the very best care modern science has to offer in a professional and ethical manner.

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Select Herbals Targeted to Eradicating Gastrointestinal Dysbiosis

by: Rachel Olivier, MS, ND, PhD

Dysbiosis is the classic term for an imbalance of gastrointestinal microflora, indicating an increase in abnormal or noncommensal flora, with a coinciding decrease in commensal or normal flora. An increase in pathogenic bacteria including *Shigella flexneri* and *Salmonella enteritidis*; opportunistic bacteria including *Klebsiella pneumoniae*, *Escherichia coli*, *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Clostridium difficile;* and yeasts, including *Candida albicans* in the lower bowel is typically associated with dysbiosis.[1] In addition to the intestinal tract, dysbiosis of the mouth is also known to occur, and is associated with dental carries.[2] There are numerous factors correlated with dysbiosis, including a poor diet, physical and/or psychological stress, and the overuse of antibiotics, which in turn results in depressed immunity. Psychological stress has been demonstrated to decrease the level of secretory IgA, resulting in decreased mucosal immunity.[3] In addition to other coinciding factors, intestinal dysbiosis has been implicated as the root cause of bowel inflammation.[4] The root cause of many chronic degenerative diseases is correlated to the health of the bowel; consequently optimizing bowel health offers a significant advantage for long lasting health benefits.

Select herbs are well recognized in promoting the synergistic healing of damaged intestinal tissue, resulting predominately from dysbiosis. These herbs include:

Stemona sessilifolia (root) - The active principals of *Stemona* are its alkaloids. These alkaloids exert antifungal, antibacterial and pesticidic properties. It is typically indicated for acute and chronic cough; cough in phthisis (wasting syndrome), whooping cough, cough occurring with or after the common cold, and for cough due to exopathogens. Its action is said to be warm in nature, rather than dry, and its use is considered calming to the entire respiratory center. It also has proven effectiveness for the eradication of louse, parasites,[5] and worms (pinworms).[6,7,8]

Artemisia absinthium, *Wormwood* (shoots, leaves) – In Traditional Chinese Medicine (TCM) *Artemisia* has been used as an antiparasitic agent for more than 1,000 years,[9] as well as an antihelmintic since primordial times. Its parasitic properties are attributed partially to its α-santonin content.[10] It is also regarded as a potent and rapidly acting antimalarial herb.[11,12] Its primary actions are noted to include cholagogue (inducing bile flow), digestive, appetite stimulating and wound healing, of which all are attributed to its essential oils and amaroids.[13] Following ingestion, the artemisinins are rapidly absorbed and subsequently penetrate the blood-brain barrier, and as in the case of malaria, accumulate into parasite infected erythrocytes. In turn these parasite infected erythrocytes are phagocytized by the leukocytes, thus subsequently eliminated.

In addition to its antiparasitic properties, the essential oil also possesses antimicrobial activity. *In vitro*, its use has been demonstrated to retard the growth of the parasite *Plasmodium falciparum*,[14] and has a confirmed 94.5% success rate in hookworm eradication.[13] It has also been demonstrated to exhibit hepatoprotective activities, partially via its inhibition of microsomal drug metabolizing enzymes (MDME).[15]

Artemisia intake has also been demonstrated to have an action in the stimulation the bitter receptors in the taste buds of the tongue, which in turn triggers a reflexive increase in stomach acid secretion. With intake a significant increase in the production of alpha-amylase, lipase, and other digestive secretions has been demonstrated.[16] Bitter taste receptor activation has been associated with a rapid change in the level of second messengers. Recent research has correlated the ingestion of bitter stimuli with an initiation of both cellular and molecular responses in the endocrine cells of the GI tract, postulating that "some elements of taste-specific signaling are operative in enteroendocrine cells."[17]

Brucea javanica (fruit) – The active constituents of *Brucea javanica* are the quassinoid compounds bruceantin and brucein C.[18] It possesses properties designated as beneficial to multiple bodily systems, including the digestive and circulatory systems, and the large intestines. Both the roots and fruits of *Brucea javanica* are used as popular agents against diarrhea, dysentery and fever.[19] *In vitro* studies have verified that *Brucea javanica* extracts are effective as amoebicides,[20] and clinical studies have shown it to be an effective agent in the treatment of amoebic dysentery[21,22] and malaria.[23] In animal studies *B. javanica* has been demonstrated to play a role in immunological regulation, as evidenced by its killing effect on the cysts associated with Pneumocys-

(Continued on page 113)

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tis carnii pneumonia. Other reports have illustrated its activity against various non-commensal organisms including *Shigella* species *(S. shiga*, *S. flexneri*, *S. boydii)*, *Salmonella* species *(S. lexington*, *S. derby*, *S. typhi* type II) and *Vibrio* species *(V. cholerae, V. inaba* and *V. cholerae ogawa)*.[25]

Pulsatilla chinensis (rhizome) – The root (rhizome) of *Pulsatilla chinensis* has been described as possessing anodyne (pain relieving), anti-inflammatory, antispasmodic, astringent and sedative properties.[26,27,28] It is noted as an effective agent for bacterial and amoebic dysentery,[27, 28] and is traditionally used in the treatment of malaria, nose bleeds and hemorrhoids, as well as externally to treat infestation with *Trichomonas vaginitis*.[27,16] It is also thought to clear toxicity and lower fever. The active compound in the root is the lactone protoanemonin, which is recognized as the bactericidal agent.[16]

Picrasma excelsa (bark) – Also referred to as Quassia, this herb is considered a powerful simple bitter, hence its use as a digestive aide. The two main ingredients are quassin and neoquassin. Traditional use is as a remedy for roundworms, as an insecticide, and as a remedy for headlice. It is also used as a remedy for digestive disorders, and for parasites.[30] Orally it is used for anorexia, indigestion, constipation, fever, or as an anthelmintic for thread worms, nematodes, and ascaris.[31] A recent study with *P. excelsa* noted a moderate inhibition of the cytochrome P450 (CYP) enzyme 1A1. This enzyme is a known activator of carcinogens.[32]

Acacia catechu (stem) – The herb *Acacia catechu* is typically utilized for its astringent and antioxidant properties. The catechins isolated from this herb have significant antioxidant and antimicrobial properties. In many parts of the world chewing sticks are made out of the stem, and because of its antimicrobial properties it is considered a valuable component for dental care.[33] The chief phytoconstituents of the heartwood are catechin and epicatechin.

Hedyotis diffusa – *Hedyotis diffusa* is one of the most popular herbs used in traditional Chinese medicine (TCM). It has been demonstrated to possess antioxidant,[34] anti-inflammatory, hepatoprotective,[35] neuroprotective,[36] and antitumor properties.[37] Its active principles include anthraquinones,[38,39] iridoid glucosides,[36,34] triterpenoids, and flavonoids.[36,34]

Achillea millefolium, **Yarrow** (leaf, flower) – The indications for the use of Yarrow, as approved by the German Commission E include loss of appetite, dyspeptic complains and liver/gallbladder issues. The actions of its flavonoids are indicated as cholagogic (bile flow stimulant), and as a vitalizer in increasing the production of stomach acid. It also possesses both anti-edema and anti-inflammatory attributes.[13] Yarrow is recognized for its relaxant property on smooth muscles, thus may aide with the relief of stomach cramps[41] associated with dysbiosis. In one study utilizing Yarrow, an anti-Staphylococcal activity was demonstrated.[42]

Anethum graveolens, **Dill** (seeds) – As a popular flavoring agent, dill has a history of use as an aromatic herb and spice exceeding 2000 years.[43] It is said to have a calming effect on both the autonomic nervous and digestive systems, as well as having carminative and stomachic properties.[44] It is also indicated as a diuretic, antispasmodic and antibacterial agent, an expectorant, and as a pancreatic stimulant.

The fruits (seeds) contain 1-4% essential oil, of which the primary compounds are corvone, limonene and α-phellandrene, representing 30-60%, 33% and 21%, respectively.[46,47] Potent antibacterial activity has been demonstrated with both aqueous and organic extracts of the seeds.[48,49,50] The compounds D-limonene and D-carvone, have been demonstrated to possess strong activity against the species *Aspergillus niger*, *Saccharomyces cerevisiae* and *Candida albicans*.[51,52,53] Its activity against both Gram negative and Gram positive bacteria, as well as fungi and molds has also been demonstrated.[54] Aside from its beneficial attributes towards eradicating these species, its primary use is for the calming action it exerts on the digestive system, and as such aids in reducing gastrointestinal irritation.

By virtue of the combination of Eastern and Western herbs, the select botanicals discussed above afford a broad anti-dysbiosic effect, even with low dosing. In addition to providing an unfriendly environment for bowel pathogens, this combination of herbs is safe for continual use for up to eight weeks, as it has a low toxicity, and affords minimal irritation to the gut lining. By providing constituents to support the healing and maintenance of the digestive epithelial lining, as well as to eradicate non-commensal flora, the above mentioned herbals affords potent healing properties.

Cautions:
Artemisia is not recommended concurrently with drugs that thin the blood, drugs that reduce stomach acid, or drugs that prevent or lessen seizures. Additionally, consumption may intensify the effects and side effects of alcohol.[55]

(Continued on next page)

Yarrow is contraindicated with blood thinners, particularly coumarin. As it contains simple coumarin components,[56,57] it may interfere with anticoagulants and blood pressure medications. Additionally, yarrow may be contraindicated concurrently with the use of drugs that minimize or reduce the production of stomach acid.[41]

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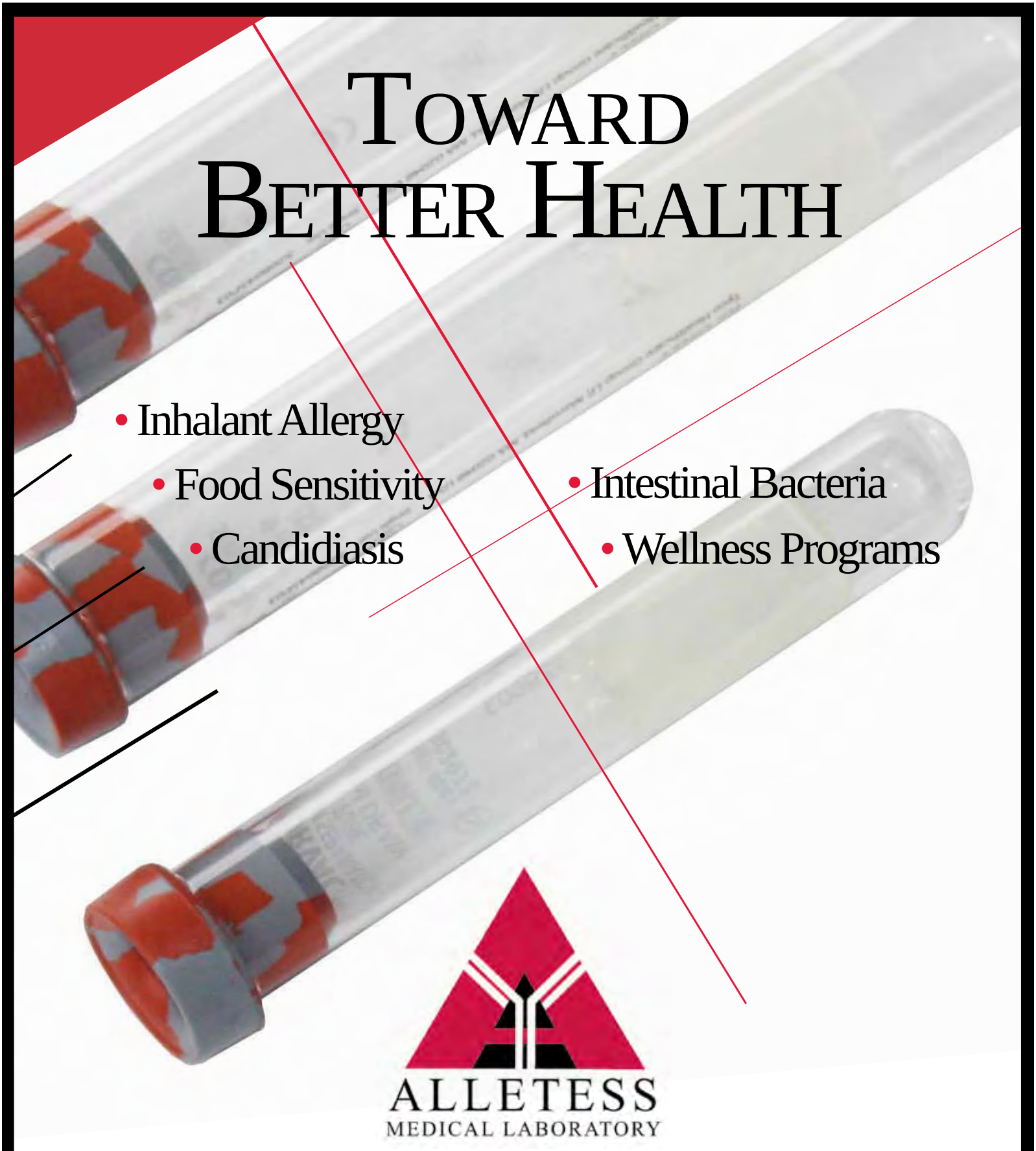

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Systemic Mycoses: An Overview for Modern Natural Health Professionals

by: Robert Thiel, Ph.D., ND

ABSTRACT

Systemic mycoses can cause a tremendous variety of health problems including digestive difficulties (diarrhea, bloating, discomfort, flatulence, constipation, colitis, etc.), skin problems (rashes, eczema, psoriasis, dry skin patches, intense itching, hives, open cut-like sores, etc.), bronchopulmonary disorders, asthma, breathing difficulties, fatigue, seasonal allergies, multiple food allergies, weight loss, fever, chronic sinusitis, irritable bowel syndrome, migraine headaches, autoimmune disorders, fibromyalgia, arthritic complaints, chills, malaise, mental cloudiness, inability to lose weight, and depression. Although Candida albicans tends to get the most attention, it is only one of 150 fungal species which are known to be pathogenic to humans. In addition to skin, respiratory, or genital areas, mycotic infections often settle in the digestive system. Understanding the various types of mycotic organisms can be helpful for health practitioners who are interested in natural interventions to help restore their infected patients to health.

INTRODUCTION

There are over 100,000 different species of fungi, of which approximately 150 are known to be pathogenic to humans.[1,2] Those which are pathogenic have been classified into three broad categories: superficial, cutaneous, and systemic. Superficial mycoses (systemic (fungal infections) normally are confined to the keratinized layer of the skin and its appendages.[3] Cutaneous/ subcutaneous mycoses enter the skin and cutaneous tissue usually in a traumatized area (such as a wound); they usually remain localized, but can spread through the lymphatics to other sites. Systemic mycoses are medically believed to usually have a pulmonary inception, but can affect most areas of the body. [1,4]

Amazingly, even though hundreds of peer-reviewed scientific articles, the *Merck Manual,*[5] and Mayo Laboratories[6] all document common problems due to systemic mycoses, **many medical practitioners 'do not believe in them', will not test for them, and will not treat them, while some others treat mycotic infections for too short of period of time to be effective.**[7] Partially due to this medical disbelief, many natural health professionals see people with a variety of mycotic infections on a regular basis.[7] Some people who have these infections them have been told that the symptoms are 'all in their head' or something just as useful.[7] Mycotic infections, though not normally fatal, are so underdiagnosed, that an autopsy-based study found that in 22% of cases where the primary diagnosis was incorrect, the deceased had some type of fungal infection.[8] Furthermore this study stated, "autopsy findings revealed a major diagnosis that, if known before death, might have led to a change in therapy and prolonged survival (class I missed major diagnoses). The most frequent class I missed major diagnoses were fungal infections."[8] In most "immunocompetent patients, systemic mycoses typically have a chronic course", instead of being life threatening.[5]

Most systemic mycoses are from opportunistic fungi. They are saprocyctes (organisms which live on decaying matter) that are usually innocuous, but become pathogenic when the host becomes abnormally susceptible to infection.[1,5,9,10] To state it less technically, some yeast are present in the body in small quantities and are considered harmless; it is only when they get out of control and multiply excessively that problems are caused.

During the last several decades there have been alarming increases in *Aspergillosis*, *Candidiasis*, *Cryptococcosis*, *Nocardiosis*, and *Zygomycosis*; which to some degree appears to be related to medical treatments such as chemotherapeutic agents, irradiation, immunosuppressive agents, broad spectrum antibiotics, and hyperalimentation as well as conditions such as malignancies, AIDS, malnutrition, metabolic diseases, receipt of multiple injections, certain surgeries, burns, intravenous hyperalimentation, and certain malignancies.[1,10-12] Heavy metals, like mercury, may contribute to these infections. Intense periods of stress or incomplete recovery from infection are other causes of yeast overgrowth. Having gall bladder surgery seems to this investigator to be a factor for some people.

Systemic mycoses can cause a tremendous variety of health problems including digestive difficulties (diarrhea, bloating, discomfort, flatulence, constipation, etc.), skin problems (rashes, eczema, psoriasis, dry skin patches, intense itching, hives, open cut-like sores, etc.), bronchopulmonary disorders, asthma, breathing difficulties, fatigue, allergies, weight loss, fever, chills,

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malaise, depression, and chronic sinusitis;[1,6,9-14] some of them may be risk factors in developing autoimmune disorders.[7,15] This investigator has also observed that many with irritable bowel syndrome, migraine headaches, autoimmune disorders, itching, fibromyalgia, alternating constipation and diarrhea, mental cloudiness, certain types of anxiety, inability to lose weight, and even certain forms of arthritis frequently appear to have some type of mycotic overgrowth--another clue is that many report multiple food intolerances (or have been told they have at least a dozen food allergies from an IgG test). Of course, it needs to be understood that nearly all the symptoms and most of the conditions listed in this paper can be caused by something other than mycotic organisms (and that most people do not have most of the symptoms).

The following conditions have also been reported (by one or more medical doctors) to be at least partially caused by fungi: "malignancies to organs including the esophagus, lung, colon, kidney, breast, uterus, blood, lymph nodes, brain and skin. Some autoimmune disorders are; scleroderma, diabetes, rheumatoid arthritis, Sjogren's syndrome, psoriasis, and systemic lupus erythematosis. Dr. Constantini also listed Raynaud's Syndrome, sarcoidosis, Duchene's muscular dystrophy and Cushing's Disease (excess secretion of adrenal hormone);" whereas a registered nurse also reported, "Multiple Sclerosis, Fibromyalgia, Chron's disease, Endometriosis, Infertility and Migraines."[5] Many mycoses are polysymptomatic[16] which means they can cause a variety of different types of problems.

It has also been reported that systemic mycoses can predispose one to develop celiac disease.[17] And while this is apparently true, it is also true that many who think that they may have celiac disease actually have some type of systemic mycotic infection. Many with Down syndrome or autism tend to have wheat sensitivities and may be more susceptible to mycotic infections than the general public.

A major clinical characteristic of virtually all mycotic infections is their chronic course.[5,9] Symptoms often develop slowly; though many are asymptomatic. Months or years often elapse before medical attention is sought.[5;18] Medical interventions for systemic mycoses include various medications, surgery, and chemotherapy.[1,5,9,10,19] Progress in the diagnosis and medical treatment of many mycoses has been unsatisfactory: [5,7,20] "Immunoserologic tests are available for many systemic mycoses, but few provide definitive diagnoses by themselves."[5] While localized yeast infections are relatively easy to treat, systemic mycoses, including those referred to as Candida Related Complex (CRC), are much more difficult.[5,7]

It needs to be emphasized that **it is not necessary to have a vaginal yeast infection to be suffering from a systemic mycotic infection.** Based on other research, Jonathan Collins, M.D., wrote, "that the bowel or digestive system is the primary site where yeast settle in the body and produce toxic by-products which bring on the vast array of symptoms throughout the body. An unhealthy lower bowel is the breeding ground for infections and inflammation and will cause illness throughout the body."[7]

Although there exists a tremendous amount of natural health literature regarding interventions to be considered for people with an overgrowth of *Candida albicans,*[i.e. 5,20-29] the literature regarding natural interventions for other mycotic organisms is less available. The purpose of this paper is to discuss selected forms of systemic mycoses and provide some information to help the naturopathic practitioner deal with them.

SYSTEMIC MYCOSES

Aspergillosis

"*Aspergillus sp* are among the most common environmental molds, frequently found in decaying vegetation (compost heaps), on insulating materials (in walls or ceilings around steel girders), in air conditioning or heating vents, in operating pavilions and patient rooms, on hospital implements, or in airborne dust."[5] *Aspergilli* are the second most common systemic mycoses and account for nearly 30% of fungal infections found at autopsy.[1] They often appear after antibiotic or antifungal therapy (to which they are usually resistant);[9] this is one distressing area of fighting systemic mycoses--sometimes when eliminating one type, another becomes prominent.[9]

"Invasive fungus infections caused by aspergillus sp. occur most frequently in immunocompromised patients. A high infection-associated death rate of up to and over 50% is attributed even today to these fungi. The disease in humans is caused mainly by Aspergillus fumigatus, Aspergillus flavus and Aspergillus niger."[30]

Clinical findings are usually nonspecific and standard sputum cultures are positive only 1/3 of the time aspergilli are present.[1] "Sputum from patients with aspergillomas often does not yield *Aspergillus* in cultures because cavities are likely to be walled off from airways."[5] They often are implicated in respiratory conditions[1,13] including sinusitis;[31] it appears that sometimes, *Candida albicans*-IgE and IgG subclasses may participate in worsening pulmonary infiltrates when bronchopulmonary *Aspergilliosis* is present.[32] *Aspergilli* are often mistaken for *Zygomycetes*.[1] As enzymes appear to play a role in the reproduction of various species of *Aspergilli,*[33] it is possible that enzyme inhibitors may play a role in diminishing their reproduction

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and growth.

Some have correlated consumption of fatty foods to asthma,[34] and it is possible that some of those people actually have an undiagnosed mycotic infection (perhaps from *Aspergillus*).

Aspergillus fumigatus is the most common form.[1,2] *Aspergillus flavus* is commonly associated with aflatoxins,[2] such as on peanuts.[31] Restrictocin and mitogillon are two other toxins produced by aspergilli--they inhibit host cell protein synthesis by degrading mRNAs.[31] "Molecular epidemiologic studies of Aspergillus isolated from opportunistic infections show many different strains of Aspergillus, suggesting that characteristics of the host are more important than characteristics of the fungi...Aspergillus has a tendency to invade blood vessels"[31] --this is probably true of most situations when a systemic mycotic infection is present. Invasive *Aspergillosis* in usually confined to immune suppressed and debilitated hosts.[31] Some with gastrointestinal upset have *Aspergillus*[1] and some with intense itching may have some version of it (superficial lesions are also a symptom[5]). *Aspergilli* "Fungus balls neither require nor respond to systemic antifungal therapy", though some other *Aspergilli* forms do.[5] Mayo Clinic researchers found it was one of the most common fungal organisms associated with fungal sinusitis.[15] This investigator's clinical experience suggest that some people with *Aspergillosis* seem to improve when dairy is removed from the diet, but whether this improvement is related to a general intolerance or is specific to any *Aspergilli* is unclear.

In 2010, German researchers concluded, "Recognition of and therapy for fungal infections of the lungs still presents problems even for the experienced clinician. The distinction between invasive mycoses of the lungs and fungal colonisations that do not require therapy is cinically difficult and can often not be made satisfactorily even with advanced microbiological diagnostics."[35] Hence there is still trouble in the identification and treatment of fungal infections that affect the breathing process.

Blastomycosis

"A disease caused by the inhalation of mold conidia (spores) of *Blastomyces dermatitidis*, which convert to yeasts and invade the lungs, occasionally spreading hematogenously to the skin or focal sites in other tissues. *Blastomyces dermatitidis* grows as a mold at room temperature. Inhaled *B. dermatitidis* conidia convert at 98.6^{F} in the lungs into invasive large yeasts."[5] It can produce dry hacking and affect the prostate, testis, kidneys, vertebrae, brain, nose, thyroid, lymph nodes, and bone marrow, but skin lesions are probably most common.[5] Men (especially over age 40[5]) are afflicted with it more than women, with wart-like lesions on the skin and sometimes internal organs.[36] There is also a South American form called *Paracoccidioidomycosis* which mostly effects men aged 20-50 who work as coffee growers.[5]

Candidiasis

Candida albicans is the most common cause of *Candidiasis*.[1,2,10] *Candidiasis* is an infection involving every part of the body. It exists in the normal flora of the oral cavity, upper respiratory tract, digestive tract, and vagina. Severe, invasive *Candidiasis* involves the kidney in 90% of cases. [31]

Candida hyphal growth (the more virulent form) requires a pH of 7.4 (slightly alkaline) for optimal growth and can be completely inhibited at a pH of 4.5 (fairly acidic) [37,38] and "is now the fourth most prevalent organism found in bloodstream infections."[5]

It can be a superficial, mucocutaneous, or systemic mycosis. Infection by any of the species of *Candida* is nearly always preceded by a compromise of the host defense mechanisms,[1,5] such as a selective defect in the functioning of T lymphocytes.[39] It can exist as both yeast forms without hyphae as well with hyphae and the transition from yeast to hyphal forms can increase problems eliminating it as the hyphae can spear their way out of cells which engulf them.[31] *Candida* has molecules on the surface that mediate its adherence to human tissues which are the main ways it negatively affects health.[31] "Pathologists studying disseminated candidiasis find tiny abscesses throughout the body. These consist of *Candida albicans* surrounded by fibrin (a protein able to clot) and a connective tissue shell. This shell isolates *Candida* from elimination by the immune system."[7]

"All forms of disseminated candidiasis should be considered serious, progressive, and potentially fatal. Predisposing conditions such as neutropenia, malnutrition, or uncontrolled diabetes should be reversed or controlled where possible."[5] All forms of *Candida* do not respond to the same medical[5] or other interventions. *Candida albicans* and *C. glabrata* tend to respond similarly, whereas *C. cruzzi* does not.[5]

However, many nutritional interventions have been reported to be effective for *Candida.*[i.e. 7,21-29] Since *Candida albicans* is often grown in a culture of various saccharides,[2] it is not surprising that reductions in the consumption of refined sugars has been effective.[13-15] Sometimes, this investigator and others[21-24] have had success having subjects also avoiding most fruits. Interestingly, it appears that *Candida albicans* cannot grow in human saliva unless it is supplemented with glucose.[37]

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It was been written that, "CRC is the most dreaded complication of fungal infections, because it is hard to recognize and even harder to treat...This spread of *Candida albicans* has been described as a domino-effect--one body system after another falls prey to CRC, unless it is stopped or reversed...Another name for CRC is mycotoxins."[11]

There have been substantial increases of candidemias caused by species other than *Candida albicans*.[40] *Candida tropicalis* is probably the second most common cause of candidiasis.[1,5] Infections with *Candida glabrata* and other Candida species are increasing with frequency.[5] *C. glabrata* can cause fungemia, urinary tract infections, sometimes pneumonia or other focal lesions.[5] *Candida paratropicalis* is quite similar to *Candida tropicalis* and is often confused with it.[1] A significant difference is that paratropicalis does not thrive with sucrose, although tropicalis does.[2] *Candida krusei* (also spelled cruzzi) seems to be less affected by refined sugars (other than dextrose) than most other Candida species, [2] thus this investigator rarely encourages reduction of fruit consumption when it is suspected. Other *Candida* species such as *C. guillermondi, C. parapsilosis, and C. pseudotropicalis* can cause infections in humans,[1,10] but (other than any differences their shape may account for[2]) this investigator is not aware of adequate reasons to differentiate the dietary restrictions from those of *C. albicans*. One of the newest discovered forms, *Candida dubliniensis*, has a lot in common with *C. Albicans*, but is still different.[41]

In the past, *Candida zeylanoides* was not considered to be much of a pathogenic yeast for humans,[42,43] but can occur in individuals who do not have the "usual risk factors for systemic candidiasis.[44] Case reports have suggested that it can cause arthritis,[45] infective endocarditis,[44] onychomycosis (nail infection), [40] and gastrointestinal disturbances.[39] It may be implicated in Scleroderma.[42] An animal study suggests that it also can cause tinea cruris (jock itch).[46] *C. zeylanoides* is a predominate form of yeast found in poultry,[47] raw sausage,[48] and some hams.[49]

Cryptococcosis

Cryptococcosis is normally due to the fungus *Cryptococcus neoformans* also called *Filobasidiella neoformans* or *Torula histolytica*. It is an encapsulated yeast and is present in soil and bird (especially pigeon) droppings. [31] Symptomatically it is quite different from the other systemic mycoses in that meningitis with headache is the way it is most commonly presented; blurred vision is also common.[9] Infection tends to occur via the respiratory route by inhalation of *Cryptococcus neoformans*[1,9] Consumption of high-dose corticosteroids is a major risk factor for it.[31] *Cryptococcosis* frequently affects the central nervous system.[36] As *Cryptococcus meningitis*, it is found in some with AIDS where it tends to increase the mortality rate.[50] The lungs, kidneys, and sometimes skin tend to be affected.[5] It is resistant to killing by alveolar macrophages.[31] It produces the enzyme phenol oxidase which tends to consume the hosts epinephrine,[31] thus adrenal support may be helpful for the sufferer. Adrenal support would not help eliminate *Cryptococci*, but at least may make the sufferer feel better through the process. Adverse reactions to medical interventions for it include gastrointestinal disturbances,[9] thus probiotic intervention possibly should be considered as an adjunct.[25,26]

Histoplasmosis and Coccidioidomycosis

Histoplasmosis and *Coccidioidomycosis* are similar fungal organisms that both produce a disease that resembles tuberculosis.[1,31] Both are caused by fungi that grow as spore producing hyphae at environmental temperatures, but as yeasts (spherules or ellipses) at body temperature within the lungs.[31] *Histoplasma capsulatum* is acquired by inhaling dust particles which contain bird or bat droppings that contain small spores (microcondia), the infectious form of the fungus.[31] "*H. capsulatum* grows as a mold in nature or...at room temperature but converts to a small...yeast cell at ...98.6...F...and when invading host cells.[5] AIDS patients are particularly susceptible to disseminated infection with *Histoplasma.*[31] *Histoplasmosis* "occurs primarily in the East and Midwest" and primarily affects the lungs[5] --in acute forms it can cause ulcers of the pharynx, spleen enlargement, and liver enlargement.[36] *Coccidioides immitis* has a high infection rate and usually resides in desert soils, and in the US is mainly confined to the Southwest.[1,5] Similar to *Histoplasma*, most primary infections with *Coccidiodes immites* are asymptomatic, but about 10% develop lung lesions, fever, cough, excess sputum, and pleuritic pains along with San Joaquin Valley fever complex.[5,31] "Once inhaled, *C. immitis* conida (spores) convert at 98.6^F to form large invasive spherules."[5] *Coccidioidomycosis* is also called "Valley Fever."[5] "Untreated disseminated coccidiodomycosis is usually fatal. Treatment for primary coccidioidomycosis is unnecessary in low-risk patients. Treatment for meningeal coccidioidomycosis must be continued for many months, probably lifelong."[5]

Mycobacilli: Nocardiosis and Actinomycosis

Although *Actinomycosis* and *Nocardiosis* are often considered together when discussing systemic mycoses, they are filamentous, gram-positive, bacteria in the order of *Actinomycetales*, and not true fungi.[1,2] These

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infections are consistently found in the U.S., but the diagnosis is difficult since they resemble other bacterial, mycobacterial, and fungal infections.[51] *Nocardiosis* and *Actinomycosis* are symptomatically similar to tuberculosis.[2] Actinomycosis affects males three times as often as females.[1] *Nocardiosis*, normally in the form of *Nocardia asteroides*, is increasingly found in patients with systemic lupus erythematosus (SLE) and is probably higher than the reported incidence of 2.8% in the SLE population.[52] "Without treatment, nocardiosis caused by *N. asteroides* is usually fatal."[5] When *actinomycosis* or *nocardiosis* is present, it is sometimes wise to avoid bovine dairy and/or refined carbohydrates. Nutritional support such as used by people with "streptococci-type" bacteria can sometimes be helpful for some with some mycobacilli.

Zygomycosis/Mucomycosis

Zygomycosis (also called *Mucomycosis*) is a generic term which refers to infections of the class *Zygomycetes* (also called *Phycomycetes*); they tend to be both opportunistic and invasive.[1] It is defined as "Infection with tissue invasion by broad, non-separate, irregularly shaped hyphae of diverse fungal species."[5] "Infection is most common in immunosuppressed persons, in patients with poorly controlled diabetes, and in patients receiving the iron-chelating drug desferrioxamine" (plus people on immunosuppressive therapies or who have chronic renal conditions).[5] It can cause pulmonary or gastrointestinal lesions.[5] The three most common areas of invasion are the sinuses, lungs, and gastrointestinal tract.[31] *Rhizopus* species may be the most common; others include *Absida corymbifera, Mucor ramosissimus, Rhizomucor pusillus*, and more.[1,2] Infection is believed to be less common than some of the other systemic mycoses mentioned in this paper, but is the third most frequent opportunistic mycoses in patients with neoplastic disease[1] as well as for ketoacidotic diabetics.[31] It appears to this investigator that some with *Rhizopus* often have problems with bile flow; as do some with intense itching. *Rhizopus nigricans* produces opportunistic infections and hypersensitivity states;[53] it seems to cause the body to produce additional IgG and IgE.[33] A recently identified strain, *Rhizopus azygosporus*, was isolated from premature Australian babies, all of which died.[54] Patients with diabetic acidosis or leukemia can be predisposed to rhinocerbral infection caused by *Rhizopus oryzae*;[1] increased consumption of most fresh fruits and vegetables has been reported to help reduce acidosis.[55]

Mold, Fungus, Yeast, and Interventions

"Mold is caused by fungus which in turn causes disintegration of organic matter. Whether it is caused by Candida albicans or any of its related species, fungus causes a weakening of the cellular structure in which it lives. This explains why patients afflicted with this type of infection become very ill and are difficult to treat; many of their cells become weak. Fungus is tenacious"[5] (it should be understood that molds are multi-cellular organisms, whereas true yeasts are single-cell organisms). These days there are many reports of homes and office buildings having mold problems which require decontamination (such decontamination measures are beyond the scope of this paper).

"Yeast, in its many varieties, is a unicellular fungus that reproduces by budding spores"[7] --it is the budding process that is one of the reasons that elimination is most difficult. This ability to froth/bud makes it difficult for mycotic infections to be controlled as the quantity of yeast can go from little to overwhelming in a rather short period of time—under optimal conditions one yeast cell can produce multiple millions of offspring in 24 hours; and 24 hours later *each of those can produce multiple millions of offspring*. Elimination of yeast is often an up and down process which makes it difficult for the one fighting it. Actually, one of the problems when mycotic infections are dealt with medically or naturopathically, is that the sufferer will sometimes feel better before the problem is gone, will skip some interventions (not take supplements, violate dietary restrictions, etc.), do fine, and then 'suddenly' notice that symptoms which had left have returned.

Another reason it is difficult to eliminate yeast is because some are dimorphic[56] and many have pleomorphic hyphae.[57] "The ability to switch between a yeast-like form and filamentous form is an extended characteristic among several fungi. In pathogenic fungi, this capacity has been correlated with virulence because along the infectious process, dimorphic transitions are often required"[57] --this dimorphic tendency may at least partially explain why changing interventions is often necessary when dealing with mycotic infections. Pheomorphic hyphae have been found to be affiliated with most types of mycotic yeasts.[57] These abilities to change shapes (dimorphism and pleomorphic hyphae) make it harder to eliminate mycotic organisms (and is one reason why the same intervention does not always work)—pH (both acid or alkaline) is also a factor.[38]

It may be of interest to note that according to at least one doctor, "Gas-forming organisms only flourish in an alkaline environment. We've been brainwashed to think that digestive acids produced in the stomach are the root of all digestive problems."[58]

The main virulent mycoses, such as *Candida* and *Aspergillus*, do not thrive in an acidic environment,[37,38]

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but some others do. Thus, the frequent consumption of anti-acids by many with 'acid reflux' (GERD) or 'irritable bowel syndrome' helps create an environment that the two major mycotic organisms can thrive in (this is not to say that there is no place for antacids, as they can help prevent ulceration and other problems). Actually, researchers have concluded that many people who think that they have food allergies actually have acid reflux [59] and the reality is that many with acid reflux actually have a mycotic infection as the real cause. Hence many "food allergies" are likely to be assumed because of how the body reacts to certain foods because of mycotic infections.

"With the continuing increase in clinically important fungal disease...the need for new and improved antifungal agents marches on."[60] This is partially because the commonly used pharmaceutical antifungal agents are not always effective:[61,62] "Emerging cases of drug resistance to currently available drugs has limited the spectrum of currently available antifungal agents."[63] "Drugs for systemic antifungal treatments include amphotercin B, various azole derivatives, and flucytosine."[5]

While drugs remain the preferred standard treatment[64] there are concerns about their safety, effectiveness, and cost.[65] "Opportunistic systemic mycoses due to yeasts and yeast-like fungi have become more common than those due to filamentous fungi, occupying fourth position in the list of bloodstream pathogens in some centers in USA. Also, their incidence, pattern of clinical presentations and species spectrum have significantly changed, largely due to more frequent and prolonged therapeutic or prophylactic use of antifungal drugs and subsequent development of resistance. Consequently, infections with resistant yeast-like fungi such as *C. lusitaniae, C. krusei, C. tropicalis, C. glabrata and Trichosporon ovoides (T. beigelii)* have recently been reported with greater frequency. Since respiratory and systemic mycoses have no pathognomonic clinical or radiologic syndrome and mycological diagnostic facilities are restricted to only some of the major metropolitan centres, these diseases may be frequently confused. Further studies should focus on the development of rapid techniques for selective isolation and identification of systemic pathogenic fungi. The problem of antifungal resistance is likely to become more serious in the future."[66]

"Innate and cell-mediated immunity are considered as the principal defense line against fungal infections in humans."[67] Thus, naturopaths tend to focus more on dietary restriction, herbs, naturopathic formulas, heavy metal detoxification, and even electricity to help their clients' immune systems overcome many of the problems associated with systemic mycotic infections. Regarding diet, as shown above, there is no single diet that helps all the people who have various types of mycotic infections. Avoiding refined sugar, as a general rule, is good for most people with systemic mycoses (even some published medical research concurs[16]): many with mycotic infections strongly crave sugar--but to submit to those cravings can make elimination more difficult (or can cause set-backs). The same can often be said for other refined carbohydrates (white flour, white rice, white pasta, alcohol, etc.). Although there are some people who need to avoid vinegar, most fruits, or mushrooms, this investigator has found that most can consume them without any apparent adverse affects. On rare occasions, some people who get severe diarrhea improve when they avoid lettuce as apparently some forms of this vegetable can contain some type of external mold spores.

There is a misconception that people with mycotic infections must always avoid yeast-containing foods--while this may be true in some cases, it is most often white flour and not the fact that bread has been leavened with yeast that is the problem. *Saccharomyces cerevisiae* (the primary yeast used in baking and brewing) is beneficial to humans and can help combat various infections,[68] including according to the German E monograph *Candida albicans*. In the text, *Medical Mycology* John Rippon (Ph.D., Mycology, University of Chicago) wrote, "There are over 500 known species of yeast, all distinctly different. And although the so-called 'bad yeasts' do exist, the controversy in the natural foods industry regarding yeast related to health problems which is causing many health-conscious people to eliminate all yeast products from their diet is ridiculous." It should also be noted, that W. Crook, M.D., perhaps the nation's best known expert on *Candida albicans*, wrote "**yeasty foods don't encourage candida growth.** Eating a yeast-containing food does not make candida organisms multiply."[21] Some people, however, are allergic to the cell-wall of yeast[21] and concerned supplement companies which have nutrient-containing yeast normally have had the cell-wall enzymatically processed to reduce even this unlikely occurrence.

There is no herb or other natural intervention that this investigator has seen which always works. Most of the substances practitioners recommend help create an environment that hyphal yeast forms do not thrive in or that the body's own defenses do.

Some of the more common natural substances this investigator has considered include aloe vera, arginase, astragalus, basil, beet root, bentonite, berberis root/ berries, betaine hydrochloride, bile, biotin and other B

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vitamins, caprylic acid, castor oil, Chinese herbs (various), cinnamon extracts,[69] chlorophyll, citrus seed extract, cinnamon, cloves, colostrum, deer antler velvet, digestive enzymes, echinacea, essential monosaccharides, flowers (various), food, multiple vitamins, garlic, goldenseal,[68] glandulars, green vegetables, homeopathic & isopathic remedies, horsetail, l-glutamine, l-valine, lactoferrin, licorice, n-acetyl glucosamine, magnesium, manganese, molybdenum, oxygen (in various forms), pau d' arco (and other South American herbs), probiotics (including non-traditional ones),[70] olive leaf[71] oregano (wild and oil forms),[72] psyllium (seeds and/or hulls), *Saccharomyces cerevisiae,*[68] silver (in various forms), thyme, tillandsia, una de gato (cat's claw), vitamin C, wheat germ, wheat grass, white fish, and zinc.

Caution about self-treatment needs to be stated: not everyone tolerates all these substances well, no one probably needs all of them, and perhaps most importantly, inadequate treatment seems to often leave the stronger fungal strains to become dominant.[5] And no matter what "experts" on the internet may claim, the reality is that there is not a single formula that is helpful for everybody struggling with yeast.

Because a compromised immune system or hormonal cycles can be involved (symptoms sometimes worsen near a woman's menstrual cycle), nutritional support for the thyroid is quite often a useful adjunct (this is true for males and females). Stress affects this condition and this is another reason that thyroid and sometimes adrenal support is helpful. Also, real food B vitamins (which are not in most vitamin formulas, including ones that say that they are "natural") can help as they decrease the overwhelming craving that some with this condition develop towards carbohydrates. Similarly, Chromium in the GTF form can help some with overwhelming sweet cravings. This investigator has also had some success using other naturopathic interventions, such as bioelectrical stimulation, proper food combining, fasting, and hydrotherapy.[73] Those with dual infections perhaps take the longest amount of time to help get back to normal, and dual-infections seem to present relatively frequently in this population.

There are normally ups and downs associated with treatment. Yet, many "treatments" have "downs" for a long time and never have the "ups" for a significant time. Hence, it is important that the health professional be highly skilled in making recommendations related to treatment.

'Die off' and other adverse reactions sometimes are encountered when interventions are successful[5]--normally these are frustrating as opposed to detrimental. Some 'holistic literature' words it, "When yeast cells are rapidly killed by the immune system, drug treatment, or dietary intervention, a 'die-off' or Herxheimer reaction occurs. This reaction is caused by the massive release of toxins from dying candida cells. Toxic proteins from the dead yeast cross cell membranes, enter the bloodstream, and trigger an intense immune reaction. Die-off reactions may last from a few days to a few weeks, but usually less than a week. A die-off reaction is especially pronounced when using powerful antifungal drugs like Nystatin that literally cause yeast cells to burst apart;"[74] whereas medical literature has stated, "For the 3 oral antifungal agents the more common adverse reactions are nausea, gastrointestinal distress, diarrhea, abdominal pain"[75] and "administration of nystatin became impossible in three patients because of vomiting."[76] Tiredness sometimes accompanies 'die-off.'

Stressful situations, 'die-off', dimorphism, and the tendency of one type of yeast to become dominant while another is being controlled, all make successful interventions complicated (as does use of antibiotics or multiple infections). But naturopathic interventions are often the most appropriate ones to help the body naturally fight the fungi itself and regain control of health. Weight-loss is difficult to sustain for overweight people while most are combating a mycotic infection and that much of the progress in this arena does not take place until the infection is controlled.

Conclusion

Into 2010, "Invasive mycoses continue to be a major problem in the growing population of immunosuppressed patients."[77] Much research in the USA and Germany is being conducted related to them.

As there are 100,000 known types of fungi,[1] there is little doubt that more will be found to be pathogenic to humans. Additional mycobacilli species are also being found to have clinical importance[78] and even mycoplasma is being investigated.[39,79] *Candida*, *Aspergillus*, and *Mucor* are ubiquitous contaminants which colonize normal skin or gut without causing illness--it is only in immunosuppressed individuals that these opportunistic fungi give rise to life threatening infections.[2]

However, even though most of the symptoms are not life threatening, overgrowths of any of them can make human life miserable. All yeast produce toxins.[31] It appears to this investigator that these toxins are responsible for symptoms such as itching, mucus, bowel difficulties, and can trigger an autoimmune reaction. Triggering of autoimmune response then seems to cause arthritic and some other pain-related symptoms.

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If one can reduce yeast populations, then the amount of toxins will be reduced and ultimately the body will be able to shut-off (or at least seriously reduce) its autoimmune responses.

Practitioners need to understand that not all pathogenic mycotic organisms are known, few are ever tested for, relatively few are ever detected through the course of most medical appointments, some are not detected when tested for, and perhaps most importantly, all do not respond to the same dietary factors. Furthermore, there is no single herb, diet, electrical device, or naturopathic formula that this investigator has ever found that will always eliminate it. Getting systemic mycoses under control is a difficult and frustrating process, but the results are worth the effort; for many who are not leading normal lives now, can live normal (or near normal) lives after control.

About the Author

Dr. Thiel was an Idaho naturopathic physician and an Alabama licensed naturopathic scientist. He received his M.S. from the University of Southern California, Ph.D. (Nutrition Science) from the Union Institute & University, and his Doctorate in Natural Health from The United States School of Naturopathy and Allied Sciences. Dr. Thiel is the author of several books, including Combining Old and New: Naturopathy for the 21st Century. He has written several papers on mycotic infections, and sees many people with mycotic infections at the Doctors' Research Clinic in Arroyo Grande, California.

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Hyperhomocysteinemia as a Result of the Methylenetetrahydrofolate Reductase (MTHFR) C677T Polymorphism Causes an Increased Risk of Cerebrovascular Disease: A Biochemical Perspective

by: Robert A. Duca, Jr. DC, MS, DABCI, DACBN, DABCSP

Introduction

There is much investigation today into the relationship of elevated homocysteine and cardiovascular disease. Homocysteinemia is widely associated with cardiovascular disease in the form of atherosclerois, myocardial infarction, peripheral arterial disease, venous thrombosis, hypertension, and cerebrovascular disease. Considerable speculation exists to the adverse effects of elevated homocysteine and neural tube defects, colorectal cancer, and dementia. This research paper is an attempt to explore homocysteinemia as a causative agent in cerebrovascular disease and stroke.

Homocysteine has the molecular formula HSCH2CH2CH(NH2)CO2H. Homocysteine cannot be obtained through the diet. It is made from the amino acid methionine by the removal of its terminal methyl group. Homocysteine can be regenerated into methionine or converted into cysteine through the utilization of folic acid and vitamin B12. Homocysteine is similar to the amino acid cysteine but differs in that it has one additional methylene group.

Elevated homocysteine in the blood is termed homocysteinemia and in the urine, homocysteinuria. Homocysteine is currently utilized as a laboratory marker of cardiovascular heart disease such as stroke, atherosclerosis, CVD, and hypertension. This cardiovascular disease risk has been documented to be readily remedied with folic acid therapy. Homocysteine is methylated to methionine by the transfer of the methyl group of methytetrahydorfolate (MTHF). Methylenetetrahydrofolate is made by the reduction of a methylene group of methylenetetrahydrofolate. The reduction of methylene of methylenetetrahydrofolate is catalyzed by methylenetetrahydrofolate reductase enzyme.[1] In this remethylation process of homocysteine, 5MTHF and methylcobalamin form a prosthetic group for the enzyme methionine synthase. Methylcobalamin is the methyl donor necessary to make homocysteine into methionine. Conversely, methionine is converted to homocystiene in the following mechanism. Methionine is converted to S-adenosylmethione (SAM) catalyzed by methionine adenosyl transferase. The methyl group is taken from SAM to form S-adenosyl homocysteine (SAH). When the adenosyl group is taken from SAH, homocysteine is formed. MTHFR enzyme normally converts dietary folate into its active cofactor in homcysteine metabolism. MTHFR converts 5,10-methylene THF to 5-methyl THF. MTHFR needs FAD of riboflavin as a prosthetic group. A genetic mutation in the MTHFR enzyme is ultimately what is implicated in cardicovascular disease and, specifically, stroke.

The mutation (677C-T) in the methylenenetetrahydrofolate reductase enzyme gene results in reduced folate dependent enzyme activity and reduced remethylation of homocysteine to methionine.[2] The number of C677T/MTHFR mutant homzygotes are classified TT genotype, mutant heterozygous CT genotype and normal homozygous CC genotype of wild type.[3] The mutation in the MTHFR gene is a C—T substitution at the base pair 677 which causes an exchange of an alanine to a valine.[4] It has been determined that the mutation is present in 35% of alleles and that the TT genotype mutant homozygotes have elevated average plasma homocysteine concentrations compared to those not carrying the mutant allele (CC genotype.) This thermolabile MTHFR causes the decreased enzyme activity that elevates plasma homocysteine levels. As a result, this common C677T/MTHFR mutation is considered to be a frequent genetic risk factor for cardiovascular disease and stroke.[5] There is speculation that MTHFR deficiency may be caused by other types of gene mutations but the current evidence is lacking.

A substantial number of studies have been conducted to investigate the relationship between the C677T/ MTHFR genetic mutation and stroke etiology, however the biochemical mechanism to explain such has been elusive.

Methods

A computerized search of The U.S. National Library of

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Medicine and the National Institute of Health PubMed was conducted. The search was conducted through the journals database, MeSH database, clinical queries, and single citations. The journal database was filtered to include journals of biochemistry, genetics, cardiovascular disease, and nutrition only. The MeSH search utilized the terms homocysteine, MTHF, MTHFR, amineoxoreductases, cardiovascular disease, stroke, and polymorphism. Included in the clinical queries and single citations were three meta-analyses, four prospective studies, three retrospective studies, four observational studies, two case reports, two editorials, and one animal model with a MTHFR deficiency. A total of 17 refereed peer-reviewed published articles were critically analyzed and comprise the basis of this research paper. This research paper will attempt to present evidence demonstrating the relationship between the MTHFR polymorphism and stroke and to address the biochemical and mechanical significance of the MTHFR polymorphism, hyperhomocysteinemia and stroke.

Data

MTHFR gene polymorphism was studied in 58 ischemic stroke patients in a tertiary care hospital setting in India.[6] In their introduction, the authors acknowledge that more than 110 inheritable, 175 genetic loci and 2050 different mutations that predispose one to stroke have been identified.[7] Ischemic stroke can result from one genetic defect or a combination of defects such as apoprotein E 4 and angiotensin coverting enzyme D/D genotypes. Other factors such as smoking, hypertension, and ethanol use in combination with these genetic mutations can provoke stroke. The authors state that the role of elevated homocysteine (Hcy) is a relatively new established risk factor for cardiovascular disease.[8] Homocysteine role in stroke is controversial although most case control studies indicate a causal relationship.[9]

Three months following stroke fasting blood was drawn and evaluated for folic acid, vitamin B12, and homocysteine. MTHFR gene analysis was conducted by extracting DNA from peripheral leukocytes. The MTHFR C-T677 substitution was identified by using enzyme digestion restriction of the polymerase chain reaction (PCR) products.[10] In the study 32.8% of patients had MTHFR C677T gene polymorphism, 3 were homozygous (TT), 16 heterozygous (TC), and the balance of patients were normal (CC).[11] Homocysteine levels were highest in TT alleles compared to TC and CC. The authors contend that the need of 5-methyltetrahydrofolate as a methyl group donor for conversion of methionine to homcystiene might account for the slight rise of serum homocysteine in homozygous patients.[12] It is known that the influence of MTHFR genotype on HCy is greater in individuals with low serum folate and B12 levels.[13] The authors suggest that that a diet high in B12 and folate could supercede a folate deficiency even in a MTHFR polymorphic patient. The authors concluded that MTHFR gene polymorphism was noted in 1/3 of patient with ischemic stroke and that the rise in homocysteine was not significantly greater than those patients without the polymorphism.

A similarly designed study published in the European Journal of Neurology in 2005 indicated a C677T MTHFR mutation was strongly associated with arterial stroke especially in young adults.[14] Sixty-nine patients with arterial stroke were studied and 49 patients with no previous history of stroke were the control group. MTHFR genotyping was conducted by PCR with specific primers and with subsequent restriction digestion and gel analysis. The authors detected 1.4% (one of 69) homozygous and 31.88% (21 of 69) heterogygous MTHFR mutant genotype.[15] The control group had only one heterozygote out of 49 (2.08%) tested. The odds ratio for the probability of the C677T MTHFR gene mutation in stroke patients compared to control group was 22.29 (95% CI 4.89-98.8). 16 The authors recommended allele evaluations in the future to reduce and prevent stroke morbidity.

A Japanese study published in 1998 showed a risk of stroke 2.05 times greater with individuals with the TT genotype and 1.35 times greater with the TC genotype than controls.[17] Their study indicated the prevalence of stroke in patients with the mutated gene to be almost two-fold relative to recent European studies.

Rates of the 677T polymorphism vary greatly among Asian, black, and white ethnic groups. These groups may, however, show different carrier rates or environmental susceptibility to polymorphisms which could account for geographical and ethnic variances in stroke risk.[18]

In a meta-analysis published in Stroke in 2005 the authors evaluated 13 papers of which only 5 categorized specific TT genotype frequencies. No association was found between TT genotype and large artery, small artery, and cardioembolic stroke.[19] In the Stroke meta-analysis of 15,000 individuals, the MTHFR 677C-T polymorphism showed an elevated risk of stroke that

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was gradient in nature and dose-dependent. Their estimate of TT homozygotes was two times that seen in heterozygotes inferring an additive influence of the T allele on stroke risk.[20] Their finding may have significant public health considerations as 3-50% of most populations are CT heterozygotes and 3-15% are TT homozygotes.[21] The authors in the Stroke meta-analysis conclude that, " there is evidence in support of a causal role for homocysteine in the etiology of ischemic stroke due to atherosclerosis and other mechanisms, as it is implausible that the 677C-T variant exerts its influence other than by impaired metabolism."[22] The authors also support folic acid fortification programs as providing benefit in preventing stroke by inhibiting the influence of the MTHFR 677C-T substitution on plasma homocysteine by facilitating homocysteine remethylation to methionine.[23]

A 2006 hospital based study of 32 acute ischemic stroke patients demonstrated four of 32 patients (12.5%) had high homocyteine levels. 24 Three of these four subjects were homozygous TT for MTHFR polymorphism and of these three, two had low serum folate. Five of all the total 32 subjects (18.8%) were heterozygous (CT) genotype. The researchers stated the hyperhomocysteinemia due to MTHFR C677T homozygous genotype is a risk factor for ischemic stroke and the folate levels may modify the presentation of the MTHFR TT genotype.[25]

Lars Brattstrom, DE, et.al., published a large meta-analysis in Circulation in which they concluded methylenetetrahydroflate reductase gene mutations cause hyperhomocysteinemia but do necessarily lead to vascular disease.[26] Although they noted those individuals that possessed TT genotype had an average of 25% higher mean total plasma homocysteine concentrations than the normal CC genotype, they noted no overall increase risk of cardiovascular disease (CVD.)[27] The authors elucidate other factors such as blood pressure, elevated total cholesterol, and lack of exercise as causative agents in hyperhomocysteinemia. Because renal function is a major marker of plasma homocysteine concentration, the authors consider aberrant renal function due to hypertension and atherosclerosis as major factors in elevated homocysteine compared with MTHFR genotype mutations.[28]

Discussion

The preponderance of evidence suggests the MTHFR mutation in the homozygous form and in the presence of folate deficiency leading to hyperhomocysteinemia is a provocative agent in cerebrovascular disease.

One meta-analysis showed that patients with the C677T TT genotype had a higher odds ratio of CVD disease compared to the CC genotype when the patients had low folate status. This demonstrated that high nutritional folate intake modifies the effect of the MTHFR T allele which in turn causes a smaller increase in homcysteine levels which could positively effect the risk of stroke associated with the polymorphism. This would propose a link of the effect of the MTHFR polymorphism on cerebovascular disease risk.

My research also displays conflicting evidence in the association between the MTHFR deficiency and some forms of cardiovascular disease. Two case studies support the association of MTHFR defect and stroke, however, two prospectives, one meta analysis, and two observational studies were contradictory. One of the observational studies suggested that plasma homocysteine increases were the result of increased homocysteine secretion and renal disease rather than cerebrovascular disease. This suggestion was supported by observations that the C677TTT genotypes had higher homocysteine levels but not comparable risk of cerebrovascular disease.

The biochemical and mechanistic etiology that is responsible for stroke provocation is more obscure in the medical literature. A number of mechanisms have been purported to demonstrate why hyperhomocysteinemia can promote atherogenesis and cerebrovascular insult. Speculation on the mechanisms by which hyperhomocysteinemia promotes cerebrovascular disease include the genesis of platelet adhesiveness and clotting, and initiating growth of smooth muscle cells. This proliferation of smooth muscle cells may cause vascular lesions in the endothelium.[29] Excess homocysteine can form homocysteine thiolactone (HCTL) which thiolates free amino groups in low density lipoproteins and causes them to be enveloped by macrophages which then form foams cells and atherosclerotic plaques in the endothelium.[30] Homocysteine is transformed into this cyclic thioester (HCTL) instead of being transferred into tRNA and made into proteins.[31]

Homocysteine is activated by the enzyme methionyl-tRNA synthetase. In a process termed N-homocysteinylation, atherogenesis may be caused in individuals with hyperhomcysteinemia. In this mechanism, homocysteine thiolactone acylates free amino groups of protein lysine remnants.[32] This changes the biochemical properties of proteins, specifically, lipoproteins and their role in atherosclerosis. Homocysteinylation of low-density lipoproteins (LDLs) favors oxidation and

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destruction by macrophages.[33]

Homocysteinylated LDL also cause a humoral immune response as anti-homocysteinyllsine antibodies have been detected in plasma of patients with cerebral stroke.[34] Homocysteine thiolactone is hydrolyzed to homocysteine by paraoxonase, a calcium-dependent esterase synthesized in the liver and contained in plasma high-density lipoproteins (HDLs).[35]

In a paper published in Amino Acids in 2007 the authors propose mechanisms of human toxicity by hyperhomocysteinemia.[36] The authors state that, by the incorporating of homocysteine into lipoproteins via disulfide or amide linkages (S-homocysteinylation or N-homocysteinylation), lipoprotein structure and function is altered.[37] The authors conclude that protein N-homocysteinylation causes cellular toxicity and promotes an autoimmune response which causes atherogenesis.[38] Due to the fact that homocysteine is pro-inflammatory, pro-thrombotic, and causes endoplasmic reticulum oxidative changes, the homocysteinylation mechanism is a very plausible explanation for inducement of cerebrovascular incidents.

Homocystiene also causes lipid peroxidation and free radical formation. A significant number of patients with atherosclerosis and stroke have been found to be deficient is cystathione synthase activity.[39] Cystathionine beta synthase is necessary to catalyze the condensation of homocysteine and serine to cystathionine in the transsulfuration pathway. Elevated levels of homcysteine and methionine caused by the cystathonine beta synthase deficiency are features of homocystinuria, an accumulation of homocysteine in the urine.[40] Homocystinuria is rare autosomal recessive disorder in which many patients have thrombolytic events before 30 years of age. It has been established that reducing elevated homocysteine levels due to cystathione beta-synthase deficiency of homocystinuria absolutely reduces cardiovascular risk.[41] Although the accumulation of homocysteine is not via the MTHFR mutation, this mechanism can explain platelet aggregation and thrombosis associated with hyperhomocysteinemia seen in stroke.

Another possible mechanism considers the fact that the production of homocysteine pathways circumvent production of glutathione and methionine, which are necessary to impede atherosclerosis and thrombotic lesions. Researchers in the Department of Clinical Chemistry University Hospital in Lund Sweden investigated the different fractions of homocysteine and their relation to different fractions of glutathione and cysteine in stroke patients and control subjects.[42] They noted that extracellular glutathione and cysteine influenced the formation of different homocysteine species. In patients with high concentrations of total plasma homocysteine they noted a lower ratio of reduced to total plasma homocysteine compared to a group of patients with lower concentration of total plasma homocysteine.[43] The low reduced to total ratio of plasma homocysteine in combination with elevated plasma homocysteine concentrations, they postulated, could demonstrate an increased pro-oxidant activity in plasma in these stroke patients. Therefore, accelerated pro-oxidant mechanisms in plasma could be a factor that could explain hyperhomocysteinemia.[44] The atherosclerotic and thrombotic nature of stroke may be explained by the increased anti-oxidant capabilities of hyperhomocysteinemia.

Another mechanism considers premature degradation of arterial elastic fibers by hyperhomocysteinemia induced elastolytic action. This was noted in hyperhomocysteinemia associated with aortic dissection.[45] Homocysteinylation via homocysteine thiolactone of elastic proteins in arterial walls termed fibrillin-1 caused reduction of arterial elasticity.[46] Fibrillin-1 is the primary ingredient of microfibrils that form a sheath surrounding the amorphous elastin in endothelial tissue. These microfibrils are composed of end-to-end polymers of fibrillin. Researchers noted that reduction defects in fibrillin-1 caused formation of elastin that was abnormally aggregated and more easily degraded by matrix metalloproteinases than normal elastin.[47] The researchers subsequently observed upregulation of synthesis of matrix metalloproteinases, progressive destruction of connective tissue by the enzymes and the development of aneurysms.[48] MTHFR defects that cause homocysteine elevations associated with stroke could presumably trigger endothelial damage via this mechanism.

Evidence suggests that homocysteine may function as a physiological mediator of the endothelial matrix.[49] Other oxidative mechanisms and the resultant decreased biological activity of endothelium-derived nitric oxide (NO) may also contribute to homocysteine associated endothelial damage.[50] Nitrous oxide deprivation is present in the pathomechanics of stroke. It is interesting to note that many cardiac patients receive NO pre-operatively as anesthesia. This nitrous oxide impairs methionine synthase inhibiting folate synthesis and causing postoperative hyperhomocysteinemia. Nitrous oxide anesthesia has been shown to promote postoperative endothelial dysfunction.

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The study of nutrigenomics may be the solution to the link between MTHFR deficiency and cerebrovascular disease. Nutrigenomics considers how nutrients modulate gene and protein expression and influence cellular metabolism.[51] Nutrigenomics is essentially the combination of molecular nutrition and genomics.[52] It is worth exploring how folic acid and vitamin B12 could potentially complete polymorphic gaps in our DNA, could work with DNA to synthesize the appropriate proteins, and, presumably, interfere with the expression of genes that cause cardiovascular disease and stroke.[53]

Conclusion

The results of this investigation support the hypothesis that impaired folate metabolism via the MTHFR genetic mutation, resulting in high homocysteine levels, is causally related to increased risk of cerebrovascular disease and stroke.

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Breast Thermography: Helping Make Breast Cancer Prevention Possible

by: Robert L. Kane, DC, DABCT

One out of every eight women will develop breast cancer during their lifetime impacting the lives of her family, friends and community. While countless dollars continue to be spent on finding a cure, women are led to believe that early detection through mammography is their best option for beating the disease. Is this really all that can be done? Something is missing from this picture and desperately needs to be added if we are really going to create a world without breast cancer.

Mammography has been controversial and has come into question in recent years. In fact, the US guidelines for screening with mammography recently changed from annual examinations at age 40 to every other year beginning at age 50. That means women under 50 currently do not have a viable method of screening for breast cancer and women over 50 will be screened less frequently. Women who develop cancer under age 50 tend to have more aggressive and more life threatening forms of the disease so it is imperative they have options for screening. For women over 50, the average sensitivity of mammography is 80-90% which sounds great unless you are one in every 5-10 women whose cancer is missed. Whether or not you approve of mammography and believe in its usage is not the point. Even advocates of the technology admit that the status quo is not good enough.

While it is obvious that our early detection strategies need to be improved, there is a glaring truth that cannot be ignored. Early detection is always too late. For cancer to be detected it must be already present and will require treatment that is generally invasive. We need to do better. When I was a child I remember the motto of medicine was "An ounce of prevention is worth a pound of cure." Complementary health care professionals along with progressive medical doctors are returning to these earlier roots by trying to find ways to help the body function better and prevent disease naturally. While early detection and finding a cure remain important, we must shift our focus to prevention if breast cancer is going to be eliminated.

To this end, enter a 40 year old imaging technology called thermography that has recently made a comeback. Thermography is a non invasive radiation free method of visualizing breast physiology by identifying vascular changes and fever at the surface of the breast. By evaluating physiology, we include an important component that has been missed by anatomical imaging such as mammography.

Here is how it works. Heat is produced in the breast by normal tissue metabolism and is carried to its surface by the blood supply. Our bodies naturally release heat to the environment in the form of infrared energy. Thermography uses a camera with a specialized infrared detector that captures this energy and produces an image. The image can then be analyzed allowing an interpreter to accurately measure the temperature of region of interest to the tenth of a degree.

Normal breast tissue will produce a characteristic temperature pattern when visualized with thermography and the measured temperature differences between breasts are within a normal range. Cancer produces heat through a combination of elevated metabolism and inflammation. The heat from a tumor travels through the circulatory system to the surface of the skin where it can be detected using a thermographic camera. In addition, cancer will dilate existing blood vessel via nitric oxide production and create its own blood supply via angiogenesis. Both of these occurrences can translate into a temperature finding at the surface of the breast and provide a means toward detection.

The thermal findings are less dependent on tumor size and depth and are more directly related with tumor growth rates and metabolic activity. The more aggressive and metabolically active the tumor, the more likely it will be seen on a thermogram. Thus a very small highly aggressive tumor is more likely to produce find-

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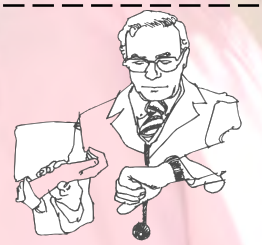

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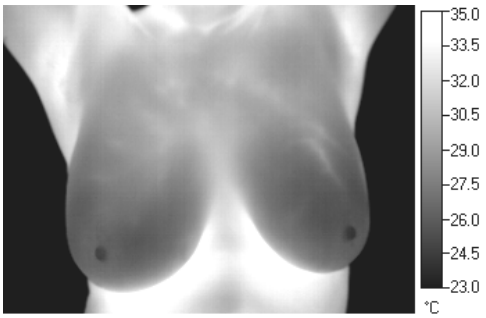
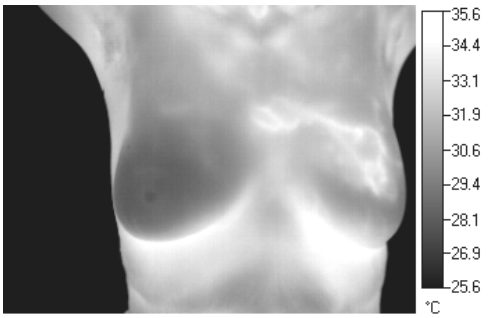
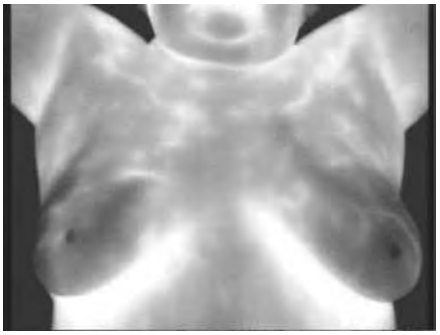


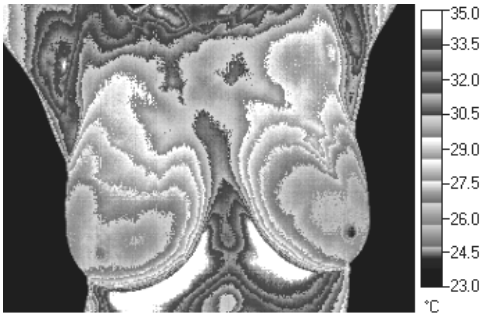
Normal Grey Scale



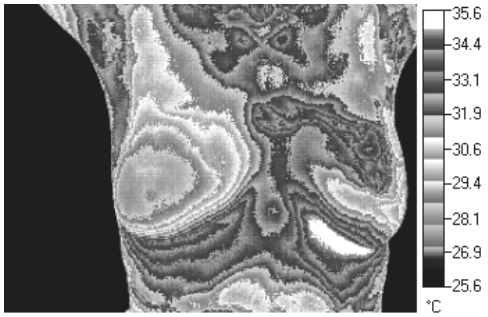
Left Breast Cancer Grey Scale



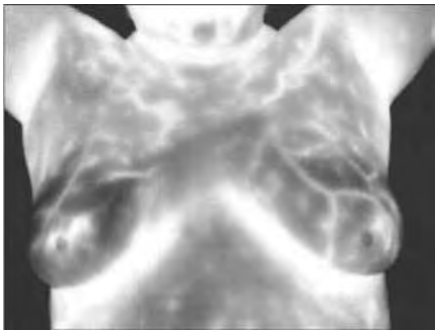
Before Intervention



Color Breast Temperature



Color Breast Temperature



After Intervention

ings on a thermogram while it may be missed by mammography because it is too small.

Perhaps more importantly, thermography provides predictive information allowing us to use it as a method to determine risk. Numerous studies have documented the presence of physiological changes consistent with cancer prior to anatomical detection with mammography. Gutherie and Gros observed that 38% of the patients with 'false positive' thermograms developed cancer within 4 years. Stark observed that 23% of the patients with 'false positive' thermograms developed cancer within 10 years. According to Gutherie and Gross, a high risk thermogram is considered to be 10x more ignificant than a first order family history of breast cancer. Hobbins further states that a sustained high risk thermogram carries with it a 22x greater likelihood of developing breast cancer than a low risk examination.

This is extremely important if we are attempting to prevent breast cancer. If thermography can be used to identify physiological signs that precede cancer and signal future risk, it can also be used to monitor the ability of therapeutic intervention to effectively lower risk.

Although a scientifically proven method to prevent breast cancer does not exist, there is a growing body of research identifying dietary and lifestyle factors that significantly contribute to risk. More importantly, many of these risk factors are modifiable and can be improved or eliminated through lifestyle, diet and natural treatment. This provides us with a starting point for creating breast cancer prevention treatments. Therapeutic interventions can then be monitored with thermography to determine if the risk has reduced.

What are the benefits of thermography over other tests that attempt to assess risk? BRCA genetic testing identifies a non modifiable risk factor that cannot be used to determine the effectiveness of treatment. Testing estrogen, estrogen metabolite (2:16 hydroxy ratios) and progesterone levels provide systemic information and do not directly evaluate the effects of these hormones on the breast tissue itself. In addition, the serum levels of hormones may not match the tissue levels. Breast tissue can have up to 50x the estrogen concentration as serum. Salivary tests have been used to assess tissue hormone levels but do not take into account that the breasts produce estrogen locally while salivary gland tissue does not. Laboratory tests such as these are still very helpful in determining a therapeutic intervention and monitoring its effects and should not be discounted. Thermography, however is a direct measure of breast physiology and ultimately needs to reflect the desired change.

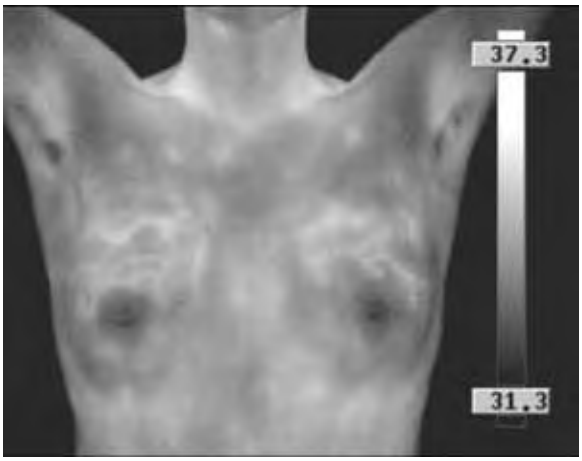
In the case of estrogen, thermography offers another unique piece of information. Thermography can help identify Breast Specific Estrogen Dominance. This is different that systemic estrogen dominance in that it occurs specifically in the breasts and may or may not be systemic. Breast Specific Estrogen Dominance produces vascular changes similar to pregnancy and

(Continued on next page)

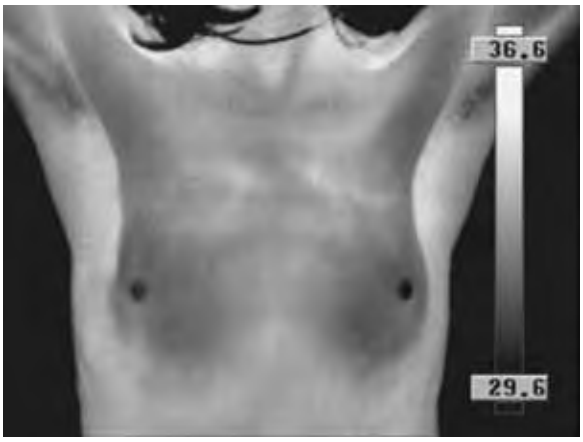
lactation. These changes are distinctly different that those of cancer and can let us know whether or not breast specific estrogen dominance exists and whether or not it is currently elevating the risk for developing breast cancer. If identified, thermography can then be used to monitor the effectiveness of intervention.

For breast cancer to be eliminated we must move beyond early detection to the realm of prevention. To create a prevention intervention we must be able to identify the modifiable risk factors and learn to improve or eliminate them. To determine if that intervention is effective, we need thermography to objectively assess the effects of that intervention on the physiology of the breasts.

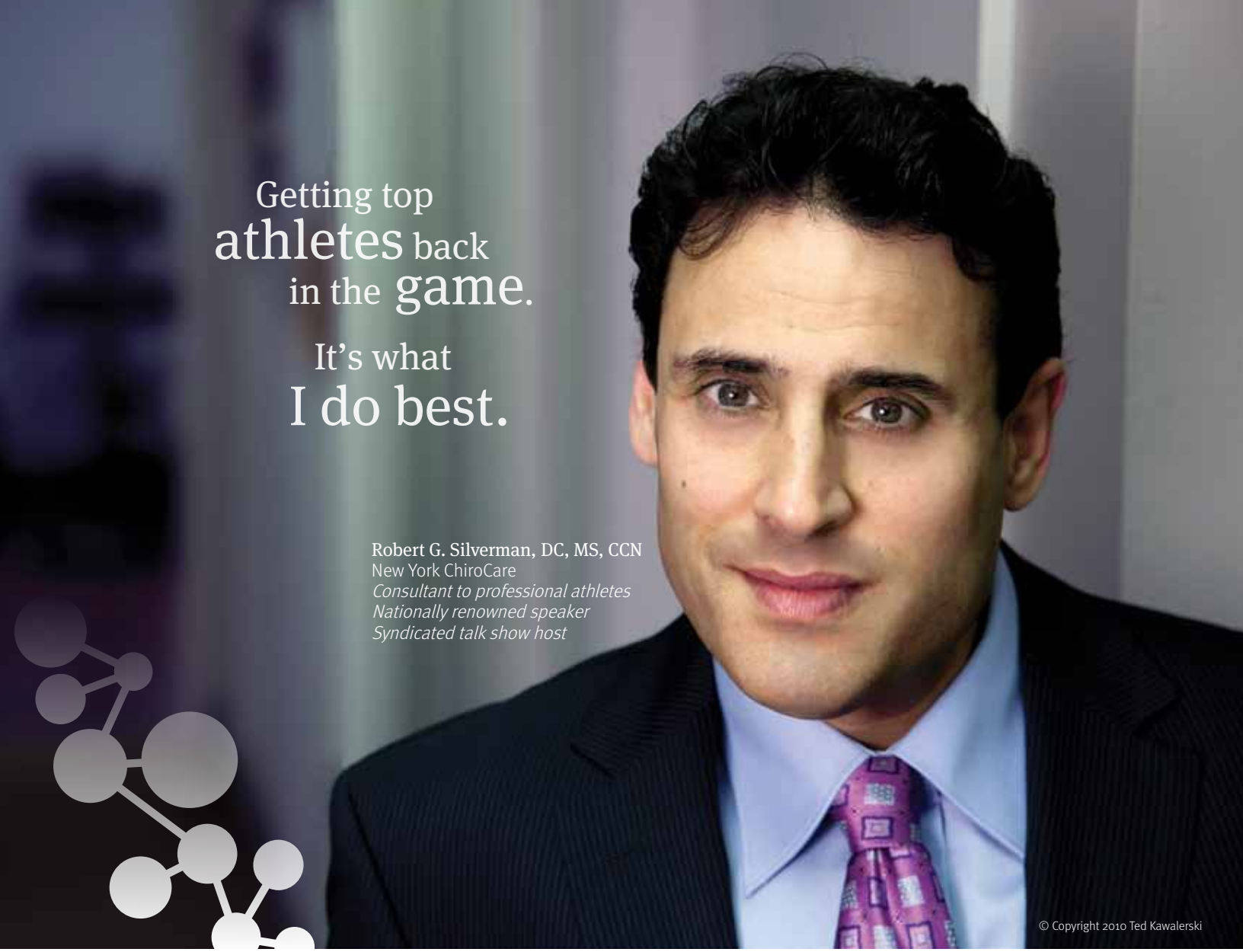
Robert L. Kane, DC, DABCT is a board certified clinical thermologist with Diplomate certification through the International Academy of Clinical Thermology and the American Board of Clinical Thermographers. Dr. Kane provides high quality training for thermography technicians and interpreters. He currently maintains a busy thermal imaging interpretation practice in Redwood City, CA. He can be reached at 650-868-0353 or rk@robertkane.com. His website is www.thermographyexpert.com. A list of references is available upon request. ◆



Normal Breast Temperature



Breast Specific Estrogen Dominance



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Abstracts of Interest

Submitted by: Emerson Ecologics

Coenzyme Q_{10} for Hypertension
by: Alan R. Gaby, M.D.

Hypertension is defined as systolic blood pressure greater than 140 mm Hg and diastolic blood pressure greater than 90 mm Hg. Hypertension is a risk factor for coronary heart disease, stroke, renal failure, and peripheral vascular disease, and lowering elevated blood pressure can decrease the risk of developing these diseases. Isolated systolic hypertension (defined as systolic blood pressure greater than 140 mm Hg with diastolic blood pressure less than 90 mm Hg) is also a risk factor for most of these conditions. A number of dietary and lifestyle modifications have been shown to be effective for lowering blood pressure. These include weight loss if overweight; regular exercise; limiting salt and alcohol intake; increasing intake of fruits, vegetables, nuts, and low-fat dairy products; and reducing stress levels. Supplementation with potassium, magnesium, calcium, and fish oil has also been found to be beneficial in some cases. Medications used to treat hypertension include thiazide diuretics, beta blockers, angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers, and calcium-channel blockers. Because non-pharmacological interventions do not always achieve satisfactory blood pressure reduction, and because antihypertensive drugs frequently cause side effects, alternative treatments for hypertension are needed.

Coenzyme Q_{10} (CoQ_{10}) is a compound that is synthesized in the body and is present in small amounts in some foods. As a cofactor in the electron-transport chain, CoQ_{10} is required for the synthesis of adenosine triphosphate (ATP), the body's main storage form of energy. CoQ_{10} has been found to lower blood pressure in five different animal models of hypertension. In addition, CoQ_{10} nutritional status (as determined by the activity of a CoQ_{10}-dependent enzyme) was significantly lower in 59 patients with essential hypertension than in controls. Thirty-two percent of the hypertensive patients, but only 6% of the controls, had CoQ_{10} deficiency. In clinical trials, supplementation with CoQ_{10} (usually in doses of 100-120 mg per day) produced substantial decreases in blood pressure in patients with essential hypertension or isolated systolic hypertension. Improvement was typically seen after one to four months of treatment.

Twenty-six patients with essential hypertension received 50 mg of CoQ_{10} twice a day for 10 weeks. Mean systolic blood pressure decreased from 165 mm Hg to 147 mm Hg and mean diastolic blood pressure decreased from 98 mm Hg to 86 mm Hg ($p < 0.001$). The fall in blood pressure was associated with a significant decrease in peripheral arterial resistance.

Eighteen patients with essential hypertension were randomly assigned to receive, in double-blind fashion, 100 mg per day of CoQ_{10} or placebo for 10 weeks. After a two-week washout period, each patient received the alternate treatment for an additional 10 weeks. Mean systolic and diastolic blood pressure fell by 10.6 and 7.7 mm Hg, respectively, during CoQ_{10} treatment ($p < 0.001$ for the difference in the change between CoQ_{10} and placebo).

Eighty-three men and women (mean age, 69 years) with isolated systolic hypertension (mean, 165 mm Hg) were randomly assigned to receive, in double-blind fashion, 60 mg of CoQ_{10} twice a day or placebo for 12 weeks. The mean fall in systolic blood pressure was significantly greater in the CoQ_{10} group than in the placebo group (17.8 vs. 1.7 mm Hg; $p < 0.01$). Fifty-five percent of the patients taking CoQ_{10} had a reduction in systolic blood pressure of 4 mm Hg or more, whereas the other 45% were nonresponders. In the responders, mean systolic blood pressure fell by 25.9 mm Hg. Mean diastolic blood pressure, which was normal at baseline, did not change.

The slow onset of action of CoQ_{10} and its known function as a cofactor in the electron-transport chain suggest that the antihypertensive effect of CoQ_{10} is mediated by an improvement in the bioenergetics of the blood vessel wall, rather than by a pharmacological effect. Other possible mechanisms of action include inhibition of the sodium-retaining effect of aldosterone and a decrease in the sensitivity of peripheral blood vessels to norepinephrine.

CoQ_{10} is generally well tolerated. Minor gastrointestinal side effects have been reported in less than 1% of patients taking 30 mg per day of CoQ_{10}. In patients with Parkinson's disease, CoQ_{10} at a dose of 1,200 mg

(Continued on next page)

per day for 16 months and in doses up to 3,000 mg per day for 8 months did not cause any serious adverse effects. There have been several case reports in which supplementation with CoQ_{10} appeared to interfere with the effect of warfarin. However, in a double-blind trial, administration of 100 mg per day of CoQ_{10} for four weeks had no effect on the International Normalized Ratio (INR) in 21 patients on long-term warfarin therapy. Thus, the sporadic case reports of an interaction between CoQ_{10} and warfarin may have been due to random fluctuations in INR values, rather than to CoQ_{10}. Because of its relatively large antihypertensive effect and its low toxicity profile, CoQ_{10} should be considered for first-line therapy of patients with hypertension who do not respond sufficiently to diet and other life-style changes.

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St. John's Wort for Menopausal Symptoms

by: Tori Hudson, N.D.

St. John's Wort products and extracts have been used for a wide range of medical conditions, the most common being depressive disorders. The most robust research is in the area of mild to moderate depression, with some additional research in anxiety, severe depression, seasonal affective disorder, premenstrual syndrome, and perimenopause/menopause. St. John's Wort is the most thoroughly researched natural antidepressant, but the majority of these studies have not been conducted on menopausal women. What follows is a review of the research on St. John's Wort for menopausal symptoms.

In the newest of St.John's Wort/menopause studies, a total of 100 Iranian women with an average age of 50 participated in a randomized, double-blind, placebo-controlled clinical trial comparing St. John's wort with placebo in women with perimenopausal/menopausal hot flashes.

50 women received 20 drops three times daily of St. John's Wort extract (Hyperin) that contained hypericin 0.2 mg/mL and 50 women received a placebo of distilled water. The study duration was two months. Clinical exams and interviews were performed at baseline, 4 weeks and 8 weeks. Treatment effectiveness was measured by the Blagg-Kupperman Index. Evaluation of frequency, duration, and severity of hot flashes was the main objective of the study.

Forty-five women in the treatment group and 43 in the control group completed the study. In women taking St. John's wort, the frequency began to decline during the first and second months, but showed more improvement during the second month. There was no statistical change in hot flash frequency during the first month of placebo but did improve during the second month. Women who used St. John's Wort showed more improvement in frequency than placebo. The decline in duration of hot flashes was statistically significant at week eight and the decline was much more evident in the St. John's Wort arm. The severity of hot flashes was relieved in the St. John's Wort during the two months of treatment and was more significant in the second month. Women in the placebo group did not show any significant decrease in severity of hot flashes during the first month, but they did have some improvement during the second month, but not as great as those women in the St. John's Wort group.

This is not the first time that St. John's Wort has been studied for hot flashes in perimenopausal/menopausal women. A recent double-blind randomized clinical trial, studied the effect of Hypericum perforatum extract (St. John's wort extract) compared with placebo on symptoms and quality of life of 47 symptomatic perimenopausal women aged 40 to 65 with three or more hot flashes per day. Women were randomly assigned to receive a St. John's Wort extract (900 mg three times per day) or placebo. After 12 weeks of treatment, a nonsignificant difference in favor of the St. John's Wort group

(Continued on page 146)

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was observed in the daily hot flash frequency and the hot flash score. After 3 months of treatment, women in the St. John's Wort group reported significantly better quality of life scores, and significantly fewer sleep problems compared to placebo.

About ten years ago, an uncontrolled, drug monitoring study was conducted in women with menopause symptoms using 900 mg of St. Johns Wort for 12 weeks. About three quarters of the women experienced improvement in both the self-rating scale and the physician rating, and significantly improved in psychological and psychosomatic symptoms as well as a feeling of sexual well-being.

The first of now three studies using St. John's Wort and black cohosh was published in 1999. This double-blind, randomized, placebo-controlled trial used St. John's Wort and black cohosh made by the makers of Remifemin. 179 women with menopause symptoms received two capsules given once daily or placebo. The Kupperman Index for the combination product decreased from 31.4 to 18.7 compared with a decrease in the placebo group from 30.3 to 22.3. Psychological symptoms also improved significantly in the black cohosh/St. John's wort combination.

In 2006, a double-blind, randomized, placebo-controlled study was done using a combination trial of black cohosh and St. John's wort. The mean Menopause Rating Scale score decreased 50% in the treatment group and 19.6% in the placebo group. The Hamilton Depression Rating Scale score decreased 41.8% in the treatment group and 12.7% in the placebo group. In both testing measures, the St. John's wort + black cohosh group was significantly superior to the placebo group.

In 2007, a second black cohosh/St. Johns wort trial was carried out in peri or postmenopausal Korean women, and was published in 2007. Healthy perimenopausal women with typical climacteric symptoms and not on HRT for at least the previous 3 months were given a 264 mg tablet containing 0.364 mL of extract from black cohosh equivalent to 1 mg terpene glycosides and 84 mg of St. Johns wort extract with 0.25 mg hypericin. 42 women completed the study in the treatment group and 35 women in the placebo group completed the study. Mean Kupperman Index scores at 4 and 12 weeks were significantly lower in the treatment group ($P \leq 0.002$). At the end of the study, the average decrease in the Kupperman Index was 20 points in the treatment group and only 8.2 points in the placebo group. ($P < 0.001$). Vaginal dryness and low libido were two symptoms that did not improve, but the average hot flash scores were significantly lower in the black cohosh/St. Johns wort group.

A third study was done in which a combination of black cohosh with or without St. John's wort was used in 6141 women at 1287 outpatient gynecologists in Germany in a prospective, controlled open-label observational study. Women were given a standard dose of black cohosh extract in the form of 2.5 mg isoproponolic extract monotherapy or a black cohosh extract of 3.75 mg isopropronolic black cohosh plus combination which also contained 70 mg of an ethanolic extract from 245 to 350 mg of St. John's wort. The main symptoms were the same in both groups: hot flashes, sleep disorders, nervousness and depressive mood and all of a moderate-severe nature. The greatest changes occurred with the combination therapy for nervousness/irritability and mood swings, but in the area of depression, there was a reduction in both treatment groups, which is a bit surprising given the potential for added anti-depressive benefits with the addition of St. John's wort.

In my experience, St. John's wort is an important clinical tool in treating perimenopausal/menopausal women for hot flashes and/or depression and/or mood swings, and as a single agent, or in combination with other therapies.

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Vitamin E Protects Patients from Cisplatin-induced Neuropathy in a Randomized Trial
by: Steve Austin, N.D.

Reference: Pace A, Giannarelli D, Galie E, et al. Vitamin E neuroprotection for cisplatin neuropathy. *Neurology* 2010;74:762–6.

Design: Randomized double blind intervention trial

Participants: Of 108 patients initially randomized in the trial, 41 were treated with cisplatin exceeding 300 mg/m^2. Of these 41 subjects, 17 had been given vitamin E and 24 had been given placebo.

Study Medication and Dosage: 596 IU d-alpha tocopherol or placebo daily, begun 1 to 8 days before the start of cisplatin therapy and continued for 3 months following discontinuation of cisplatin therapy

Outcome Measures: Development of peripheral neuropathy as a result of cisplatin therapy and measurement of the severity of the neurological damage through the use of Total Neuropathy Score (TNS)

Key Findings: Neurotoxicity occurred in 6% in the vitamin E group versus 42% in the placebo group ($p<0.01$). The severity of neurological damage was also far lower in the vitamin E group (1.4 TNS versus 4.1; $p<0.01$).

Practice Implications: Cisplatin use is limited by severe neurotoxicity. When the cumulative dose exceeds 300 mg/m^2, in some reports most patients suffer significant neurological damage. Cisplatin is known to increase oxidative stress, vitamin E is an antioxidant, and current evidence suggests that the antioxidant actions of vitamin E do not interfere with the therapeutic effect of chemotherapy (*Int J Cancer* 2003;104:243–50). Neuropathy has been reported to occur as part of the presentation of vitamin E deficiency. As a result, researchers are interested in exploring the ability of vitamin E to protect against cisplatin-induced neuropathies.

In 2007, a Cochrane review concluded that previous evidence linking the use of vitamin E to protection against neuropathy in patients given cisplatin was encouraging but needed additional support from randomized clinical trials (*Cochrane Database Syt Rev* 2007;1:CD005228). The findings of the new trial take us a significant step in that direction. Until more is known, all patients given cisplatin should concomitantly take vitamin E supplements beginning several days before the start of the chemotherapy.

Oral Magnesium Again Reported to Help Asthma Sufferers in a Randomized Trial
by: Steve Austin, N.D.

Reference: Kazaks AG, Uriu-Adams JY, Albertson TE, et al. The effect of oral magnesium supplementation on measures of airway resistance and subjective assessment of asthma control and quality of life in men and women with mild to moderate asthma: a randomized placebo controlled trial. *J Asthma* 2010;47:83–92.

Design: Randomized double blind intervention trial

Participants: 55 adults with mild to moderate asthma who used only beta-agonists or inhaled steroids for asthma control

Study Medication and Dosage: 170 mg magnesium (Mg) from Mg citrate given b.i.d., or placebo for 6.5 months

Primary Outcome Measures: Methacholine challenge test, a variety of pulmonary function tests, subjective quality of life scores, C-reactive protein (CRP), and exhaled nitric oxide (NO)

Key Findings: Peak expiratory flow rates increased 6% in the Mg group ($p=0.03$), though several additional parameters of lung function did not change significantly in the Mg group. The concentration of methacholine required to cause a 20% reduction in force expiratory volume increased in the Mg group while slightly declining in the placebo group ($p=0.02$; specific data not provided). On a 7-point quality of life index, the Mg group experienced a 0.5 point improvement ($p=0.004$) versus a 0.2 point improvement with placebo (NS). Indicators of inflammation, NO and CRP levels, did not improve significantly in either group.

Practice Implications: While most Americans consume less than recommended amounts of Mg, some but not all previous research indicates that asthma sufferers tend to have even lower Mg status than do other Ameri-

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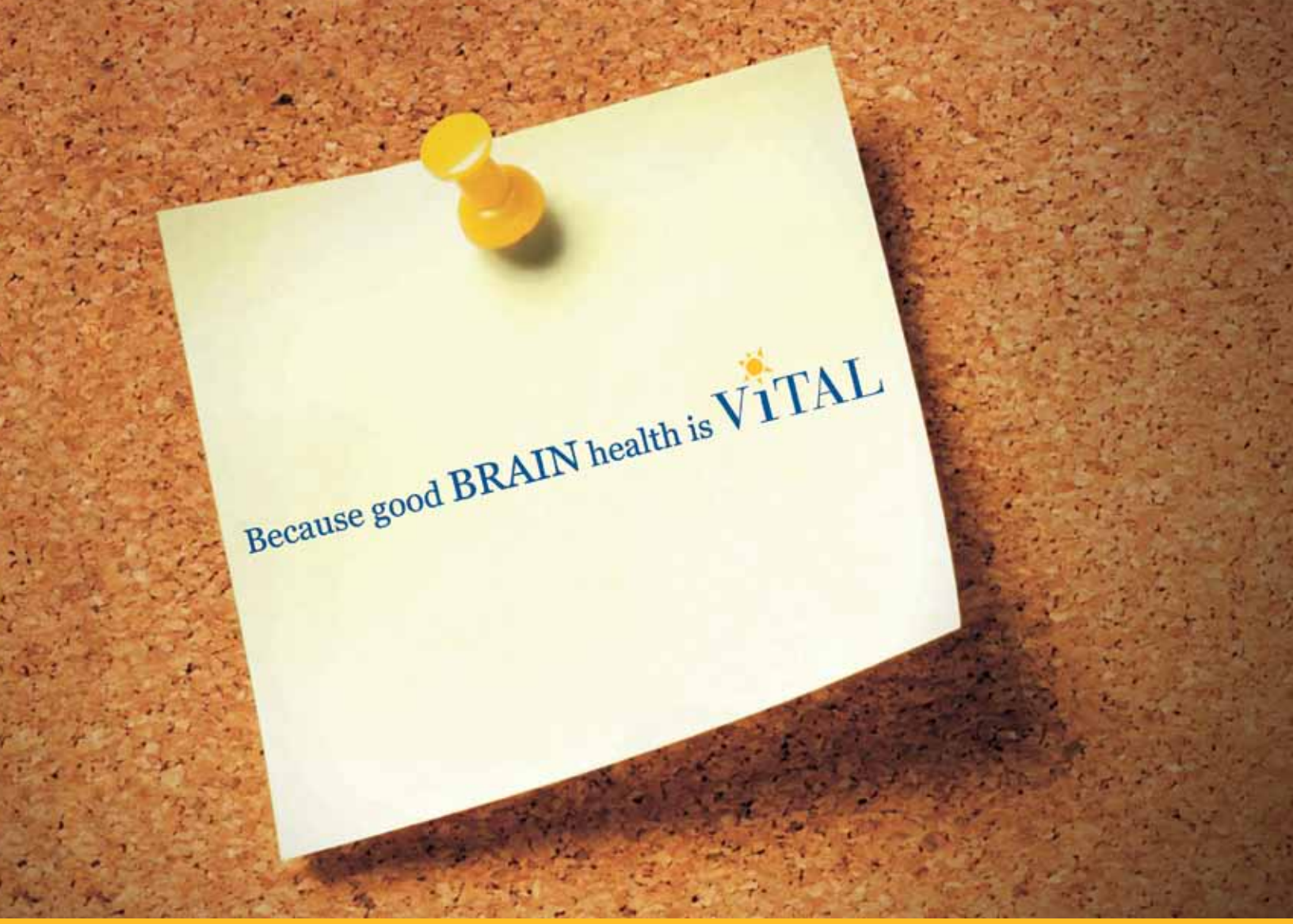

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cans. Mg is believed to have bronchodilatory and anti-inflammatory effects that might help asthma patients. However, previous Mg trials studying therapeutic effects in asthmatics have produced only mixed results.

The primary factor separating the current trial from previous negative reports is duration. The 6.5 month intervention used by these researchers was significantly longer than the 3 to 12 week interventions employed elsewhere. The authors show that previous trials studying bronchial responsiveness have produced positive results when interventions have lasted several months, but negative results when they only occurred for several weeks. These new findings should change our view of the appropriate length of time needed to evaluate the effects of Mg supplementation in people with asthma. ◆

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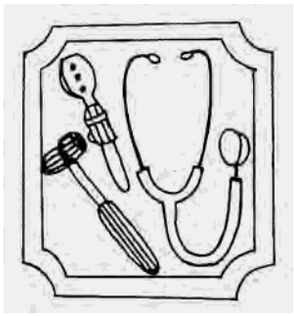

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