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*The Original Internist* is published quarterly. Publication months are March, June, September and December, barring any unusual or unforeseen circumstances.

News items and/or letters pertaining to natural health care are welcome. The editorial staff reserves the right to edit and/or reject all material received. Letters to the editor may be condensed in order to fit the allotted space. An address and telephone number where the author may be reached during normal business hours should also be included for verification purposes. Deadline for article submission is the 15th of the month preceding publication.

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Pediatrics  
Instructor: *Frank Strehl, DC DABCI*

**June 16, 2007** (Chicago, IL)  
Session 5 - Physical Examination Workshop  
Instructors: *Cindy Howard, DC DABCI & Frank Strehl, DC DABCI*

**June 23-24, 2007** (Kansas City, MO)  
Evaluating Vascular & Venous Disorders by Instrumentation  
Instructor: *Jack Kessinger, DC DABCI*

**July 7-8, 2007** (Houston, TX)  
Allergy Part 2 - Management of the Hypertensive Patient  
Instructor: *Jack Kessinger, DC DABCI*

**July 14-15, 2007** (Charlotte, NC)  
Spirometry and Pulmonary Disease  
Instructor: *Jack Kessinger, DC DABCI*

**July 20-22, 2007** (Las Vegas, NV)  
Symposium

**July 28-29, 2007** (Kansas City, MO)  
Peripheral Vascular Disease Workshop  
Instructor: *Tim McCullough, DC DABCI*

**August 4-5, 2007** (Houston, TX)  
Common Diseases Affecting the Arterial System  
Instructor: *Jack Kessinger, DC DABCI*

**August 11-12, 2007** (Charlotte, NC)  
Geriatrics  
Instructor: *Jack Kessinger, DC DABCI*

**August 25-26, 2007** (Kansas City, MO)  
Facts of Neoplastic Process & Examining the Cancer Patient  
Instructor: *Jack Kessinger, DC DABCI*

**September 8-9, 2007** (Houston, TX)  
Evaluating Vascular & Venous Disorders by Instrumentation  
Instructor: *William Kleber, DC DABCI*

**September 15-16, 2007** (Charlotte, NC)  
Urinary Disorders and Hair Biopsy Assessment  
Instructor: *Frank Strehl, DC DABCI*

**September 29-30, 2007** (Kansas City, MO)  
Malignant Diseases, AIDS, & Their Management & Treatment  
Instructor: *William Kleber, DC DABCI*

**October 6-7, 2007** (Houston, TX)  
Peripheral Vascular Disease Workshop  
Instructor: *Tim McCullough, DC DABCI*

**October 13-14, 2007** (Charlotte, NC)  
Immunology and Allergy, Part 1  
Instructor: *Jack Kessinger, DC DABCI*

**October 20-21, 2007** (Kansas City, MO)  
Upper Gastrointestinal Disease  
Instructor: *Jack Kessinger, DC DABCI*

**October 27-28, 2007** (Chicago, IL)  
Introduction to Chiropractic Internal Disorders  
Instructor: *Jack Kessinger, DC DABCI*

**November 3-4, 2007** (Houston, TX)  
Facts of Neoplastic Process & Examining the Cancer Patient  
Instructor: *Jack Kessinger, DC DABCI*

**November 10-11, 2007** (Charlotte, NC)  
Allergy Part 2 - Management of the Hypertensive Patient  
Instructor: *William Kleber, DC DABCI*

**November 17-18, 2007** (Kansas City, MO)  
Lower Gastrointestinal Disease  
Instructor: *Frank Strehl, DC DABCI*

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History Taking  
Instructor: *Jack Kessinger, DC DABCI*

**December 1-2, 2007** (Houston, TX)  
Malignant Diseases, AIDS, & Their Management & Treatment  
Instructor: *William Kleber, DC DABCI*

**December 8-9, 2007** (Charlotte, NC)  
Common Diseases Affecting the Arterial System  
Instructor: *Jack Kessinger, DC DABCI*

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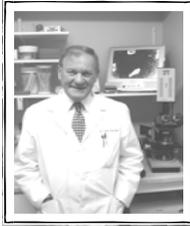
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# From the Editor's Desk

by Jack Kessinger, DC, ND, DABCI  
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Dr. Jack Kessinger

## ***Where Am I Supposed To Get My Calcium?***

If I can't have cow's milk, where am I supposed to get my calcium? The next question should be, "Where does a cow get calcium?" Naturally, it's from the food it eats. Likewise, we should concentrate on foods rich in calcium. There are numerous foods which provide calcium — green leafy vegetables, almonds, parsley, beet greens, broccoli, spinach, green beans, just to name a few.

Nature provides the protein linkages, fat molecules, and hormones intended to bring calves to maturity in two years, at about 2,000 pounds and with a low IQ. Humans are the only mammals who do not wean their young (or drink milk from another species).

Calcium is the most abundant mineral in our bodies and has numerous essential functions. The National Academies Press reported in 1997, that in addition to building bones and teeth, calcium is needed for blood vessel contraction and expansion and secretion of hormones and enzymes, as well as sending messages through the nervous system. A constant level of calcium is maintained in body fluid so these vital body processes function efficiently. Bone undergoes continuous remodeling, with constant resorption and deposit of calcium in new bone formation. The balance between resorption and deposition changes as people age. During childhood, there is a higher amount of bone formation and less breakdown. In early and middle adulthood, these processes are relatively equal. In aging adults, particularly among postmenopausal women, bone breakdown exceeds its formation, resulting in bone loss, which increases risk for osteoporosis.

There are several types of calcium; calcium carbonate, lactate, gluconate, and citrate, are the ones usually associated with human needs. Calcium carbonate is the most abundant type of calcium, and the most difficult of all calciums for humans to absorb. Calcium carbonate is

the type of calcium found in milk and often is described as "closely resembling chalk." A very low gastric pH is essential for absorption of milk. Dairy tends to reduce HCl, thereby reducing the body's ability to absorb the type of calcium found in cow's milk.

In addition to the natural hormones found in all mammals' milk, the latest high-tech onslaught on the cow is the addition of bovine growth hormone, better known as BGH. This genetically engineered drug is provided to stimulate increased milk production. A related problem is that it causes a marked increase (50-70%) in mastitis in cows. This, in turn, requires regular antibiotic therapy. These antibiotics and hormones are consumed with the milk by humans.

All lactating mammals excrete toxins through their milk. These include antibiotics, pesticides, chemicals, and hormones. Also, all cow's milk is reported to contain blood. According to *The Milk Letter: A Message to My Patients*, by Robert M. Kradjian, MD, the USDA allows milk to contain from one to one and a half million white blood cells per milliliter (1/30 of an ounce). White blood cells found where they are not supposed to be found are called "pus cells." If the cow has mastitis, there is pus in the milk. However, there are ways to camouflage that fact with language such as "macrophages containing many vacuoles and phagocytosed particles," etc.

The dairy industry has done such an excellent job of marketing their product that the general public has the common belief that milk is its only source of calcium. We have been told that milk is an important source of calcium that helps kids grow up big and strong. Milk is said to contain vital nutrients and to help prevent osteoporosis. A well orchestrated \$180 million annual advertising budget has convinced the US Department of Agriculture of the importance of dairy. Its dietary guidelines report that everyone should receive between 2-3 servings of dairy every day. Milk is advocated by various agencies of the US government, legions of physicians and white-mustached celebrities (e.g., Britney Spears, Carson Daly, Naomi Campbell, Spike Lee, and Rudy Giuliani).

A 12-year prospective study, reported in the *American Journal of Public Health* (1997; 87:992-7) was performed among 7,761 women (aged 34-59) who had never used a calcium supplement. Women who drank two or more glasses of milk per day had relative risks of 1.45 for hip fractures and 1.05 for forearm fracture, when compared with women consuming one glass or

*(Continued on next page)*

less per week. Higher intakes of total dietary calcium (or calcium from dairy products) were not associated with decreased risk of hip or forearm fracture.

A large Harvard study, reported in the *Journal of Nutrition* (1997; 127:1782-7) and the *American Journal of Public Health* (1997; 87:992-7), of male health professionals and female nurses reported that individuals who drank one glass of milk (or less) a week were at no greater risk of breaking a hip or forearm than those who drank two or more glasses per week.

However, another study, published in the *New England Journal of Medicine* (1997; 337:670-6), reported supplementation with calcium along with vitamin D significantly reduced bone loss and the incidence of non-vertebral fractures

Very early in my chiropractic career, I serendipitously discovered that cow's milk, if consumed regularly, will commonly contribute to a wide array of health problems including chronic back pain, a compromised immune system, and delayed food allergies. According to Amy Lanou, PhD, the nutrition director of the Physicians Committee for Responsible Medicine (PCRM), "Besides prostate cancer, milk has been linked to asthma, anemia, allergies, juvenile-onset diabetes mellitus, obesity, heart disease, and ovarian and breast cancer." A study in the *American Journal of Epidemiology* (May 1, 2007) reported a study of more than 130,000 US adults, followed for nine years, confirmed a relationship between large amounts of dairy products and increased rates of Parkinson's Disease.

Just a few weeks ago, a mother brought her 15-year-old son to our clinic for examination and treatment for chronic lower back and hip pain. His mother reported that he loves and excels at sports and "hurt his back" playing football during the seventh grade. Furthermore, she reported that following the injury, they had spent several thousand dollars trying to find what caused the pain and fix it. During the history I learned he consumed milk every day, and with most meals. On the physical examination, in addition to subjective pain reported to the lower back, the kidney flank was reported to be tender. He reported relief following a spinal diversified adjustment to correct the subluxation. I predicted that if he would avoid dairy, he would be well in a week. Two weeks later, his mother reported him to be pain free, and he played in the season opening baseball game for his high school.

Two years ago a 45-year-old female fell down her basement stairs on a Monday and was then transported to the

hospital via ambulance. Numerous MRIs showed two large bulging discs between C6 and C7. Surgery was scheduled for the following Monday, with the plan being to replace the two ruptured discs with discs from a cadaver. Recovery time was expected to last a year. She got some good advice to try alternative therapies first. Rather than the planned surgery on Monday, she called for an appointment and arrived at our clinic with her right arm resting in a sling, and her left hand supporting her right elbow. She reported the pain was intense and radiated from her lower cervical spine to the right shoulder and into her arm and fingers. She was unable to drive due to the unrelenting pain. She brought copies of the MRIs, which revealed two of the largest bulging discs I had ever seen. While conducting the history, I learned she had long suffered from sinusitis and experienced frequent sore throats, colds, and influenza. Her diet was not the best, and she reported consuming milk daily. A very conservative treatment approach, with dietary counseling, nutritional support, light manipulation, and ultrasound was initiated. Effectiveness of treatment was certainly slow for the first two weeks. However, five weeks later she asked when could she play golf again. I advised, that in order to allow for maximum healing, to wait until the next summer. The next week she reported that she had played golf, and it didn't hurt. We now see her occasionally for adjustments. Her diet has improved, and she avoids dairy. She reports no sinusitis, colds, or flu. She also plays volleyball, reportedly at 75% effort.

In my experience and humble opinion, the consumption of dairy is responsible for numerous health problems and has increased the financial rewards of the health care profession exponentially. ♦

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TO SUBMIT ARTICLES

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# *The Legacy Continues*

by A. Jay Kessinger IV, DC ND  
jay@drkessinger.com

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“Never hit a lick that doesn’t count.” “A rolling stone gathers no moss.” “The only ones out after midnight are hoodlums, hooligans, and cops.” These are just a few of the mantras that I’ve heard enough times to have become a part of who I am. “Every action has a reaction, and there are like consequences for each.” “If you don’t focus you won’t be able to concentrate.” “If we don’t get enough rest (‘keep our batteries charged’), we’ll never be able to achieve our potential.” And, “there will always be those that either think we’re on drugs, or we should be.”

Personal perception of an individual conception that leads to an accurate interpretation with a consecutive succession of successful applications is the ultimate desire of most. However, in order to be truly satisfied with the whole process, one need mostly feel it’s his/her own idea. It is here that the doctor/patient relationship can be most likened to a marriage, whether blissful or miserable. The doctor, like a good spouse, will direct the patient in the direction of correction, and in a healthy relationship there will be mutual trust and appreciation. The patient most commonly made the initial overture which led to this relationship and ultimately, makes any decisions on his/her own. If the patient chooses to continue allowing the doctor the privilege of treatment, then he/she has permission to mentor the partner toward the attainment, and then maintenance, of the optimal level of health.

In the practice of professional health care, like the lessons learned and wisdom applied in life, we all are in search of the techniques and methods that work for us. The lessons already learned by those we trust is an excellent starting point in finding our own way, but we must all find our own way. Living life is like the practice of professional health care delivery in that they are both dynamic and constantly change, whether in growth or in deterioration. The latter consequence, a negative yet albeit potentially real outcome, is the result of poor planning and energy expenditure.

We need to not only find what works for us, then test it, and try it out for competence and endurance, and modify it diligently until the final product is the best we have to use, but most importantly, after finding what works for

us, we need to add to it. Never take away from, but always add to, what works!

Hitting licks that don’t count many times could be considered “trying to reinvent the wheel.” Most costly and foolish is an attempt to prove that wheels aren’t round. Why in the world would any sane person do this? The answer is, “Because we’re human.” What a cop out! Write down your goals, and meditate/pray over them often, and focus! “If you build it, they will come.”

Did you ever wonder why one of the prerequisites to being President of the United States is that you have to be at least 35 years old? In my younger days, I thought this was a form of discrimination. However, since I’ve passed that phase, I can see that until we’re 35, we never realize that not getting enough sleep made *us* the problem.

Convince your patients through documentation that there is hope. Maintain your own spiritual and physical well-being by practicing what you preach. Gain their trust, and then you can show them how to enjoy life optimally through your recommendations.

These are just a few of the mantras that I’ve written enough times to have become a part of who *you* are. ♦

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# *Systemic Mycoses: An Overview for Natural Health Professionals*

by R. Thiel, PhD, NHD

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This is an updated version of an article originally printed in *The Original Internist* in December 2002.

## **Introduction**

There are over 100,000 different species of fungi, of which approximately 150 are known to be pathogenic to humans.<sup>1,2</sup> 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.<sup>3</sup> 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.<sup>1,4</sup>

Amazingly, even though hundreds of peer-reviewed scientific articles, the *Merck Manual*<sup>5</sup>, and Mayo Laboratories<sup>6</sup> 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 a period of time to be effective.<sup>7</sup> Partially due to this medical disbelief, many natural health professionals see people with a variety of mycotic infections on a regular basis.<sup>7</sup> Some people with them have been told that the symptoms are “all in their head” or something just as useful.<sup>7</sup>

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.<sup>8</sup> 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.”<sup>8</sup> In most “immunocompetent patients, systemic mycoses typically have a chronic course,” instead of be-

ing life threatening.<sup>5</sup>

Most systemic mycoses are from opportunistic fungi. They are saprocytes — organisms which live on decaying matter — which are usually innocuous but become pathogenic when the host becomes abnormally susceptible to infection.<sup>1,5,9,10</sup> 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 AIDS, malnutrition, metabolic diseases, receipt of multiple injections, certain surgeries, burns, intravenous hyperalimentation, and certain malignancies.<sup>1,10-12</sup> 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, malaise, depression, and chronic sinusitis<sup>1,6,9-14</sup>; some of them may be risk factors in developing autoimmune disorders.<sup>7,15</sup> 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

*(Continued on next page)*

esophagus, lung, colon, kidney, breast, uterus, blood, lymph nodes, brain and skin, some autoimmune disorders ... scleroderma ... diabetes ... rheumatoid arthritis ... Sjogren's syndrome ... psoriasis ... and systemic lupus erythematosus. 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 ... Crohn's disease ... endometriosis ... infertility ... migraines."<sup>5</sup> Many mycoses are polysymptomatic<sup>16</sup> 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, MD, 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."<sup>7</sup>

Although there exists a tremendous amount of natural health literature regarding interventions to be considered for people with an overgrowth of *Candida albicans*,<sup>5,20-29</sup> 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, found frequently 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."<sup>5</sup> Aspergilli are the second most common systemic mycoses and account for nearly 30% of fungal infections found at autopsy.<sup>1</sup> They often appear after antibiotic or antifungal therapy (to which they are usually resistant);<sup>9</sup> this is one distressing area of fighting systemic mycoses — sometimes when eliminating one type, another becomes prominent.<sup>9</sup>

Clinical findings are usually nonspecific and standard sputum cultures are positive only 33% of the time aspergilli are present.<sup>1</sup> "Sputum from patients with aspergilomas often does not yield *Aspergillus* in cultures because cavities are likely to be walled off from airways."<sup>5</sup> They often are implicated in respiratory conditions,<sup>1,13</sup> including sinusitis;<sup>30</sup> it appears that sometimes, *Candida albicans* (IgE and IgG subclasses) may participate in worsening pulmonary infiltrates when bronchopulmonary aspergilliosis is present.<sup>31</sup> Aspergilli are often mistaken for zygomycetes.<sup>1</sup> As enzymes appear to play a role in the reproduction of various species of *Aspergilli*,<sup>32</sup> it is possible that enzyme inhibitors may play a role in diminishing their reproduction and growth.

*Aspergillus fumigatus* is the most common form.<sup>1,2</sup> *Aspergillus flavus* is commonly associated with aflatoxins,<sup>2</sup> such as on peanuts.<sup>30</sup> Restrictocin and mitogillon are two other toxins produced by aspergilli — they inhibit host cell protein synthesis by degrading mRNAs.<sup>30</sup> "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."<sup>30</sup> This is probably true of most situations when a systemic mycotic infection is present. Invasive aspergillosis is usually confined to immune-suppressed and debilitated hosts.<sup>30</sup> Some with gastrointestinal upset have *Aspergillus*,<sup>1</sup> and some with intense itching may have some version of it (superficial lesions are also a symptom).<sup>5</sup> Aspergilli "fungus balls neither require nor respond to systemic antifungal therapy," though some other aspergilli forms do.<sup>5</sup> Mayo found it was one of the most common fungal organisms associated with fungal sinusitis.<sup>15</sup> This investigator's clinical experience sug-

(Continued on next page)

gests 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.

**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.”<sup>5</sup> It can produce dry hacking and affect the prostate, testes, kidneys, vertebrae, brain, nose, thyroid, lymph nodes, and bone marrow, but skin lesions are probably most common.<sup>5</sup> Men (especially over age 40<sup>5</sup>) are afflicted with it more than women, with wart-like lesions on the skin and some times internal organs.<sup>33</sup> There is also a South American form called *Paracoccidioidomycosis* which mostly effects men aged 20-50 who work as coffee growers.<sup>5</sup>

**Candidiasis:** *Candida albicans* is the most common cause of Candidiasis.<sup>1,2,10</sup> 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.<sup>30</sup>

*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)<sup>34,35</sup> and “is now the fourth most prevalent organism found in bloodstream infections.”<sup>5</sup>

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<sup>1,5</sup> such as a selective defect in the functioning of T-lymphocytes.<sup>36</sup> It can exist as 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.<sup>30</sup> *Candida* has molecules on the surface that mediate its adherence to human tissues which are the main ways it negatively affects health.<sup>30</sup> “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.”<sup>7</sup>

“All forms of disseminated candidiasis should be considered serious, progressive, and potentially fatal. Predis-

posing conditions such as neutropenia, malnutrition, or uncontrolled diabetes should be reversed or controlled where possible.”<sup>5</sup> All forms of candida do not respond to the same medical<sup>5</sup> or other interventions. *Candida albicans* and *C. glabrata* tend to respond similarly, whereas *C. cruzi* does not.<sup>5</sup>

However, many nutritional interventions have been reported to be effective for candida.<sup>7,21-29</sup> Since *Candida albicans* is often grown in a culture of various saccharides,<sup>2</sup> it is not surprising that reductions in the consumption of refined sugars has been effective.<sup>13-15</sup> Sometimes, this investigator and others<sup>21-24</sup> 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.<sup>34</sup>

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.”<sup>11</sup>

There have been substantial increases of candidemias caused by species other than *Candida albicans*.<sup>37</sup> *Candida tropicalis* is probably the second most common cause of candidiasis.<sup>1,5</sup> Infections with *Candida glabrata* and other candida species are increasing with frequency.<sup>5</sup> *C. glabrata* can cause fungemia, urinary tract infections, sometimes pneumonia or other focal lesions.<sup>5</sup> *Candida paratropicalis* is quite similar to *Candida tropicalis* and is often confused with it.<sup>1</sup> A significant difference is that paratropicalis does not thrive with sucrose, although tropicalis does.<sup>2</sup> *Candida krusei* (also spelled cruzi) seems to be less affected by refined sugars (other than dextrose) than most other candida species,<sup>2</sup> thus this investigator rarely encourages reduction of fruit consumption when it is suspected. Other candida species such as *C. guilliermondi*, *C. parapsilosis*, and *C. pseudotropicalis* can cause infections in humans,<sup>1,10</sup> but (other than any differences their shape may account for<sup>2</sup>) 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.<sup>38</sup>

*Candida zeylanoides* used to not be considered to be a pathogenic yeast for humans,<sup>39,40</sup> but can occur in individuals who do not have the “usual risk factors for systemic candidiasis.”<sup>41</sup> Case reports have suggested that it

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can cause arthritis,<sup>42</sup> infective endocarditis,<sup>41</sup> onychomycosis (nail infection),<sup>40</sup> and gastrointestinal disturbances.<sup>39</sup> It may be implicated in scleroderma.<sup>39</sup> An animal study suggests that it also can cause jock itch.<sup>43</sup> *C. zeylanoides* is a predominate form of yeast found in poultry,<sup>44</sup> raw sausage,<sup>45</sup> and some hams.<sup>46</sup>

**Cryptococcosis:** Normally due to the fungus *Cryptococcus neoformans*, also called *Filobasidiella neoformans* or *Torula histolytica*, cryptococcosis is an encapsulated yeast and is present in soil and bird (especially pigeon) droppings.<sup>30</sup> Symptomatically, it is quite different from the other systemic mycoses, in that meningitis with headache is the way it most commonly presents; blurred vision is also common.<sup>9</sup> Infection tends to occur via the respiratory route by inhalation of *Cryptococcus neoformans*.<sup>1,9</sup> Consumption of high-dose corticosteroids is a major risk factor.<sup>30</sup> Cryptococcosis frequently affects the central nervous system.<sup>33</sup> As *Cryptococcus meningitis*, it is found in some with AIDS and tends to increase their mortality rate.<sup>47</sup> The lungs, kidneys, and sometimes skin tend to be affected,<sup>5</sup> and it is resistant to killing by alveolar macrophages.<sup>30</sup> It produces the enzyme phenol oxidase which tends to consume the hosts epinephrine,<sup>30</sup> thus adrenal support may be helpful for the sufferer (it 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,<sup>9</sup> thus probiotic intervention possibly should be considered as an adjunct.<sup>25,26</sup>

**Histoplasmosis and Coccidioidomycosis:** Similar fungal organisms, *Histoplasmosis* and *Coccidioidomycosis* can both produce a disease that resembles tuberculosis.<sup>1,30</sup> 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.<sup>30</sup> *Histoplasma capsulatum* is acquired by inhaling dust particles which contain bird or bat droppings that contain small spores (microconidia), the infectious form of the fungus.<sup>30</sup> "*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.<sup>5</sup> AIDS patients are particularly susceptible to disseminated infection with *Histoplasma*."<sup>30</sup> *Histoplasmosis* "occurs primarily in the East and Midwest" and primarily affects the lungs.<sup>3</sup> In acute forms it can cause ulcers of the pharynx, spleen enlargement, and liver enlargement.<sup>33</sup> *Coccidioides immitis* has a high infection rate and usually resides in desert soils. In the US it is mainly confined to the Southwest.<sup>1,5</sup> Similar to *Histoplasma*, most primary infections with *Coccidioides immitis* are asymptomatic, but about 10% develop lung

lesions, fever, cough, excess sputum, and pleuritic pains along with San Joaquin Valley fever complex.<sup>5,30</sup> "Once inhaled, *C. immitis* conidia (spores) convert at ... 98.6° F to form large invasive spherules."<sup>5</sup> *Coccidioidomycosis* is also called "Valley Fever."<sup>5</sup> "Untreated disseminated coccidioidomycosis 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."<sup>5</sup>

**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.<sup>1,2</sup> These infections are consistently found in the US, but the diagnosis is difficult since they resemble other bacterial, mycobacterial, and fungal infections.<sup>48</sup> Nocardiosis and actinomycosis are symptomatically similar to tuberculosis.<sup>2</sup> Actinomycosis affects males three times as often as females.<sup>1</sup> 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.<sup>49</sup> "Without treatment, nocardiosis caused by *N. asteroides* is usually fatal."<sup>5</sup> 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.<sup>1</sup> It is defined as an "infection with tissue invasion by broad, non-separate, irregularly shaped hyphae of diverse fungal species."<sup>5</sup> "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).<sup>5</sup> It can cause pulmonary or gastrointestinal lesions,<sup>5</sup> and the three most common areas of invasion are the sinuses, lungs, and gastrointestinal tract.<sup>30</sup> *Rhizopus* species may be the most common; others include *Absidia corymbifera*, *Mucor ramosissimus*, *Rhizomucor pusillus*, and more.<sup>1,2</sup> 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 mycosis in patients with neoplastic disease<sup>1</sup> as well as in ketoacidotic diabetics.<sup>30</sup> It appears to this investigator

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that some with *Rhizopus* often have problems with bile flow, as do some with intense itching. *Rhizopus nigricans* produces opportunistic infections and hypersensitivity states;<sup>50</sup> it seems to cause the body to produce additional IgG and IgE.<sup>32</sup> A recently identified strain, *Rhizopus azygosporus*, was isolated from premature Australian babies, all of which died.<sup>51</sup> Patients with diabetic acidosis or leukemia can be predisposed to rhinocerebral infection caused by *Rhizopus oryzae*;<sup>1</sup> increased consumption of most fresh fruits and vegetables has been reported to help reduce acidosis.<sup>52</sup>

### 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.”<sup>5</sup> 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.”<sup>7</sup> 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. 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<sup>53</sup> and many have pleomorphic hyphae.<sup>54</sup> “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.”<sup>53</sup> 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.<sup>54</sup> 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). The body’s pH (both acid or alkaline) is also a factor.<sup>35</sup>

The main virulent mycoses, such as candida and aspergillus, do not thrive in an acidic environment,<sup>34,35</sup> but some others do. Thus, the frequent consumption of antacids 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).

“With the continuing increase in clinically important fungal disease ... the need for new and improved antifungal agents marches on.”<sup>55</sup> This is partially because the commonly used pharmaceutical antifungal agents are not always effective.<sup>56,57</sup> “Emerging cases of drug resistance to currently available drugs has limited the spectrum of currently available antifungal agents.”<sup>58</sup> “Drugs for systemic antifungal treatments include amphotericin B, various azole derivatives, and flucytosine.”<sup>5</sup>

While drugs remain the preferred standard treatment,<sup>59</sup> there are concerns about their safety, effectiveness, and cost.<sup>60</sup> “Opportunistic systemic mycoses due to yeasts and yeast-like fungi have become commoner than those due to filamentous fungi, occupying the fourth position in the list of bloodstream pathogens in some centers in the US. 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. beigeli*) 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.”<sup>61</sup>

“Innate and cell-mediated immunity are considered as the principal defense line against fungal infections in humans.”<sup>62</sup> Thus, naturopaths tend to focus more on dietary restriction, herbs, naturopathic formulas, heavy

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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<sup>16</sup>): many with mycotic infections strongly crave sugar but to submit to those cravings can make elimination more difficult or cause setbacks. The same can often be said for other refined carbohydrates (e.g., white flour, white rice, white pasta, alcohol, etc.). Although there are few people who need to avoid vinegar, most fruits, or mushrooms, this investigator has found that most can consume them without any apparent adverse affects.

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,<sup>63</sup> including according to the German E monograph *Candida albicans*. In the text, *Medical Mycology* John Rippon (PhD, 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, MD, 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."<sup>21</sup> Some people, however, are allergic to the cell-wall of yeast,<sup>21</sup> 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, astragalus, basil, beet root, bentonite, berberis root/berries, betaine hydrochloride, bile, biotin and other b vitamins, caprylic acid, castor oil, Chinese herbs (various), cinnamon ex-

tracts,<sup>64</sup> chlorophyll, citrus seed extract, cinnamon, cloves, colostrum, deer antler velvet, digestive enzymes, echinacea, essential monosaccharides, flowers (various), food multiple vitamins, garlic, goldenseal,<sup>63</sup> 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),<sup>65</sup> olive leaf,<sup>66</sup> oregano (wild and oil forms), psyllium (seeds and/or hulls), *Saccharomyces cerevisiae*,<sup>63</sup> 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.<sup>5</sup> 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 often a useful adjunct (this is true for males and females). This investigator has also had some success using other naturopathic interventions, such as bioelectrical stimulation, proper food combining, fasting, and hydrotherapy.<sup>67</sup> 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.

"Die off" and other adverse reactions sometimes are encountered when interventions are successful.<sup>5</sup> 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."<sup>68</sup> Whereas medical literature has stated, "For the three oral antifungal agents the more common adverse reactions (are)...nausea, gastrointestinal distress, diarrhoea, abdominal pain,"<sup>69</sup> and "administration of nystatin became impossible in three patients because of vomiting."<sup>70</sup> Tiredness sometimes accompanies die-off.

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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 much of the progress in this arena does not take place until the infection is controlled.

### Conclusion

As there are 100,000 known types of fungi,<sup>1</sup> 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<sup>71</sup> and even mycoplasma is being investigated.<sup>36,72</sup> *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.<sup>2</sup>

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.<sup>30</sup> It appears to this investigator that these toxins are responsible for symptoms such as itching, mucus, and bowel difficulties, and can trigger an autoimmune reaction. Triggering of autoimmune response then seems to cause arthritic and other pain-related symptoms. 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.

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### REFERENCES

- 1) Chandler F and Watts J. "Mycotic, actinomycotic, and algal infections." In: *Anderson's Pathology*, 9th edition. Kissane J (ed). C.V. Mosby, St. Louis, 1996; 391-432.
- 2) Larone DH. *Medically Important Fungi*. American Society for Microbiology, Washington, DC, 1993.
- 3) Assaff RR and Weil ML. "The superficial mycoses." *Dermatol Clin*, 1996; 14(1):57-67.
- 4) Body BA. "Cutaneous manifestations of systemic mycoses." *Dermatol Clin*, 1996; 14(1):125-135.
- 5) *The Merck Manual of Diagnosis and Therapy*. Lane KAG (ex. ed). Merck Research Laboratories, Whitehouse Station, NJ, 1996.
- 6) Ponikau JU, et al. "The diagnosis and incidence of allergic fungal sinusitis." *Mayo Clin Proc*, 1999; 74:877-884.
- 7) Lahoz SC. *Conquering Yeast Infections*. S. Cotel Lahoz, St. Paul (MN), 1996.
- 8) Roosen J, et al. "Comparison of premortem clinical diagnoses in critically ill patients and subsequent autopsy findings." *Mayo Clin Proc*, 2000; 75:562-567.
- 9) *The Merck Manual of Diagnosis and Therapy*, 14th ed. Merck & Company, Rahway, NJ, 1982; 148-159.
- 10) Shepherd MG, Poulter RTM, and Sullivan PA. "Candida albicans: Biology, genetics, and pathogenicity." *Ann Rev Microbiol*, 1985; 39:579-614.
- 11) Kawahata N, Yokuchi M, and Ohtomo E. "A clinicopathological study of candidemia in the elderly." *Nippon Ronen Igakkai Zasshi*, 1990; 27(2):45-51.
- 12) Rolston K. "Overview of systemic fungal infections." *Oncology*, 2001; 15(11 Supp 9):11-14.
- 13) Gupta BK. "Study of fungi associated with bronchopulmonary disorders." *Indian J Med Sci*, 1996; 50(9):333-336.
- 14) Verhoeff AP and Burge HA. "Health risk assessment of fungi in home environments." *Ann Allergy Asthma Immunol*, 1997; 78(6):544-554.
- 15) Vojdani A, et al. "Immunological cross reactivity between *Candida albicans* and human tissue." *J Clin Lab Immunol*, 1996; 48:1-15.
- 16) Santelmann H, Laerum E, et al. "Effectiveness of nystatin in polysymptomatic patients." *Fam Pract*, 2001; 18(3):258-265.
- 17) Nieuwenhuizen WF, Pieters RH, et al. "Is *Candida albicans* a trigger in the onset of coeliac disease?" *Lancet*, 2003; Professional Books, Jackson, TN, 1986.
- 18) Hay RJ. "Fungal infections." *Clin Dermatol*, 2006; 24(3):201-212.
- 19) Chin NX, et al. "In vitro activity of fluvastatin, a cholesterol lowering agent, and synergy with flucanazole and itraconazole against candida species and *Cryptococcus neoformans*." *Antimicrob Agents Chemother*, 1997; 41(4):850-852.
- 20) Martino P and Girmenia C. "Are we making progress in antifungal therapy?" *Curr Opin Oncol*, 1997; 9(4):314-320.
- 21) Crook W. *The Yeast Connection: A Medical Breakthrough*.

(Continued on page 66)

- Professional Books, Jackson, TN, 1986.
- 22) Rochlitz S. *Allergies and Candida with the Physician's Rapid Solution*. Human Ecology Balancing Sciences, New York, 1991.
  - 23) Carlsen G. *The Candida Yeast Answer*. Healthology Center, Provo, UT, 1997.
  - 24) Rockwell S. *Coping with Candida Cookbook*. Sally J. Rockwell, Seattle, WA, 1984.
  - 25) Gibson G and Roberfroid MB. "Dietary manipulation of human colonic microbiota: Introducing the concept of probiotics." *J Nutr*, 1995; 125:1401-1412.
  - 26) Duarte A. *Health Alternatives*. Mega Systems, Morton Grove, IL, 1995.
  - 27) *Citricidal: Grapefruit Seed & Pulp Extract*. Bio/Chem Research, Lakeport, CA, 1994.
  - 28) Ionescu G, et al. "Oral citrus seed extract in atopic eczema: In vitro and in vivo studies on intestinal microflora." *J Ortho Med*, 1990; 5(3):72-74.
  - 29) Rodriguez R, et al. *The Application of Dioxychlor to Clinical Practice*. Bradford Research Institute, Tijuana, 1989.
  - 30) Cotran RS, Kumar V, and Collins T. *Pathologic Basis of Disease*, 6th ed. WB Saunders, Philadelphia, PA, 1999.
  - 31) Roig E, et al. "Anti-*Candida albicans* IgE and IgG subclasses in sera of patients with allergic bronchopulmonary aspergilliosis (ABPA)." *Allergy*, 1997; 52:394-403.
  - 32) Reichard U. "Expression pattern of aspartic proteinase antigens in aspergilli." *Mycoses*, 1996; 39 (3-4):99-101.
  - 33) "Fungal infections of humans and animals." In: *Columbia Electronic Encyclopedia*, Columbia University Press, 2000.
  - 34) Werbach MR. "Candidiasis." In: *Textbook of Nutritional Medicine*. Third Line Press, Tarzana, CA, 1999; 222-228.
  - 35) Konno N, Ishii M, et al. "Mechanism of *Candida albicans* trans formation in response to changes of pH." *Biol Pharm Bull*, 2006; 29(5):923-926.
  - 36) Benjamini E, Sunshine G, and Leskowitz S. *Immunology: A Short Course*, 3rd ed. Wiley-Liss, New York, 1996.
  - 37) Horrobin DF. "Essential fatty acids, immunity, and viral infections." *J Nutr Med*, 1990; 1:145-151.
  - 38) Anane S, Kallel K, et al. "*Candida dubliniensis*: A novel emerging specie." *Ann Biol Clin (Paris)*, 2007; 65(1):13-19.
  - 39) Levenson D, Pfaller MA, et al. "*Candida zeylanoides*: Another opportunistic yeast." *J Clin Microbiol*, 1991; 29(8):1689-1692.
  - 40) Crozier WJ. "Two cases of onychomycosis due to *Candida zeylanoides*." *Australas J Dermatol*, 1993; 34(1):23-25.
  - 41) Whitby S, Madu EC, and Bronze MS. "Case report: *Candida zeylanoides* infective endocarditis complication infection with the human immunodeficiency virus." *Am J Med Sci*, 1996; 312(3):138-139.
  - 42) Bisbe J, Vilardell J, et al. "Transient fungemia and candida arthritis due to *Candida zeylanoides*." *Eur J Clin Microbiol*, 1987; 6(6):668-669.
  - 43) Liao WQ, Li ZG, et al. "*Candida zeylanoides* causing candidiasis as tinea cruris." *Chin Med J*, 1993; 106(7):542-545.
  - 44) Deak T. "Identification of yeasts isolated from poultry meat." *Acta Biol Hung*, 2001; 52(2-3):195-200.
  - 45) McCarthy JA and Damoglou AP. "The effect of substrate on the radiation resistance of yeast isolated from sausage meat." *Lett Appl Microbiol*, 1996; 22(1):80-84.
  - 46) Nunez F, Rodriguez MM, et al. "Yeast population during the ripening of dry-cured Iberian ham." *Int J Food Microbiol*, 1996; 29(2-3):271-280.
  - 47) Van der Horst CM, et al. "Treatment of cryptococcal meningitis associated with the acquired immunodeficiency syndrome. National Institute of Allergy and Infectious Diseases Mycoses Study Group and AIDS Clinical Trials Group." *NEJM*, 1997; 337(1):15-21.
  - 48) Warren NG. "Actinomycosis, nocardiosis, and actinomycoetoma." *Dermatol Clin*, 1996; 14(1):85-95.
  - 49) Mok CC, Yuen KY, and Lau CS. "Nocardiosis in systemic lupus erythematosus." *Semin Arthritis Rheum*, 1997; 26(4):675-683.
  - 50) Alonso A, et al. "Interstitial pneumonitis induced in guinea pigs by the antigens of *Rhizopus nigricans*." *J Invest Allergol Clin Immunol*, 1997; 7(2):103-109.
  - 51) Schipper MA, Maslen MM, et al. "Human infection by *Rhizopus azygosporus* and the occurrence of azygospores in Zygomycetes." *J Med Vet Mycol*, 1996; 34(3):199-203.
  - 52) Airola P. *How to Get Well*. Health Plus Publishers, Sherwood, OR, 1974; 284-285.
  - 53) Sanchez-Martinez C and Perez-Martin L. "Dimorphism in fungal pathogens: *Candida albicans* and *Ustilago maydis* — similar inputs, different outputs." *Curr Opin Microbiol*, 2001; 4(2):214-221.
  - 54) Brandwein M. "Histopathology of sinonasal fungal disease." *Otolaryngol Clin North Am*, 1993; 26(6):949-981.
  - 55) Muller RJ. "A brief review of antifungal therapy for deep fungal infection." *Oncology*, 2001; 15 (11 Supp 9):21-25.
  - 56) Hossain MA and Ghannoum MA. "New developments in chemotherapy for non-invasive fungal infections." *Expert Opin Investig Drugs*, 2001; 10(8):1501-1511.
  - 57) Abuhammour W and Habte-Gabr E. "Systemic antifungal agents." *Indian J Pediatr*, 2001; 68(7):655-668.
  - 58) Pranav Kumar SK and Kulkarni VM. "Insights into the selective inhibition of *Candida albicans* secreted aspartyl protease: A docking analysis study." *Bioorg Med Chem*, 2002; 10(4):1153-1170.
  - 59) Loo DS. "Systemic antifungal agents: an update of established and new therapies." *Adv Dermatol*, 2006; 22:101-124.
  - 60) Schedel I. "New medications for treatment of systemic mycoses." *Internist (Berl)*, 2005; 46(6):659-670.
  - 61) Randhawa HS. "Respiratory and systemic mycoses: An overview." *Indian J Chest Dis Allied Sci*, 2000; 42(4):207-19.
  - 62) Cramer R and Blaser K. "Allergy and immunity to fungal infections and colonization." *Eur Respir J*, 2002; 19(1):151-157.
  - 63) *PDR for Herbal Medicines*, 3<sup>rd</sup> ed. Gruenwald, et al (ed). Thomson PDR, Montvale, NJ, 2004.
  - 64) Ooi LS, Li Y, et al. "Antimicrobial activities of cinnamon oil and cinnamaldehyde from the Chinese medicinal herb *Cinnamomum cassia* Blume." *Am J Chin Med*, 2006; 34(3):511-522.
  - 65) Hatakka K, Ahola AJ, et al. "Probiotics reduce the prevalence of oral candida in the elderly — A randomized controlled trial." *J Dent Res*, 2007; 86(2):125-130.
  - 66) Markin D, Duek L, and Berdicevsky I. "In vitro antimicrobial activity of olive leaves." *Mycoses*, 2003; 46(3-4):132-136.
  - 67) Thiel R. *Combining Old and New: Naturopathy for the 21st Century*. Whitman Publishing, Warsaw, IN, 2000.
  - 68) Anon. "Candida yeast protection program, Part 2." *Intelegem*, 2002; [http://intelegem.com/nutrients/candida\\_yeast\\_protection\\_program.htm](http://intelegem.com/nutrients/candida_yeast_protection_program.htm)
  - 69) Gupta AK and Shear NH. "A risk-benefit assessment of the newer oral antifungal agents used to treat onychomycosis." *Drug Saf*, 2000; 22(1):33-52.
  - 70) Van Delden C, Lew DP, et al. "Antifungal prophylaxis is severely neutropenic patients." *Clin Microbiol Infect*, 1995; 1(1):24-30.
  - 71) Butler WR, et al. "*Mycobacterium celatum* sp. nov." *Int J Syst Bacteriol*, 1993; 43(3):539-548.
  - 72) Nicolson GL and Nicolson NL. "Diagnosis and treatment of mycoplasmal infections in Persian Gulf War Illness — CFIDS patients." *Int J Occup Med Immunol Tox*, 1996; 5:69-78. ♦

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# The Gatekeepers to Good Health

by Michael G. Rehme, DDS, CCN

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After practicing dentistry for over two decades, I've witnessed some interesting changes that have taken place in our profession. Although modern technologies continue to improve the ways in which we practice and develop our technical skills, there is a new concept that is slowly developing in dentistry that has caught my eye and requires a bit more attention on my part.

In the last several years, numerous dental articles and medical reviews have been published regarding connections made between the oral cavity and the rest of the body:

- "Oral inflammation." *Colgate-White Papers*, 2005
- "The oral-systemic link: How much do we know?" *Inside Dentistry*, December 2005
- "Periodontal disease and diabetes." *Journal of Periodontology*, November 2002

These articles, as well as many others, are being published much more frequently throughout the health care industry. However, I don't believe this topic is receiving as much attention or credence as it deserves in the field of dentistry. Sometimes, I think as dentists, we focus too much on the mechanics of dentistry (i.e., dental procedures, esthetics and materials used) and not enough on the biologic issues of dental health that may actually help our patients improve their overall health and wellness.

About ten years ago, I met a dentist who introduced me to a new concept in dentistry. It wasn't just another one of your "drill, fill and bill" type of practices. He told me the key to his success was helping his patients *feel better* and providing a service that was deeply appreciated by those people who came to see him. I could see the passion in his eyes as he shared this information with me, and what I learned next, certainly changed my life and the direction in which I was heading. It also provided me with a new career opportunity that I will forever be grateful for.

The message that I received here was that there's more to dentistry than just trying to be the best restorative den-

tist possible. The oral cavity provides us with so much information; we just need to know what to do with it.

When I see a patient with compromised periodontal conditions, tooth decay and generalized inflammation, what is this telling me? Conventional wisdom says we treat this *local* condition typically with more frequent dental visits, improved home care techniques (i.e., better flossing and brushing of your teeth), usually some antibiotics, root plane and scaling, and sometimes even surgical procedures. However, I believe these are all signs of a *systemic* imbalance going on in the body, that definitely should attract a dentist's attention. If we don't improve these imbalances, won't the same problems come back to haunt our patients over and over again? Can there be an alternative approach? Let's think about *evaluating the chemistry* of the body; try balancing the system, and you'll soon begin to observe healthier, long-term effects that you never thought possible.

If you really think about it, *dentists* can be the "gatekeepers" to good health! Everything begins in the oral cavity — what we eat and what we drink enters our bodies through the mouth. Our first line of defense and our initial immune responses begin in the mouth. In 1943, Dr. Reinhold Voll, the father of electroacupuncture according to Voll (EAV), stated, "Eighty to ninety percent of all illnesses originate from the mouth."

Maybe there *is* more going on in the mouth than we ever imagined. As dentists look into their patients' mouths each and every day, the message should become quite clear. If there are imbalances in the mouth — inflammation, red, puffy, bleeding gums, tooth decay, sensitive teeth, halitosis, root canals, missing teeth — you can probably expect imbalances in other parts of the body as well. Therefore, if we're going to *fix* the mouth, let's *fix* the rest of the body as well.

## Tooth and Body Connections

The following are just a few examples to help explain how the oral cavity is connected to the rest of the body. Many more examples can certainly be found that will continue to support this concept, and I believe this could be a new start as we begin creating somewhat of a paradigm shift in dentistry today.

**Periodontal Disease and Heart Disease:** Oral bacteria can affect the heart when they enter the bloodstream, attaching to fatty plaques in the coronary arteries (heart blood vessels) and contributing to clot formation. Blood clots obstruct abnormal blood flow, restricting the amount of nutrients and oxygen required for the heart to

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function. This may lead to heart attacks. Researchers have found that people with periodontal disease are almost twice as likely to suffer from coronary artery disease as those without periodontal disease. (*The American Academy of Periodontology*, July 23, 2003)

Since the early 1970s, when the American Heart Association issued a health alert to the dental community to premedicate all patients with heart disease, rheumatic fever and MVP, dentists have faithfully administered the proper antibiotics to protect their patients from infectious complications that could effect the heart itself. Although this has been considered the “gold standard of care” for prophylactic coverage in dentistry for years, there is still ample research, both past and present, that indicates we must not overlook the consequences of periodontal disease and its connection to, or effects on, the rest of the body. Bacterial infections do not discriminate as to the sight of their attack. *The whole body* is susceptible, and usually the weakest organ system will be the target. This is commonly referred to as a *focal infection* — a bacterial infection localized in a specific part of the body, such as the mouth, that may spread to another part of the body.

Streptococcus and other pathogens found in the mouth can cause inflammatory and infectious events to occur throughout the entire body. Streptococcus has been linked to the following diseases: endocarditis, pericarditis, myocarditis, rheumatism, articular and muscular, gastric and duodenal ulcer, appendicitis, pneumonitis, bronchitis, asthma, non-TB pleuritis, nephritis, thyroid, poliomyelitis and multiple sclerosis.<sup>1</sup>

Dr. Westin Price’s research studied indigenous people who ate native, whole food diets. These cultures remained relatively free of dental and periodontal disease, and there was a lack of degenerative diseases, too. Those who converted or were exposed to the Western refined diet, manifested increased susceptibility to dental malformations and dental decay as well as infertility, miscarriages, birth defects, TB, arthritis, cancer and other chronic degenerative disorders.<sup>2</sup>

“Disease is not due to the presence of bacteria, but rather to the body being out of balance in such a way that the bacteria responsible for the inflammation are breeding out of control. Killing the bacteria is not the answer, placing the body back in balance is a much more effective method of treatment.”<sup>3</sup>

“Over the last 25 years, the field of periodontology has witnessed remarkable changes in the relationship to the body as a whole. The focus on inflammation of the

gingiva and periodontium, as important solely for disease of the oral cavity, *has shifted* to include significant associations with the health of other body systems. In recent years, increasing evidence has supported the concept that the relationship between systemic and oral health is bi-directional.”<sup>4</sup>

Medical and dental research needs to spend more time investigating this concept of the tooth and body connection.<sup>5</sup>

Vitamin C is a powerful antioxidant that is required for tissue growth and repair, adrenal gland function, and healthy gums. Deficiencies include gums that bleed when brushed, increased susceptibility to infection, joint pains, lack of energy, poor digestion, prolonged healing time, a tendency to bruise easily, and tooth loss.<sup>6</sup>

“People with diabetes are more likely to have periodontal disease than people without diabetes, probably because diabetics are more susceptible to contracting infections. Poorly controlled Type II diabetic (adult-onset) patients are more likely to develop periodontal disease than well-controlled diabetics are.”<sup>7</sup>

This form of Type II diabetes accounts for 90 - 95% of all the diabetes cases in America today and is considered to be almost entirely diet related.”<sup>8</sup>

During the last 40 years, the strong association between periodontal disease and thyroid disorders, especially hypothyroidism has been documented many times over (i.e., Riedel and Ordeltz, 1966; Abate, 1968; Baba, *et al*, 1972; Pencea, *et al*, 1978; Saburova and Isaeva, 1971; Schneider, 1969; Shkoliar, *et al*, 1967; Pashaev, 1982; Danilevskii, *et al*, 1988; Kerimov, 1989; Puzin, *et al*, 1996) in the world literature, yet it has received little or no attention in the US.

### **The Next Step**

Dental problems can influence systemic problems, and systemic problems can lead to dental problems. Sometimes it’s like asking yourself, “Which came first, the chicken or the egg?” I’m not sure, but either way, if we learn how to balance the system, chances are we’ll fix both problems at the same time.

Today, more than ever, people are beginning to take responsibility for their own health and wellness. Safe, natural, and conservative approaches are what people are looking for, and I believe that dentistry has an opportunity to embrace this *tooth and body connection* and pro-

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mote a health-oriented, biologic approach for treating their dental patients.

How can we implement this concept into dentistry? All that's needed is a genuine desire for dentists to help their patients, a basic nutritional program to follow, and the willingness to assist patients in achieving their goal of optimum health. If we understand the importance of improving body chemistry and balancing the system, the dental profession will soon witness an incredible phenomenon that can create a win-win situation for everyone — nutritional therapy for the dental patient. New interest will generate a new source of patients for the dentist, and health oriented people will begin to seek out their services.

It is a given that dentistry will continue to improve on the mechanical aspects of our profession, but we should also remember to focus on the bigger picture. Learn to incorporate this biologic approach as well, and then dentistry can truly offer a more valuable, health-oriented service for those who need our help.

#### **About the Author**

*Dr. Micahel Rehme is a general dentist, as well as a Certified Clinical Nutritionist who provides nutritional therapies and whole food supplements for his dental patients.*

#### **More Information on this Topic**

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#### **REFERENCES**

- 1) Price W. "Peripheral diseases of proven etiology 1902-1938." *Nutrition and Physical Degeneration*, 1945.
- 2) Price W. "Native, whole food diets." *Nutrition and Physical Degeneration*, 1945.
- 3) Page M. *Dental Problems are the Result of Nutritional Deficiencies*. 1949.
- 4) "Oral inflammation." *Colgate-White Papers*, 2005.
- 5) "The oral-systemic link: How much do we know?" *Inside Dentistry*, December 2005.
- 6) Balch JF. *Prescription for Nutritional Healing*. Avery Publishing Group, 1997.
- 7) "Periodontal disease and diabetes." *Journal of Periodontology*, November 2002.
- 8) Gittleman. *Get The Sugar Out*. Three Rivers Press, 1996.
- 9) Ferber D. *The Checkup That Can Save Your Life*. ♦

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# *The Use of Probiotics as part of a Comprehensive Approach to Optimal Health*

by Rachel Oliver, MS, ND, PhD

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In humans the organ considered to be the most diverse and the most metabolically active is the gastrointestinal tract. An integral part of an optimally functioning gastrointestinal tract is the microbial community. The initial inoculum for microbial colonization is assumed to be during the birth process, at which time the gastrointestinal tract is colonized via the delivery process. Consequently, those born via a Caesarean delivery are exposed to a different environment than would those born via vaginal delivery, and are thus considered to have a gut flora more representative of an adult. In humans the gastrointestinal tract is said to contain 100 trillion viable bacteria. Classically defined probiotics are “a preparation of, or a product containing viable, defined microorganisms in sufficient numbers, which alter the microbiota (typically by colonization) in a compartment of the host, and by that exert beneficial health effect in this host.” The benefits attributed to the use of probiotics include improved digestion, enzymatic activity and overall health.

The gut microflora is viable living system. The resistance of the host against the invasion of microorganisms, termed the ‘barrier effect,’ is dependent upon the stability of the indigenous gut microbiota. Consequently, a significant contribution of the commensal intestinal microflora is the protective host mechanism it exerts, specifically against invading microorganisms. Functionally, the intestinal microbiota takes part in the fermentation of both endogenous and exogenous growth substrates, which includes participation in the fermentation of oligosaccharides and sugar varieties, as well as non-absorbable sugar alcohols. These metabolic end-products of carbohydrate fermentation subsequently play an integral part in human health. The gut microflora also participates in a number of other important commensal functions, including the production of short chain fatty acids (SCFA), which serves primarily as a component for energy metabolism of the large intestinal mucosa, as well as a component for the growth of the colonic cells. The SCFAs also serve as a reducing component, which functions to lower the local pH. The intesti-

nal microflora also functions as a producer of B vitamins and vitamin K, as well as a source for the production of secretory IgA, thus acting as non-inflammatory immune stimuli, and as a barrier against the invasion of foreign bacteria and viruses.

Documented research has indicated important benefits associated with the use of probiotic bacteria, including the management of lactose intolerance, as a functional component in lowering both cholesterol and blood pressure, and significantly increasing HDL cholesterol, resulting in a desired LDL/HDL cholesterol ratio, as well as increasing IgA producing plasma cells, T-lymphocytes and Natural Killer cells.’ Probiotics have also shown benefits in the treatment of respiratory tract infections, irritable bowel syndrome, ulcerative colitis, and have been suggested as an adjuvant to detoxification, specifically in regard to the treatment of autistics.

Be it that the gut microflora is viable living system, it is highly sensitive to antibiotic therapy. Functionally, the gut microflora produces hydrolytic enzymes, which act to split relevant conjugates, thus allowing the reabsorption of steroids from the GI tract. Use of the antibiotics ampicillin and neomycin were shown to reduce the excretion of biliary steroids by 70%, which was correlated to a reduction in the gut microflora. Consequently, the use of antibiotic therapy, and specifically that of broad-spectrum antibiotic therapy, which is often the therapy of choice in chronic illness and immune suppression, can severely alter the gut microflora. This alteration in microbial flora has documented detrimental consequences on health, as the balance between beneficial and harmful microorganisms is disturbed. Such consequences include the disturbance of the normal microflora, such that opportunistic microorganisms or other pathogens such as yeasts or parasites are allowed to flourish, thus increasing the risk of iatrogenic disease.

As a consequence of depleted microflora due to the use (or overuse) of antibiotic therapy, the use of probiotics has gained popularity in recent years, as a means to reestablish or alter gut flora. Symptomatology classically associated with depletion of commensal microflora includes the infestation of non-commensal organisms, including *H. pylori* and/or *Candida* species. It has been suggested that probiotics interfere with the ability of *H. pylori* to adhere to gut epithelial cells.

Traditionally, probiotics have consisted of species from the genus *Lactobacillus* and *Bifidobacterium*. Analysis of human rectal and oral mucosa has predominantly identified the taxa *L. plantarum*, *L. rhamnosus*, and *L. paracasei*, in which *L. plantarum* was recognized as the

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response to antibiotic therapy. For example, *Lactobacillus plantarum* was shown to ‘decrease translocation, improve mucosal status, improve liver status, improve the immunologic status of the mucosa, and to reduce mucosal inflammation’. Subsequently, *L. plantarum* has been considered to be an integral part in the host’s immunologic defense. In a recent study comparing the immune cell response to the commensal bacteria *B. infantis* and *L. salivarius*, versus the pathogenic bacteria *Salmonella typhimurium*, *S. typhimurium* was shown to increase both NF-kappaB and IL-8 gene expression, along with thirty-four other immune associated genes, of the eight hundred forty-seven assayed. The commensal bacteria, however, did not alter the expression of any of the genes screened. Despite a lack of effect on expression, these commensal organisms “attenuated both IL-8 secretion at baseline, and *S. typhimurium*-induced pro-inflammatory responses.” The commensal bacteria were also documented to act in an immunostimulatory manner via alteration of the secretion of both IL-10 and TNF-alpha by dendritic cells.

Probiotics have also shown to have a modulating effect on the immune system. Dendritic cells, demonstrated to play a role in both early bacterial recognition and tolerance induction, as well as in influencing the response of T cells, have been utilized as immunomodulatory markers. In respect to dendritic cells, a probiotic combination of Lactobacilli species (4 strains), Bifidobacteria species (3 strains) and Streptococcal strains (1 strain) was shown to be immunomodulating by virtue of both upregulation of IL-10 production by dendritic cells, and decreased production of interferon-gamma by T-cells. The authors also noted a “diminished proinflammatory effect” via a decrease in the lipopolysaccharide production of IL-12. Probiotics have also demonstrated success in the case of chronic allergic reactions, with their use indicated in the reduction of atopic eczema. In a separate study the administration of *L. plantarum* was correlated with a decrease in pain and flatulence associated with IBS.

Like probiotics, prebiotics have also shown to play a beneficial role in gastrointestinal health, via their alteration of the gut flora composition favor of beneficial bacteria. Prebiotics are categorized as “a non-digestible food ingredient that beneficially affects the host by selectively stimulating the growth and/or activity of one or a limited number of bacteria in the colon.” Inulin is considered to be a prebiotic, and has shown beneficial results by virtue of its ability to stimulate the enumeration of the colonic bifidobacte-

ria population, thus improving the composition of the gut flora.

Therefore, in considering overall health, the combination of a pre- and probiotic is a judicious choice, as both have documented success in improving gastrointestinal health. Additionally, it is highly likely that by utilizing a prophylactic approach gastrointestinal complaints may be addressed in a means that will promote optimal wellness.

## REFERENCES

- 1) B. O’Grady and GR Gibson. Microbiota of the Human Gut. *Probiotic Dairy Products*. A.Y. Tamime, ed. Blackwell Publishing Ltd. 2005.
- 2) Havenaar R and Huis In’t Veld, MJH. 1992 Probiotics: a general view. *Lactic acid Bacteria in Health and Disease*, Vol.1 (ed. B.J.B. Wood), pp. 151-170, Elsevier Applied Science Publishers, Amsterdam.
- 3) Alderberth, I,et.al. Mechanisms of colonisation and colonisation resistance of the digestive tract. *Microbial Ecol of Health and Disease*. 12, 223-239.
- 4) Cummings, J.H. and G.T. Macfarlane, *Role of intestinal bacteria in nutrient metabolism*. JPEN J Parenter Enteral Nutr, 1997. 21(6): p. 357-65.
- 5) GT Macfarlane & AJ McBain. The human colonic microbiota. *Colonic Microbiota, Nutrition and Health*, (eds. GR Gibson and MB Roberfroid), pp. 1-25, Kluwer Academic Publishers, Dordrecht. 1999.
- 6) Hooper LV, Midtvedt T and Gordon JL. How host-microbiota interactions shape the nutrient environment of the mammalian intestine. *Annual Review of Nutrition*, 2002;22, 283-307.
- 7) Moreau MC. Flore intestinale, prebiotique et effets sur la reponse immunitaire intestinale a IgA. *Archives de Pediatrie*, 2000; 247-248.
- 8) Sanders ME. Considerations for use of probiotic bacteria to modulate human health. *J Nutr*. 2000;130:384S-390S.
- 9) Sanders ME. Considerations for use of probiotic bacteria to modulate human health. *J Nutr*. 2000;130:384S-390S.
- 10) Long-term consumption of fermented dairy products over 6 months increases HDL cholesterol.
- 11) Reid G, Jass J, Sevulsky MT, McCormick JK. Potential uses of probiotics in clinical practice. *Clin Microbiol Rev*. 2003;16:658-72.
- 12) Ouwehand AC, Salminen S, Isolauri E. Probiotics: an overview of beneficial effects. *Antonie Van Leeuwenhoek*. 2002;82:279-89.
- 13) Hatakka K, Savilahi E, Ponka A, Meurman JH, Poussa T, Nase L, Saxelin M, Korpela R. Effect of long term consumption of probiotic milk on infections in children attending day care centres: double blind, randomized trial. *BMJ*. 2001;322:1327
- 14) Whorwell PJ, Altringer L, Morel J, Bond Y, Charbonneau D, O’Mahony L, Kiely B, Shanahan F, Quigley EM. *Am J Gastroenterol*. 2006 Jul;101(7):1581-90.
- 15) Kerr, Martha. Probiotics Significantly Reduce Symptoms of IBS, Ulcerative Colitis.
- 16) Brundnak MA. Probiotics as an adjuvant to detoxification protocols. *Medical Hypotheses*. 2002; 25(5), 382-385.
- 17) ML Orme and DJ Back. Therapy with oral contraceptive steroids and antibiotics. *J Antimicrob Chemother*, 1979. 5 (2): p. 124-6.

(Continued on next page )

- 18) C. Felley and P. Michetti. Probiotics and Helicobacter pylori. Best Pract Res Clin Gastroenterol. 2003 Oct;17(5):785-91.
- 19) Ahrne S, Nobaek S, Jeppsson B, Adlerverth I, Wold AE, Molin G. The normal Lactobacillus flora of healthy human rectal and oral mucosa. J Appl Microbiol. 1998 Jul; 85(1):88-94.
- 20) Madden, J. A., et.al. Effect of probiotics on preventing disruption of the intestinal microflora following antibiotic therapy: a double-blind, placebo controlled pilot study. Int Immunopharma col. 2005;5(6):1091-7.
- 21) Molin G. Probiotics in foods not containing milk or milk constituents, with special reference
- 22) O'Hara AM, O'Regan P, Fanning A, O'Mahony C, Macsharry J, Lyons A, Bjenenstock J, O'Mahony L, Shanahan F. Functional modulation of human intestinal epithelial cell responses by Bifidobacterium infantis and Lactobacillus salivarius. Immunology. 2006 Jun;118(2):202-15.
- 23) Ruiz PA, Hoffmann M, Szcesny S, Blaut M, Haller D. Innate mechanisms for Bifidobacterium lactis to activate transient pro-inflammatory host responses in intestinal epithelial cells after the colonization of germ-free rats. Immunology. 2005 Aug;115(4):441-50.
- 24) Hart AL, Lammers K, Brigidi P, Vitali B, Rizzello F, Gianonchetti P, Campieri M, Kamm MA, Knight SC, Stagg AJ. Modulation of human dendritic cell phenotype and function by probiotic bacteria. Gut. 2004 Nov; 53(11):1602-9.
- 25) Isolauri E, Arvola T, Sutas Y, Moilanen E, Salminen S. Probiotics in the management of atopic eczema. Clin Exp Allergy. 2000 Nov;30(11):1604-10.
- 26) Furrle E. Probiotics and allergy. Proc Nutr Soc. 2005 Nov;64(4):465-9.
- 27) Nobaek S, Johansson ML, Molin G, Ahrne S, Jeppsson B. Alteration of intestinal microflora is associated with reduction in abdominal bloating and pain in patients with irritable bowel syndrome. Am J Gastroenterol. 2000 May;95(5):1231-8.
- 28) Gibson GR. Dietary modulation of the human gut microflora using the prebiotics oligofructose and inulin. J Nutr. 1999 Jul;129(7 Suppl):1438S-41S.
- 29) Gibson GR and Roberfroid MB. Dietary modulation of the human colonic microbiota – introducing the concept of prebiotics. J of Nutrition. 1995. 125, 1401-1412.
- 30) Manning TS, Gibson GR. Microbial-gut interactions in health and disease. Prebiotics. Best Pract Res Clin Gastroenterol. 2004 Apr;18(2):287-98. ♦

**On a sad note, we would like to express our condolences to the friends and family of Dr. Edward Kaszans. We lost a fabulous doctor and a good friend in February of this year. Ed was always supportive of alternative care.**

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# *Evidence of Defective Cellular Oxidation and Organification of Iodide in a Female with Fibromyalgia and Chronic Fatigue*

by Guy E. Abraham, MD and J. D. Flechas, MD

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## **Introduction**

Orthiodosupplementation is the daily amount of the essential element iodine needed for whole body sufficiency and is assessed by an iodine/iodide loading test.<sup>1,2</sup> The test consists of ingesting four tablets of a solid dosage form of Lugol (Iodoral®), a total of 50 mg iodine/iodide. Then urinary iodide levels are measured in a subsequent 24-hour collection. During orthiodosupplementation, a negative feedback mechanism is triggered that progressively adjusts the excretion of iodine to balance the intake. When whole body sufficiency for iodine is achieved, the absorbed iodine/iodide is quantitatively excreted as iodide in the urine, and 90% or more of the iodine load is recovered in the 24-hour collection. The body retains around 1.5 g of elemental iodine at sufficiency.<sup>2</sup> Baseline serum inorganic iodide levels 24 hours after the last dose of iodide in eight normal subjects with normal body weight who achieved whole body iodine sufficiency had a mean  $\pm$  SD of  $1.1 \pm 0.18$  mg/L.<sup>3,4</sup> We have defined whole body sufficiency for iodine and a normally functioning iodine retention mechanism as a baseline serum inorganic iodide level from 0.85 to 1.3 mg/L when the serum sample is obtained 24 hours after the last dose of 50 mg iodine in a subject who excretes 90% or more of the ingested iodine.<sup>4</sup>

In patients with normal gastrointestinal absorption of iodine but with a very defective iodine retention system, the absorbed iodine is quantitatively excreted in the urine with little or no retention.<sup>5</sup> In these cases, the loading test will suggest whole body iodine sufficiency (90% or more excreted), but the serum inorganic iodide levels 24 hours after the iodine load will remain low (less than 0.13 mg/L). The inefficient iodine retention mechanism could be due to either an inefficient cellular iodine transport system or to an inefficient oxidation and organification of the intracellular iodide, or possibly both.<sup>6,7</sup>

We previously evaluated one hypothyroid patient with pre-supplementation high urinary excretion of the iodine load.<sup>5</sup> The patient, a 52-year-old woman (height 64 inches; weight 140 lbs.), had a past history of hyperthy-

roidism followed by hypothyroidism and had taken Synthroid 50 mg/day for five years. She developed side effects to orthiodosupplementation and could tolerate only half a Lugol tablet/day (6.25 mg iodine/day) due to the detoxification of elevated bromide levels by the iodine supplementation.

She was evaluated with serial serum samples for 11 hours post-load, before and after three months on a sustained-release form of vitamin C at 3 g/day. Pre-vitamin C loading test showed 90% of the load excreted in the urine, but her baseline serum iodide level was only 0.016 mg/L, compared to the expected levels of 0.85-1.3 mg/L in those with whole body iodine sufficiency. Prior to intervention with vitamin C, a sharp peak of serum iodide at 32 mg/L at one hour post-load was followed by a rapid drop. This suggested that the gastrointestinal absorption of iodine was very efficient, but the transfer of the serum iodide to the target cells was not efficient. After three months on vitamin C, the same test was repeated. The data revealed a normal profile of serum inorganic iodide levels. Her baseline serum inorganic iodide level increased from 0.016 mg/L to 0.42 mg/L, and she retained 50% of the iodine load, compared to 10% of the load prior to vitamin C supplementation.

To our knowledge, this was the first case report of a patient with evidence of a very defective retention mechanism for iodine who was studied with serial serum iodide levels prior to and following intervention. A combination of orthiodosupplementation in amounts of iodine the patients could tolerate and administration of the antioxidant vitamin C via the oral route improved the performance of the iodine retention mechanism.

The milder forms of inefficient iodine retention and utilization, due either to inefficient cellular uptake of peripheral iodide or to inefficient oxidation and organification of intracellular iodide, will probably be overlooked until a more refined procedure is worked out to assess accurately the efficiency of the iodine transport and utilization mechanisms. Obviously, serial serum measurement of iodide would not be practical on a routine basis to evaluate patients with a high percentage of the iodine load excreted prior to supplementation. A simple test was needed for the combined assessment of whole body sufficiency for iodine with the assessment of the efficiency of the body to utilize peripheral iodide.

## **Assessing the Cellular Uptake and Utilization of Peripheral Iodide**

Currently, radioiodide is used to assess the uptake of

*(Continued on next page)*

**Table 1—Some Clinical Data on the 6 Female Volunteers**

SS#	Age	Height	Weight	SBP	DBP	COMMENT
1	48	67"	178 lbs	106	63	
2	47	62"	126 lbs	116	73	*CFS-FM
3	47	69"	170 lbs	123	81	
4	56	61"	148 lbs	132	79	**Mild Hypothyroidism
5	22	65"	125 lbs	122	88	
6	47	66"	130 lbs	130	89	

\* Undiagnosed Fibromyalgia and chronic fatigue detected at the initial evaluation.

\*\*Undiagnosed hypothyroidism detected at initial evaluation based on thyroid function tests.

SBP=Systolic blood pressure; DBP=Diastolic blood pressure

iodide by the thyroid gland.<sup>7</sup> In conjunction with potassium perchlorate, radioiodide is used to evaluate the oxidation and organification of iodide by the thyroid gland. However, there is no available procedure to assess whole body iodine uptake and utilization. The salivary glands use a mechanism similar to the thyroid gland and the other target cells to concentrate peripheral iodide with subsequent oxidation and organification of iodide. Although the salivary glands can incorporate iodine in thyrosine to form mono- and di-iodothyrosine, they cannot couple iodinated thyrosine to form thyroid hormones.<sup>1,3</sup>

We previously reported a procedure to measure saliva and serum inorganic non-radioactive iodide levels 24 hours following ingestion of 50 mg of iodine in the form of Lugol tablets.<sup>6</sup> The saliva/serum iodide ratio measures the ability of the salivary glands to concentrate peripheral iodide. The assumption made is that the saliva/serum ratio of iodide is an index of iodide uptake and utilization by target cells throughout the whole body. Inefficient cellular uptake of iodide is associated with a low saliva/serum iodide ratio whereas inefficient organification of intracellular iodide with normal cellular iodide uptake is associated with an elevated ratio.<sup>8</sup> The normal range of saliva/serum ratios was 28-74 with a mean  $\pm$  SD of 44.2 $\pm$ 12.7 in 14 normal subjects. Low saliva/serum iodide ratios were observed in breast cancer patients with high serum bromide levels. Orthoiodosupplementation at 50-100 mg/day resulted in decreased serum bromide and increased saliva/serum ratio.<sup>6</sup>

#### **Effect of the Daily Amounts of Lugol**

After three months of supplementation with 50 mg iodine/iodide/day, most non-obese subjects with normal iodide retention mechanisms achieved whole body iodine sufficiency, defined as 90% or more of the iodine load excreted in the 24-hour urine collections and 24-

hour post-load serum inorganic iodide level between 0.85 and 1.3 mg/L.<sup>1,2</sup> Adult subjects retained approximately 1.5 g of iodine when they reach sufficiency.<sup>3</sup>

We previously discussed the possibility of achieving whole body sufficiency for iodine in less than three months if greater amounts of iodine/iodide were ingested.<sup>9</sup> In balance studies of amiodarone, a sustained release form of iodine, whole body sufficiency for iodine was achieved at seven weeks when given orally at 112 mg iodine/day, and the patients retained 1.5 g of iodine at sufficiency. Based on these balance studies, we postulated that with a daily intake of Lugol tablets at 100 mg iodine, whole body sufficiency could be achieved in six weeks.

Quoting Abraham and Brownstein:<sup>9</sup> "The above comparison of the data obtained from amiodarone administration and orthoiodosupplementation is suggestive of an important role of inorganic iodine released from amiodarone in the therapeutic effect of this drug, and that whole body sufficiency for iodine is a requirement for optimal cardiac function. Since the amount of iodine used in the amiodarone study is twice the amount of iodine used in orthoiodosupplementation, the time required for whole body iodine sufficiency was only seven weeks for amiodarone and 12 weeks for orthoiodosupplementation. In order to achieve whole body sufficiency for iodine in six weeks using orthoiodosupplementation, the daily intake required would be 100 mg."

To validate the above statement, one of us (JDF) recruited six female volunteers. This project was performed under contract at Flechas Family Practice and supported by a grant from Optimox Corporation. The

*(Continued on next page)*

clinical data on these six subjects are displayed in Table 1. Following informed consent, the six volunteers received 100 mg of elemental iodine (two tablets of Iodoral<sup>®</sup> 50 mg daily) for six weeks. The iodine/iodide loading test was performed with two tablets of Iodoral<sup>®</sup> 50 mg for a total of 100 mg prior to; two weeks; four weeks; and six weeks following a daily ingestion of two tablets of Iodoral<sup>®</sup> (50 mg). At 24 hours post-load, serum and saliva samples were collected as previously described<sup>7,8</sup> to measure the saliva/serum inorganic iodide ratio. The subjects were asked to abstain from the iodine supplement for 48 hours prior to the loading test in order to prevent a carryover effect. Thyroid function tests were performed pre-intervention and after six weeks on Iodoral<sup>®</sup> at 100 mg/day. Pre-intervention thyroid function tests were normal in five of the subjects (Table 2). Volunteer #4 had mild hypothyroidism with a TSH level of 6.4 IU/L (normal: 0.35-5.5) and T4 value of 4.4 µg/dL (normal: 4.5-12).

**Evidence for Inefficient Oxidation and Organification of Peripheral Iodide**

One of the female volunteers (subject #2 in Table 1) suffered from fibromyalgia based on the American College of Rheumatology 1990 criteria<sup>10</sup> and also chronic fatigue. The diagnosis was made at the initial evaluation by one of us (JDF). This manuscript presents the data obtained in volunteer #2 pre- and post-intervention for markers of iodine metabolism and for the self-assessed severity of her symptoms. Data on the other five female volunteers will be published separately.

During the 6-week study, she reported significant improvement of her symptoms and she decided to continue orthiodosupplementation at 50 mg iodine/day. Following 40 weeks at 50 mg/day, thyroid function tests were repeated again, and her daily intake was increased to 100

mg/day combined with 1,000 mg of vitamin B<sub>3</sub> (Inositol Hexanicotinate - Vitamin Research Product). The loading test and saliva/serum ratio of inorganic iodide was repeated after 40 weeks on Iodoral<sup>®</sup> at 50 mg/day and after eight weeks on Iodoral<sup>®</sup> at 100 mg/day combined with Vitamin B<sub>3</sub> at 1,000 mg/day. She was asked to score her symptoms on an analogue scale from 1 to 10; 1 being the worst she had ever felt, and 10 being the best (Table 3). From a pre-intervention score of 3 for myalgia, the score improved to 5 at six weeks post-Iodoral<sup>®</sup> 100 mg/day; 7 after 40 weeks at 50 mg iodine/day; and 6 following eight weeks on iodine 100 mg plus B<sub>3</sub> 1,000 mg/day. However, the addition of vitamin B<sub>3</sub> resulted in a significant improvement of muscle pain post-exercise. The scores for this symptom were 2 pre-intervention; 6 after 46 weeks on iodine at 50-100 mg/day; and 9 after 8 weeks on 100 mg iodine plus 1,000 mg B<sub>3</sub>/day. The score for fatigue was 2 pre-intervention and increased to 5 after six weeks at 100 mg iodine/day; to 8 after 40 weeks at 50 mg iodine/day; and to 9 following eight weeks on 100 mg iodine and 1,000 mg B<sub>3</sub>/day.

Prior to intervention, the total score for the 24 symptoms was 121, that is 50% of the maximum overall wellbeing. Following six weeks at 100 mg iodine/day, her total score improved to reach 62% of maximum. Following 40 weeks at one tablet Iodoral<sup>®</sup> 50 mg/day, a marked improvement was observed, scoring 83% of the maximum. The addition of vitamin B<sub>3</sub> to iodine 100 mg/day for 8 weeks resulted in a further improvement with a total score of 87% of the maximum value. Out of 24 symptoms evaluated, the subject gave scores of 9 or 10 for 11 symptoms following 40 weeks on Iodoral<sup>®</sup> at 50-100 mg/day, compared to scores of 9 or 10 for 18 symptoms following 8 weeks on Iodoral<sup>®</sup> and vitamin B<sub>3</sub> 1,000 mg/day. Overall, the addition of vitamin B<sub>3</sub>

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**Table 2—Self-Results of the Initial Thyroid Function Tests in the 6 Female Volunteers**

SS#	TSH (IU/L)	T4 (µg/dl)	FT4 (ng/dl)	T3 (ng/dl)	FT3 (pg/ml)
1	2.4	7.4	1.1	83	2.5
2	4.0	7.7	1.09	120	3.2
3	1.8	6.6	1.19	118	3.6
4	6.4	4.4	0.83	81	2.6
5	1.1	6.9	1.05	109	2.9
6	4.0	7.2	0.99	119	3.3
Normal range	0.35-5.5	4.5-12.0	0.6-1.8	85-205	2.3-4.2

to orthiodosupplementation resulted subjectively in a significant amelioration of her symptoms.

Pre-intervention TSH, T3, T4, free T3, and free T4 were within the normal range. TSH level increased above normal at six weeks of Iodoral<sup>®</sup> 100 mg/day. After 40 weeks at 50 mg/day, TSH decreased from 8.0 to 3.6 (Table 4). TPO antibodies were not detected.

The results of the loading tests and the saliva/serum iodide ratios are displayed in Table 5. Prior to supplementation with Iodoral<sup>®</sup>, the volunteer excreted 107% of the 100 mg ingested, and the original saliva/serum iodide ratio was elevated at 103, the normal range being 28-74.<sup>6</sup>

After two weeks ingesting 100 mg Iodoral<sup>®</sup>/day, the saliva/serum iodide ratios decreased to 43 and remained within the normal range during the rest of the study, fluctuating between 36 and 46. Pre-intervention, she excreted the total iodine load in the 24-hour urine collection. Following two weeks on Iodoral<sup>®</sup> at 100mg/day, a sharp drop in the percentage of the load excreted was observed — 18% of the 100 mg ingested. The percentage load excreted thereafter increased progressively to 66% at four weeks and to 88% at six weeks of supplementation with 100mg/day. She excreted 96% of the load after 40 weeks at 50 mg Iodoral<sup>®</sup>/day and 102% after eight weeks on 100 mg Iodoral<sup>®</sup> plus 1,000 mg B<sub>3</sub> daily.

**Table 3—Self-Assessed Effects of Lugol Tablets (Iodoral<sup>®</sup>) at 50-100 mg/day with or without the Addition of Vitamin B<sub>3</sub> on Symptomatology in a Female Subject with Fibromyalgia and Chronic Fatigue**

	Pre-Intervention	100 mg Iodoral <sup>®</sup> /day for 6 weeks	50 mg Iodoral <sup>®</sup> /day for 40 weeks	100 mg Iodoral <sup>®</sup> and 1,000 mg B <sub>3</sub> per day for 8 weeks
Muscle Pain	3	5	7	6
Pain w/Exercise	2	3	6	9
Joint pain	3	4	7	8
Joint swelling	2	4	8	9
Leg cramps	10	10	10	8
Restless legs	4	6	9	10
Stiffness	4	5	7	9
Fatigue	2	5	8	9
Insomnia	1	4	8	9
Brain fog	7	7	8	9
Dizziness	6	6	10	10
Headaches	9	9	4	6
Frequent urination	10	6	10	10
Burning urination	5	7	9	10
Abdomen cramping	7	7	9	10
Constipation	6	7	8	8
Nasal congestion	4	6	7	7
Anxiety	1	5	7	9
Irritability	8	9	10	10
Hostility	10	10	10	10
Depression	3	7	8	9
Panic attack	2	4	10	9
Flushing	6	6	10	10
Fever	6	6	10	10
Total Score	121	148	200	214
<b>Percentage of Maximum Score</b>	<b>50</b>	<b>62</b>	<b>83</b>	<b>87</b>

\* Score from 1-10; 1 being the worst and 10 being the best; Maximum score for overall best response = 240

## Discussion

Fibromyalgia is a common clinical syndrome of generalized musculoskeletal pain, stiffness, and chronic aching, characterized by reproducible tenderness on palpitation of specific anatomical sites, called tender points.<sup>10</sup> Fibromyalgia is nine times more common in middle-aged women (between the ages of 30 and 50 years) than in men. The association of fibromyalgia with chronic fatigue syndrome has been reported.<sup>11</sup> We previously proposed that fibromyalgia is caused by deficiencies of substances needed in ATP synthesis.<sup>10</sup> The role of iodine in ATP synthesis and in normal functions of striated muscles is presently unknown. In severely iodine-deficient individuals, the thyroid gland takes the lion's share (70-80%) of the total body iodine pool. However, in iodine-sufficient individuals, the maximum iodine content of the thyroid gland (50 mg) represents only 3% of the total body iodine of 1,500 mg at sufficiency.<sup>3</sup> Striated muscles contain 33% of the total body iodine in iodine sufficient individuals.<sup>17</sup>

The synthesis of ATP by intact respiring mitochondria requires the presence of oxygen, magnesium, ADP, inorganic phosphate, and the substrates from the metabolism of carbohydrates, lipids, and amino acids.<sup>10</sup> When all substances are present in optimal concentrations, the integrity of the mitochondrial membrane and the capacity of the enzymatic system in the respiratory chain become rate limiting. Defects in carbohydrate metabolism have been reported in fibromyalgia patients.<sup>12,13</sup> ATP levels are low in muscle tender points<sup>14</sup> and in the red blood cells of fibromyalgia patients.<sup>15</sup>

ATP is the universal currency of the energy used in biological systems to maintain an organism in a state that is far from thermodynamic equilibrium with the environment, that is far from death.<sup>18</sup> The active form of ATP is

a complex of ATP with mainly magnesium, but also with manganese. In cases of manganese deficiency, magnesium can replace manganese. The turnover of ATP is extremely high. For example, a human at rest consumes one half of his/her weight of ATP daily. The synthesis of ATP from ADP plus a high energy phosphate group is called oxidative phosphorylation and is dependent on the electron flow through the electron transport chain via electron carriers. NADH and FAD H<sub>2</sub> are the major electron carriers in the synthesis of ATP. The B vitamins, niacin and riboflavin, are the precursors of the cofactors NADH and FAD H<sub>2</sub>. These cofactors play an important role also in the oxidation and organification of iodide by generating hydrogen peroxide via the NADPH oxydase system.<sup>1</sup>

Iodine may play an important role in the protection of the cell and mitochondrial membranes against free radical damage by iodination of unsaturated lipids in the membranes.<sup>16</sup> The iodination of lipids of the cell membrane requires the oxidized form iodine, not iodide. The only plausible explanation for the elevated saliva/serum iodide ratio prior to intervention in the volunteer with fibromyalgia is a deficient oxidation and organification of intracellular iodide, combined with a normal cellular uptake of peripheral iodide.<sup>7</sup> Therefore, the enhancing effects of vitamin B<sub>3</sub> iodine supplementation may be due to increased iodination of lipids and proteins involved in the synthesis of ATP and also in cell function and membrane integrity. The importance of magnesium and the vitamins B<sub>2</sub> and B<sub>3</sub> in ATP synthesis and overall well-being emphasizes the need for a complete nutritional approach<sup>1</sup> in the implementation of orthiodosupplementation program for best results. We are planning to study patients with fibromyalgia following the same protocol but with a more extensive clinical evaluation pre- and post-supplementation.

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**Table 4—Results of Thyroid Function Tests Pre- and Post-Supplementation of 100 mg Iodine/day for 6 weeks and 50 mg iodine/day for 40 weeks in a Female Volunteer with Fibromyalgia and Chronic Fatigue**

	Pre-Supplementation	After 6 weeks	After 46 weeks	Normal Range
<b>TSH (IU/L)</b>	4	8	3.6	0.35 – 5.5
<b>T4 (µg/dl)</b>	7.7	6.2	6.5	4.5 – 12.0
<b>T3 (ng/dl)</b>	120	98	120	85 – 205
<b>FT4 (ng/dl)</b>	1.1	0.86	0.9	0.6 – 1.76
<b>FT3 (pg/ml)</b>	3.2	2.2	2.5	2.3 – 4.2

## About the Author

Guy E. Abraham, MD, is a former Professor of Obstetrics, Gynecology, and Endocrinology at the UCLA School of Medicine. Some 35 years ago, he pioneered the development of assays to measure minute quantities of steroid hormones in biological fluids. He has been honored as follows: General Diagnostic Award from the Canadian Association of Clinical Chemists, 1974; the Medaille d'Honneur from the University of Liege, Belgium, 1976; the Senior Investigator Award of Pharmacology, Sweden, 1980. Dr. Abraham's current research interests include the development of assays for the measurement of iodide and the other halides in biological fluids and their applications to the implementation of orthoiodosupplementation in medical practice.

Jorge D. Flechas, MD MPH, is the medical director of Flechas Family Practice in Hendersonville, North Carolina, specializing in hormonal therapy for treatment of fibromyalgia and chronic fatigue and immune dysfunction syndrome (CFIDS) since the late 1980s. He also specializes in iodine therapy for hypothyroidism and fibrocystic breast disease.

## REFERENCES

- 1) Abraham GE. "The safe and effective implementation of orthoiodosupplementation in medical practice." *The Original Internist*, 2004; 11(1):17-36.
- 2) Abraham GE. "The historical background of the Iodine Project." *The Original Internist*, 2005; 12(2):57-66.
- 3) Abraham, GE. "The concept of orthoiodosupplementation and its clinical implications." *The Original Internist*, 11:29-38, 2004.
- 4) Abraham, GE. "Serum inorganic iodide levels following ingestion of a table form of Lugol solution: Evidence for an enterohepatic circulation of iodine." *The Original Internist*, 11(3):29-34, 2004.
- 5) Abraham GE and Brownstein D. "Evidence that the administration of Vitamin C improves a defective cellular transport mechanism for iodine: A case report." *The Original Internist*, 2005; 12(3):125-130.
- 6) Abraham GE, Brownstein D, and Flechas JD. "The saliva/serum iodide ratio as an index of sodium/iodide symporter efficiency." *The Original Internist*, 2005; 12(4):152-156.
- 7) Abraham, GE and Brownstein D. "A simple procedure combining the evaluation of whole body sufficiency for iodine with the efficiency of the body to utilize peripheral iodide: The triple test." *The Original Internist*, 2007; 14(1):17-23.
- 8) Abraham, GE. "The combined measurement of the four stable halides by the ion-selective electrode procedure following their chromatographic separation on a strong anion exchanger resin: Clinical applications." *The Original Internist*, 2006; 13(4):172-195.
- 9) Abraham, GE and Brownstein D. "Validation of the orthoiodo-supplementation program: A rebuttal of Dr. Gaby's editorial on iodine." *The Original Internist*, 2005; 12(4):184-194.
- 10) Abraham GE and Flechas JD. "Management of fibromyalgia: rationale for the use of magnesium and malic acid." *Journal of Nutritional Medicine*, 1992; 3, 49-59.
- 11) Goldenberg DL, Simms RW, et al. "High frequency of fibromyalgia in patients with chronic fatigue seen in a primary care practice." *Arthr Rheum*, 1990; 33(3):381-7.
- 12) Eisinger J and Ayavou T. "Transketolase stimulation in fibromyalgia." *J Am Coll Nutr*, 1990; 9:56-7.
- 13) Eisinger J, Plantamura A, and Ayavou T. "Glycolysis abnormalities in fibromyalgia." *J Am Coll Nutr*, 1994; 13:144-8.
- 14) Bengtsson A, Henriksson KG, and Larsson J. "Reduced high energy phosphate levels in the painful muscles of patients with primary fibromyalgia." *Arthritis Rheum*, 1986; 29:817-21.
- 15) Russell IJ, Vipraio GA, and Abraham GE. "Red cell nucleotide abnormalities in fibromyalgia syndrome (abstr)." *Arthritis Rheum*, 1993; 36:S223.
- 16) Cocchi M and Venturi S. "Iodine antioxidant function and omega-6 and omega-3 fatty acids: A new hypothesis of a bio chemical cooperation?" *Prog Nutr*, 2000; 2:15-19.
- 17) Broekhuysen J, Laruel R, and Sion R. "Recherches dans la serie des benzofurannes XXXVII. Etude compare du transit et du metabolisme de l'amiodarone chez diverses especes animales et chez l'homme." *Arch Int Pharmacodyn*, 1969; 177(2):340-359.
- 18) *Biochemistry*, 2nd edition. Stryer and Lubert (ed). Freeman, New York, 1975; 240-246. ♦

**Table 5—Effects of Iodine Supplementation and Vitamin B<sub>3</sub> on Some Parameters of Iodine Metabolism in a Female Subject with Fibromyalgia and Chronic Fatigue**

	Percentage of Load Excreted*	Saliva/Serum Iodide Ratio **
<b>Pre-Intervention</b>	107	103
<b>Iodoral® 100mg/day for 2 weeks</b>	18	43
<b>Iodoral® 100mg/day for 4 weeks</b>	66	39
<b>Iodoral® 100mg/day for 6 weeks</b>	88	36
<b>Iodoral® 50mg/day for 40 weeks</b>	96	44
<b>Iodoral® 100mg and B<sub>3</sub> 1,000mg/day for 8 weeks</b>	102	46

\* Percent of 100mg iodine/iodide excreted in 24hr urine collection

\*\* The ratio of inorganic non-radioactive iodide in mixed saliva over the serum level

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# ABSTRACTS OF INTEREST

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## *Curcumin Helps Maintain Remission in Patient with Ulcerative Colitis*

by Donald Brown, ND

**Design and Duration:** Randomized, double-blind, placebo-controlled trial with a treatment period of six months followed by a six-month observation period during which patients continued on sulfasalazine or mesalamine.

**Participants:** 89 patients (ages 13-65 years) with a diagnosis of ulcerative colitis (UC) as confirmed by radiologic, endoscopic, or histological criteria. Patients were required to have a clinical activity index (CAI) of  $\leq 4$ , to have been stable for the previous four weeks, to also have achieved remission with a corticosteroid  $\geq 20$  mg/day or an alternative medication, and had successfully stopped corticosteroid treatment. Finally, patients were required to have a hemoglobin of  $\geq 10$  g/dL.

**Study Medication and Dosage:** All patients were given either sulfasalazine (1.0-3.0 g/day; median 2.0 g/day) or mesalamine (1.5-3.0 g/day; median 2.25 g/day). Patients were then randomized to receive 1 g of curcumin (API Co, Ltd., Gifu, Japan) b.i.d. or placebo. Other than sulfasalazine or mesalamine, patients were required to stop all other medications.

**Outcome Measures:** CAI was measured at entry (two weeks before randomization) and then every two months and at the conclusion of the trial. Patients who had a CAI  $\leq 4$  were considered to be in remission, while relapse was defined as CAI  $\geq 5$ . Endoscopic index (EI) was also measured at entry and at the conclusion of the study.

**Key Findings:** Eighty-two patients completed the study (43 in the curcumin group and 39 in the placebo group). In the curcumin group, two patients relapsed during the six months of therapy (4.65%) compared to 8 in the placebo group (20.51%) ( $p=0.04$ ). Recurrence rates evaluated on the basis of intention-to-treat analysis showed significant difference between curcumin and placebo groups ( $p=0.049$ ). Comparing mean CAI before and after treatment, the curcumin group decreased (improved) from  $1.3 \pm 1.1$  to  $1.0 \pm 2.0$  ( $p=0.38$ ) compared to an increase (deterioration) in the placebo group from  $1.0 \pm 1.1$  to  $2.2 \pm 2.3$ . Patients in the curcumin group also

had a significantly improved EI from baseline to the end of treatment ( $p=0.0001$ ) while there was no change in the placebo group. At the 6-month follow-up after the end of treatment, eight patients in the curcumin group had relapsed compared to six in the placebo group. A total of nine mild and transient adverse events were reported in seven patients. These included sensation of abdominal bulging, nausea, transient hypertension, and transient increase in stools.

**Practice Implications:** This interesting study completed in Japan suggests that 2 g/day of curcumin safely and effectively improves remission rates in patients with UC taking sulfasalazine or mesalamine compared to those taking the drugs alone. The study also suggests that curcumin therapy should be considered, as there was a significant relapse in patients during the 6-month observation following treatment. Considering the high rate of side effects with sulfasalazine or mesalamine, it would be prudent for future studies to examine the efficacy of curcumin alone in maintaining remission in UC patients.

Hanai H, Iida T, Takeuchi K, *et al.* "Curcumin maintenance therapy for ulcerative colitis: Randomized, multicenter, double-blind, placebo-controlled trial." *Clin Gastroenterol Hepatol*, 2006; 4:1502-6.

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## *Magnesium Builds Bones in Pre-pubertal and Adolescent Girls*

by Alan R. Gaby, M.D.

Consuming adequate amounts of magnesium is essential for building strong bones during childhood, according to the results of a new study. The study also found that many girls between the ages of eight and 14 years are not getting enough magnesium in their diet to assure optimal bone health. The results of this study add to the body of evidence that magnesium plays an important role on bone health and osteoporosis prevention.

In the new study, dietary magnesium intake was estimated from three-day diet records in 122 healthy Caucasian girls (aged 8-14 years) living in Connecticut. From

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that group, 50 girls were chosen who were consuming less than 220 mg per day of magnesium (mean intake, 184 mg per day). They were randomly assigned to receive, in double-blind fashion, 150 mg of magnesium (as magnesium oxide) twice a day or placebo for one year. Compared with placebo, magnesium supplementation significantly increased bone mineral content (BMC) of the hip and nonsignificantly increased BMC of the lumbar spine. The authors of the study concluded that magnesium supplementation had a positive effect on accrual of bone mass in peri-pubertal Caucasian girls with suboptimal dietary magnesium intake. The study also found that at least 41% of healthy girls are obtaining less-than-optimal amounts of magnesium from their diet.<sup>1</sup>

Other studies have also demonstrated the importance of magnesium for building and maintaining healthy bones. As a cofactor for the enzyme alkaline phosphatase,<sup>2</sup> magnesium is essential for bone mineralization, and a deficiency of this mineral appears to result in abnormal bone crystal formation. In a study of 19 women with osteoporosis, 16 were found to have laboratory evidence of magnesium deficiency. Interestingly, magnesium status appeared to have a major influence on the type of crystals present in the bones of these women. Of the 16 women with magnesium deficiency, every one had abnormally large bone mineral crystals, the shapes of which were also abnormal. In contrast, each of the three women with adequate magnesium status had normal bone mineral crystals.<sup>3</sup>

In addition to having a positive effect on bone quality, magnesium may help prevent or reverse age-related bone loss. In one study, 31 postmenopausal women received magnesium supplements in doses of 250 to 750 mg per day for two years. Bone mineral density increased between 1% and 8% in nearly three-quarters of the women and remained unchanged in most of the others. In contrast, 17 women who did not take magnesium supplements had bone density losses of 1% to 3%.<sup>4</sup> This study shows that magnesium supplementation can prevent or reverse the loss of bone mass that typically occurs around the time of menopause. While this study was small and did not include a control group, it is noteworthy that the magnitude of the effect was greater than that achieved in most studies of calcium supplementation.

Numerous studies have demonstrated that many Americans do not consume adequate amounts of magnesium. In a nationwide survey conducted by the US Department of Agriculture, average magnesium intake was found to be below the Recommended Dietary Allowance (RDA) for all age and sex classes, with the exception of children younger than five years. Magnesium consumption was particularly low among adolescent females, adult fe-

males, and elderly men, with 75-85% of the people in these groups failing to meet the RDA.<sup>5</sup>

While the role of calcium in building and maintaining bone mass is widely recognized, the importance of magnesium is not generally appreciated. That is unfortunate, because taking calcium supplements without additional magnesium can make an existing magnesium deficiency worse.<sup>6</sup> It is therefore important in most circumstances to balance calcium with magnesium in a supplement program. Some practitioners recommend a two-to-one ration of calcium to magnesium, whereas others recommend ratios closer to one-to-one. Most of these recommendations are based on opinion or personal preference, since there are no good data available that address the question of what the optimal calcium-to-magnesium ratio is.

A reasonable dosage range for magnesium supplementation is 200 to 600 mg per day for adults, with lower doses for children, in rough proportion to body weight. The most common adverse effect of magnesium is loose bowels, which can be corrected by reducing or splitting the daily dose. Major food sources of magnesium include whole grains, nuts, and green leafy vegetables.

1 Carpenter TO, DeLucia MC, Zhang JH, et al. A randomized controlled study of effects of dietary magnesium oxide supplementation on bone mineral content in healthy girls. *J Clin Endocrinol Metab* 2006;91:4866-72.

2 Johnson RB. A new fluorometric method for the estimation or detection of total and fractionated alkaline phosphatase. *Clin Chem* 1969;15:108-123.

3 Cohen L, Kitzes R. Infrared spectroscopy and magnesium content of bone mineral in osteoporotic women. *Isr J Med Sci* 1981;17:1123-1125.

4 Stendig-Lindberg G, Tepper R, Leichter I. Trabecular bone density in a two year controlled trial of peroral magnesium in osteoporosis. *Magnes Res* 1993;6:155-163.

5 Morgan KJ, Stampely GL, Zabik ME, Fischer DR. Magnesium and calcium dietary intakes of the U.S. population. *J Am Coll Nutr* 1985;4:195-206.

6 Smith KT, Luhrsen KR. Trace mineral interactions during elevated calcium consumption. *Fed Proc* 1986;45:374.

(Abstracts continued on page 86)

## ***Saccharomyces boulardii* Reduces the Frequency and Duration of Acute Diarrhea in Children**

by Donald Brown, ND

**Design:** Randomized, controlled trial

**Reference:** Billoo AG, Memon MA, Khaskheli SA, et al. Role of probiotic (*Saccharomyces boulardii*) in management and prevention of diarrhea. *World J Gastroenterol* 2006; 12:4557-60.

**Participants:** 100 children (ages 2 months to 12 years old) with acute watery diarrhea of mild to moderate severity.

**Study Medication and Dosage:** All children were treated with oral rehydration therapy and nutritional support. Half the children also received 250 mg of *Saccharomyces boulardii* (Biocodex, France) b.i.d. in either water or a semi-solid food.

**Duration:** Five days of active treatment with a 2-month follow-up

**Outcome Measures:** All children in the study were examined on days 0 (inclusion day), 3, and 6 during the treatment phase and every month for two months following treatment. Data collected on the first visit included date and onset of diarrhea, previous treatment, weight of the child, number and consistency of stools, vomiting, body temperature, and signs of dehydration. Stool cultures were also analyzed during the first visit for bacteria and rotavirus. The second and third visit information variables included date of stoppage of diarrhea in case of inter-current recovery, weight, daily record of frequency and consistency of stools, and tolerance and acceptability of treatment. The monthly observational follow-ups included the weight of the child and any new episodes of diarrhea.

**Key Findings:** Bacteria were isolated in 26% of the *S. boulardii* group and 12% of the control group at baseline. Rotavirus was detected in 16% and 20%, respectively. On day 3, the mean number of stools per day was reduced to 2.8 in the *S. boulardii* group and 4.4 in the placebo group ( $p = 0.01$ ). On day 6, the difference was 1.6 and 3.3, respectively ( $p = 0.001$ ). The mean duration of diarrhea was 3.5 days in the *S. boulardii* group and 4.8 days in the control group ( $p = 0.001$ ). In the following two months, the median number of episodes of diarrhea was 0.2 in the *S. boulardii* group and 0.64 in the control group at month 1 ( $p = 0.001$ ) and 0.32 and 0.56, respectively at month 2 ( $p = 0.04$ ). *S. boulardii* treatment

was well accepted and there were no reports of any side effects during the treatment period.

**Practice Implications:** Completed in Pakistan, this study finds that the addition of *S. boulardii* to standard oral rehydration and nutritional intervention reduces stool frequency and duration of illness in children with acute diarrhea. Additionally, the study demonstrates a 50% reduction in the number of episodes of diarrhea in the subsequent two months following treatment—a difference most notable in the first month. Although an earlier trial found that *S. boulardii* reduced the duration of acute diarrhea in children,<sup>1</sup> this is the first to show that short-term use also leads to long-term benefits with regard to recurrence of diarrhea. Readers interested in a more extensive overview of the use of probiotics in pediatric gastrointestinal disease should seek out the excellent review article published in the *Journal of Pediatric Gastroenterology and Nutrition* last year.<sup>2</sup>

### **References:**

- 1 Kurugöl Z, Koturoglu G. Effects of *Saccharomyces boulardii* in children with acute diarrhea. *Acta Pediatrica* 2005; 94:44-7.
- 2 Szaewska H, Setty M, Mrukowicz J, Guandalini S. Probiotics in gastrointestinal diseases in children: Hard and not-so-hard evidence of efficacy. *J Ped Gastroenterol Nutr* 2006; 42:454-75

## ***Safety Update: Red Clover Extract Does Not Cause Endometrial Hyperplasia in Postmenopausal Women***

by Donald Brown, ND

**Reference:** Imhof M, Gocan A, Reithmyr F, et al. Effects of a red clover extract (MF11RCE) on endometrium and sex hormones in postmenopausal women. *Maturitas* 2006;55:76-81.

**Design:** Randomized, double-blind, placebo-controlled, crossover study.

**Participants:** 109 postmenopausal women ( $\geq 40$  years old and amenorrhea  $> 12$  months). Participants had moderate to severe menopausal symptoms (Kupperman index  $\geq 15$ ) and were not allowed to be on HRT.

**Study Medication and Dosage:** 80 mg of MF11RCE red clover (*Trifolium pratense*) extract (Melbrosin International, Vienna, Austria) per day. MF11RCE red clover extract is standardized to contain 40 mg of aglyconic isoflavones (biochanin A, formononetin, genistein and daidzein).

(Continued on page 88)

**Duration:** Participants were randomly assigned to receive either red clover extract or placebo for 90 days. After a 7-day washout period, they were switched to receive the opposite treatment for another 90 days.

**Outcome Measures:** Ultrasound examination was performed to determine changes in endometrial thickness. Additionally, serum levels of testosterone (T), 17 $\beta$ -estradiol (E<sub>2</sub>), follicle stimulating hormone (FSH), luteinizing hormone (LH), and sex hormone binding globulin (SHBG) were measured.

**Key Findings:** Overall comparison of red clover extract versus placebo by Wilcoxon rank test, found that serum t levels were significantly increased with red clover ( $p = 0.003$ ) and endometrial thickness was significantly decreased ( $p = 0.001$ ) compared to placebo. Compared to baseline, there was a decrease in LH in the placebo group compared to the red clover group ( $p = 0.04$ ). No significant differences were noted for E<sub>2</sub>, FSH and SBHG.

**Practice Implications:** Although only 3 months in duration, this safety study suggests that a standardized red clover extract does not lead to endometrial hyperplasia and may actually reduce endometrial thickness. These results mimic those found for soy isoflavones<sup>1</sup> as well as the BNO 1055 black cohosh extract.<sup>2</sup> To date, only one study found that use a

very high dose of soy isoflavones (150 mg/day) for 5 years lead to endometrial hyperplasia in 6 of 154 women studied.<sup>3</sup> The authors suggest that while testosterone depletion is not menopause-specific, there is data that suggests that a decline androgens may play a role in mood and mental state during perimenopause and menopause.<sup>4,5</sup> The finding of an increase in testosterone is interesting and may have implications for future studies looking at the effect of red clover isoflavones on depression in postmenopausal women

#### References

1. Scambia G, Mango D, Signorile PG, et al. Clinical effects of a standardized soy extract in postmenopausal women: a pilot study. *Menopause* 2000;7:105-11.
2. Rauš K, Brucker C, Gorkow C, Wuttke W. First-time proof of endometrial safety of the special black cohosh extract (*Actea* or *Cimicifuga racemosa* extract) CR BNO 1055. *Menopause* 2006;13:678-91.
3. Unfer V, Cassini ML, Costabile L, et al. Endometrial effects of long-term treatment with phytoestrogens: a randomized, double-blind, placebo-controlled study. *Fertil Steril* 2004;82:145-8.
4. Burd ID, Bachmann GA. Androgen replacement in menopause. *Curr Womens Health Rep* 2001;1:202-5.
5. Rohr UD. The impact of testosterone imbalance on depression and women's health. *Maturitas* 2002;41:S25-S46. ♦

# ***Message from the ACA CDID***

*by Cindy M. Howard, DC, DABCI, FIAMA  
President, ACA Council on Diagnosis and Internal Disorders*

## ***My Last Words***

In July of this year, the Council on Diagnosis and Internal Disorders will have a new President. Dr. Steve Zaeske will take over the office, and with all certainty, he will do the council proud. He is dedicated to the type of medicine that we practice, and he is committed to our council. I am proud to work with him as a past president and as his friend and wife.

These last two years have been great. We continue to provide top-notch educational symposiums every July, and we are growing the DABCI classes around the country. In October 2007, we will have four classes running in four major cities in the US. There are many people responsible for how far the CDID has come while I served as your President.

Thank you to Dr. Phil Arnone who works non-stop on the symposium, to Dr. Robert Duca who put together our message board and to Dr. Jack Kessinger and Virginia Kessinger who continue to run the DABCI classes without thinking twice about giving up their weekends to further educate more doctors. The instructors in these programs also deserve a word of thanks: Dr. Tim McCullough, Dr. Frank Strehl, Dr. Michael Cessna, Dr. Bill Kleber, Dr. Richard Santelli and Dr. Dan Richard-

son.

Thank you to the ABCI: Dr. Delilah Anderson, Dr. Jonathan Williams, and Dr. Todd Smith who have worked endless amounts of hours to improve our board exam, organize the test, and to start the ball rolling on national accreditation for our diplomate.

Each year that I have been doing the DABCI work, I see things improve. People are looking for doctors that practice and care for them the way we do. I know this because my practice continues to grow through referrals while others are struggling around me. We offer medicine that is truly unique in its abilities to diagnose, treat, prevent disease through proper health and lifestyles.

I am thankful for the opportunity to serve as your President. My only request, which comes every year at this time, is to reach out to those of you who we do not see regularly at the symposium. The weekend of July 20-22 in Las Vegas will be the best weekend you spend learning and visiting with peers of intelligent and like minds. The education you will receive is unmatched by any other organization, and the camaraderie is priceless. Please check out the website at [www.councildid.com](http://www.councildid.com) for more information on this year's symposium (*see page 89 for details*). I would like to see all of you there.

My service as your President has ended, but my commitment to you and the CDID has not. I look forward to many more years with all of you. ♦

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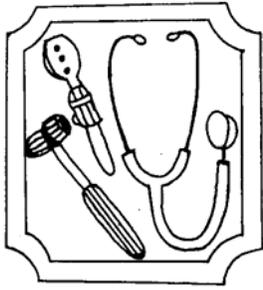
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