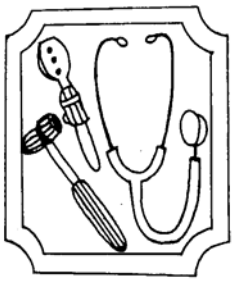

THE Original Internist

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THE ORIGINAL INTERNIST

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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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CALENDAR OF EVENTS

January 5-6, 2008 *(Houston, TX)*
Upper Gastrointestinal Disease
Instructor: *Jack Kessinger, DC DABCI*

January 12-13, 2008 *(Charlotte, NC)*
Evaluating Vascular & Venous
Disorders by Instrumentation
Instructor: *Jack Kessinger, DC DABCI*

January 19-20, 2008 *(Chicago, IL)*
The General Examination and Associated Pathology.
Instructor: *Jack Kessinger, DC DABCI*

January 26-27, 2008 *(Kansas City, MO)*
Reports, Clinical Documentation & Drug Reactions
Instructor: *Jack Kessinger, DC DABCI*

February 2-3, 2008 *(Houston, TX)*
Lower Gastrointestinal Disease
Instructor: *Frank Strehl, DC DABCI*

February 9-10, 2008 *(Charlotte, NC)*
Peripheral Vascular Disease Workshop
Instructor: *Jack Kessinger, DC DABCI*
A. Jay Kessinger, DC DABCI

February 16-17, 2008 *(Chicago, IL)*
Diseases and Exam of the Pelvis and Associated Pathology
Instructor: *Frank Strehl, DC DABCI*

March 1-2, 2008 *(Houston, TX)*
Reports, Clinical Documentations & Drug Reactions
Instructor: *Jack Kessinger, DC DABCI*

March 8-9, 2008 *(Charlotte, NC)*
Facts of Neoplastic Process & Examining the Cancer Patient
Instructor: *Jack Kessinger, DC DABCI*

March 15-16, 2008 *(Chicago, IL)*
Multi-Channel Blood Chemistries, CBC, Thyroid Thyroid Panel, TSH
Instructor: *Jack Kessinger, DC DABCI*

March 28-30, 2008 *(St. Louis, MO)*
DABCI GETAWAY WEEKEND
St. Louis Airport Marriott

April 12-13, 2008 *(Charlotte, NC)*
Malignant Diseases, AIDS, & Their Management & Treatment
Instructor: *Jack Kessinger, DC DABCI*

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April 19-20, 2008 *(Chicago, IL)*
Additional Blood Tests/Tumor Markers for Internal Disorder Pt.
Instructor: William Kleber, DC DABCI

April 26-27, 2008 *(Dallas, TX)*
Introduction to Chiropractic Internal Disorders
Instructor: *Jack Kessinger, DC DABCI*

May 3-4, 2008 *(Charlotte, NC)*
Upper Gastrointestinal Disease
Instructor: *Jack Kessinger, DC DABCI*

May 17-18, 2008 *(Chicago, IL)*
Blood Interpretation Workshop
Instructor: *Jack Kessinger, DC DABCI*

May 31- June 1, 2008 *(Dallas, TX)*
History Taking
Instructor: *Jack Kessinger, DC DABCI*

June 7-8, 2008 *(Charlotte, NC)*
Lower Gastrointestinal Disease
Instructor: *Frank Strehl, DC DABCI*

June 21-22, 2008 *(Chicago, IL)*
Cardiovascular Disease: Prevention / Diagnosis / Management
Instructor: *Jack Kessinger, DC DABCI*

June 28-29, 2008 *(Dallas, TX)*
The General Examination and Associated Pathology
Instructor: *Jack Kessinger, DC DABCI*

July 12-13, 2008 *(Charlotte, NC)*
Reports, Clinical Documentations & Drug Reactions
Instructor: *Jack Kessinger, DC DABCI*

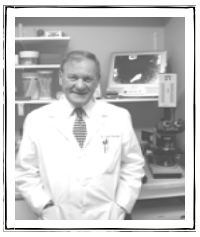
July 26-27, 2008 *(Chicago, IL)*
Electrocardiography and Photocardiography
Instructor: William Kleber, DC DABCI

July 26-27, 2008 *(Dallas, TX)*
Diseases and Exam of the Pelvis and Associated Pathology
Instructor: *Frank Strehl, DC DABCI*

August 16-17, 2008 *(Chicago, IL)*
Pharmacognosy (Herbal therapy)
Instructor: *Daniel L. Richardson, MSc, DN, PhD*

August 23-24, 2008 *(Dallas, TX)*
Multi-Channel Blood Chemistries, CBC, Thyroid Thyroid Panel, TSH
Instructor: *Jack Kessinger, DC DABCI*

From the Editor's Desk



Dr. Jack Kessinger

by A. Jack Kessinger, DC, ND, DABCI
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Caution: Pharmaceutical Procedures May Be Hazardous to Your Patients' Health and Often Delay Proper Treatment

Occasionally, I receive a response from a medical "specialist" discouraging the patient from using the alternative products I have prescribed — often suggesting they are possibly not only ineffective, but in fact, hazardous. The communication is commonly by the way of a carbon copy to a family practitioner. Even though it does take time (that is valuable to a busy practitioner), I do believe it is important to educate our obviously uninformed counterparts on such occasions.

Such an opportunity came recently, thus fodder for my "Editor's Desk" report for this issue of *The Original Internist.* This particular patient reported to our clinic for examination and treatment on May 16, 2007. His major complaint was severe malaise. He also reported several associated health problems including an ongoing fibril illness associated with night sweats, chills and upper thoracic pain. On further questioning, it was elicited that he also experienced a sensation in his head described as a "weird feeling." He denied experiencing a dry mouth or a sandy sensation in his eyes but did report some gastrointestinal bloating. Bowel movements were reported to be daily. He stated that he was never was a good sleeper and described his diet as average with too many bakery products, soft drinks and "probably not enough fruits and vegetables."

Physical examination showed the following: height — 68 1/2 inches; weight — 250 lbs; blood pressure — 120/80 bilaterally; temperature — 99.5°F taken in the ear; strong and regular heart, no bruits or murmurs; and lungs and bronchials clear, vocal fremitus normal. Orthopedic and neurologic examinations to the upper and lower extremities were all within normal limits, including deep tendon reflexes and dermatomes. Subluxations with multiple fixations were identified by clinical palpation to the upper thoracic and cervical spine bilaterally. Mild lymphadenopathy was noted to the cervical lymph nodes bilaterally. The abdominal examination elicited tenderness to the upper right abdominal quadrant by clinical palpation indicating hepatomegaly.

Complete blood chemistries were ordered, which included a multichannel profile, CBC with differential, thyroid screen, ferritin, and c-reactive protein. The profile showed an AST of 42 and ALT of 86; the ferritin and c-reactive protein were extremely elevated, at 805 and 7.58 respectively. Clinical values for ferritin are between 10 and 325 with optimum values between 25 and 225. Clinical values for c-reactive protein are below 0.8. The serum iron was, in fact, low at 43. Greatly elevated ferritin and c-reactive protein levels are found in inflammatory processes including life-threatening conditions such as cardiovascular disease, cancer, and diabetes mellitus. In the event that the elevated ferritin is not from inflammation, hyperhomocystemia must be ruled out.

Our treatment plan included diversified chiropractic adjustments to the cervical and thoracic spine to help restore nerve flow to the visceral organs as well as for the associated neuritis. Diet recommendations consisted of avoiding greasy and fried foods, dairy, chocolate, soft drinks, pork, chips, crackers and lunch meats. All meat should be boiled, broiled, baked or stewed. He was prescribed the following nutritional products: Bio FTS (known for its actions to thin bile viscosity), Lipo-Key (a dessicated liver protomorphogen) for liver support, essential fatty acids at 6 g/day and vitamin C at 4,000 mg/day. The multichannel was repeated on 06/29/07 and showed the liver enzymes to be within homeostatic range and the c-reactive protein also well within normal range at 0.16. The ferritin had dropped to nearly half, at 486. A third blood test showed continued improvement in every category, except a persistently elevated ferritin at 553. The patient's weight dropped to 228. Since I was concerned about the possibility of homochromatosis, I suggested referral to a hematologist for possible phlebotomy.

A few weeks later I received a letter, by way of carbon copy, from the hematologist that reported the patient was doing quite well, had lost between 20 and 30 pounds,

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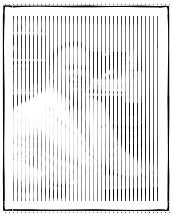
Apology to J.D. Flechas, MD

In the September 2007 issue of The Original Internist, Dr. Flechas solely authored a paper entitled *Alternative Treatment of Fibromyalgia Using the Oxytocin-Hormonal-Nutrient Protocol to Increase Nitric Oxide*. The paper was listed on the front cover with a co-author.

The Legacy Continues



by A. Jay Kessinger IV, DC, ND, DABCI
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It's not that it's so hard to teach an old dog new tricks. This has been shown to not only be possible, but also plausible, and ultimately profitable. Especially if the old dog of which we speak is a person that avoids change for no other reason than the dread of having to leave the comfort zone of the present. The saying, "You can't make a silk purse out of a sow's ear, but you can make a hotdog," is a good example of how something old can be made new and improved by enthusiastically applying innovative and revitalized re-tooling. Another example is that pesky tag in the collar of a good Fruit of the Loom T-shirt that has been replaced by a permanent stamp-on advertisement. Re-tooling is taking a good idea then injecting it with more practicality.

Ever since Ponce de Leon attempted to find the single magic bullet - the fountain of eternal youth - humanity in its finite wisdom has been searching for the same. Both chiropractors and medical physicians have fallen into this same pit proclaiming to have found the fountain of youth. In reality we are living in a more and more polluted, nutritionally depleted, and physically less demanding world as a result of industrialization, commercialization, and modernization. We need to keep reworking the old "what works" into the modern ever changing world of which we are a part, rather than prematurely proclaiming, "We found it!! Over here!! The fountain of youth!!"

Shortsightedness can thwart advancement. A study from the University of New Mexico School of Medicine introduced "the tomato effect" in *JAMA* (May 11, 1984). The name was derived from the history of the tomato in America. It was shunned until the 1800s in this country because we knew it was poisonous. Everyone at that time knew tomatoes belong to the nightshade family, and the leaves and fruit of several plants in this family can cause death if ingested. The fact that Europeans were eating tomatoes without harm was irrelevant. It simply did not make sense to eat poisonous food. What we can now glean from this study is that life is certainly uncertain, and our wisdom is limited. Deductive reasoning in its superficial sense can be a downfall. If we had continued to avoid tomatoes, in this instance, we would continue to blindly be void of a ready source of lycopene.

Another example of our collective mind set being throttled is in our receiving professional health care. If we expected as much out of our car insurance as we expect out of our health insurance, we'd be doomed to public transportation because we couldn't afford a policy that changes our oil and our tires and keeps our tanks full. We still enjoy the freedom of hopping in the car and going wherever we want, whenever we want, but that flies in the face of our health care model of, "If my insurance won't cover it, then I can't afford it." A re-tooling of our thinking — a paradigm shift — in regards to our health delivery system is obviously necessary for us to maintain our American individuality and endowed unalienable rights in the pursuit of happiness, etc.

The old adage that penicillin, *et al*., will cure "all that ails you" has fallen flat in light of the new *mersa* staph scare, etc. However, the original premise of the old adage was not wrong in its entirety. The error of "the germ theory" (thus, treatment of disease) is the notion of its all inclusiveness. Since germs, according to the original premise, cause all disease, then their eradication will cure all disease. Time, clinical examples, and the "super bugs" (i.e., drug resistant bacteria, etc.) humanity now faces have provided undeniable evidence that a paradigm shift is necessary once again.

For those "chiropractic at heart," the aforementioned germ theory of disease should ring the same as, "if the production of that bump caused deafness (i.e., all disease), then the reduction of that bump will restore hearing (i.e., optimal health)." Nerve tone was the original single foundation of chiropractic, and upon this principle came the cycle of keeping the producer in the product, hand only/spine only, *ad nauseum*. Once again, humanity, in her finite mental understanding of all things, has found the notion of all-inclusiveness flawed. A re-tooling is again required.

Both the germ theory and specific chiropractic spinal adjusting are flawed in their shortsightedness. However, in reality, both can, without a doubt, be beneficial when used for certain conditions although certainly not all-inclusively for any condition. Utilizing antibiotics before attempting to allow and/or bolster the innate immune system is wrong, in that biological weakness will ensue. Chiropractic adjusting without maintaining a healthy lifestyle (i.e., exercise, nutritionally clean diet, individually tailored nutritional supplementations) is

(Continued on page 187)

SOUND OFF

by Frank Strehl
DC, DABCI

Gardasil: Pros and Cons

As I contemplated this month's hot topics, I tried to think of the hottest topic facing today's physicians, and I settled on Gardasil. It is true that when effective, it can prevent 70% of cervical cancers caused by the viruses that carry a high risk for becoming cervical cancer and is 90% effective against the viruses that carry a low to moderate risk for becoming cervical cancer (this type is generally responsible for genital warts). As I tell my students at National, my job is not to unduly influence their thought process or their decision for their patients but rather to provide them with the salient facts on which to base their decision. So, the question for you this month is, "Would you break from the profession's tradition of opposing vaccinations and recommend this potentially life-saving vaccine for your patients and their daughters?"

Perhaps some of you would like a little more information about Merck's Gardasil vaccine. The human papillomavirus (HPV) quadrivalent vaccine will be sold under the trade name Gardasil. It contains no live virus and is given in three separate intramuscular injections in the upper arm over a 6-month period. A Merck news release says that the vaccine will cost about $120 per dose — for a total of $360 for immunization. The actual cost to the physician is a little more than initially estimated.

The FDA emphasized to reporters that the product does not protect women if they have already been infected with HPV before vaccination. And yet, some gynecologists recommend and administer the vaccine to women with cervical dysplasia secondary to the HPV virus. Regulators say this fact "indicates the importance of immunization before potential exposure to the virus." The product is also not designed to protect against less common HPV types, and routine Pap screening will therefore remain critical moving forward.

A background document provided to an FDA advisory committee flags a potential problem for a small subset of women. The briefing refers to the possibility that the HPV quadrivalent vaccine enhances disease among a subgroup of subjects with evidence of persistent infection with vaccine-relevant HPV types at baseline.

In the briefing document, the review team raises a second concern with cases of CIN 2/3 or worse from HPV types not included in the vaccine, "These cases of disease due to other HPV types have the potential to counter the efficacy results of Gardasil for the HPV types contained in the vaccine."

The National Vaccine Information Center (NVIC) called on the CDC's Advisory Committee on Immunization Practices (ACIP) to just say "no" on June 29 to recommending "universal use" of Gardasil in all preadolescent girls. NVIC maintains that Merck's clinical trials did not prove the HPV vaccine designed to prevent cervical cancer and genital warts is safe to give to young girls.

The FDA allowed Merck to use a potentially reactive aluminum containing placebo as a control for most trial participants, rather than a non-reactive saline solution placebo. A reactive placebo can artificially increase the appearance of safety of an experimental drug or vaccine in a clinical trial. Gardasil contains 225 mcg of aluminum and, although aluminum adjuvants have been used in vaccines for decades, they were never tested for safety in clinical trials. Merck and the FDA did not disclose how much aluminum was in the placebo.

Animal and human studies have shown that aluminum adjuvants can cause brain cell death and that vaccine aluminum adjuvants can allow aluminum to enter the brain as well as cause inflammation at the injection site leading to chronic joint and muscle pain and fatigue. Nearly 90% of all Gardasil recipients and 85% of aluminum placebo recipients reported one or more adverse events within 15 days of vaccination, particularly at the injection site. Pain and swelling at injection site and fever occurred in approximately 83% of Gardasil and 73% of aluminum placebo recipients. About 60% of those who got Gardasil or the aluminum placebo had systemic adverse events including headache, fever, nausea, dizziness, vomiting, diarrhea, and myalgia. Gardasil recipients had more serious adverse events such as headache, gastroenteritis, appendicitis, pelvic inflammatory disease, asthma, bronchospasm and arthritis.

Merck and the FDA have not publicly revealed several important aspects of the trials:

(Continued on next page)

- How many girls aged 9-15 participated in the clinical trials?
- How many of the participants received Gardasil and hepatitis B vaccine at the same time?
- How many of the participants experienced serious adverse events after injection of either Gardasil or the aluminum placebo?

For example, if fewer than 1,000 girls were actually injected with three doses of Gardasil, it is extremely important to know how many had serious adverse events and how long they were followed for chronic health problems, such as juvenile arthritis.

In May 2007, it was reported that over 1,600 adverse reactions, including three deaths, had been linked to Gardasil. Among those reactions, 371 were classified as serious, and of the 42 women who received the vaccine while pregnant, 18 experienced side effects including spontaneous abortion and fetal abnormalities.

It appears the number of reactions (and deaths) is steadily rising. A review of the National Vaccine Information Center revealed the following statistics about this vaccine: 2,207 adverse reactions to Gardasil have been reported including:

- 5 girls died
- 31 were considered life-threatening
- 1,385 required a visit to the emergency room
- 451 of the girls have not recovered as of July 2007
- 51 of the girls were disabled

Gardasil "may be more dangerous than consumers have been led to believe" according to one public interest group, and an editorial in the *New England Journal of Medicine* has also raised questions about the vaccine's effectiveness.

It should be kept in mind that HPV types 6 and 11 are considered as low risk and are only two of approximately nine viral types in that group that can cause genital warts. Also, HPV types 16 and 18 are considered high risk for becoming cervical cancer and are only two of approximately 11 known to be in the group that are considered to be high risk for becoming cervical cancer.

Now, doctor, would your recommendation still be the same as when I asked the question at the beginning of this article? Perhaps it is time for us to take up our swords and fight this battle from the inside rather than from the outside of conventional medicine. Perhaps it is time for us to acquire a license to "unprescribe." ◆

Prolotherapy: Synergism with Chiropractic Treatment

by E. W. McDonagh, DO

For the past 18 years I have utilized a non-surgical treatment to successfully reverse severe conditions of joints, tendons, and ligaments. Prolotherapy is a unique treatment method that enables the doctor to call up the body's latent powers to repair damaged, torn, or slack ligaments and tendons without prescription drugs or surgery. Joint spacing is normalized. Bones do not rub and grind on each other. The underlying bone damage to joints and vertebrae is relieved and pain is eliminated. Ligaments and tendons are made tighter and stronger than ever before.

I believe I know of what I speak. Five years ago my labrador retriever lassoed my ankles one night. I fell backwards into the rock garden, striking my lumbar spine against a sharp, pointed rock. I had fractured a lumbar vertebra. At the hospital the radiologist told me that:

1) I had a fracture of L2.
2) I needed to check into the hospital.
3) I needed an MRI (magnetic resonance imaging).
4) I would need to have surgery in the morning, or I would never walk again.

At this point I was not sure what was worse: the severe pain or the prognosis of never walking again.

Knowing the great results I had consistently seen in severely damaged lumbo-sacral spine patients, I opted to have my partner do prolotherapy on my lower back. I took 10 treatments in 11 days. Two weeks after the accident, I was walking at a medical meeting in Florida. I have been doing a gymnasium workout three days per week ever since. I have no back problems or trouble walking.

Prolotherapy, or as we commonly call it reconstructive injection therapy, is accomplished by the injection of multiple minute droplets of a non-drug solution into the ligaments/tendons of the damaged joint. Many tiny inflammatory areas along the ligament/tendon cause a chemical change that attracts immune system fibroblast cells to migrate to these areas and lay down new tendon or ligament material. Subsequent treatments continue the process, until the joint is fully functional, strong again, and pain-free. It might take 4-5 treatments or possibly 10. Although rarely, it could take more than 10 depending on any other underlying pathology — poor circulation to the joint, osteopenia, osteoporosis, diabetes, anemia, etc. — that can slow the process of joint healing.

When we test for these things, we treat them also, to obtain best results with minimum prolotherapy treatments. Patients getting great treatment results will refer several new patients, especially if they were originally advised to have surgery by their physician.

For chiropractors who can do injections, this can be a wonderful opportunity to generate improved reputation in your community. There is a veritable hoard of patients available for this treatment already in your practice.

We always advise our patients to continue the treatment schedule outlined by their chiropractor. He can realign the bones, relieve pressure of spastic muscles and fascia but cannot repair ligaments/tendons. Prolotherapy will repair and rebuild these, a great treatment synergy.

Prolotherapy is compatible with most other treatments. At our clinic, we do chelation therapy, hyperbaric oxygen treatments, etc. We have seen no incompatibilities. I could cite many wonderful, advanced cases relieved and reversed by prolotherapy over the years, but I think that a few cases will suffice.

Miss P, a 44-year-old partially disabled Caucasian female presented with lumbar discopathy. She had a failed surgery for disc rupture at L5-S1. Pain recurred almost immediately. When first seen at this clinic in March 1991, she had severe pain radiating down the left leg and was able to ambulate only with the use of a push-along walker. She received no concommitant therapy other than that provided by this institution, and excellent results were obtained. Currently, she has no restrictions of locomotion and does not experience any pain. Studies by MRI were obtained both before and after our treatments and were performed on a General Electric Signa Advantage MRI.

After the initial examination, the patient received a course of 21 infusions of intravenous EDTA, together with multivitamins and trace mineral supplements. In addition, the patient received 14 injection treatments, each consisting of multiple small injections into the ligamentous structures surrounding the paraspinal muscles of the lower dorsal and lumbar spines over a period of

(Continued on next page)

12 months. After treatment, the patient's abnormal disc protusion was reduced 60% from 5mm to 2mm as demonstrated by pre- and post-treatment MRI. Subsequently she divorced and moved to the east coast. A year after we treated her, she returned to Kansas City and brought a copy of a recent (third) MRI. The radiologist stated he could find no evidence of disc herniation.

DS, a Caucasian gentleman, age 70, had the diagnoses of atherosclerosis, lumbar disc compression syndrome, and arthritic deterioration and instability of the spine. This patient gave a history of multiple falls and injuries to his back and hips during his career as a farmer, resulting in spurring, arthritic deterioration, disc compression, and destabilization of the vertebral column. For the three months prior to his first visit to this clinic, he could not sleep uninterrupted more than one hour due to recurring pain. If he attempted to lie on his left side, additional excruciating pain would radiate into his left side and down the left leg.

He elected to have reconstruction injection therapy. After one treatment of multiple small amounts of proliferating solution into the tendons and ligaments of the dorsal and lumbar spine and sacroiliac joints, he was pain free. He stated the next morning, "Doc, I slept the whole night through!" He was given no prescription drugs, manipulation, physiotherapy, physical restriction advice, or any other treatment.

Another patient, RM, a 70-year-old cattle farmer complained of constant muscle spasm and pain in the cervical spine, right shoulder, and dorsal spine. He walked abnormally favoring his right leg. He had been suffering for 50 years in spite of regular chiropractic care, which afforded minimal relief. He had had a 70-pound hay bale fall on his head, which he carried at a 45 degree angle from vertical. Prolotherapy was begun with the stipulation that he continue with his regular chiropractor as before. He is now asymptomatic after a year of off-and-on treatment.

Summary

This treatment, in a step-wise progression, can rebuild the tendons, ligaments, and joints of the body. The pain is eliminated, strength and function return, pain medication is eliminated, and the patient can resume his/her life with no functional limitation. Costs rarely exceed 10-15% of the surgical treatment approach. Reconstructive injection therapy restores the strength as Mother Nature intended and is permanent. Surgical joint replacement is rarely permanent, but needs to be repeated periodically. Many side effects — deterioration, infection, etc. — are also seen with the surgical treatment. Prolotherapy is very effective, easy to learn, and is inexpensive. It generates great patient gratitude. It is a wonderful practice builder. Most importantly: prolotherapy treatment results are better than any other.

About the Author

Dr. McDonagh has practiced alternative and complementary medicine including cardiovascular chelation and hyperbaric oxygen therapy for 45 years in Kansas City, Missouri. McDonagh Medical Center has published more scientific papers showing the benefits of EDTA Chelation therapy than any other physicians in the country. His prolotherapy results are enhanced by his unique treatment method.
see www.mcdonaghmed.com

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Sarcopenia: When Weight Loss is Counter-Productive

by Harry G. Preuss MD, MACN, CNS, Debasis Bagchi PhD, FACN, CNS, Nicholas Perricone MD, Samuel C. Keith, BS, Steven C. Boyd, MD, and Gilbert R. Kaats PhD, FACN

While chiropractors and other health care providers are universally aware of the increased risk for osteopenia and osteoporosis as a consequence of the age-related decline in bone density, fewer are cognizant of the deleterious effects of sarcopenia, the progressive loss of muscle tissue with age. Skeletal muscle mass and strength generally peak between 20 and 35 years of age.[1] Thereafter, 3-8% of the achieved muscle mass may be lost per decade, a loss rate that has been reported previously to accelerate after the age of 60.[2-6] Using a cross-sectional analysis of the 20,000 DEXA tests of body composition in his database, one of the co-investigators (GRK) found that the rate of lean mass loss increases with each passing decade, a pattern virtually identical for men and women as shown below.

Sarcopenia differs from involuntary muscle depletion as seen in inadequate nutritional intakes, starvation, cancer, or AIDS. It also is distinct from cachexia, a cytokine-driven loss of lean body mass, that occurs despite maintenance of body weight which is sometimes seen in patients with rheumatoid arthritis, congestive heart failure, or renal failure.[7] "Sarcopenia is not a disease … but is the backdrop against which the drama of disease is played out: a body already depleted of protein because of aging is less able to withstand the protein catabolism that comes with acute illness or inadequate protein intake."[8]

While cachexia generally connotes a state of advanced malnutrition and wasting, we now know that this term refers, more specifically, to a loss of body cell mass. Studies of starvation, critical illness and normal aging have found that a decrease in body cell mass greater than 40% is fatal.[9] Even with as little as 5% loss of body cell mass, there are demonstrable changes in morbidity, including loss of muscle strength, altered energy metabolism, and increased susceptibility to infections.[10] Accompanying the muscle loss is a reduction in voluntary strength (about 30% between 50 and 70 years of age).[11]

Derived from the Greek root words: "sarx" meaning flesh and "penia" meaning loss, sarcopenia has important consequences for balance, metabolism, physical appearance, general well-being, and quality of life. As the current length of the human life span increases, the projected number of people suffering from sarcopenia will also increase. As sarcopenia progresses, activities of daily living and mobility are further impaired, which may result in osteoporosis, falls, fractures, thrombophlebitis, pulmonary embolism, isolation, depression, and other adverse consequences. It is estimated that 14% of persons 65-75 require assistance with activities of daily living, and this figure increases to 45% for persons over 85 years of age.[12] The medical effects and economic costs of sarcopenia are profound. As one reviewer has reported, "Sarcopenia is an important cause of frailty, disability, and loss of independence in the elderly, and recent estimates suggest that it costs the United States over $18 billion per year, a sum on par with the economic consequences of osteoporosis."[14]

Some studies suggest that sarcopenia is the major predictor of function limitations while others suggest that it is the combination of obesity and sarcopenia that is the primary cause of disability. The implications are that sarcopenic obesity in old age is more strongly associated with disability in daily living than either sarcopenia or obesity alone.[15]

The widespread use of scale weight as a measure of wellness or the effectiveness of weight loss interventions obscures what may be the most important aspect of weight loss — it is not the amount of weight one loses that is the most important measure of well-being but rather the kind of weight one loses. While it is understandable that the general public would be unaware of these consequences, it is troubling that many large, randomized, placebo-controlled, double-blinded studies performed at leading medical centers have used scale weight changes almost exclusively as their primary end point to interpret the safety and efficacy of weight loss interventions.

To address this problem, two of the investigators (GRK & HGP) have begun using a body composition improvement (BCI) index as the criterion for a safe and effective weight loss intervention or change. The BCI is the result of scoring all gains in fat-free mass and decreases in body fat as positive outcomes, and losses in fat-free mass and increases in body fat as a negative outcome. Thus, a person gaining two pounds of lean and losing two pounds of fat would have no change in scale weight but would have a BCI of +4 since both the changes in lean and fat are positive outcomes. Conversely, a person

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losing two pounds of lean and gaining two pounds of fat would have no change in scale weight but would have a -4 BCI since both of these changes are considered negative outcomes. Using only scale weight instead of the BCI would lead one to conclude that both interventions were ineffective while one was, in fact, effective while the other was ineffective. To examine the difference between using changes in scale weight as opposed to the BCI, these two authors compared scale weight changes with BCI in over 15,000 scale weight changes in the former's database. Differences were calculated for five possible outcomes:

1) No change in scale weight but a positive BCI
2) No change in scale weight but a negative BCI
3) A positive change in scale weight with no change BCI
4) A positive change in scale weight but a negative BCI
5) A negative change scale weight but a positive BCI

A comparison of the changes that occurred in the roughly 15,000 interventions revealed that in 84% of the cases, there were differences between scale weight changes and the BCI. Thus, different conclusions would be drawn 84% of the time using these five types of comparisons suggesting the potential for misinterpreting the safety and efficacy of the intervention was widespread. To calculate the average magnitude of these differences, a difference was calculated between the BCI and the scale weight change for each of the five comparisons. For example, comparing a scale weight change of -4.0 lbs with a BCI of +3.0 lbs was recorded as a difference score of 7.0 lbs (using the absolute value). These comparisons were completed for ~15,000 pre- and post-intervention changes. The average magnitude of the difference between the scale weight changes and the BCI was 7.1 lbs. These data suggest that there are widespread significant, and sometimes profound, differences between using scale weight change as an outcome measure as opposed to using the Body Composition Improvement index which could lead to very different conclusions about the safety and efficacy of weight loss interventions.

For overall beneficial health, we do not pretend to know how many pounds of muscle it takes to offset gains in body fat, or how much fat one has to lose to offset losses in muscle. But what is clear is that loss of lean and gains of body fat are clearly negative treatment outcomes irrespective of any changes in scale weight. Thus, as we said above, the litmus test of a safe and efficacious weight loss program is the preservation of lean and the depletion of excess body fat — in short, the kind, not amount, of weight that is lost.

Thus, sarcopenia is a significant problem to be addressed by chiropractors and other healthcare providers. As one reviewer has concluded, "It appears clear that for the clinician, the most important message is that sarcopenia exists in all older individuals. In the face of acute or chronic illness, maximizing muscle mass and protein stores through adequate nutritional support, aggressive physical therapy, and exercise programs become all the more important if muscle function and quality of life are to be preserved in the older population."[16]

What is behind the increasing rate of obesity/sarcopenia plaguing the world? No doubt, there are multiple reasons for it, so let us consider a few possibilities. It is readily recognized that the primary treatment for improving the muscle/fat ratio is exercise and diet. In the case of the first, it is generally accepted that most individuals get too little exercise. No one should argue that such a state leads to a smaller muscle mass and a larger fat mass. As for the second, dietary indiscretion is recognized everywhere. The public has been trained to focus on "saturated fat," and now "trans fat," content of foods to avoid atherosclerosis and heart disease. Never mind that trans fats were originally ignored, and the excess calories and refined carbohydrates that often replaced the avoided saturated fats were also ignored. In his well-documented and scholarly researched book, *Good Calories, Bad Calories,*[17] Gary Taubes, a correspondent for *Science* magazine concludes:

> "The problem is the carbohydrates in the diet, their effect on insulin secretion and the hormonal regulation of homeostasis — the entire harmonic ensemble of the human body. Insulin is the primary regulator of fat storage. When insulin levels are elevated — either chronically or after a meal — we accumulate fat in our fat tissue. When insulin levels fall, we release fat from our fat tissue and use it for fuel. The more easily digestible and refined the carbohydrates, the greater the effect on our health, weight, and well-being. Fattening and obesity are caused by an imbalance — a disequilibrium — in the hormonal regulation of adipose tissue and fat metabolism. By stimulating insulin secretion, carbohydrates make us fat and ultimately cause obesity. The fewer carbohydrates we consume, the leaner we will be."

Taubes, the only print journalist to have won three Science in Society Journalism awards given by the National Association of Science Writers, presents compelling evidence that it is the effects of easily-digestible simple car-

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bohydrates, not dietary fat, saturated or not, that is the major cause of obesity, heart disease, and many other chronic diseases of civilization, perhaps even sarcopenia. The message of reducing calories and avoiding fat has gained so much popularity one may question as to the extent to which it may lead to even more sarcopenia.

We recently found that positively affecting the glucose-insulin system might also be helpful, i.e., proper insulin metabolism in contrast to insulin resistance favors fat loss and muscle gain. Fortunately, many safe natural substances can favorably influence the glucose insulin system. We found this out in a recent study using trivalent chromium in the form of niacin-bound chromium. Niacin-bound chromium is a substance known to overcome peripheral insulin resistance.[18] Twenty overweight African-American women participated in a randomized, double-blinded, placebo-controlled, crossover study.[18] They received placebo three times a day during the control period and niacin-bound chromium, 200 mcg three times a day during the test period for two months. Body weights and fat and non-fat body masses were estimated. Body weight losses were essentially the same in both the placebo and chromium-receiving ladies. However, fat loss was significantly greater and non-fat body mass loss significantly less with chromium intake. Niacin-bound chromium given to modestly dieting and exercising African-American women caused a significant loss of fat and sparing of muscle compared to placebo even though scale weight losses were essentially similar. A frequent complaint during the study was the lack of a significant decrease in scale weight, but often in the same breath, the subject would describe a decrease in dress size.

At this point, let us focus more on maintaining muscle mass. With typical, everyday movement and exercise, skeletal muscle in the human body is under constant renovation.[19-21] An equal balance between synthesis and degradation of muscle proteins results in the size of muscle tissue remaining essentially unchanged. If, however, there is a shift in this balance, the resulting state will be atrophy (breakdown is greater than synthesis) or hypertrophy (synthesis is greater than breakdown). It follows that changes in muscle mass are correlated with modifications in protein synthesis, protein degradation, or both.[22] During and immediately following exercise, degradation of muscle protein is augmented. The released amino acids emanating from protein breakdown have the potential to produce glucose and ATP that are then used to produce energy throughout the body. The result is that there is less protein when the stressed muscles come to rest.[23]

In addition, damage to some muscle cell membranes may take place, creating delayed-onset muscle soreness. This is especially associated with eccentric exercise such as downhill running. Injury can similarly occur in the connective tissue as well, diminishing performance for some time.[24] Obviously, lessening the protein breakdown would ameliorate, at least to some extent, this situation, i.e., shortening the period of increased protein degradation would be advantageous for people wishing to maintain, and even improve, their muscle mass.[24] The fact that sarcopenia is a multi-factorial condition may explain why it can be treated by several different means other than resistance training. These include hormonal interventions, increased protein in the diet, and nutritional supplements.

The most accepted hormonal treatment of sarcopenia uses growth hormone (GH). This is based on the fact that reduced levels of growth hormones are found in patients with sarcopenia.[25] One study where patients with sarcopenia were treated with GH supplements showed a significant increase in plasma IGF-1 levels and small increases in lean body mass.[26] Unfortunately, serious side effects followed, like increased levels of circulating glucose and systolic blood pressure. Other adverse side effects included carpal tunnel syndrome, gyneocomastia, and hyperglycemia. Although GH therapy significantly increases lean muscle mass, the frequent occurrences of serious side effects, as well as the high monetary costs, outweigh the clinical benefits. A potential option to replace GH therapy is the use of growth hormone-releasing hormone intervention (GHRH) supplements. GHRH appears to maintain the benefits of GH with fewer side effects than GH therapy.[26]

Testosterone and estrogen supplements offer additional hormonal intervention in elderly people with sarcopenia. One study showed that testosterone replacement therapy increased grip strength and decreased low-density lipoprotein cholesterol with no detectable changes in weight, body fat, or lean muscle mass.[26] Unfortunately, there exists too much variability in results to confidently recommend testosterone therapy for sarcopenia.[27] Similar to testosterone, estrogen or estradiol replacement therapy also yields variable results that require further elucidation.[28] The variability may be due to biological differences among people. Along similar lines, dihydroepiandrosterone (DHEA), an adrenal androgen, is the biological precursor of active androgenic hormones such as testosterone and dihydrotestosterone.[26] DHEA has been used for replacement therapy to increase anabolic hormones that increase levels of lean muscle mass in the elderly people with sarcopenia. It is generally recognized that DHEA supplementation requires more information and testing before its therapeutic benefits can be

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Table 1

Percentage Rate of Loss of Lean by Decade from Age 35

Women		Decade	Men	
N	% Decline	From 35 years	N	% Decline
3,383	1%	**45**	1,014	1%
4,647	2%	**55**	1,259	2%
2,671	3%	**65**	909	4%
858	6%	**75**	425	6%
163	8%	**85**	65	7%

ascertained definitively. To sum up, hormone replacement therapy appears to have nominal and variable effects on lean muscle mass and muscle strength with frequent unfavorable side effects.

The branch chain amino acids, in particular leucine, appear to play a significant role in muscle building. Scientists over many years have been impressed with the ability of leucine and other branched-chain amino acids, such as valine and isoleucine, to augment muscle mass.[29-34] These amino acids yield anabolic effects similar in many respects to those of insulin.[30] Leucine is not synthesized by the human body and must be acquired through diet placing it in the category of an essential amino acid. When passing through the liver, leucine loses its amino moiety to form its keto analogue, alphaketoisocaproate (KIC). The majority of KIC is further metabolized by the mitochondrial enzyme, KIC-dehydrogenase. Eventually, acetoacetate and acetyl CoA are formed. Through another minor pathway, beta-hydroxy-beta-methylbutyrate (HMB) is made from KIC in the presence of cytosolic KIC-dioxygenase.[35-42]

In 1982, Tischler, *et al*.,[43] assessed whether degradation of leucine is necessary for stimulation of protein synthesis. KIC, the product of leucine transamination, reduced proteolysis without stimulating protein synthesis. When cycloserine was used to block transamination of leucine, the inhibition of proteolysis was prevented, however, the stimulation of protein synthesis was not affected. The authors concluded that some intermediate(s) or product(s) of leucine catabolism inhibits proteolysis. Over the years, it has been found that KIC and HMB are two very useful breakdown products of leucine catabolism that inhibit proteolysis of muscle. Accordingly, branched-chain amino acids and their breakdown products, such as KIC, have been used extensively to increase protein synthesis and reduce protein degradation in muscles.[44-52]

What do we know about HMB? Approximately 5% of the dietary intake of leucine is converted to form HMB.[53] Nevertheless, this amount of endogenous HMB in the body (estimated to be approximately 0.2-0.4 g/day for an average 70 kg person[53]) does not appear to be sufficient to produce a significant effect on muscle protein. Exogenous sources of HMB, such as alfalfa, corn silage, grapefruit, and catfish, also exist. However, these indirect sources do not significantly contribute to the amount of HMB necessary to produce the proposed effect. Almost 20 years ago, Kuhlman, *et al*.,[54] reported that supplemental HMB had an important metabolic role. Although many findings have subsequently corroborated this assumption, the exact mechanism by which HMB slows muscle protein degradation remains uncertain.[55] It is important to note that beneficial effects of HMB supplementation on body composition will not occur to any great extent without exercise. With sedentary subjects, HMB produced no changes in muscle strength or body composition. When resistance training was initiated using the same volunteer group (three times a week for four weeks), increases in lean body mass and upper body strength were noteworthy. The proposed reason to account for differences between sedentary versus exercise effects is that available endogenous HMB cannot accommodate the activity of muscle during exercise. The body uses the excess HMB to restore and protect original muscle protein and membranes.[23] In contrast, supplementation of additional HMB to sedentary subjects is not needed by the body and is excreted by the kidney. The safety of HMB has been shown clearly.[23,56]

In conclusion, overweight/obese and sarcopenic states

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are prevalent worldwide. Many individuals from all walks of life must become aware of their dangers and take the necessary precautions to prevent, ameliorate, or overcome these disabling states. More details on this subject can be found in the recently published book, *The Natural Fat Loss Pharmacy*.[57]

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The Dangers of Magnesium Deficiency in Endurance Athletes

by Terry J. Nelson, DC, DABCI

The weekend of October 6-7, 2007, was one of the biggest marathon weekends of the year. *Marathon Guide*, an online everything-you-want-to-know-about-marathons site listed 23 marathons on their calendar for that weekend and estimated that one of every six people who would complete in a marathon in 2007 would do it that weekend. Included among those numerous races were several of the most popular yearly events including the marathons in Chicago, Twin Cities (Minneapolis-St. Paul), and Portland, Oregon. These three races alone would attracted nearly 70,000 runners of all levels.

Several weeks prior to this weekend the national weather forecasters began to warn of a heat event that might move into the northern US and pose a risk to participants, especially those in Chicago. While participating in the Portland marathon, I met a runner from New Mexico who had canceled his plans to run in Chicago when he heard the weather forecast and came to Portland instead.

I had barely crossed the finish line in Portland when the stories began to filter in about Chicago, the heat, the confusion, and the fact that a runner had died on the course. It would be another day before it was announced in the press that an autopsy showed a heart condition, not record-setting heat, had killed the police officer from Michigan. "'Chad Schieber … had a mitral valve prolapse and did not die from the heat,' the medical examiner's office said."[1]

Is it possible that Chad Schieber developed this "heart condition" as a result of his running and a failure to supplement his diet with adequate amounts of magnesium? Is it possible that thousands of other runners are making the same mistake and running a similar course toward danger?

Mitral valve prolapse (MVP) is a common condition occurring in 4-18% of the population. It is an abnormality of the mitral valves of the heart in which one or both of the mitral valve flaps close incompletely, often producing either a click or murmur.[2] It is the most common valvular disorder in industrialized nations, and a familial tendency has been demonstrated.[3] While there is a familial or genetic tendency for MVP, there is a significant body of evidence suggesting that magnesium deficiency is at least a symptom of MVP and that many of the symptoms of MVP syndrome are reduced or resolved by magnesium supplementation.[3-8] Several authors suggest however that there is more evidence and logic supporting the theory that mitral valve prolapse is likely to be a symptom of magnesium deficiency itself.[2,2]

Magnesium

Magnesium belongs in a category of minerals called electrolytes because they conduct electrical signals in the body. It is found in all of the body's cells, although it is most concentrated in the bones, muscles, and soft tissues. It's a necessary element in more than 300 enzyme reactions involving nerve transmission, muscle contraction, and especially adenosine triphosphate (ATP) production. (ATP is the fundamental energy currency of the body. All other fuels — glucose, fats, etc. — are broken down to produce ATP, which in turn stimulates muscle contraction.)

All of the important electrolytes except calcium are lost through perspiration. Heavy sweat loss can therefore interfere with the important functions for which magnesium and other electrolytes are responsible. Low blood magnesium levels during exercise have been cited as a cause of muscle fatigue and irregular heartbeat."[9]

Studies have confirmed that serum and urinary magnesium concentrations decrease significantly while running a marathon — enough to produce magnesium deficiency.[10] One can imagine then that a runner training through a summer that was unusually hot, especially in the northern sections, could develop a magnesium deficiency if (s)he didn't consciously work to replace the loss of magnesium that occurs with exercise over time. Add this to the fact that a 1996 study reported, "Since the turn of the century, there has been a steady and progressive decline of dietary magnesium intake to where much of the Western World population is ingesting less than an optimum RDA. Geographic regions low in soil and water magnesium demonstrate increased cardiovascular morbidity and mortality."[11]

A research paper from France notes that a marginal primary magnesium deficit affects 15-20% of the population, and another French study showed that 77% of women and 72% of men had dietary magnesium intakes lower than recommended dietary allowances.[2]

There are other symptoms of magnesium deficit that might alert one to a problem. Common conditions such

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as migraines, attention deficit disorder (ADD), fibromyalgia, asthma, allergies, and type-2 diabetes as well as MVP, have been linked to magnesium deficiency.[12]

In seeking to evaluate the magnesium status of an athlete, one should request or the doctor should order an erythrocyte Mg level since a number of studies have found normal levels of serum magnesium in patients with MVP but a significantly lower erythrocyte Mg level in those same subjects.[3]

Most runners depend on electrolyte replacement products to replace the fluid and electrolytes lost during exercise. A good sports drink should contain carbohydrate and sodium which maximizes fluid absorption and helps to provide muscles with energy. It should also provide potassium, chloride, and magnesium, all lost in sweat.[9,13] A review of seven commercially available sports drinks showed only three contained any magnesium at all. Gatorade and Powerade, products often supplied during races and given to runners at water stations contain no magnesium at all. It was found that Accelerade has the highest amount of Mg at 120 mg per serving.[14]

It should be noted that the RDA for magnesium is 420 mg/day (males) and 320 mg/day (females).[15] Endurance activity and training will increase this. Sources of magnesium include meats, and for heart-healthy sources, try steamed or broiled halibut and mackerel, rice bran, nuts, seeds, and green leafy vegetables such as spinach and Swiss chard.[16] Supplementation of magnesium, if not done on a daily basis, may be helpful for a week to 10 days before and after an event to support the increased demand of this activity.

While mitral valve prolapse is a commonly occurring condition, it appears that it presents with, and/or may be caused by, a chronic magnesium deficiency. People participating in endurance sports should consider having their erythrocyte magnesium level tested a couple times a year as prevention of any of the diseases associated with Mg deficiency, including MVP. Medical consultants to events and race directors should seek electrolyte replacement products for their events that include magnesium and the other electrolytes in proper ratios. Athletes should consider magnesium supplementation as a part of their regular nutritional program and be aware of the products they use before, during, and after training or actual events.

About the Author

Terry J. Nelson, DC, DABCI, graduated from Park University with a BS in Nutrition and Cleveland Chiropractic College in 1987. He received his Diplomate degree from the American Board of Chiropractic Internists in 1997. He served as a member of the Kansas Chiropractic Association's committee on education 1989-1991. He currently is in private practice in Independence, Missouri. He has been a chiropractic physician for the Kansas City Wizards professional soccer team since 2003. He has completed 25 marathons in 21 states and is currently on a quest to run a marathon in every state and Washington, DC.

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Neutrophils – A Potent Source of Immune Enhancement

by Rachel Olivier, MS, ND, PhD

Introduction

Neutrophils, one of the three types of granulocytic white blood cells (leukocytes), are the hallmark of acute inflammation.[1] They serve as key components in the defense against infection and are the most abundant of white blood cells, accounting for ~60% of all leukocytes. They are primarily associated with acute bacterial inflammation, and their reaction time is immediate, typically within one hour of tissue injury. Neutrophils are phagocytes, capable of ingesting microorganisms or particles. Each phagocytic event results in the formation of a phagosome into which reactive oxygen species and hydrolytic enzymes are secreted. Due to the consumption of oxygen during this event, a "respiratory bust" ensues. This "respiratory bust" in turn activates the enzyme NADPH oxidase, resulting in the production of large amounts of superoxide. The superoxide anion is subsequently converted to hypochlorous acid (HOCl) via the enzyme myeloperoxidate, which is presumed to result in the elimination of the phagocytized bacteria.[2]

The other two classes of granular leukocytes are the eosinophils and basophils. Eosinophils are activated during allergic diseases, infections, or other medical conditions, and elevated levels are associated with an allergic response or parasitic infestation. They contain coarse, cytoplasmic granules of uniform size and make up 1-3% of the total circulating leukocytes. Basophils have a nuclei size similar to eosinophils, however, when activated, they degranulate and release cellular components, including histamine and proteoglycans, stored within the granules, along with proteolytic enzymes.[2] They represent approximately 0.01-0.3% of the circulating leukocytes.

Interestingly, neutrophil activity has also been correlated with coronary artery disease. Särndahl, *et al*., reported a significant reduction in neutrophillic ROS production in patients with stable coronary artery disease, noting a 30% ±17 mean reduction, compared to matched controls. It was also noted that as a consequence of non-receptor mediated response, a significantly lower neutrophillic ROS production ensued (a reduction of 21% ±12).[3] A separate study also noted a reduced capacity in the ability to up-regulate CD11b cells as well as to produce H_2O_2, both functional aspects of neutrophil activation, compared to healthy controls.[4]

All leukocytes have a minimal life span, and thus a high turnover rate. As such, they are extremely vulnerable to mineral, vitamin, and antioxidant deficiencies. The complex interplay between multiple cell types and intercellular messengers makes nutritional status both subtle and far-reaching, particularly when it involves immune activation.

Minerals Associated with Neutrophil Activation

Chronic low-grade type infections, associated with neutrophil influx, are characteristic of a number of disease processes, ranging from periodontal infections and asthma to cardiovascular disease, pulmonary complications, and rheumatoid arthritis. Certain minerals play important roles in supporting optimal immune function. Zinc, copper, calcium, and phosphorous are particularly important mineral participants in this role.

***Zinc*:** As an essential cofactor for over 70 enzymes, zinc is a vital component in immunocompetence. It is particularly important for highly proliferating cells including those of the immune system. Overt signs of zinc deficiency are numerous and have been well documented. They include atrophy of the thymus, spleen, and lymph nodes; decreased or delayed hypersensitivity response and allograft rejection; lowered production of B- and T-lymphocytes; decreased natural killer cell activity; decreased phagocytosis; and dcreased thymic hormone activity.[5,6] A deficiency in zinc results in a rapid and extensive effect on the immune system[7] including decreased function of monocytes and macrophages, decreased phagocytosis of the neutrophil granulocytes, and decreased cytotoxicity of natural killer cells.[8] Additionally, the number and activity of NK cells have shown to be dependent upon the level of serum zinc.[9] Zinc malabsorption is evidenced by poor wound healing as well as an increased susceptibility to infections. Short periods of zinc supplementation have shown to substantially improve immune defenses, particularly in children, the elderly, and individuals with certain diseases including chronic gastrointestinal disorders.[10]

Copper: Anemia and neutropenia are two known hematological manifestations of copper deficiency. The phagocytic capacity of the neutrophil is markedly modified in copper insufficiency.[11] Neurological manifestations of copper deficiency have also been observed, a typical presentation being myelopathy.[12] Deficiencies in copper, in conjunction with other vitamin and mineral deficiencies, have been correlated with a loss of thymic cellularity, which results in a diminished differentiation of T-lymphocytes. Subsequently, a maturational defect

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in T-lymphocytes ensues, observed by a decrease in both total T-cells (T3 and rosette-forming T-cells), and in T4 helper/inducer cells. In mice deficient in copper, iron, and zinc, cytotoxic T-lymphocyte (CTL) activity is impaired. *In vitro* studies have also reported a reduced number and function of T-cells with experimental deficiencies in copper, zinc, iron, vitamin E, and vitamin A.[13]

Calcium: Calcium mobilization plays a critical role in the activation of cytokine gene expression in helper T-cells, as it is an integral part of calcineurin, the calcium-dependent phosphatase, which is essential for the activation of cytokine gene expression in helper T-cells. Calcineurin activation is required for lytic granule exocytosis in cytotoxic T-lymphocytes.[14] Intracellular calcium signals (Ca^{2+}) also play an essential role in the signaling of Interleukin-8 (IL-8), which is an important constituent in neutrophil activation.[15]

Phosphorous: Cellular phosphorous is closely linked to calcium, typically referenced as being between the ratio of 1:2 and 2:1, calcium to phosphorous. Phosphorous is an essential constituent of nucleic acids, ATP, and phospholipids. Studies have demonstrated an association of the phospholipid phosphatidylinositol with the activated neutrophil.[16] In animal studies the effect of dietary phosphorous on inflammation has also been demonstrated. As an example, in a study with pigs, the effect on cellular and humoral immune response was assessed when associated with an inflammatory challenge. A linear increase in the average daily gain ($P<0.02$) was associated with an increase in dietary phosphorous, which was correlated to enhanced cell-mediated immune response with a corresponding reduction in humoral response.[17]

Vitamins Associated with Neutrophil Activation

Vitamin A: Vitamin A provides immunosupport via its action on cellular immunity in response to challenges, as well as by means of its role in the support of mucosal cell surfaces.[18] It also aids in maintaining the integrity of lymphatic tissues and the level of antibodies, especially that of secretory IgA. Antibody production is effected by hypovitaminosis A, evidenced by a 55% reduction in NK cell activity ($P\leq0.05$) in animals presented with an immune challenge. Vitamin A repletion restored function either partially or completely. Additionally, when spleen cells were assessed, the deficiency in vitamin A was correlated to significantly less Interferon production ($P\leq0.05$), theoretically implicating a decreased immune response and an increased susceptibility to disease.[19] In pediatric patients with irritable bowel disease (IBD), low vitamin A status (<20 mcg/dL) was determined to be a common occurrence (16% of the population), which was correlated to the severity of the disease.[20] In adults with IBD, in addition to other deficiencies a 26% inadequacy in vitamin A was observed.[21] Deficiency, however, appears to be only partially alleviated by the consumption of dark green, leafy vegetables,[22] which has been correlated to a low bioavailability of vitamin A in fruits and vegetables.[23] Natural mixed carotenoids containing alpha and beta carotene, lutein, and zeaxanthin seem to be more readily absorbed and to be more effective antioxidants than synthetic (all trans) beta carotene.[24]

Vitamin C: Vitamin C is a potent water-soluble antioxidant and functions as an active electron donor and acceptor.[25] It dynamically participates in immune support as it acts as both an anti-inflammatory mediator and an immunomodulator. The neutrophil is a known concentrator of vitamin C, increasing its intracellular concentration as much as 10-fold upon activation.[26,27] Neutrophils have also been designated as ascorbate recyclers, having the capability to enhance their intracellular vitamin C concentration as much as 30-fold in the presence of microorganisms, which was correlated to extracellular ascorbate concentration.[28] Ascorbic acid (2 g/day for 5 days) supplementation has been demonstrated to significantly increase the cellular ascorbic acid content of both granulocytes and platelets.[29] Conversely, with a deficiency in ascorbate, a defect in the clearance and apoptosis of macrophages has been observed, which was correlated to macrophage recognition inability, implicated as a "novel and important function for vitamin C in inflammatory cells." The mechanism ascribed was the upregulation of the hypoxia-inducible factor-1α (HIF-1α).[30] This mechanism has also been attributed to the depletion of ascorbate by nickel(II) and cobalt(II).[312]

Vitamin E: Vitamin E acts as a potent oxygen free-radical scavenger and has shown to be protective against injury to the gut mucosal.[32,33] It is also the major membrane and lipid antioxidant of the body. In healthy individuals, short-term vitamin E supplementation was shown to improve immune responsiveness, as evidenced by a decrease in lipid-peroxidation products, including PGE2.[34] Supplemental vitamin E has also been associated with a reduced incidence of respiratory tract infections in the elderly,[35] and has shown to significantly reduce both the incidence and number of common colds in the elderly.[36] Animals deficient in vitamin E were shown to have a 90-fold depletion in alpha-tocopherol, which was correlated to a significant decline in antioxidants, as well as to the accumulation of lipid peroxidation products, both of which were as-

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sociated with a greater incidence of inflammation.[37]

Nucleotides and Immune Activation

RNA: Nucleotides, along with their metabolites, are important to many bodily processes and have documented efficiency in optimizing function. Nucleotides are considered essential for both cell-mediated immunity and T-lymphocyte function.[38,39] The need for dietary nucleotides is particularly evident in times where there is a high physiological demand such as rapid growth, metabolic stress, recovery from a major surgery or trauma, or with inadequate liver function.[40] An adequate supply of nucleotides in the form of purines and pyrimidines, which comprise RNA and DNA, allows for rapid cell proliferation and protein synthesis. In tissue culture studies coculturing nucleotides with specific antigens were demonstrated to have an influence on both immune cell growth and cytokine secretion.[41] Other studies have indicated that cellular immunity is significantly depressed when animals are maintained on a nucleotide-free diet.[42] When reversed, a nucleotide-supplemented diet was shown to upregulate Th1 immune response via the enhancement of IL-12 production with a noted corresponding suppression of antigen-specific IgE response. This was correlated to a significantly higher production of antigen-specific interferon-γ by spleen cells.[43]

Botanicals Associated with Neutrophil Activation

***Maitake mushroom (Grifola frondosa*):** Maitake, an edible mushroom, is a source of complex carbohydrates (glucans), polysaccharides, and minerals. A standardized beta-glucan polysaccharide (beta-1,6 glucan and beta 1,3 glucan) from Maitake, termed the D-fraction, has been extensively studied. In these studies administration has demonstrated immunomodulatory effects, including enhanced humoral immunity, and increased production in nitric oxide, interleukin (IL) 10,[44] and IL-12, which resulted in enhanced cytotoxicity of NK cells.[45] Others have noted an increased NK cell activity with Maitake intake.[46] In a separate study animals given a 20% Maitake-fortified diet were observed to have an altered lipid metabolism, which was attributed to both inhibition of lipid accumulation and deterrence of lipid elevation.[47] These observations correlated with previous studies, which also noted a beneficial effect of Maitake on lipid metabolism.[48,49] Maitake has also shown favorable outcomes in animals studies of hypertension[47,50] and diabetes mellitus.[51-53] In one animal study administration of Maitake, along with vancomycin, resulted in macrophage activation and a 2.7-fold increase in the production of IL-1β. Enhanced bactericidal activity of splenic T-cells was also observed, denoting a 2.6-fold increase in activity with Maitake intake, as compared to non-treated cells.[54] Both observations indicate Maitake's potent action on immunocompetent cells.

Chrysanthemum morifolium: Although traditionally used as a beverage component, specifically in tea, the flower portion of Chrysanthemum has documented medicinal benefits. The flower contains flavonoids, amino acids, vitamins and trace elements, as well as caffeoylquinic acids,[55] luteolin and apigenin, the latter two being ascribed as the primary bioactive components.[56] Evidence of the beneficial attributes of *Chrysanthemum morifolium* (*Cm*) was demonstrated in one *in vitro* study, in which cardioprotective effects were observed in isolated rat heart following ischemia/anoxia and reperfusion/reoxygenation. *Cm* was shown to have a protective effect on the ventricular myocytes by virtue of its attenuation of the reduction of left ventricular pressure and coronary flow caused by ischemia/reperfusion.[57] The Chinese literature documents *Cm* for prevention of sore throat and promoting fever reduction when drunk as a tea.[58] The Chinese *Materia Medica* also indicates its effectiveness against *Staphlococcus aureus*, B-hemolytic *Streptococcus* and *Shigella sonnei*.[59]

Loquat (Eriobotrya japonica): In an animal study administration of *Eriobotrya japonica* (*Ej*) was demonstrated to exert a significant hypoglycemic effect, as evidenced by a lowered blood glucose level in both normal and/or alloxan-diabetic mice.[60] *Ej* is known to contain triterpene acids, and in a separate study the inflammatory response of experimentally induced chronic bronchitis was investigated. Animals given *Ej* were noted to have a significantly decreased level of inflammatory cytokines, including TNF-alpha, IL-1, NF-kB, PGE2, and leukotriene B4 {LTB(4)} expression, as compared to the control group. The investigators concluded that *Ej* "inhibited NF-kB activation" and "led to down-regulation of TNF-alpha, IL-1, PGE2, and LTB(4) expression" in a dose-dependent manner,[61] thus demonstrating immunosupportive properties.

Dyer's-Woad (Isatis indigotica): *Isatis* has noted antibacterial actions, demonstrating effectiveness against strains of staphylococci, pneumococci, and meningococci. It is also considered an effective agent against viruses including influenza. Its actions are described as antipyretic and anti-inflammatory, and it demonstrates choleretic actions.[62]

Prickley Ash (Zanthoxylum americanum): In traditional Chinese medicine, *Zanthoxylum* is utilized to increase blood flow and to promote the circulation of

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qi.[62] Its actions are considered warming and stimulating, thus benefiting circulation.[63] The native Indians of North America have long utilized it for rheumatism and toothaches (odontalgic).[64] The bark is considered an irritant and demonstrates anti-rheumatic properties.[65] Both the roots and bark have been utilized as a tonic in debilitating conditions of the stomach and digestive organs.[66]

Thyme (Thymus vulgaris): Thyme's medicinal properties are associated with its actions as a bronchial antispasmodic, an antibacterial agent, and an expectorant. The chief components of thyme are thymol (20-55%) and carvacrol, along with other minor ingredients.[67] Carvacrol has demonstrated effectiveness as both an antimicrobial[68] and an antifungal agent.[69]

Mullein (Verbascum thapsus): Verbascum's use is correlated to its effectiveness as an expectorant, in stimulating the expulsion of phlegm, and in association with reducing mucus formation. Its classical use is in the management of tracheitis and bronchitis.[64] The leaves and flowers are claimed to be anodyne, anti-inflammatory, antiseptic, antispasmodic, astringent, demulcent, diuretic, emollient, expectorant, and vulnerary.[66,70-76] Extracts contain various derivates, including arabinogalactans, iridoids (catalpol derivatives), saponins (verbasosaponin), flavonoids (kaempferol, luteolin, rutin, apigenin), and phenolic acids (caffeic acid, ferulic acid). In folk medicine Verbascum has been utilized in supporting wound healing, bronchial function, and immune responses, perhaps as a function of the antioxidant activity of the flavonoids and phenolic acids.[77]

Reed Herb (Phragmites communis): Common compounds isolated from *Phragmites* include tricine, luteolin, chrysoeriol, rutin, and isoquercitrin.[67] The root properties are noted as being beneficial in asthma, as an aid for nausea and vomiting (antiemetic), as an aid in reducing fevers, as a cough suppressant (antitussive), as a cleansing agent, as a diuretic, as a fever reducer (febrifuge), as an aid in the prevention of stone formation (lithontripic agent), as an aid in increasing the flow of saliva (sialogogue), as a stomach toner (stomachic), and as a sedative.[78-81] It is traditionally used for diarrhea, fevers, cough, vomiting, coughs with phlegm, urinary tract infections, and food poisoning, especially from sea foods.[81,82]

Barberry (Berberis vulgaris): *Berberis* is a source of vitamin C and isoquinoline alkaloids including berberine, berbamine, and oxyacanthin. It is documented to have antipyretic and cholagogue effects,[67] and the root bark has been shown to aid in normalizing blood pressure. Other properties attributed to the bark include antiseptic, astringent, cholagogue, hepatic, purgative, stomachic and tonic qualities.[66,73,76,83-86] Berberine has also shown effectiveness as anti-diarrheal agent.[87,88]

Oregon Grape Root (Mahonia aquifolium): The chemical components of *Mahonia aquifolium* (*Ma*) consist of isoquinolin and isoquinoline derivates including berberine, berbamine, and oxyacanthine.[67] The traditional use of *Ma* by North American Indian tribes was for the treatment of appetite loss and debility.[64] Its uses have included treating gastritis, general digestive weakness, and catarrhal problems as well as stimulating kidney and gallbladder function.[64,82] Its immunosupportive properties have been attributed to its synergistic antibacterial, anti-inflammatory, and bile-stimulating characteristics.[89] Earlier studies demonstrated that *Ma* exhibited a strong ability to quench the superoxide anion radical, contrary to a strong radical scavenging ability. Recently, *Ma* was demonstrated to have an inhibitory effect on lipoxygenase, indicating its potential role in modulating inflammatory processes.[90]

Summary

Regulation of the neutrophil is critical for immunocompetence. A dynamic balance in neutrophillic sequestration exists, that being between the bone marrow and hepatic sequestration,[91] implicating a complex system of neutrophillic regulation. Supporting neutrophillic activation and regulation with an appropriate nutritional regimen affords a healthy immune system, particularly important in the elderly and those with compromised immune systems. Thus, support of neutrophil activation in these patients, as well as in those with cardiovascular diseases, affords reduced illness and a healthy immune system. The mentioned components serve to provide both general immune support and the support of immune response.

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The Importance of Leptin in Managing Patients with Dysglycemia & Metabolic Syndrome

by Kevin K. Bodling, DC, DACBN

Nearly one-fourth of all Americans age 20 or older are obese, meaning that they are 30 or more pounds over their ideal weight. Obesity is linked to five of the 10 leading causes of death in the US, including diabetes. Nationwide, costs of obesity are estimated at nearly $100 billion. Over the past several years there have been numerous research papers correlating leptin and obesity. However, few people, including health care professionals, realize the importance of leptin in regulating blood sugar in the body and why it is important to test for leptin when treating patients that have blood sugar regulation disorders such as type 2 diabetes and metabolic syndrome.

Abdominal fat stores are really the key in the development of type 2 diabetes and CVD (cardiovascular disease), two of the major symptoms of metabolic syndrome. In the US, the prevalence of metabolic syndrome is 6.7% for 20- to 30-year-olds but rises to 43% between the ages of 60 and 70. In medicine, it is common practice to run tests such as fasting glucose, insulin, or hemoglobin A1c to assess these conditions, but serum leptin may prove to be a powerful tool in the early detection of metabolic syndrome and serve as a guide in helping individuals at risk of developing this condition to transition their metabolism from a sugar-burning metabolism to a fat-burning metabolism. By increasing consumption and supplementation of healthy fats like olive oil and fish oil, L-carnitine, and CLA, restricting the consumption of simple and refined carbohydrates, and exercising, these individuals will actually burn fat and subsequently reduce the burden on the pancreas, liver, and other organs.

Leptin is a 16-kDa protein hormone produced by white adipose tissue. Smaller amounts of leptin are also secreted by cells in the epithelium of the stomach, brown adipose tissue, placenta (syncytiotrophoblasts), ovaries, skeletal muscle, stomach (lower part of fundic glands), mammary epithelial cells, bone marrow, pituitary and liver. Prior to leptin's discovery, adipose tissue was simply viewed as an energy storage reserve. After it was discovered that adipocytes produced hormones like leptin, fat became an endocrine organ like the ovaries, thyroid, pancreas, and pituitary, influencing the rest of the body, especially the brain. Leptin tells the brain what to do about life's two main biological goals: eating and reproduction.

One of the main roles of leptin is to help control appetite. When it was first discovered, it was found that humans and mice with defects in the gene that codes for leptin would become morbidly obese due to lack of CNS stimulation by leptin, which would normally cause suppression of appetite and help control energy expenditure. When these obese mice were given exogenous leptin, they would lose weight. Ongoing research indicates that leptin not only plays an important role in the body's response to food deprivation but is also involved in a diverse array of physiological functions including angiogenesis, hematopoiesis, immunity, bone formation, sexual development, reproduction, and blood sugar regulation. Studies have shown that humans and mice with genetic absence of leptin do not complete puberty, and increased leptin levels lead to early puberty. Leptin levels also affect fertility in females and the development of a normal pregnancy via excretion of leptin by the placenta in the second and third trimesters. Other studies show that leptin also plays a profound role in inflammation, cardiovascular disease, diabetes, osteoporosis, and all diseases of aging, and perhaps aging itself. There is also evidence that leptin plays a role in hyperemesis gravidarum (severe morning sickness), polycystic ovary syndrome, and fetal lung maturation.

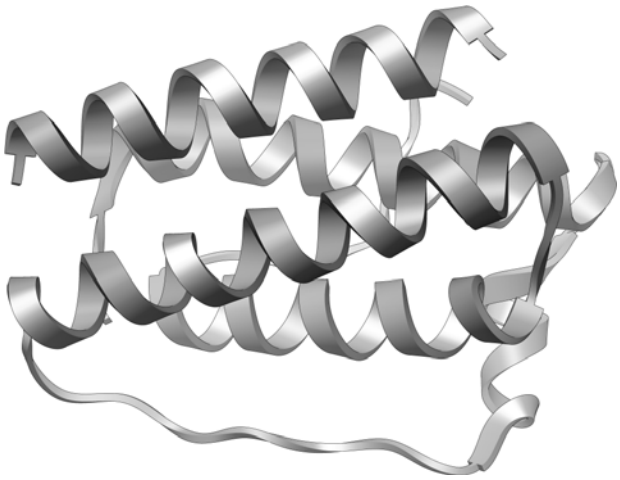
In regards to blood sugar regulation and metabolism, insulin and leptin work synergistically to control the quality and rate of one's metabolism through the nervous system control. Insulin works mostly at the individual cellular level, signaling the cell whether to burn or store fat and whether to utilize that energy for maintenance and repair or reproduction. Leptin then communicates with the brain via the hypothalamus about how much energy (fat) the body has stored, and whether it needs more, should burn some, and whether it is an advantageous time for the body to reproduce. The proposed reference ranges for serum leptin based on current research are:

- Adult Males: 1.2-9.5 ng/mL
- Adult Females: 4.1-25.0 ng/mL

Increasing leptin levels in the blood correlate with increasing body fat. Therefore, obese individuals have the highest levels of leptin, suggesting that these individuals are insensitive to leptin ("leptin resistant") as opposed to suffering from leptin deficiency. Leptin resistance is caused by the same general mechanism as "insulin resistance." As the hormone concentration level in the blood

Figure 1

Leptin



becomes elevated, the corresponding receptor cells become resistant to the hormone over time, thereby requiring an even higher concentration of hormone to produce the desired effect. In the case of leptin, as fat storage increases, more leptin is produced, which over time causes the receptor cells in the hypothalamus and other target tissues to become "resistant" to it. The effect is a reduction in the feeling of satiation after meals, similar to the effect caused by low leptin levels. Therefore, these individuals feel like they are hungry all the time so they eat more and gain more body fat perpetuating this cycle. One proposed mechanism of leptin resistance is the desensitization or down-regulation of the signaling form of the leptin receptor (Ob/Rb) and the induction of a protein called SOCS3, which inhibits leptin signaling. Both Ob-Rb and SOCS3 can be detected in blood monocytes.

In lean individuals leptin, along with adiponectin, is pivotal in preventing ectopic lipid accumulation, which is the accumulation of fat outside the usual fat stores (i.e., in places other than adipose tissue). A drop in adiponectin coupled with leptin resistance leads to ectopic lipid accumulation in various tissues and organs, especially in the abdomen. When this occurs in muscle, it leads to insulin insensitivity (a relative inability of insulin to facilitate the disposal of glucose in tissues). Insulin insensitivity is the first step towards the development of both type 2 diabetes and heart disease. Lipid accumulation in pancreatic beta cells, the site of insulin production, contributes to the development of type 2 diabetes and in cardiomyocytes contributes to cardiovascular disease. Furthermore, in obesity the release of growth hormone declines, exacerbating the decline normally seen with aging and perpetuating obesity through the loss of the hormone's muscle-building and fat-burning effects.

Therefore, the key to interrupting ectopic lipid accumulation and the resulting dysglycemia and metabolic syndrome is to re-sensitize leptin receptors and lower the leptin levels in the blood by transitioning the metabolism to a fat-burning metabolism via omega-3 fatty acids, CLA, L-carnitine, lowering simple and eliminating refined carbohydrates, and exercising in adequate amounts.

Since its discovery in 1994, leptin has radically changed the way science looks at fat, nutrition, and metabolism. Now it's time for medicine to catch up and start looking at leptin when assessing these patients and stop promoting a "low fat" diet to combat obesity and blood sugar dysregulation.

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ABSTRACTS OF INTEREST

Submitted by Emerson Ecologics

Do Many IBS Patients Really Have Celiac Disease?

By Steve Austin, ND

Design & Participants: Non-randomized intervention trial of 145 patients with diarrhea-predominant irritable bowel syndrome (IBS), 74 patients with celiac disease, and 57 patients with active inflammatory bowel disease (IBD)

Intervention: Gluten-free diet in the subset of patients with IBS lasting six months

Outcome Measures: Baseline celiac disease-associated IgA and IgG antibodies against gliadin and tissue-transglutaminase, and HLA-DQA1*0501/DQB*0201 expression were measured. The same indices were measured again in the IBS patients following the six-month intervention as were before-and-after clinical symptoms.

Key Findings: Over half of the celiac patients who had been avoiding gluten and almost all of the celiac patients who had not been avoiding gluten had elevated levels of IgG (though not IgA) antibodies. More interestingly, 37% of the IBS patients also tested positive for specific IgG levels, and HLA-DQ2 expression was found in 39% of the IBS patients. In contrast, these numbers were only 18% and 23% respectively in IBD patients.

A majority (60%) of the IBS patients who were positive for HLA-DQ2 and gliadin-specific IgG became symptom-free as a result of the gluten-free diet. Even 12% of the IBS patients who tested negative for HLA-DQ2 and gliadin-specific IgG became symptom-free as a result of the gluten-free diet. Arguably this subgroup may have had a simple wheat allergy or food sensitivity.

Practice Implications: This work is a follow-up to investigations by the same research team, confirming and extending their previous findings (*Ann NY Acad Sci*, 1998; 859:280-4; *Gastroenterol*, 2007; 2001:121:1329-38). Celiac disease patients often, (and by definition, diarrhea-predominant IBS patients always) suffer with diarrhea. These researchers suggest that sensitivity to gluten might create symptoms in the absence of villous atrophy. Indeed, in the diarrhea-predominant IBS patients who did not have classic celiac disease, evidence of reactions against gliadin were far higher than would normally be expected — a finding supported by the much lower numbers that appeared in the IBD control group.

The findings of this report should alter the standard work-up for IBS. When patients report predominantly symptoms of diarrhea, it's time to test for anti-gliadin antibodies and possibly to conduct HLA testing. Positive findings demand an extended gluten-free therapeutic trial. The data from this new report suggest that it will take only a handful of such patients for the doctor and a patient to arrive at a definitive and treatable diagnosis. Avoiding gluten is hard work for the patient. Many patients with significant symptoms are likely to be compliant if doing so will render them symptom-free.

Wahnschaffe U, Schulzke J-D, *et al*. "Predictors of clinical response to gluten-free diet in patients diagnosed with diarrhea-predominant irritable bowel syndrome." *Clin Gastroenterol Hepatol*, 2007; 5:844-50.

Combined Supplements Improve Visual Acuity in Subjects with Dry Macular Degeneration

By Steve Austin, N.D.

Design & Participants: This was a controlled, double blind (though not randomized) intervention trial. The subjects included 37 adults with age-related macular degeneration (AMD). The control was a constructed cohort of similar patients from a previous report matched for both inclusion and exclusion criteria.

Study Medication & Dosage: Study subjects received retinol (10,000 IU/d), beta carotene (28,640 IU/d), vitamin C (452 mg/d), vitamin E (200 IU/d), zinc (56 mg/d), copper (1.6 mg/d), taurine (400 mg), EPA (180 mg/d), DHA (120 mg/d), lutein (8 mg/d), and zeaxanthin 400 mcg/d). The control subjects had also received vitamin C (400 mg/d), vitamin E (200 IU), zinc (40 mg), and beta carotene (300 IU/d), but none of the other supplements.

Primary Outcome Measures: Changes from baseline

in visual function were measured using a variety of standard research tools (e.g., Best-Corrected Visual Acuity [BCVA] via the Early Treatment Diabetic Retinopathy Study [ETDRS] chart, contrast sensitivity, and retinal imaging).

Key Findings: 77% of subjects receiving the full complement of supplements demonstrated stabilization or improvement of BCVA at six months. These same subjects saw small improvements in visual acuity that achieved statistical significance ($p<0.05$). As expected, mean visual acuity declined in the control group.

Practice Implications: Conventional medicine has little to offer many patients with this all-too-common disease (incidence: >15,000,000 Americans >60 years of age), now the leading cause of irreversible blindness in the elderly. Progressive deterioration is the norm, though the rate of deterioration can vary significantly. Dry AMD is the most common form. Previous research has suggested the possibility that a wide variety of nutritional supplements help patients with AMD, such as lutein, fish oil, zinc, and antioxidants, or some combination thereof. This trial combines most of these supplements at easily attainable dose levels. The outcome — a halting of progression and the beginnings of a reversal — give health care practitioners a potential treatment plan from which to get started.

Interestingly, a standard control group was not part of the trial design. This was not due to the cost, but rather because the independent review board determined that evidence supporting nutritional supplementation is now so strong that the standard of care demands some nutritional supplementation.

Cangemi FE. "TOZAL study: An open case control study of an oral antioxidant and omega-3 supplement for dry AMD." *BMC* Ophthalmol, 2007; 7:3-12.

Vitamin D Supplements Reduce Mortality in a Meta-Analysis of 18 Randomized Trials

By Steve Austin, N.D.

Design & Participants: This is a meta-analysis of 18 randomized trials of which 13 were blinded or partially blinded. It includes 57,311 combined subjects from the 18 trials, of whom 4,777 died during a mean follow-up of 5.7 years

Study Medication & Dosage: Vitamin D, mostly as cholecalciferol (D_3) at 300-2,000 IU/day (mean dose: 528 IU/day), mostly from oral supplementation, with two trials using injectable vitamin D

Primary Outcome Measure: Mortality data
Key Findings: The risk of dying during the course of follow-up declined a statistically significant 7% in those assigned to vitamin D supplementation (95% CI, 0.87-0.99). An 8% reduction was found in trials lasting at least three years.

Practice Implications: Gone are the days in which irradiated ergosterol (D_2) and D_3 are considered interchangeable, with D_3 coming out the clear winner in terms of bioavailability. Gone are the days when 400 IU/day was considered an adequate supplemental dose, increasingly being replaced with calls for doses in the 1,000-2,000 IU/day range. Most importantly, gone are the days in which vitamin D is viewed as simply a way to affect calcium metabolism and bone health.

Evidence showing immune-enhancing and anti-cancer actions have been accompanied by new data proving the existence of extra-renal bioconversion from semi-activated $25(OH)D_3$ to fully-activated $1,25(OH)_2D_3$. These new findings paired with the fact that vitamin D receptors exist in many parts of the body outside of bone have excited the research community, and rightly so. The consequences are of potentially great importance, and one way to gauge those consequences is through measurement of the one end-point no one can argue with — mortality.

Perhaps the worst way to gauge a drop in mortality, particularly cancer mortality, is through relatively short-term trials, as was the case with most of the trials that were studied in this meta-analysis. Yet despite this severe limitation, a significant reduction in mortality was reported. In an accompanying editorial, noted Harvard researcher Edward Giovannucci reminds us, "If vitamin D had an additional effect on the development of chronic diseases, which tend to have long latencies, these studies would have underestimated the total benefit of vitamin D supplementation. For cancer, there is some evidence for an influence of vitamin D on both incidence and survival." He goes on to remind us that 800 IU/day is insufficient to achieve even the bottom end of optimal serum $25(OH)D_3$ levels, yet most of the subjects in these trials received far lower doses. He concludes by wondering, "Would even a greater reduction in mortality accrue than that suggested in this meta-analysis if intakes of vitamin D were higher, if compliance was improved, if higher levels of 25-hydroxyvitamin D were attained, and if the duration of supplementation was longer?"

(Continued on next page)

Unfortunately, the meta-analysis did not separate data on the basis of cause of death. Nonetheless, we have reason to expect that despite the relatively short follow-up, much of the reduction may well have been due to a drop in cancer-related deaths.

Arguably — at least for Americans — vitamin D has rapidly become the single most important component in multivitamins (with the possible exception of folic acid for women who could become pregnant). Vitamin D has also become the vitamin for which doses typically found in multis is the most inadequate. This deficit is likely to change rapidly.

Autier P and Gandini S. "Vitamin D supplementation and total mortality — a meta-analysis of randomized controlled trials." *Arch Intern Med*, 2007; 167:1730-7.

Acne Found Treatable With Low-Glycemic Index Diet

By Steve Austin, ND

Design & Participants: Single-blind intervention trial of 43 men with acne

Study Medication & Dosage: The intervention consisted of a 12-week diet that included 25% protein and 45% low-glycemic index (GI) carbohydrates (CHO) or a control diet emphasizing high-CHO without regard to GI. The intervention diet replaced high-GI foods (e.g., potatoes and refined CHO) with lean high-protein foods (e.g., fish and fowl) and with low-GI/high-CHO foods (e.g., pasta, beans, and fruit).

Primary Outcome Measure: Lesion counts

Key Findings: Lesion counts declined 24% in those assigned to the low-GI diet versus a decline of 12% while on the control diet (P=0.03). Body weight decreased 6.4 pounds versus 1.1 pounds on the control diet (P<0.001). Body mass index (BMI) declined 0.9 kg/m^2 on the low-GI diet versus no decline in the control group (P=0.001). Insulin sensitivity also improved during the low-GI diet alone (P<0.03).

Practice Implications: Acne has appeared for the first time in populations shortly after they are initially exposed to Western diets, causing epidemiologists to suspect that poor diet is involved in the etiology. Years ago, however, when researchers attempted to pin the problem onto isolated foods and failed to do so, the idea was dropped. Nonetheless, a difference this writer has observed between the apparent acne prevalence seen in teenagers outside of convenience stores and those seen outside of health food stores has been striking.

These researchers decided to study the effects of glycemic load on young men because high-GI foods cause surges in serum insulin, which in turn could exacerbate acne by increasing both androgen bioavailability and free levels of insulin-like growth factor I.

Although the results were reasonably impressive, the small print provides hope for even better results with a more extended intervention. The decline in the total lesion count during the third month was just as impressive as it was during the second, leaving us little room to expect that the full potential of the dietary intervention had been explored in just 12 weeks.

The findings of this report suggest that one of the natural treatments for acne should clearly include nutrition. While it may seem reasonable to assume that a switch from a "bad" diet to a "good" one is the key (because most low-GI foods are healthful and many high-GI foods are not), keeping one's eye on GI seems potentially important, given the link between GI and androgen load on one hand and between androgen load and acne on the other.

Smith RN, Mann NJ, *et al*. "A low-glycemic-load diet improves symptoms in acne vulgaris patients: a randomized controlled trial." *Am J Clin Nutr*, 2007; 86:107-15.

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EDITOR'S DESK *continued from page 151*

and, except for the ferritin, his laboratory values had returned to normal. The specialist reported that he had suggested the patient discontinue the Bio TCP and Lipo-Key due to their "very high elements of iron." The letter also stated that vitamin C "increases the absorption of iron."

My follow-up letter to the specialist, and carbon copy to the doctor was as follows:

"Dear Doctor:

"As you may recall, I referred Mr. P to you due to his continually elevated ferritin. However, his iron stores continue to be well within normal range. As you are no doubt aware, an elevated ferritin is most often due to liver overload, an inflammatory process, or hemochromatosis.

"As you can see by the blood chemistries, his liver enzymes were elevated out of "clinical range," suggesting some sort of hepatitis or fat congested liver cells. The liver was palpated swollen and tender.

"As your letter stated, since our management of Mr. P's initial health concern, his condition has continually and satisfactorily improved. Due to the fact that he reported that hemochromatosis has been diagnosed in his family, it is known to be familial, and his continually elevated ferritin, I was a little concerned he may have inherited the condition. This was the purpose for our referral.

"According to *Guyton's Physiology*, the liver has certain basic functions, including bile formation and protein synthesis, without which we could survive only a very few days. By considering the history and blood chemistries, it is obvious Mr. P had a fat congested liver, greatly contributing to his health problems. Bile is essential for efficient uptake of oily substances, including the fat soluble vitamins A, D, E and K. It is reported that vitamin C is responsible for the first step in converting cholesterol to bile acids. Vitamin C deficiency has been shown to cause cholesterol to accumulate in the liver and blood resulting in increased gall stones and elevated cholesterol levels in the blood.

"By your letter, I see you suggested Mr. P discontinue two of the nutrients responsible for his improvement, Lipo-Key and Bio TCP, due to your assumption that they contain iron. In fact, none of the nutrients prescribed contain *any* iron.

"Bio TCP is a source of whole beet concentrate, which has proven to reduce bile viscosity. The ingredients found in this particular product are: taurine (an organic acid that is a major constituent of bile); pancrelipase (a combination of lipase, protease, and amylase, three enzymes essential for digestion of fats, proteins, and sugars); superoxide dismutase (an important antioxidant in defense of nearly all cells exposed to oxygen); catalyse (an enzyme found in living organisms, catalyzing decomposition of hydrogen peroxide to water and oxygen); cellulose (often referred to as dietary fiber); stearic acid (a saturated fat used for stability and has been shown to have little effect on cholesterol); and magnesium state (often used as a filling agent in medical tablets and capsules). So you can readily see Bio TCP has no iron or protomorphogens.

"Lipo-Key has three nutrients essential for liver health — inositol, choline, and methonine. It has several B vitamins (B1, B2, B3, B6, and B12), in addition to the glandular protomorphogens, liver concentrate, and desiccated liver. Glandular protomorphogens are commonly used to stimulate function. Liver glandulars do not enhance iron absorption.

"In view of Mr. P's continued health improvement, in conjunction with his blood chemistries and the knowledge of the nutrients, the prescribed supplements cannot be described as anything but beneficial. I strongly recommend to Mr. P that he continue his nutritional support."

I also carbon copied the letter to my patient. He has since returned for follow-up testing and reports feeling well. ◆

LEGACY *continued from page 153*

also wrong, in that biological ineptness will ensue. Even 100% nerve supply is only a part of the whole picture. In actuality, a person presenting with optimal health achieved through proper nutritional intake, a healthy genetic expression, daily exercise, personal satisfaction in life, etc., would with little doubt be in need of a chiropractic or medical physician for no more than routine scorekeeping examinations and coaching. Thus, the necessary paradigm shift here is in our society's understanding that, optimally, a health care provider is best used to steer us clear of disease rather than sitting in the waiting rooms of our lives until there is a crisis.

By no means am I intending to imply that the practice of medicine is limited to the prescription of antibiotics or that chiropractic is limited to treating neuromusculoskeletal lesions. As their names imply, medicine and chiropractic have separate foundations (i.e. medicinal and chiropractic therapies, respectively) but, ultimately, the same end goal. The end goal is optimal health for those we have the privilege to serve. Let us not wait until the founding fathers die before freeing ourselves from antiquated dogma. Find what works, make sure it works, and then add to it; re-tool a good idea with another. Never take away from what works. Always add to it. ◆

SARCOPENIA *continued from page 167*

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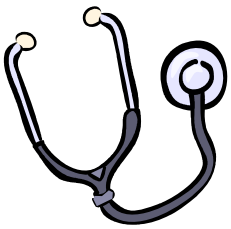
Message from the ACA CDID

by Dr. Robert A. Duca
Clinical Message Board Moderator

New Clinical Message Board

The council on Diagnosis and Internal Disorders of the ACA has established a clinical message board for DABCI's, DABCI students and practitioners of functional medicine. We invite all interested parties to participate. Our clinical message board will be in a new Yahoo! Group format.

Topics of discussion will include:



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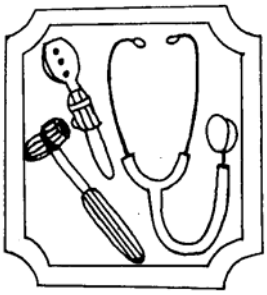

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