## CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>CALENDAR OF EVENTS</td>
<td>154</td>
</tr>
<tr>
<td>FROM THE EDITOR’S DESK</td>
<td>155</td>
</tr>
<tr>
<td>Jack Kessinger, DC, ND, DABCI</td>
<td></td>
</tr>
<tr>
<td>THE LEGACY CONTINUES</td>
<td>157</td>
</tr>
<tr>
<td>A. Jay Kessinger IV, DC, ND, DABCI</td>
<td></td>
</tr>
<tr>
<td>NUTRITIONAL CONSIDERATIONS IN THE MANAGEMENT OF ATTENTION DEFICIT HYPERACTIVITY</td>
<td>159</td>
</tr>
<tr>
<td>Robert A. Duca, Jr. DC, MS, DABCI, DACBN, DACBSP</td>
<td></td>
</tr>
<tr>
<td>SOUND OFF</td>
<td>172</td>
</tr>
<tr>
<td>Forest Mapes, DC</td>
<td></td>
</tr>
<tr>
<td>WHAT IS FAT SOLUBLE THIAMINE?</td>
<td>174</td>
</tr>
<tr>
<td>Derrick Lonsdale, MD</td>
<td></td>
</tr>
<tr>
<td>CHiroprACTIC MANAGEMENT OF CHRONIC HYPTERTENSION: COMPREHENSIVE DIFFERENTIAL DIAGNOSIS</td>
<td>181</td>
</tr>
<tr>
<td>Alex Vasquez, DC, ND, DO</td>
<td></td>
</tr>
<tr>
<td>ABSTRACTS OF INTEREST</td>
<td>191</td>
</tr>
<tr>
<td>DABCI{s AND WHERE THEY ARE</td>
<td>199</td>
</tr>
</tbody>
</table>

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VOL. 17, NO. 4  ·  ISSN 1529-4722  ·  DECEMBER 2010
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Clint Publications
720 Oak Knoll
Rolla, MO 65401
Telephone: (573) 341-8448
Fax: (573) 341-8494
E-mail: virginia@drkessinger.com

www.clinpublications.com

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

News items and/or letters pertaining to natural health care are welcome. The editorial staff reserves the right to edit and/or reject all material received. Letters to the editor may be condensed in order to fit the allotted space. An address and telephone number where the author may be reached during normal business hours should also be included for verification purposes. Deadline for article submission is the 5th of the month preceding publication.

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December 4 - 5, 2010  Session # 17 (Los Angeles, CA)
Immunology & Allergy Part 1
Instructor: Jack Kessinger, DC DABCI

December 4 - 5, 2010  Session # 12 (Hartford, CT)
Chronic Degenerative Disease
Instructor: Ben Bowers, DC DABCI

December 4 - 5, 2010  Session # 6 (Denver, CO)
Multi-Channel Blood Chemistry, CBC, Thyroid
Instructor: Bill Kleber, DC DABCI

December 11 - 12, 2010  Session # 13 (Kansas City, MO)
Pediatrics
Instructor: Cindy Howard, DC DABCI

January 8 - 9, 2011  Session # 13 (Hartford, CT)
Pediatrics
Instructor: Cindy Howard, DC DABCI

January 8 - 9, 2011  Session # 7 (Denver, CO)
Additional Blood Tests, Tumor Markers
Instructor: Bill Kleber, DC DABCI

January 15 - 16, 2011  Session #14 (Kansas City, MO)
Spirometry and Pulmonary Disease
Instructor: Ben Bowers, DC DABCI

January 22 - 23, 2011  Session # 18 (Los Angeles, CA)
Allergy Part 2, Management of the Hypertensive Patient
Instructor: Ben Bowers, DC DABCI

January 29 - 30, 2011  Session # 22 (Portland, OR)
Facts of Neoplastic Process & Examining the Cancer Pt
Instructor: Bill Kleber, DC DABCI

February 5 - 6, 2011  Session # 14 (Hartford, CT)
Spirometry and Pulmonary Disease
Instructor: Ben Bowers, DC DABCI

February 5 - 6, 2011  Session # 8 (Denver, CO)
Blood Interpretation Workshop
Instructor: Bill Kleber, DC DABCI

February 12 - 13, 2011  Session # 19 (Los Angeles, CA)
Common Diseases Affecting the Arterial System
Instructor: Jack Kessinger, DC DABCI

February 19 - 20, 2011  Session # 15 (Kansas City, MO)
Geriatrics
Instructor: Jack Kessinger, DC DABCI

February 26 - 27, 2011  Session # 23 (Portland, OR)
Malignant Diseases, AIDS, & Their Management
Instructor: Bill Kleber, DC DABCI

March 5 - 6, 2011  Session # 15 (Hartford, CT)
Geriatrics
Instructor: Jack Kessinger, DC DABCI

March 5 - 6, 2011  Session # 9 (Denver, CO)
Cardiovascular Disease: Prevention/Diagnosis/ Management
Instructor: Bill Kleber, DC DABCI

March 5 - 6, 2011  Session # 16 (Kansas City, MO)
Urinary Disorders and Hair Biopsy Assessment
Instructor: Ben Bowers, DC DABCI

March 12 - 13, 2011  Session # 20 (Los Angeles, CA)
Evaluating Vascular & Venous Disorders by Instrumentation
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The cold and flu season is just arriving, accompanied by the annual pharmaceutical intimidations to “be sure and get vaccinated.” This year an additional flu, the swine flu (aka H1N1), virus is reported. April marks the 1-year anniversary since the swine flu virus (H1N1) was first recognized. In a press conference on April 01, 2010, Dr. Anne Schuchat, director of CDC’s National Center for Immunizations and Respiratory disease announced, “the virus is still circulating, and people continue to get sick.”

The announcements strongly suggest that vaccination alone will provide total protection against this annual threat. According to the Centers for Disease Control (CDC), 5% to 20% of the U.S. population contacts the flu annually. It’s also true that more than 200,000 of those individuals are hospitalized annually for flu complications, and approximately 36,000 people die of flu-related complications.

Influenza is an extremely contagious viral disease that occurs most frequently in winter and early spring. It is well recognized that those at highest risk for the flu, and its complications, include individuals over fifty, children between six months and five years of age, people with heart or lung disease, pregnant women and those with a compromised immune system.

Flu complications can range from mild to life-threatening. Symptoms range all the way from coughing and sneezing, headaches, sinusitis, ear infections, sore and aching muscles, dehydration, to worsening of chronic and debilitating health problems. These include congestive heart failure, asthma and diabetes.

While preliminary reports show that approved swine flu vaccines have a low incidence of serious side effects, I continue to read the research that the flu shots still contain mercury or aluminum. How can that lead to a healthy future? We have been recommending a homeopathic preparation to our patients who are worried about contracting the flu bug.

No medications are effective for preventing colds or flu. However, there are several natural therapies that are known to be beneficial for prevention, as well as for their therapeutic value. It is well recognized that a healthy immune system is essential to protect from foreign invaders, including the influenza virus. Natural therapies include ensuring adequate rest, consuming a healthy diet, maintaining regular exercise, and proper nutritional supplementation. Doctors of chiropractic also promote the positive benefits of a healthy and un-impaired nervous system necessary for supporting overall health, including our immune system.

In addition to the above mentioned natural therapies ensuring protection from the flu virus, there are several well documented nutrients that are effective against the flu virus.

Monolauric acid (Lauric acid) works by melting away the fatty camouflage effectively exposing the virus to the immune system. Lauric acid is basically non-toxic, which gives it a distinct advantage over modern pharmaceuticals that are typically used to fight viruses. Mother’s milk provides numerous benefits for infants, including a rich source of monolauric acid, essential for providing a natural, non-toxic, antiviral agent that possesses a wide range protection against fungus and viruses. Unlike many medications, monolauric acid has no reported unwanted side effects.

Since antibiotics are totally ineffective for viral conditions we, as physicians, should consider using lauric acid for all those conditions. It is bad medicine to continue prescribing antibiotics for many childhood conditions (ear infections, colds, etc) that would respond quickly with a natural approach of lauric acid. I have averted many tube implants in ears of children by simply recommending NO DAIRY in their diet and a round of lauric acid.

Dr. Jack Kessinger and Dr. Jay Kessinger have a local weekly radio program called “A Healthy Concept” every Tuesday morning from 9:30-10. The programs are available on their website www.drkessinger.com or by going directly to www.drkessinger.com/radio.html.
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Preventive health care, true to its definition, is what the professional health care consumers want and are willing to pay for. Pitfalls in all endeavors are unfortunate and can be devastating; however, their effects can be minimized if not thwarted altogether with appropriate planning. Just as one prepares, with much careful thought and professional insight, for their financial future, most modern intellectual free thinkers would rather minimize if not avoid future health pitfalls by being taught to take responsibility for their own destiny. They don’t want to have one more person or group of persons to dictate their choices, including when and in what condition they will spend “the golden years” of their lives.

I practice in a college/university town. Many students that I have the privilege to serve have had enough foresight in their educational planning that by the time they reach senior 5 (or whatever it takes to finally reach that last semester before a lifetime of work), their class load is minimal with no hard classes. This is planning ahead. All of our patients should be taught to be long range planners. Becoming older is inevitable and unavoidable if they live long enough; however, being sick and disabled in our senior years is not a given. It’s not even natural. It is unfortunately all too often normal, but being sick in those golden years is not natural. As is the colloquial saying of today, it’s best to die while sliding sideways into home base.

If you were a football coach, you wouldn’t want to wait until the last two minutes of the last quarter of the last game of the season to check the scoreboard. You’d keep yourself apprised of the situation every play within every quarter of every game for the whole season long with a keen eye for identifying and eliminating any problems and implementing any solutions along the way in order to attain the best possible season. How is it so different in health than it is scholastically, monetarily, or athletically. The goals should be parallel, in that success in whatever endeavor we set our sights on is the ultimate optimum.

Genetics play a role in our well being; however, for the majority, the genetic role is minor. I like to tell my patients that heredity is 85-90% habit and only 10–15% genetic, so rather than spend your efforts on things you can’t change, let us investigate the things you can change, and then monitor the results.

We, as professional natural health care providers, have the daunting task of re-educating our patients. They have, for generations been taught to trust their doctors because they know best. They are fully aware of how drugs will interact in their patients bodies.

We have to teach our patients that preventive health care is not treating a disease early. Preventive health care is preventing a disease from developing. Medications are toxic and all have side effects. Our patients need to be taught to investigate their own treatment options, understand and keep copies of their own diagnostic tests.

Everyone that we have the privilege to serve needs to be enabled to live their lives as active, vibrant, hopeful, and happy as possible.

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Nutritional Considerations in the Management of Attention Deficit Hyperactivity

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

Attention deficit hyperactivity disorder (ADHD) is the term used to describe children and adults who are inattentive, impulsive, and hyperactive. The cause is uncertain and is thought to be multifactorial. This paper will attempt to provide a broad review of the available literature on nutritional and environmental aspects of the epidemiology, etiology, prevention, and treatment of ADHD.

Attention deficit hyperactivity disorder is a developmental disability with a childhood onset of symptoms that usually results in a chronic and persistent pattern of impairment in learning, and in school, social, and daily adaptive functioning. In adulthood, ADHD manifests via maladaptions in behavior, work, social life, and marital relationships. Typically, ADHD is characterized by deficits in sustained attention and resistance to distraction. Impulsive behavior with a lack of inhibition are also cardinal features of the disorder. A percentage of these individuals exhibit restlessness and hyperkinetic activity.

In many ways, ADHD reflects an exaggeration of what is considered normal behavior. Individuals with ADHD demonstrate either too much or not enough of what is conventionally expected in certain settings.

All of these characteristics can be observed in children, adolescents, and adults. The majority of adolescents and adults with ADHD acknowledge having great difficulty in focusing, meeting deadlines, following tasks through to completion and sustaining effort and motivation, particularly with tasks that are considered boring or irrelevant. Additionally, ADHD individuals that have been surveyed report being easily sidetracked, having frequent forgetfulness, displaying inconsistency in work and school performance, a tendency to being disorganized, failing to plan ahead, and having mood swings. Issues related to anger, difficulties in social relationships, and marriage strife have also been reported. ADHD in childhood and adolescence is associated with an increased likelihood of cigarette smoking and substance abuse later in life.

The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition–Text Revision (DSM-IV-TR) diagnostic criteria for ADHD are made up of nine inattentive and nine hyperactive/impulsive symptoms. In addition to demonstration of a minimum of six symptoms of either inattention or hyperactivity/impulsivity a child or adult must have evidence of functional impairment in two settings. Although a citation is made in the DSM-IV-TR that persons with ADHD may demonstrate variability in IQ, the diagnostic criteria reflect behavioral rather than cognitive or neuropsychological manifestations. Some researchers believe that there is emerging research that suggests that cognitive processes could be added to the DSM-IV diagnostic criteria for ADHD.

Conservative estimates indicate that 3%–7% of school-age children are affected with ADHD. Other studies estimate the prevalence as high as 7%–12%. Approximately 60%–85% of children diagnosed with ADHD continue to have symptoms in adolescence, and 60% experience impairments into and continuing through adulthood.

Children with ADHD often have comorbid diagnoses such as learning disorders, oppositional defiant disorder (ODD), anxiety, and depression. About 40% to 80% of autistic spectrum children also have ADHD as a comorbid disorder. The high rate of comorbid conditions makes for a complex process in the differential diagnosis and treatment of ADHD. Throughout the mid-1980s and into the 1990s, many children and adolescents affected with ADHD went without referral, undiagnosed, and untreated, and rarely were neuro-psychological deficits proposed as explanatory factors of the condition or related impairments.

Nutritional management of ADHD is one aspect that has been relatively neglected to date. Nutritional factors such as food additives, refined sugars, food sensitivities/allergies, and fatty acid deficiencies have all been linked to ADHD. There is increasing evidence that many children with behavioral problems are sensitive to one or...
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more food components that can negatively impact their behavior.

ETIOLOGY AND PATHOGENESIS

Information linking nutrients with ADHD has been explored and examined carefully in the last 15 years. An increased understanding and application of biochemistry and its relation to the standard American diet (SAD) has permitted researchers to consider multiple etiologies in the development of ADHD. It has become more evident that nutrition plays a major role in the genesis of ADHD. The evidence suggests that the physiological and anatomic brain abnormalities in ADHD are not preprogrammed, or inevitable, but are instead an expression of the genetic vulnerabilities to noted risk factors, particularly malnutrition. Family studies and twin studies suggest a genetic component in the disorder. It is possible that early childbearing and low academic achievement are themselves symptomatic of ADHD in mothers, with genetic transmission contributing to the diagnosis of ADHD in their children.

Certain individuals may have genetically-imposed, heightened requirements for certain nutrients. If such individuals are not provided with optimal nutrition, they may be significantly more vulnerable to the physiological and anatomical brain abnormalities associated with ADHD and its symptoms.

Other studies of children with ADHD support the hypothesis that impaired catecholamine regulation is involved in the development of ADHD. The ratio of omega-3 to omega-6 polyunsaturated fatty acids affects transmission in nerve networks controlled by serotonin and catecholamines. Impaired catecholamine regulation of brain areas involved in ADHD may reduce the energy available to brain cells.

Choline is one of the building blocks of acetylcholine and an important neurotransmitter involved in memory. Glutamate and glutamine are amino acids involved in the production of GABA (gamma-aminobutyric acid), a neurotransmitter that inhibits certain nerve impulses and may affect hyperactivity. Choline supplementation is theoretically supposed to be beneficial for diminished memory and learning. Unfortunately oral supplementation of choline is hindered by its un-pleasant taste. As a result, children prefer the administration of dimethylenonoethanol (DMAE). DMAE is a supplement that increases brain levels of choline. DMAE may speed up production of acetylcholine in the brain and it has been used in ADHD treatment for reduced attention span, learning and reading problems, hyperactivity, and poor coordination.

Phospholipids have also been shown to have a role in behavior. Phospholipids include any of the various phosphorous-containing lipids that are composed of fatty acids, a phosphate group, and a simple organic molecule. Phosphatidylserine is involved in the production of neurotransmitters via its role in nerve cell synaptic membrane transmission. A small study of 21 ADHD cases showed improvement in more than 90% of subjects with supplementation of 200-300 mg per day of phosphatidylserine over a 4 month period. Phosphatidylcholine, from which the neurotransmitter acetylcholine is derived, appears to have a stimulant effect on the brain and has been used effectively in reducing hyperactivity and improving learning disorders.

Some studies suggest that abnormal genes may interact with environmental culprits to trigger or aggravate ADHD in susceptible individuals. The science of epigenetics holds great promise for a better understanding of these triggers. Among these potential triggers: food additives; food intolerances; sensitivities to chemicals, molds, and fungi; heavy metals such as lead; pollutants such as polychlorinated biphenyls (PCBs); and nutritional deficiencies.

All learning disabilities such as attention deficit hyperactivity disorder, developmental delays, and emotional and behavioral problems are increasing in prevalence and are drawing increasing concern. Interacting genetic, environmental, and social factors are important determinants of childhood brain development and function. Extensive laboratory and clinical studies of several neurodevelopmental toxicants, including lead, mercury, polychlorinated biphenyls, alcohol, and nicotine, demonstrate the unique vulnerability of the developing brain to environmental agents at exposure levels that have no lasting effect in adults. Throughout history, understanding the effects of these toxicants on the developing brain has emerged slowly while generations of children are exposed to unsafe levels. Unfortunately, with few exceptions, neurodevelopmental toxicity data is missing for most industrial chemicals in widespread use, even when populationwide exposures are documented.

Many studies have indicated that childhood lead exposure can reduce IQ and attention and cause hyperactivity at levels below the current 10 g/dL blood lead standard. A U.S. study of 97 children with ADHD and 53 control children reported that
higher blood lead levels were associated with significantly higher levels of ADHD symptoms.\textsuperscript{21}

A large randomized double blind crossover controlled trial was designed to determine whether artificial food colorings and benzoate preservative in the diet of more than 1800 three year old children in the general population could influence hyperactive behavior. The authors of the trial concluded that there existed a general adverse effect of artificial food coloring and benzoate preservatives on the behavior of 3 year old children which was detectable by parents but not by a simple clinic assessment.\textsuperscript{22}

A meta-analysis of 15 double-blind, placebo-controlled studies in patients with ADHD reported that artificial colors such as tartrazine significantly increased ADHD symptoms.\textsuperscript{23} Of the 15 individual studies, 5 showed that food colors were associated with significantly increased ADHD, 8 showed that food colors were associated with nonsignificant increases in ADHD symptoms, and 2 showed that food colors were associated with nonsignificant decreases in ADHD symptoms.\textsuperscript{23} More recent studies suggest that avoiding food dyes and preservatives can improve ADHD symptoms.\textsuperscript{24,25} Food coloring agents potential role in the pathogenesis of ADHD remains controversial.

Besides food colors, other dietary components that may possibly worsen ADHD symptoms include other food preservatives such as nitrates and monosodium glutamate as well as food that naturally contains salicylates (such as almonds, oranges, raspberries, apples, cherries, grapes, peaches, strawberries, cucumbers, plums, and tomatoes).\textsuperscript{26}

A robust study was done to determine whether a standard elimination diet can decrease the ADHD-symptoms in a heterogeneous group of young children with ADHD. Forty children, 36 boys and 4 girls, aged 3-7 (average 4.8 years), who met the DSM-IV-criteria for ADHD, followed their usual diet for two weeks and thereafter for two weeks an elimination diet which permitted only a few foods in the diet (rice, turkey, pear and lettuce). The behavior of the children was evaluated at study entry, after the baseline period, and at the end of the diet. Parents completed the 10-item Conners list, the ADHD Rating Scale, and a physical complaints list. The teachers completed the 10-item Conners list and the ADHD Rating Scale twice, at the beginning and at the end of the diet. The study reported the following. According to the parent-ratings, 25 children (62\%) showed an improvement in behavior of at least 50\% on both the Conners list and the ADHD Rating Scale at the end of the elimination diet. Among the 15 children with both parent and teacher ratings, 10 responded favorably both at home and in school. The authors concluded that an elimination diet can lead to a statistically significant decrease in symptoms in young children with ADHD.\textsuperscript{27}

A large systemic review included identification of suboptimal levels of nutrients and sensitivities to certain foods and food additives. This review presented an overview of all research studies published before April 2008. The review provided an up-to-date account of clinical trials that have been conducted with zinc, iron, magnesium, pycnogenol, omega-3 fatty acids, and food sensitivities. Although the authors of the review acknowledged that further research is required, they concluded that current evidence supported the utilization of nutrition and confirmed the effect of dietary influences on behavior and learning in children with ADHD. The strongest support to date was reported for the use of omega-3s and the presence of behavioral food reactions.\textsuperscript{28}

The effect of refined sugar and food additives on ADHD patients is controversial, with some, but not all, studies showing that sugar and food additives can trigger worsened ADHD symptoms. A review of 16 studies with ADHD children found that sugar challenges were associated with worsened symptoms of inattention and hyperactivity in 4 studies, little change in 11 studies, and improvement in ADHD symptoms in 1 study.\textsuperscript{29} Additional studies, particularly those examining the effects of sugar and sweeteners exclusively, are inconclusive.\textsuperscript{18,30}

A challenge design was used in two separate studies to investigate the effects of sucrose ingestion on the behavior and learning of hyperactive boys. In both studies, 16 boys were admitted to a clinical research center for 3 successive days, on each of which they were given a sucrose-free diet. On subsequent days the boys were given a challenge drink of either sucrose 1.75 gm/kg or a placebo (aspartame in equivalent sweetness). The results of both studies revealed no differences between the boys' performance on the two challenge days. These findings undermine the hypothesis that sucrose plays a major role in accounting for the inappropriate behavior of hyperactive boys. However, a potential weakness of both of these studies was the administration of aspartame in the placebo drink. The active ingredient in aspartame, phenylketonurics, have been implicated in neurologic dysfunction and headache.\textsuperscript{31}

(Continued on page 164)
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Homeonutritional therapy using nutritional and homeopathic formulas provides immediate response and long term benefit.
Some features of ADHD may relate to abnormalities of fatty acid metabolism. Several studies have identified abnormal membrane fatty acids in individuals with ADHD.\textsuperscript{32} Preliminary data suggests that some patients with ADHD have higher rates of oxidative breakdown of omega-3 polyunsaturated fatty acids. This observation provides an additional rationale for supplementation with omega-3 essential fatty acids in the management of ADHD.\textsuperscript{32}

A preliminary study of 50 children showed some improvements in ADHD-like symptoms (compared to placebo) from four months of treatment with a polyunsaturated fatty acid supplement providing a daily dose of 480 mg of DHA, 80 mg of EPA (eicosapentanoic acid), 40 mg of arachidonic acid, 96 mg of GLA (gamma-linoleic acid), and 24 mg of alpha-tocopherol acetate (vitamin E). Areas of improvement included conduct and behavior reported by parents, poor attention as rated by teachers, and rebellious attitudes and acting-out episodes.\textsuperscript{33}

Several studies have found that ADHD patients have significantly lower levels of omega-3 fatty acids in their blood phospholipids and red blood cell membranes.\textsuperscript{22,34} Compelled by the work of others, a group of researchers hypothesized that some children with ADHD have altered fatty acid metabolism. In their study they found that 53 subjects with ADHD had significantly lower concentrations of key fatty acids in the plasma polar lipids (20:4n-6, 20:5n-3, and 22:6n-3) and in red blood cell total lipids (20:4n-6 and 22:4n-6) than did 43 control subjects. A subgroup of 21 subjects with ADHD exhibiting many symptoms of essential fatty acid (EFA) deficiency had significantly lower plasma concentrations of 20:4n-6 and 22:6n-3 than did 32 subjects with ADHD with few EFA-deficiency symptoms. The precise reason for lower fatty acid concentrations in some children with ADHD is not clear.\textsuperscript{35}

A pilot study evaluated the effects of supplementation with polyunsaturated fatty acids (PUFA) on blood fatty acid composition and behavior in children with ADHD symptoms also reporting thirst and skin problems. Fifty children were randomized to treatment groups receiving either a PUFA supplement providing a daily dose of 480 mg DHA, 80 mg EPA, 40 mg arachidonic acid (AA), 96 mg GLA, and 24 mg alpha-tocopherol acetate, or an olive oil placebo for 4 months of double-blind parallel treatment. Supplementation with the PUFA led to a substantial increase in the proportions of EPA, DHA, and alpha-tocopherol in the plasma phospholipids and red blood cell (RBC) total lipids as was also noted in the plasma phospholipid proportions of 18:3n-3 with olive oil supplementation. Significant improvements in multiple outcomes (as rated by parents) were noted in both groups, but a clear benefit from PUFA supplementation for all behaviors characteristic of ADHD was not observed. PUFA supplementation led to a greater number of participants showing improvement in oppositional defiant behavior from a clinical to a nonclinical range compared with olive oil supplementation. Also, significant correlations were observed when comparing the magnitude of change between increasing proportions of EPA in the RBC and decreasing disruptive behavior.\textsuperscript{36}

Double-blind, placebo-controlled studies have reported that supplements of carnitine\textsuperscript{38} and zinc\textsuperscript{39,40} have been helpful in treating ADHD symptoms. Some studies have found that magnesium and vitamin B6 supplements significantly reduce excitability and improve concentration in ADHD children. Magnesium levels in blood plasma and red blood cells may be low in patients with ADHD.\textsuperscript{41} Vitamin B6 was also shown to increase low levels of serotonin in hyperactive children.\textsuperscript{42} Zinc deficiency in children with ADHD is associated with decreased blood levels of fatty acids.\textsuperscript{43}

In a well-controlled, randomized study of 400 children with ADHD, zinc sulfate was statistically superior to placebo in reducing symptoms of hyperactive or impulsive behavior and impaired socialization.\textsuperscript{42}

In a randomized, controlled study, 13 of 24 boys treated with carnitine improved 20-65% in measures of behavior parameters in home and in a school setting. The authors concluded that treatment with carnitine significantly decreased attention problems and aggressive behavior in boys with ADHD.\textsuperscript{44}

Blood levels of iron are often low in ADHD patients. A French study reported that serum ferritin levels averaged only 23 ng/mL in 53 children with ADHD but averaged 44 ng/mL in 27 control children.\textsuperscript{45}

CONVENTIONAL TREATMENT OF ADHD

Conventional medical management promotes the combined use of medical, behavioral, and environmental techniques as the most effective short-term interventions for ADHD. Certainly, extensive literature attests to the benefits of medicine, specifically stimulants, in reducing
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key symptoms of ADHD and thus improving daily functioning across the life span.

It has been demonstrated that cognitive behavioral therapy for ADHD is an effective intervention, reducing symptom severity and impairment, particularly when paired with medication treatment.1

Nutritional management of ADHD is one aspect that has been relatively neglected to date. Typically, a child is followed by a treatment team, consisting of physicians, behavioral specialists, teachers, and parents with little or no regard for the nutritional implications in the etiology, development, or therapeusis of the condition.

Stimulant medication has been the mainstay of treatment for pediatric ADHD. 90% of all children diagnosed with ADHD will receive some form of medication as a treatment regimen, including approximately 70% who will receive a stimulant as a first-line treatment. To date, stimulant medications have been the single most effective pharmaceutical treatment for ADHD. These medications have been proven to reduce the primary symptoms of hyperactivity, impulsivity, and inattention associated with ADHD.3 The drugs in this stimulant class include methylphenidate (Ritalin), d-methylphenidate (Focalin), amphetamine, (Dexedrine), and mixed amphetamine salts (Adderall). Stimulants are sympathomimetic agents that are similar in structure to endogenous catecholamines. They work by enhancing transmission of dopamine and norepinephrine in the brain.

Risperidone and aripiprazole are two atypical neuroleptic agents used in the treatment of complicated ADHD. Neuroleptics are tranquilizing drugs that exert a calming effect on a individual. There is some data supporting their use in the treatment of severe ADHD or its comorbidities of aggression, ODD, and conduct disorder. Unfortunately, the majority of this data comes from short-term, open-label, and anecdotal reports.4 Common side effects of these Risperdone and aripiprazole medications include sedation and weight gain.4 The totality of the adverse effects of the use of these medications are numerous and have yet to be fully elucidated. The most commonly reported adverse events of the stimulant medications are loss of appetite and insomnia. Other side effects can include affective flattening, mood disturbances (irritability, depressive symptoms, mania/hypomania), headaches, and abdominal pain.4 Ritalin has demonstrated the potential for addiction.46,47

Lethargy, and fatigue are also common side effects. “Rebound” effects are transient deteriorations in behavior that may be seen between doses, as the effect of the medication declines, or following abrupt discontinuation of the medication. Mild increases in pulse and blood pressure have also been consistently documented; however, the clinical significance of these varies depending on the cardiovascular history of the child and his/her family.

Long-term effects of the stimulants on growth are also concerns for patients and clinicians. For the majority of patients treated with stimulants into adolescence, decreases in weight and height are seen. The reductions in expected height and weight are relatively small, and they do attenuate with time in the majority of cases. It is also thought that patients with ADHD may potentially have a different growth trajectory than patients without the condition.4 Studies demonstrate that approximately one third of children with ADHD either do not respond to stimulant drugs or cannot tolerate their side effects.48,49 Therefore, a novel nutritional approach may have the potential for better symptom management with less risk. In addition, side effects appear slightly greater and benefits slightly fewer in young children.1

Although stimulant medication has been the cornerstone of treatment for attention deficit hyperactivity disorder (ADHD), numerous nonpharmacological treatments can be employed in combination with medication to assist in the lifelong management of the disorder.50 A comprehensive multimodal treatment combining education about the disorder, medication, psychotherapy, compensatory behavioral/self-management skills, nutritional therapy, supplemental therapy, and environmental considerations are likely to result in the most favorable treatment outcome.

Much of the conventional medical and psychologic community believe that nontraditional treatments such as dietary manipulation, nutrient supplementation, and food and environmental chemical elimination used to permanently alter the symptoms of ADHD have not stood the test of scientific research, and thus, should not be advocated as first-line treatments of choice for children with ADHD. However, they do acknowledge that these approaches can be effective when targeted to specific problems and impairments in the classroom for all students, with or without diagnoses.1

It should be noted that little controlled research has been undertaken on psychosocial treatments of (Continued on next page)
ADHD. The psychologic community remains unable to draw firm scientific conclusions regarding the efficacy of psychologic counseling in the management of ADHD. To date, only one empirical study investigating the usefulness of cognitive therapy in treating adults with ADHD has been published. In the clinical practice setting ADHD treatment options remain largely at the level of anecdotal evidence, clinical experience, common sense, and, in some cases, extrapolations from child interviews.\(^5\)

It is this author’s intent to demonstrate that a nutritional and environmental treatment paradigm is an essential component of the clinical management of ADHD patients. A nutritional and environment approach to the treatment also possesses a superior relative safety index and therapeutic index.

PREVENTION

From a nutritional perspective the process for prevention of ADHD begins early in life. Ninety percent of the total human brain growth occurs in the first three years of life. The nutrients required for brain development must be supplied to support optimal brain health in these early years. However, fewer than 1% of US children and adolescents receive the US recommended daily allowance (RDA) for the five food groups (fruits, vegetables, meat, dairy, and grains), and 16% do not consume the RDA of any of the five food groups, according to a US Department of Agriculture survey of 3,300 participants.\(^5\)

In a controlled study of nearly 500 school children who were randomly assigned to receive daily multi-vitamin/mineral supplements containing 50% of the RDA for four months had a 47% lower rate of antisocial behavior than did those who received placebo.\(^5\)

A study by the USDA on the dietary intakes of school aged children found that the average school aged child in the mid 1990's consumed over six servings of refined grains and only one serving of whole grain. These children consumed almost one and a half servings of soda and just under one serving of fruit flavored drinks. The children in the study ate only one tenth of a serving of dark green leafy vegetables, one serving of other vegetables and less than one and a half servings of fruit. There was no distinction made between fresh fruit and vegetable intake and canned, frozen or otherwise processed versions of these foods. Fish and nuts and seeds accounted for only one tenth of a serving each. This type of diet is obviously deficient in essential fatty acids due to insufficient fish, nut and seed intake, zinc and B vitamins due to low intake of whole grains and legumes, minerals due to low intake of vegetables, vitamin C due to low intake of fruit and vegetables, and is very low in fiber. In addition, the six plus servings of grain were likely to be predominantly wheat, the most common grain in American diets. The diets averaged two servings of dairy products daily.\(^5\)

Clearly, based on the research that supports nutrient imbalances as an underlying contributor in the etiology of ADHD, school aged children in the United States are at high risk. Understanding that certain individuals may have genetically imposed, heightened requirements for certain nutrients, when they eat the average American diet, as represented by the USDA study, they become significantly more vulnerable to the manifestation of symptoms of ADHD.

TREATMENT

The clinician trained in nutritional medicine should compile a detailed family and individual health and diet history of the ADHD patient. The nutritional clinician will perform a comprehensive physical examination to assess the patient for physical manifestations of nutritional deficiencies or impaired physical growth and development. Laboratory testing is utilized to elucidate and confirm diagnostic suspicions. Specialized nutritional and toxological testing will assist in the clinician’s laboratory assessment. This testing will help the nutritionist, and the family, tailor the nutritional program for that individual patient.

A nutritional strategy for individuals with ADHD should begin with a balanced diet free of potential allergens and food preservatives as well as high amounts of processed sugars. A diet rich in organically grown whole foods, abundant in mineral and phytonutrients, rich in fresh vegetables and fruits, and plentiful in reliable sources of essential fats is important to optimize the long term success of a nutritional intervention in a comprehensive ADHD treatment program. As been previously discussed, avoidance of nutrient depleting refined foods and sugars, trans fats and chemical additives, colorings and preservatives promises further benefit. The patient should follow a diet low in refined carbohydrate and high in quality protein. Proper hydration is essential for the transport of chemical messengers and nutrients to the brain and to facilitate optimal elimination of waste products.

When considering specific nutritional supplementation in the management of ADHD, omega-3 essential fatty acids...
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The fairly recent advances in understanding the biochemical and neurophysiological roots of ADHD show how supplementation may allow for symptomatic resolution without the adverse side effects of medications, particularly stimulants. Therapeutic dosing of the following nutrients based on age and weight of the patient should be considered. These nutrients are B vitamins, choline, phosphatidylserine, amino acids, iron, acetyl-L-carnitine, zinc, and herbal nutrients that have potential to improve brain circulation. Because of the numerous and potentially adverse consequences of ADHD if left untreated, nutritional supplementation should be started as soon as possible after diagnosis and at the earliest stages of physical and emotional development. The nutrients zinc, magnesium, iron, B vitamins, and essential fatty acids are commonly deficient in the diet of the average American child and should be prescribed as soon as feasible.

Parris M. Kidd, PhD conducted a recent review of the scientific literature regarding nutrient deficiencies and ADHD and concluded without equivocation, "Nutrient deficiencies are quite common in ADHD; supplementation with minerals, the B vitamins, omega-3 and omega-6 essential fatty acids, flavonoids, and the essential phospholipid phosphatidylserine can ameliorate ADHD symptoms."36

Encouraging scientific evidence demonstrates that sedating herbs such as passionflower, valerian, or lemon balm may help control symptoms of hyperactivity in children with ADHD. The mechanism of action of these herbs are well documented and they are safe to administer for long periods of time.37 At present, herbal therapies that improve blood flow, such as ginko biloba, are being investigated and appear especially promising.

CONCLUSION

Attention deficit hyperactivity disorder is caused by many complex factors and requires a multifaceted treatment approach.28 An abundance of research has investigated causes and treatments for attention deficit hyperactivity disorder (ADHD).28

ADHD involves a broad range of genetic, prenatal, social, developmental, nutritional, and environmental factors, and it is unlikely that only 1 single cause will be found for the disorder.44 Multiple treatment modalities are needed to treat patients with ADHD and should include nutritional, environmental, pharmacologic, and psychosocial interventions. Potential negative environmental associations with ADHD are problematic and demand a need for more research. A preventive public health response based upon evidence that establishes the plausibility of harm will require mitigation of exposures to potential neurodevelopmental toxicants.55

The nutritional management of ADHD needs to be conducted with a strategy that approaches each child or adult as uniquely biochemical and a unique psychosocial organism. Their treatment should be designed on the basis of these unique and varying circumstances. The individual response is an important factor for determining the proper approach in treating children with ADHD. In general, dietary modification, with supplementation, plays a central, primary, and the major role in the management of ADHD.56

It is the opinion of this author that an integrated approach in the management of ADHD using diet, nutritional supplements, and detoxification is consistently effective in benefiting individuals with ADHD. Children are far better served by modifying the diet and using nutrients first and turning to pharmaceuticals only as a last resort. Hopefully, continued research and clinical trials will deepen our understanding and enhance our abilities to manage ADHD from a nutritional perspective.

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(Continued on next page)

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42) Bhagavan HN, Coleman M, Coursin DB. The effect of pyridoxine hydrochloride on blood serotonin and pyridoxal (Continued on page 173)
Several years ago Blue Cross Blue Shield of Illinois decided to authorize a few chiropractors to be primary care physicians. This was a plan to see if chiropractors would qualify and eventually, all chiropractors could possibly be approved. Could you do it? If people with pneumonia and idiopathic hypertension, diabetes, infections and all the things that are the mainstay of the typical medical practice started showing up in your office, could you handle it? Would you know what to do? And, vertebral manipulation, by itself, doesn’t always correct some of these things!!

I read a recent report that over 90% of chiropractors are using nutritional supplements. I suspect that many of them are using botanicals and herbal preparations also. Several questions come to my mind. Do you know the difference between a botanical and a drug? Do you know when a botanical is preferred or when a drug would be a better choice?

For the sake of simplicity, let’s call all nutritional supplements, botanicals, herbal preparations, homeopathic remedies, and all other non-drug treatments that chiropractors might use alternative therapies. Can you name at least one alternative therapy that works as an antibiotic, or to lower hypertension treat diabetes, bacterial infections, and viral infections? Were you aware that these things exist? If a patient comes into your office and is in shock, can you determine the blood sugar level immediately and if it is low, give the patient sugar to revive him? Do you have an EKG machine in the office and can you at least screen it to see if there is a possible eminent myocardial infarction? If you answered yes to all of these questions you are probably ready to be a primary care physician.

The most important question is this: Why bother treating patients with alternative medicine when you can refer them to the allopath for drugs? I believe that the answer to that is: allopathic medicine is in a bind; adverse drug reactions are the fourth leading cause of death in this country, according to the AMA. Why would anyone want to refer a patient to something so dangerous?

For those of you that are in the less than 10% of chiropractors that use any supplements in your practice please tell me: 1.) If a patient comes into your office with a vitamin D deficiency, how many vertebral adjustments does it take to correct this deficiency? Are you aware that vitamin D is deficient in almost all of your patients? I suspect that this is true even if you have adjusted them many times. 2.) Can you name a vitamin that is known to be very effective treating tuberculosis and leprosy, yet you as a chiropractor can’t use it because when it is used that way, the medical doctors call it a drug? It is vitamin C, which is much better, safer and less expensive than any drug on the market, yet almost never used for this purpose.

A simple way to limit good health care is to call something medicine when it is not. It might be different if the medical profession was using it for that purpose but they are not going to use it and they are going to try to prevent us from using it too. Who suffers? You do. But your patients suffer more, because they are being prevented from getting the therapy they need. They are being forced to take drugs and subjecting themselves to the fourth leading cause of death. All the while, they are putting up billboards accusing us of causing strokes with cervical manipulation!

Are you aware of an alternative therapy for treating strokes that has been proven to be more effective than the therapy you can get in the ER of your local hospital? It is legal for chiropractors to use it and I know some who are using it in their offices as you read this.

Medical doctors get almost no education in nutrition, chiropractors get a lot more, yet many of us are not using it to the level we could. If you are one of those that would like to expand their practice, here are seven action steps that I recommend.

1. I personally believe that every chiropractor should take the DABCI course. They are giving it in many cities around the country.
2. Learn to read an EKG. “Rapid Interpretation of EKG’S” by Dale Dubin MD is easy and simple.
3. Buy an EKG machine and start doing one on all new patients. It’s like learning to bat; first you pick up

(Continued on next page)
the bat and then start swinging at the ball. Pretty soon you will start getting good.

4. Start examining your patients the way you learned in school. You will become good at everything that you do regularly.

5. Start doing lab work on all new patients. Even if you only do a CBC and blood sugar, you will be surprised at what you will learn. As you keep learning, your practice will start growing.

6. Support those states that are not restricting chiropractors: New Mexico, Missouri and Oklahoma. Petition your state to follow those policies.

7. Remain cognizant of the powers of manipulation and all mind-body therapies. We are still in the early stages of discovering the potentials of manipulation. It is what sets us apart and it is our gift from our forefathers and our gift to pass on to the world.

As wonderful as medicine is, it is not very good at treating chronic disease and promoting wellness. It is good at treating acute trauma and life threatening problems. But qualified chiropractors are in a better position to provide excellent general health care.

About the Author

Dr. Mapes was a hospital corpsman in the US Navy where he received training in Laboratory and Blood Bank technology. He graduated from National College of Chiropractic and has practiced in the Chicago area since 1983. He is married to Adorable Patrico from the Philippines and they have four children. Dr. Mapes completed the DABCI program and his practice is primarily focused on Internal Medicine.

ROBERT DUCA, DC DABCI: continued from page 171


About the Author

Dr. Robert A. Duca, Jr. is a chiropractic internist and nutritionist who has been in private practice for 22 years. Dr. Duca holds an MS degree in human nutrition and is a diplomate of the American Board of Chiropractic Internists, the American Clinical Board of Nutrition, and the American Chiropractic Board of Sports Physicians. Dr. Duca currently serves as an executive board officer of the Council on Diagnosis of Internal Disorders of the American Chiropractic Association.

THE ORIGINAL INTERNIST DECEMBER 2010 173
Thiamine, vitamin B1, is found in food in its free form and its absorption is a very complex process requiring one or more transporters. When it gets into cells it has to go through several reactions that make it into its active forms. At some stage of synthesis a phosphate group is added to make thiamine monophosphate (TMP) whose action(s) is/are still poorly understood. Another phosphate is added to create thiamine diphosphate, or pyrophosphate (TPP). This is the ester that is best known and understood as cofactor to the decarboxylating component of the pyruvic, the branched chain and alpha ketoglutarate complexes and transketolase. Transketolase occurs twice in the hexose monophosphate shunt and since this pathway occurs in erythrocytes it is used to test for biological evidence of thiamine deficiency\(^1\).

This test is by far the most definitive method of ascertaining thiamine deficiency, although laboratories that perform it are hard to find, even though thiamine deficiency is widespread, due largely to the inordinate consumption of sugar in our culture. The first part of the \textit{in vitro} study is to ascertain the concentration of the product of the enzyme, measured in units and reported as transketolase activity (TKA). The second part is reported as the thiamine pyrophosphate effect (TPPE) after the addition of TPP to the reaction. If the TPPE reveals an acceleration of transketolase activity, it is measured as a percentage increase over baseline. Thus it is reported usually in two parts, the TKA and TPPE. Prolonged deficiency severe enough to cause deterioration of the TKA is relatively uncommon, whereas an abnormal TPPE is very common in our experience.

There is yet another component of thiamine, requiring three phosphates to form thiamine triphosphate (TTP), whose actions are still only partly understood. It is known, however, to have a vital action in brain and nervous system. It is in high concentration in the electric organ of the electric eel, \textit{Eletrophorus Electricus}, an adaptation of a synaptosome that depends on acetylcholine as the neurotransmitter.\(^2\) It also has some connection with the opening of chloride channels\(^3\) and has recently been reported to be synthesized from TPP with energy derived from the respiratory chain\(^4\). Thus, any mitochondrial disruption is likely to be associated with deficiency of TTP. At present there is no way of ascertaining TTP deficiency in the clinical laboratory.

Thiamine deficiency is therefore extremely complex and has a major effect on many different aspects of energy metabolism, depending on which of the three esters are affected. In any patient shown to have an abnormal TPPE with or without deterioration in TKA, the symptoms are extremely diverse. The test should always be applied for people that are polysymptomatic and admit to craving for sugar. Its relationship with alcohol is well known. The symptoms generated are mostly restricted to the brain, nervous system and the heart, organs that are affected in beriberi and are known to have the heaviest consumption of oxygen. Thiamine therefore has a vital place in oxidative metabolism. Beriberi is the prototype for dysautonomia in its early stages and degeneration of autonomic nerves and ganglia in its later stages\(^5\). The symptoms are seldom severe enough to make a firm diagnosis of beriberi, although the full blown disease will occasionally be encountered. It is usually missed because it is generally believed that such a classic nutritional disease does not occur in developed countries. For example, an anesthesiologist was admitted to a hospital with an enlarged heart. His medical student son recognized it and the cardiologists in charge of the case rejected the diagnosis as absurd. His diet history started with no breakfast because of “the dry heaves”, anesthetics for about 10 cases, followed by consumption of a large piece of chocolate cake. On return home he was too tired to eat dinner and would go to bed. Eventually the cardiologists treated him too fast with thiamine and he died (Lonsdale D. Unpublished observation). This is an important point because it was long ago noted that beriberi recovered if the blood sugar was normal and usually if it was elevated. Recovery with the administration of thiamine did not occur if there was hypoglycemia.

Thiamine is water soluble and there is virtually no storage in the body so, like all water soluble vitamins, a daily intake is required, the RDA. This has been estimated as about 1 to 1.5 mg/day, although this only applies to biochemically healthy people. Beriberi is accepted as the recognized thiamine deficiency disease and has been a scourge for thousands of years, particularly in Eastern cultures where rice has been the staple diet. In the early years of discovery, however, it was found that several hundred milligrams of thiamine

\textit{(Continued on next page)}
would have to be given in the treatment of beriberi, suggesting that deficiency of the cofactor for a long enough time would cause deterioration in the activity of the corresponding enzyme(s).

This principle of megadoses of vitamins appears to be an important fact in the modern era and our experience has shown repeatedly that a prolonged deficiency of thiamine cannot be addressed by physiological RDA doses in patients with an abnormal thiamine pyrophosphate effect (TPPE).

Magnesium is a cofactor for transketolase and its deficiency is also very common, giving rise to symptoms that are similar to those that arise from thiamine deficiency. Wernicke encephalopathy is a manifestation of both. Mild to moderate deficiency particularly affects the limbic system and gives rise to symptoms that are similar to those from mild hypoxia. Normal mechanisms that control the endocrine and autonomic nervous systems are grossly exaggerated. One of the best known survival reflexes is fight-or-flight and mild oxygen or thiamine deficiency often gives rise to this spontaneously. They are referred to as panic attacks that have been reproduced experimentally by inhalation of carbon dioxide. A very old experiment in human subjects showed that an artificially constructed thiamine deficient diet caused typical symptoms that are generally given the label of psychosomatic disease. The symptoms were abolished by restoring sufficient thiamine. Over many years of clinical study, it has been our experience that patients with emotional and psychosomatic disease can usually be helped by dietary removal of all forms of sugar and supplying vitamin supplements that include thiamine and magnesium in particular.

There is, therefore, a great deal of information that indicates that thiamine is an important therapeutic nutrient, tied inevitably to the huge consumption of sugar in the modern era. Measurement of TKA and TPPE constitutes the best guide to the clinician. It is important, however, to note that simply applying large doses of non caloric nutrients does not work unless dietary discipline is enforced. Recovery is slow and often causes paradoxical worsening of symptoms in the early stages of treatment.

Many different thiamine derivatives were carefully studied by Japanese investigators. Investigation of the active principles in garlic had revealed that thiamine in the garlic bulb was acted on by an enzyme that converted it to a disulfide derivative when the bulb was cut or crushed. They had found that this new substance had a much more powerful biologic effect than water soluble salts of thiamine in animal experiments and they called it allithiamine after the fact that it occurred in many of the allium species of plants. It was eventually synthesized and has become a prescription item in Japan under the trade name of Alamin. It is also sold elsewhere under the name of Fursultiamine. The experimental work quoted in the reference chapter is extremely detailed and of great interest in modern therapy.

These investigators had found that they could synthesize several different disulfide forms and a number of non disulfides (Figures 1 and 2). All of these derivatives are absorbed from the intestine easily so that the circulating blood level of thiamine is

(Continued on page 178)
**Tentative Schedule of Events**

**Friday, March 25, 2011**

6:00 – 7:00 P.M.  Virtual Tours of Diagnostic Clinics & Floor plans

7:00 P.M.  Welcome Reception

**Saturday, March 26, 2011**

8:00 A.M.  Welcome & Introduction ~ Jack Kessinger, DC DABCI

8:05 – 9:00 A.M.  Success Stories & Business Building

Ben Bowers, DC, DABCI & Chris Murray, DC, DABCI

9:00 – 9:45 A.M.  Weight Loss - A Never Ending Revenue Source

Annette Copeland, CNHP & Virginia Kessinger

9:45 – 10:15 A.M.  Food Break

10:15 — 11:00 A.M.  5 Easy Steps to Social Media Marketing

Kelley Kirchner, DC, DABCI

11:00 A.M. — NOON  Internal Marketing

Phil Arnone, DC, DABCI

Noon — 1:30 P.M.  Luncheon

1:30 – 3:30 P.M.  Using the Law to Make Money & Protect Your Assets

Lee Phillips, JD

3:30 – 4:00 P.M.  Food Break

4:00 – 5:00 P.M.  Riding the Wave - Taking advantage of Current Health Trends & Advantages of Private Labeling

Virginia Kessinger & Annette Copeland, CNHP

5:00 – 6:00 P.M.  Managing a Profitable Front Office & Outside Marketing

Annette Copeland, CNHP & Virginia Kessinger

7:00 P.M.  Social Hour & Banquet & Special Entertainment

~ Dr. Jack Kessinger

**Sunday, March 27, 2011**

8:00 A.M.  Introduction ~ Jack Kessinger, DC DABCI

8:05 – 9:30 A.M.  The Importance of Allergy Testing

Michelle Clark, MBA & Veronica Kent, BS

9:30 — 10:00 A.M.  Food Break

10:00 — 11:00 A.M.  Marketing Nutritional Supplements

Ben Bowers, DC, DABCI

11:00 A.M. — NOON  Marketing - Drug Screens DOT Physicals and Paternity Testing
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Marketing & Managing a Diagnostic Practice

March 25-27, 2011

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increased over a similar dose of water soluble salts. The major difference between them is that the disulfides, the most modern of which is TTFD (Fig 1) is hydroyzed on contact with cell membranes and the open ring thiamine passes into the cell without requiring the physiological transport system. The thiazolium ring closes and the cell can build up a high concentration of functional thiamine. Its ability to introduce a closed ring thiamine molecule to the cell through the lipid barrier of the cell is why it has been referred to as “fat soluble”. Strictly speaking, TTFD, like other disulfide derivatives, is soluble in water and can be given intravenously. The physiology is different in the S-acyl derivatives that require an enzyme in liver or kidney to hydrolyze the open ring form. The prosthetic mercaptan group of the disulfides remain outside the cell and their fate has been well studied14-17.

The prosthetic group derived from S-acyl derivatives has not been studied to my knowledge. There has been recent interest in the therapeutic action of benfotiamine (BTMP Fig 2) in diabetic neuropathy18, but it has been shown that it does not cross the blood brain barrier as opposed to TTFD that does19. Several publications exist on the clinical use of TTFD20-23.

Much experimental work was published by the members of the Vitamin B Research Committee of Japan5,13. They found that the disulfides had many effects that were not exhibited by the S-acyl derivatives. Much of their work was done with thiamine propyl disulfide (TPD) and TTFD was eventually synthesized because it eliminated the powerful garlic odor that emanated from humans and animals exposed to TPD. Thus the Takeda Chemical Industries produced TTFD under the trade name of Alinamin (Odorless). It is now a prescription item in Japan and is used also to enrich food substances.

TPD partially protected mice from the lethal effect of cyanide and the toxic effect of carbon tetrachloride on liver13. Its use in humans is extremely wide and Japanese literature records success in treating postoperative ileus, nerve deafness and some inflammatory conditions. Although the concentration of thiamine in plasma was similar with both TPD and BTMP, the intracellular concentration of the disulfide was higher than that induced by the S-acyl derivatives. They concluded that it was the disulfide that made the difference.

Perhaps the greatest potential for thiamine disulfide, best exemplified by TTFD is in brain metabolism. It has been shown that exposure to sulbutiamine improves memory in mice and produced a significant increase in hippocampal sodium-dependent high affinity choline uptake24. It has also shown some benefit in treatment of Alzheimer disease although the dose used by the investigators was only 100 mg/day.25. It showed great benefit in nutritional polyneuropathy26 and there is evidence that thiamine has a central cholinergic effect27. In autopsych studies, the findings demonstrated that the morphological changes in the mamillary bodies due to thiamine deficiency and those due to hypoxia may be identical28.

A case report of a woman with proven thiamine deficiency had hemocentration that suggested an effect produced in the oxygen-chemosensitive sites in brainstem. Treatment with nutrients that included TTFD produced normal hemoglobin and erythrocyte count, disappearance of symptoms and a decrease in TPPE11,29. Increase in erythropoietin appears to be the mechanism30.

It has been hypothesized that functional dysautonomia is a common polysymptomatic condition caused by oxidative stress, particularly in the limbic system controls of the autonomic endocrine axis and that it explains its association with a number of organic diseases. This association has never been explained and most of the information exists as case reports where the dysautonomia is reported as an interesting example of two diseases occurring at the same time in one individual31. Although the most definitive textbook on dysautonomia concentrates on the genetic aspect, there is no discussion on the association with a modern diet that contains foods with an excess of empty calories32. Since acetyl choline is a major neurotransmitter in brain and both branches of the autonomic nervous system depend on it, any interruption of pyruvate into the citric acid cycle would be expected to interfere with its synthesis. Another possibility that was considered over 40 years ago is that thiamine plays a part in acetyl choline release33. A recent article has implicated the role of thiamine in cholinergic neurotransmission via its effect on the metabolism of acetyl-CoA and acetyl-choline34. Recent studies have supported the concept that thiamine deficiency is tied to the ingestion of excess carbohydrate35.

CONCLUSION

There seems to be little doubt that the dietary mayhem of simple sweet carbohydrate ingestion in the modern world carries with it hidden risks that are often categorically refuted by patients to whom the intake of sugar in all its different forms represents an addiction. Even if the patient believes the physician’s advice, often after prolonged and difficult discussion, it may be very hard to discontinue, since the craving is not satisfied by the intake of sweet fresh fruit. It is hypothesized

(Continued on next page)
that it is the hedonistic input of a unilateral sweet stimulus from the tongue to the brain that creates the addictive tendency, for the brain is programmed to receive permutations and combinations of input involving sweet, sour, bitter, salt, astringent and metal that provide the different flavors from organic food. The same thing can be said of the craving for salt that is extremely common in my experience. It can be compared with the fact that the brain is also programmed to receive the input effects of full spectrum white light. Added to this effect is the stimulation of the brain from caffeine whose action is to give an impression of increased energy, whereas it is consuming cellular energy that the patient can often ill afford. I have used TTFD since 1973 under an Independent Investigator License and have treated hundreds of patients. I have never seen the slightest sign of toxic effect and I have good reason to believe that it is a therapeutic agent that is of great consequence in the modern era of dietary mayhem.

Thiamine tetrahydrofurfuryl disulfide has been used under Independent Investigator License IND 11019.

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(Continued on next page)
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Chiropractic Management of Chronic Hypertension: Comprehensive Differential Diagnosis

by: Alex Vasquez, DC, ND, DO

Given the high clinical importance and prevalence of hypertension (HTN) in general clinical practice, Doctors of Chiropractic (DC) should remain aware of its differential diagnosis so that proper patient management can be implemented and so that the underlying causative factors can be addressed when possible. Listed below are many of the more common primary causes of hypertension with a brief sketch of their classic clinical characteristics, including physical examination and laboratory findings. This information is excerpted and updated from the recently published guidelines “Chiropractic Management of Chronic Hypertension” published by Vasquez.

 Major Differential Diagnoses of Chronic Hypertension:

Characteristics of secondary hypertension include therapeutic recalcitrance, onset at an early age (< 30y) or at a more advanced age (>50y), and the typical associated features of the causative disorder, such as hypokalemia with hyperaldosteronism, depression or musculoskeletal pain with hypovitaminosis D, and cold intolerance, bradycardia, and delayed Achilles reflex return with hypothyroidism.

- **Aortic coarctation**: Classic presentation includes upper extremity hypertension with lower extremity hypotension/hypoperfusion/claudication in a child or young adult; secondary activation of the renin-angiotensin system due to renal hypoperfusion exacerbates the HTN and complicates this focal anatomic disorder by adding a systemic neurohormonal component. Aortic coarctation is diagnosed by imaging the aorta with computed tomography (CT), magnetic resonance imaging/angiography (MRI/MRA), or echocardiogram (echo) or transthoracic ultrasound (US). Treatment includes antihypertensive interventions (reviewed in the section on Therapeutic Considerations) to manage the hypertension until surgery corrects the coarctation.

- **Cocaine use**: Cocaine use can cause acute and chronic elevations in blood pressure. Drug cessation is the key to treatment; urine drug testing is appropriate for patients suspected of undisclosed drug use or noncompliance with cessation. In hospital practice, patients presenting with hypertensive disorders, chest pain, and other cardiovascular syndromes are routinely tested for acute (serum drug screen) and chronic (urine drug screen) drug exposure; an impressive number of these tests come back positive even among patients who swear to have never used or not recently used recreational drugs.

- **Cushing’s disease/syndrome**: Excess glucocorticoids whether endogenous or exogenous promote sodium retention directly via their mineralocorticoid effect and by causing hyperinsulinemia via induction of peripheral insulin resistance; both of these pathophysiologic processes contribute to HTN. Useful tests include measurements of serum adrenocorticotropic hormone (ACTH), urinary/salivary cortisol in addition to looking for the clinical characteristics of moon facies, striae, sarcopenia, and abdominal obesity. Treatment is withdrawal of exogenous steroids (if possible) for iatrogenic Cushing’s syndrome, or surgical removal of the ACTH-producing pituitary corticotroph adenoma (classically) in cases of endogenous Cushing’s disease. An additional type of Cushing’s syndrome can result from ectopic ACTH production from tumors such as small cell carcinoma of the lung or a carcinoid tumor.

- **Estrogen, oral contraceptives**: As a group of various hormones with divergent effects, estrogens generally tend to promote sodium and water retention, which promotes volume overload and the development of HTN. For women with “estrogen dominance” due to excess endogenous production or exogenous administration of estrogens, supplementation with pyridoxine 50-250 mg/d (nearly always co-administered with magnesium 600-1,200 mg/d or to bowel tolerance) and/or natural progesterone (rather than a synthetic progestin, since many of these preparations have inherent glucocorticoid/mineralocorticoid activity) can frequently offset the HTN-inducing effects of estrogens. Clinicians desiring a more comprehensive anti-estrogen pro-tocol within a context of practical hormone optimization (“orthoendocrinology”) may find helpful the review in chapter 4 of Integrative Rheumatology.

- **Ethanol**: Excess ethanol consumption raises blood pressure and makes HTN more difficult to treat. Many patients fail to accurately disclose the extent and duration of their alcohol consumption.

- **Hypercalcemia**: Easily diagnosed by routine labor-
ary testing, hypercalcemia may be caused by hyperparathyroidism, malignancy, sarcoidosis, Paget’s disease of bone, or rarely by nutritional excesses of calcium and/or vitamin D. Most hypercalcemia (80-90%) is due to hyperparathyroidism or malignancy; the most common cause of hypercalcemia in the outpatient setting is hyperparathyroidism, while in the hospital setting the most common cause is malignancy, particularly lymphoma/lymphosarcomas, multiple myeloma, and metastatic disease. While other differential diagnoses also need to be considered, some endocrinologist particularly advocate testing 24-h urinary calcium levels as a test for familial hypercalcemic hypercalcemia. When primary hyperparathyroidism is suspected, the serum level of intact parathyroid hormone (iPTH) is tested. When malignancy is suspected (particularly from the finding of an unexplained serum calcium > 13 mg/dL [> 3.25 mmol/L]), patient-centered evaluation is performed, which often includes initial chest radiograph followed by pan-scanning with CT for occult malignancies in the thorax (e.g., lung cancers), abdomen and pelvis (e.g., gastrointestinal tumors).

- **Insulin resistance and hyperinsulinemia:** Insulin promotes renal retention of sodium which leads to water retention and the subsequent volume overload and systemic hypertension which logically follow in sequence. This explains the well proven and replicable benefit of low-carbohydrate diets in treating “idiopathic” HTN in the general population. Elevated or high-normal serum insulin along with “idiopathic” HTN in the general population. Elevated or high-normal serum insulin along with chronic hyperglycemia is most suggestive of insulin resistance; the most effective treatments for insulin resistance are integrative nutritional interventions as detailed in *Chiropractic and Naturopathic Mastery of Common Clinical Disorders*.4

- **Neurogenic hypertension:** In the context of discussing HTN, “neurogenic” was historically interchangeable with “essential”, “primary”, and “idiopathic”; since neurogenic is no longer generally used for this purpose, and because new research advocates the term’s reinstitution, neurogenic hypertension should be exonerated from its previous identification with idiopathy and given revised meaning. For the purposes of this discussion and as detailed later in this chapter, the term “neurogenic hypertension” will mean what its name implies, namely chronic HTN induced principally by the nervous system due to irritation or functional disturbance rather than overt pathology. Given its basis in physiology rather than pathology per se, the term “functional neurogenic hypertension” would serve to further emphasize the functional and therefore largely reversible mechanism of the disorder.

Foci of neurogenic hypertension can reside in the central nervous system (CNS) or peripheral nervous system (PNS). In this text, “central neurogenic hypertension” is used to describe hypertensive states induced by irritation of the central nervous system, in particular at the level of the brainstem (i.e., medulla oblongata in general and the root entry zones [REZ] of cranial nerves 9 and 10 as well as the nucleus tract solitarius [NTS] in particular) as will be reviewed in a following section on surgical interventions for the treatment of medullary neurovascular compression. The first use of the term central neurogenic hypertension of which the current author is aware was published by Reis in a 1981 review, mostly of animal research; in this review, Reis included the hypothesis that irritation of the CNS by either mechanical or neurochemical means could serve as a predisposition or antecedent to the manifest development of clinical HTN. The diagnosis of central neurogenic hypertension is generally based upon 1) MRI/MRA or CT findings of neurovascular compression of the left medulla oblongata in conjunction with 2) reduction in blood pressure following decompressive intervention. “Peripheral neurogenic hypertension” as an entity is more theoretical, less studied, and might be exemplified by irritation of spinal nerve roots and sympathetic ganglia as discussed primarily in the chiropractic6,7,8,9,10 and osteopathic literature.11,12 Functional compromise in general and facilitation13 in particular of the nerve roots and sympathetic ganglia as a potential cause of or contributor to chronic HTN supports the rationale for the use of spinal manipulation and manual medicine for the treatment of HTN and other nonmusculoskeletal disorders. Peripheral neurogenic hypertension may be diagnosed based on clinical/electrographic/vasodynamic evidence of functional PNS compromise/facilitation/irritation and alleviation of HTN following appropriate regional intervention such as manual manipulative treatment of the spine and adjacent neuromusculoskeletal structures applied to effect restoration of proper nervous system function and balance. Central and peripheral types of neurogenic HTN will be discussed in more detail later in this chapter within the context of their surgical and manipulative treatments, respectively.

- **Nonsteroidal anti-inflammatory drugs (NSAIDs):** NSAIDs in general and COX-2 inhibitors (coxibs) in particular reduce endogenous production of vaso-dilating prostacyclin and thus cause pharmacologic/iatrogenic renal artery constriction, which leads to varying degrees of HTN via activation of the renin-angiotensin system. This explains, in part, the inter-
increased cardiovascular mortality due to overutilization of coxibs such as rofecoxib/Vioxx, withdrawn from the US market in 2005 by the US FDA due to its causal role in increasing cardiovascular deaths. Evidence of increased cardiovascular morbidity and mortality secondary to coxib use was widely publicized for several years before rofecoxib/Vioxx and a similar drug valdecoxib/Bextra were belatedly withdrawn from the consumer market, the multiple failures involved in this politicopharmaceutical phenomenon include 1) failure of Merck to act on data showing that it’s popular and profitable new drug was harming and killing an unacceptable proportion of patients who took it, 2) failure of the US FDA to regulate the pharmaceutical Industry, 3) failure of the medical profession as a whole to police itself and call for a ban on the use of this drug before either Merck or the FDA took action. See Eric Topol’s “Failing the public health—rofecoxib, Merck, and the FDA” published in the October 21, 2004 issue of New England Journal of Medicine for authoritative discussion.

- Pheochromocytoma: Exceedingly rare in contrast to the frequency with which it is covered in textbooks and licensing board exams, pheochromocytoma’s classic presentation is episodic HTN, headache, and diaphoresis; it is diagnosed with increased 24-hour urinary catecholamines, metanephrines, and/or vanillylmandelic acid followed by CT/MRI to localize the secreting neuroendocrine tumor. Treatment is surgical excision of the adrenal/extra-adrenal mass.

- Gestational hypertension and preeclampsia: Pregnancy-induced (after week 20 of gestation) hypertension without proteinuria is termed gestation-al hypertension; gestational hypertension with concomitant proteinuria is termed preeclampsia, while the addition of seizures advances the diagnosis to eclampsia. Preeclampsia can accelerate rapidly and cause life-threatening complications for the mother and/or fetus; treatment requires parenteral therapy (intravenous magnesium sulfate for seizure prophylaxis; hydralazine and/or labetolol for HTN control) and/or emergency interventions—namely, delivery. Some evidence suggests that the incidence of preeclampsia can be reduced via increased intake of aspirin, ascorbate, calcium, tocopherol(s), and magnesium, and by pre-pregnancy treatment/cure of obesity, diabetes mellitus, and HTN. Acute HTN of 160 mm Hg systolic or 110 mm Hg diastolic requires urgent treatment; acute-onset HTN can cause stroke at pressures generally tolerated in chronic HTN because in the latter vascular adaptations accommodate higher pressures, while in the former, the cardiovascular system has not had time to adapt, thus leaving the patient particularly vulnerable. Acute-onset HTN from any cause should be treated urgently when pressures approximate or exceed 160-180 mm Hg systolic or 110 mm Hg diastolic, especially but not exclusively if accompanied by complications such as angina (test serum cardiac enzymes), shortness of breath (consider pulmonary edema and auscultate for crackles), vision changes, papilledema, headache/confusion/seizures (which suggest cerebral edema or cerebral vasospasm), proteinuria, or edema of the face, peripheral extremities, or of the general body (anasarca, check for sacral edema and weight gain).

- Primary hyperaldosteronism (Conn’s syndrome): Primary hyperaldosteronism is caused by a unilateral adrenal adenoma or bilateral adrenal hyperplasia. The typical finding is HTN with hypokalemia, occasionally with slight hypernatremia, and diagnosis is by increased urine or serum aldosterone or by the more specific elevated serum aldosterone:renin ratio. Per The Merck Manual, “Initial laboratory testing consists of plasma aldosterone levels and plasma renin activity (PRA). Ideally, tests are done with the patient off of drugs that affect the renin-angiotensin system (e.g., thiazide diuretics, ACE inhibitors, angiotensin antagonists, β-blockers) for 4 to 6 wk. PRA is usually measured in the morning with the patient recumbent. Patients with primary aldosteronism typically have plasma aldosterone > 15 ng/dL (> 0.42 nmol/L) and low levels of PRA, with a ratio of plasma aldosterone (in nanograms/dL) to PRA (in nanograms/mL/h) > 20.” Curative treatment is laparoscopic removal/resection of the hypersecreting adrenal tumor; for patients who are not surgical candidates, drug treatment with an aldosterone-blocking drug (i.e., spironolactone or eplerenone) is used. Pseudohyperaldosteronism can be caused by overconsumption of Glycyrrhiza glabra (licorice) because glycyrrhizin inhibits 11-beta hydroxysteroid dehydrogenase thus preventing cortisol’s inactivation to cortisone in the kidney and thereby potentiating the mineralocorticoid effect of endogenous cortisol. Another cause of pseudohyperaldosteronism is Liddle’s syndrome, a genotropic disorder causing increased sodium reabsorption, characterized by early onset (<35y) HTN with hypokalemia, low urinary sodium levels, and normal serum aldosterone levels.

- Renal artery (renovascular) stenosis: Classically caused by fibromuscular dysplasia in young adult women (<25y) and by atherosclerosis in older adults (>50y), renovascular stenosis is suggested by elevation of creatinine following administration of an ACEi. Diagnosis is by renal ultrasound or contrast arteriography; treatment is with stent placement.
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- **Renal parenchymal disease:** Renal disease can both lead to and result from HTN. Chronic HTN causes renal parenchymal damage, and parenchymal damage (whether due to HTN or another cause such as glomerulonephritis, pyelonephritis, polycystic kidneys, etc) leads to water retention and activation of the renin-angiotensin-aldosterone system, thus promoting a vicious cycle of progressive HTN and renal failure. The clinical picture commonly includes edema, elevated BUN and creatinine, anemia due to insufficient production of erythropoietin, and osteomalacia/osteodystrophy due to hyperphosphatemia, hypocalcemia, and insufficient renal formation of 1,25-dihydroxyvitamin D3. The diagnosis of renal disease is suggested by the finding of elevated BUN and creatinine on routine chemistry/metabolic panel blood tests; the diagnosis is further verified and refined by the use of CT, MRI, or US imaging, followed if necessary by renal biopsy. The Cockcroft-Gault formula has commonly been used for bedside estimation of renal function based on the patient’s age, weight, gender, and serum creatinine (sCr); the formula is provided below in two versions, one using American-favored mg/dL as the unit for sCr, and the other using the international units of micromol/L—note that the later formula employs a different constant value per gender in the numerator of the equation. The Cockcroft-Gault formula estimates creatinine clearance, which in turn is an estimate of the glomerular filtration rate (GFR), a measure of kidney function; thus creatinine clearance and GFR are somewhat interchangeable from a practical clinical perspective. Clinicians should appreciate the importance of the patient’s age in determining GFR; sCr in the upper end of the normal range may indicate renal insufficiency in a patient of advanced age.

- Although the Cockcroft-Gault formula is the best known and longest used formula for the estimation of GFR, currently the best equation for more accurately estimating GFR from serum creatinine is the Modification of Diet in Renal Disease (MDRD) Study equation, which is available on-line at http://www.nkdep.nih.gov/professionals/gfr_calculators/. Finally on this topic, clinicians should be aware of measuring serum cystatin C to assess renal function. Cystatin C is a cysteine protease inhibitor produced by all nucleated cells, and its serum level is not affected by diet or muscle mass (unlike serum creatinine). The normal range for cystatin C when measured by particle-enhanced nephelometric immunoassay (PENIA) is <0.28 mg/L or <0.95 mg/L when measured by other immunologic methods. Cystatin C is a more sensitive indicator of declining renal function than is serum creatinine, and—like elevating serum creatinine or declining GFR (or elevated CRP for that matter)—cystatin C predicts risk and severity of CVD, CHF, and CKD; furthermore, cystatin C is directly involved in the pathogenesis of atherosclerosis.

- **Sleep apnea:** Obstructive sleep apnea (OSA) is a risk factor for HTN, and treatment for OSA with continuous positive airway pressure (C-PAP) can produce modest reductions in BP that are proportionate to the severity of the HTN and compliance with treatment. Diagnosis is generally by history and physical exam confirmed with an overnight sleep study (polysomnography).

- **Systemic sclerosis:** HTN in general and treatment-resistant HTN in particular are seen in systemic sclerosis, a disease in which cardiopulmonary disease (e.g., pulmonary hypertension, congestive heart failure) and renal compromise (e.g., acute renal crisis heralded by nephrogenic hypertension) are the most common causes of death. Abnormalities disclosed on history and physical exam may include Raynaud’s phenomenon, sclerodactyly, mask-like face, telangiectasia, and esophageal dysfunction. Laboratory findings typically include some combination of positive antinuclear antibodies (ANA), anticientromere antibodies, anti-SCL-70 antibodies, and (more rarely) anti-fibrillarin antibodies. Treatment for scleroderma and other common autoimmune disorders is reviewed in *Integrative Rheumatology*.

- **Thyroid disease, including both hyperthyroidism and hypothyroidism:** Assess clinically (e.g., pulse rate, physical exam, weight loss/gain, Achilles reflex return speed, body temperature), and with laboratory testing: serum TSH, free T4, free T3 and/or total T3; strongly consider testing reverse T3 when assessing for functional hypothyroidism. Some integrative

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\text{Estimated Creatinine Clearance (mg/dL)} = \frac{(140 - \text{age in years}) \times \text{Weight in kilograms}}{72 \times \text{serum creatinine in mg/dL}} (0.85 \text{ if female})
\]

\[
\text{Estimated Creatinine Clearance (micromol/L)} = \frac{(140 - \text{age in years}) \times \text{Weight in kilograms}}{72 \times \text{serum creatinine in micromol/L}} (1.23 \text{ for men or 1.04 for women})
\]

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Tobacco smoke constituents cause arterioconstriction which promotes HTN. Constituents and free radicals in tobacco smoke are more pathogenic than nicotine, while the latter in isolation indeed causes adverse cardiovascular effects.

- **Upper cervical spine dysfunction/subluxation:** A remarkable clinical trial published in Journal of Human Hypertension in 2007 by Bakris et al23 showed that *correction of upper cervical spine subluxation/dysfunction* by chiropractic spinal manipulation causes “marked and sustained reductions in BP [blood pressure] similar to the use of two-drug combination therapy.”

- **Vitamin D deficiency:** Vitamin D deficiency is common in the general population—often up to 90-100% of subjects in large population-based studies—and causes intracellular hypercalcinosi24 via elevated PTH levels and contributes to chronic HTN25 via endothelial dysfunction, systemic inflammation, insulin resistance, and activation of the reninangiotensin-aldosterone system.26 *Correction of vitamin D deficiency can cause a reduction in elevated blood pressure comparable to that which can be achieved by single-drug oral antihypertensive medication*27 while also providing numerous collateral benefits (including reductions in depression, pain, and risks for autoimmune and malignant diseases) at lower cost and greater safety than can be achieved with pharmaceutical drugs.28,29

About the author: Dr. Alex Vasquez is a researcher and lecturer for Biotics Research Corporation. This article is copyrighted by Dr Alex Vasquez in 2010 and published with permission in The Original Internist.

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Abstracts of Interest

Submitted by: Emerson Ecologies

DIETARY AND ENVIRONMENTAL FACTORS FOR BREAST CANCER PREVENTION

by Alan R. Gaby, M.D.

Breast cancer is one of the most common types of cancer among women in Western societies. Although the cause is unknown, there is evidence that the risk of developing breast cancer may be influenced by certain dietary and environmental factors.

Environmental factors
Bisphenol A (BPA) is a compound used in the production of a wide variety of food and beverage containers. This compound leaches from the containers into foods and beverages, and BPA has been found in the urine of 95% of people in the United States. BPA has estrogenic activity, and therefore has the theoretical potential to promote the development of estrogen-dependent cancers, including breast cancer. Various organochlorine pesticides also have estrogenic activity, and some (but not all) observational studies have found an association between pesticide exposure and breast cancer incidence. 2,3 Although the evidence is not definitive, it would be prudent for people to use containers that are free of BPA and to consume organically grown foods whenever possible.

Obesity
Being overweight is associated with an increased risk of developing breast cancer. It has been estimated that excess body weight accounts for about 5% of all cancers in Europe. 4 Maintaining an ideal body weight is therefore recommended.

Chlorinated water
Chlorine is added to many municipal water supplies as a disinfectant. Chlorine in water reacts with organic matter to form a number of organic compounds, some of which are carcinogenic, such as trihalomethanes and chloroform. In observational studies, consumption of chlorinated drinking water was associated with an increased risk of breast cancer. 5,6 Chlorine can be removed from tap water by water filters or by boiling or adding a pinch of vitamin C crystals to the water. Alternatives to chlorination include ultraviolet irradiation and ozonation.

Alcohol
In observational studies, consumption of large amounts of alcohol was associated with an increased risk of breast cancer. In many studies, the association between alcohol consumption and cancer risk was dose-related. However, the association between moderate alcohol intake and cancer risk is less clear than that for higher intake. The increased risk of breast cancer associated with alcohol consumption was attenuated by higher folate intake. 7,8

Fruits and vegetables
Numerous observational studies have found that consumption of fruits and vegetables protects against the development of breast cancer. The protective effect of fruit and vegetable consumption may be due in part to the high fiber content of these foods (fiber is believed to influence estrogen metabolism). Other compounds in fruits and vegetables that have anticancer activity include lignans, indole-3-carbinol, allium compounds, isoflavones, isothiocyanates, protease inhibitors, saponins, phytosterols, inositol hexaphosphate (phytate), and flavonoids.

Heavily cooked meat
Observational studies have shown that consumption of very well-done meat, meat cooked at high temperatures (as in grilling), and fried meat is associated with an increased incidence of various types of cancer, including breast cancer. These associations are presumably due to the fact that high-temperature cooking of animal foods results in the formation of carcinogens such as heterocyclic amines and polycyclic aromatic hydrocarbons. Some studies have also shown an association between meat consumption per se (without reference to cooking method) and various types of cancer. However, the results have been inconsistent.

Soy foods
Observational studies suggest that soy consumption may reduce the risk of developing breast cancer. 9,10 Potentially protective substances in soy include isoflavones (which have an anti-estrogenic effect), lignan precursors, beta-sitosterol, and phytate.

Iodine
One investigator hypothesized that inadequate iodine intake may lead to increased estrogenic activity, potentially increasing the risk of estrogen-dependent cancers such as breast, endometrial, and ovarian cancer. In support of this hypothesis, he stated that there is an inverse association between dietary iodine intake in various parts of the world and the incidence of these cancers. 11 However, in areas of sub-Saharan Africa where dietary iodine deficiency is common, the incidences of cancer of the breast, endometrium, and ovary are very low. 12 While maintaining adequate iodine intake (such as 150 mcg per day for adults) is important, supplementing with supraphysiological doses for the purpose of preventing cancer is not risk-free, and based on the available evidence seems unwarranted.

(Continued on next page)
Conclusion
Most of the evidence regarding dietary and environmental factors and breast cancer risk is preliminary. However, making certain dietary and lifestyle changes as discussed above could improve overall health and might decrease the risk of developing breast cancer.

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Pycnogenol® IMPROVES CHRONIC VENOUS INSUFFICIENCY IN A RANDOMIZED TRIAL

by Steve Austin, N.D.


Design: Randomized unblinded intervention trial

Participants: 98 patients with severe chronic venous insufficiency (CVI)

Study Medication and Dosage: 150 mg/d Pycnogenol (administered 50 mg t.i.d.), compression stockings, or both for 8 weeks

Primary Outcome Measures: Rate of ankle swelling (RAS), symptom scores, disease-related disability scores, and resting flux scores (RF)

Key Findings: In all indices, Pycnogenol produced better results than did compression stockings. With Pycnogenol alone, RAS declined 35%, symptom scores declined 58%, disease-related disability scores and RF both declined 48%. The differences favoring Pycnogenol alone compared with compression stockings alone were statistically significant. In the groups using both interventions, outcomes were mostly (but not entirely) slightly better than with Pycnogenol alone.

Practice Implications: Pycnogenol is a standardized extract derived from maritime pine bark. It contains approximately 70% procyanidins previously reported to reduce capillary fragility.

For decades, research from controlled trials (some of which have been blinded and randomized) has found Pycnogenol effective in treating patients suffering from CVI. However, much of this research has either remained unpublished, appeared only in French language journals, appeared in uncommonly-read English language journals, or is now old enough to be difficult to access through internet databases such as PubMed. As a result, some practitioners remain unaware of this relatively consistent evidence. The high cost of Pycnogenol has discouraged others from recommending therapeutic trials in patients with CVI.

Hopefully, this new report in a standard botanical medicinal journal will help remind healthcare professionals that Pycnogenol is indeed a useful agent for CVI patients who can afford the cost, and a more effective treatment than compression stockings (when each therapy is used alone).

Given the discomfort many patients experience in wearing compression stockings and the small gains achieved by adding that intervention to Pycnogenol (when compared with Pycnogenol alone), many practitioners and patients may wish to explore potential benefits with Pycnogenol as monotherapy.

Horsechestnut seed extracts (HCSE) cost significantly less than Pycnogenol. As a result, HCSE is likely to remain the first-line therapy in treating patients with CVI. However, in patients for whom HCSE does not produce an acceptable therapeutic response, Pycnogenol remains a viable scientifically-proven alternative.

Flaxseed lignan lowers cholesterol and liver enzymes in a randomized blinded trial

by Steve Austin, N.D.


Design: Randomized double blind intervention trial

Participants: 30 men with total cholesterol (TC) levels (Continued on page 194)
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**Study Medication and Dosage:** Subjects were given 20 mg/day of flaxseed lignan (secoisolaricresinol diglucoside [SDG]), 100 mg/day SDG, or placebo for 12 weeks.

**Outcome Measures:** Total cholesterol (TC), LDL cholesterol, HDL cholesterol, triglycerides (TG), and hepatic enzymes AST, ALT, and GGTP

**Key Findings:** Results were clinically insignificant at the 20 mg/day dosage, but at 100 mg/day, SDG caused a 7% decline in TC, an 11% decline in LDL cholesterol, a 6.5% increase in HDL cholesterol, and a drop in TC/HDL ratio from 4.0 to 3.5. No change in TG levels occurred. When LDL/HDL ratios were compared, an improvement of 16% was observed (P<0.05). The trial was underpowered to attain statistical significance for the other clinically-relevant lipid level improvements that were observed.

As with lipid levels, changes induced at the 20 mg/day level were neither statistically significant nor clinically relevant. At the 100 mg/day dosage, however a 15% decline in ALT (P=0.003) and a 20% decrease in GGTP (P=0.008) occurred, though AST levels did not decline from baseline.

**Practice Implications:** Previous trials using SDG to successfully lower TC have used doses varying from 200 to 600 mg/day. The purpose of the new trial was to evaluate whether a clinically relevant lowering of TC and LDL cholesterol could be achieved with doses likely to gain a higher level of compliance.

These findings suggest that a dosage of 100 mg/day of SDG can induce clinically important reductions in lipid levels while a dosage of 20 mg/day cannot. SDG supplementation may now be considered a potential adjunctive cholesterol-lowering agent to accompany dietary and lifestyle changes.

Previous evidence from rodent research had suggested that SDG might also protect against fatty liver induced by diet. Those findings interested these researchers in exploring the effects of SDG on liver function in humans.

When hepatic enzyme levels are elevated, seeking the cause of the problem is often more important than initially instituting therapeutic interventions. The etiology, whether related to alcohol, hepatitis or drugs, will usually affect treatment considerations.

However, sometimes isolated liver enzyme elevations cannot be attached to a cause. Such elevations (particularly if minor) may remain clinically non-relevant. Nonetheless, patient and practitioner alike prefer to see these elevations drop back into the normal range. The findings reported in this new trial suggest that elevated ALT and GGTP levels may decline in response to SDG supplementation, though the specific circumstances under which this intervention might best produce these positive results remain unknown. These findings further suggest that isolated elevations in AST will not likely respond to SDG supplementation.

**DIABETES IN MIDLIFE WOMEN AND SELECTED BOTANICALS**

by Tori Hudson, N.D.

Type 2 diabetes mellitus (T2DM) affects an estimated 24 million individuals in the US, almost 8% of our population.1 More than 9 million of those with diabetes in the US are women and nearly one-third of these women are undiagnosed.2 As women (and men) age, the incidence of diabetes increases and approximately 20% of individuals older than 65 will have diabetes. Given the increasing life expectancy of women, the number of women in the US who will acquire diabetes or who will be at high risk for diabetes will rise.

Menopause appears to be associated with a decrease in pancreatic insulin secretion as well as increased insulin resistance.3 These changes are thought to contribute to the increased risk for developing T2DM after menopause although it is not clear whether this is due to only to the postmenopausal lower estrogen status of with aging. However, two landmark studies in women’s health, the Heart and Estrogen/progestin Replacement Study (HERS) and the Women’s Health Initiative (WHI), both suggest that estrogen alone or combined estrogen-progestogen therapy reduces the incidence of new-onset Diabetes.4,5

The connection between estrogen and insulin resistance and T2DM is further strengthened by a meta-analysis quantifying the effects of hormone therapy on metabolic syndrome in postmenopausal women. This meta-analysis found that hormone therapy in peri and post menopausal women improved insulin resistance and fasting glucose in women with diabetes and improved insulin resistance, lipid levels, blood pressure and abdominal obesity in women without diabetes.6 These 3 studies strongly suggest that normal physiologic low estrogen levels that occur in menopause do indeed influence the development of insulin resistance, metabolic syndrome and T2DM in peri and postmenopausal women.

The metabolic changes related to the loss of estrogen in the postmenopausal woman do not bode well for her future health. Increased central body fat, increased low-density lipoproteins (LDL), increased triglycerides, decreased high-density lipoproteins (HDL), increased glucose and insulin resistance substantially increases her risk for cardiovascular disease. As a result, treatment

(Continued on page 196)
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should focus not only on optimizing glucose control and improving insulin sensitivity, but also treating all of her cardiovascular disease (CVD) risk factors rigorously. Just tightening glycemic control in individuals with T2DM will not reduce the cardiovascular risk in those with established CVD or those at high risk for CVD.7,8,9

It is essential that the practitioner appreciate not only the diagnosis of T2DM, but also the various definitions and criteria for the diagnosis of metabolic syndrome and its’ origins from insulin resistance, and the subsequent oxidative stress and vascular inflammatory mechanisms, vascular endothelial dysfunction, thrombosis and atheroembolic disease.

**Lifestyle Intervention**

Intensive lifestyle modification including weight reduction, dietary restrictions, and exercise was compared with placebo or metformin in 3200 overweight adults with impaired glucose metabolism in the Diabetes Prevention Program (DPP) of 2002.10 Compared with placebo, the incidence of diabetes during 3 years was reduced by 58% in the intensive lifestyle intervention group compared with 31% in the metformin group. A 6-year follow-up study was just published of the patients who completed the DPP study.11 The net reduction in diabetes incidence during the full 10 years was 34% in the lifestyle group and 18% in the metformin group when compared with the placebo group, demonstrating that lifestyle interventions can lower the incidence of diabetes for many years.

The key features of most effective lifestyle interventions follow the Total Lifestyle Change (TLC) guidelines. In addition, sixty minutes of daily physical activity that maintains the heart rate at 60% to 80% of 220 minutes minus one’s age is a recommended guide.

**Glycemic Support with Selected Botanical Therapies**

While the remainder of this article will focus on a few selected natural ingredients to improve insulin sensitivity and/or stabilize blood glucose, again, I would like to stress the importance of being aggressive for patients with metabolic syndrome or T2DM, and not only treating their hyperglycemia and their insulin resistance, but also in terms of treating their hypertension, dyslipidemia, endothelial dysfunction, vascular inflammation and obesity.

Berberine has been shown to significantly lower fasting glucose, hemoglobin A1C, triglycerides and insulin levels in patients with T2DM. Berberine, an alkaloid from either goldenseal, goldenthread, Oregon grape, barberry or tree turmeric, has been shown to lower elevated blood glucose equivalent to the effects of metformin.12 In study A, 36 adults with new T2DM were randomly assigned to berberine at 500 mg 3x/day or metformin 500 mg 3x/day. Fasting glucose decreased from 190.8 mg/dl to 124.2 mg/dl and hemoglobin A1c decreased from 9.5% to 7.5% in the berberine group. Postprandial glucose and triglycerides also reduced significantly. In study B, 48 adults with poorly controlled T2DM were given berberine for 3 months. Fasting glucose and postprandial glucose declined significantly within the first two weeks, 190.8 to 124.2 mg/dl and 356.4 to 199.8 mg/dl respectively. Hemoglobin A1c, fasting insulin, total cholesterol and LDL-C also decreased significantly. Another study in 2008, also used berberine for T2DM. One hundred sixteen individuals with T2DM and dyslipidemia were given either 1.0 gm/daily or placebo for 3 months.13 The berberine group had a decrease in fasting glucose from 126 to 100.8 mg/dl and a decreased of HbA1c from 7.5% to 6.6%. Triglycerides, total cholesterol and LDL-C also decreased significantly.

Bitter melon’s ability to lower blood sugar has been demonstrated in both animal and human studies. The fruit has the ability to increase the storage of glucose, decrease the body’s production of glucose, increase insulin production and decrease insulin resistance.14,15 Bitter melon’s actions are similar to that of many well-known conventional anti-diabetic medications, like metformin, gliburide, and acarbose. Unfortunately, head-to-head studies have not been done to investigate whether treatment with bitter melon can be considered equivalent to these standard medications and, as such, one should not replace current medications with bitter melon without consulting a physician.

Most of the studies on bitter melon have been carried out in animals or in the lab, demonstrating hypoglycemic effects, total cholesterol and triglyceride lowering, and weight loss, but the few human trials do offer preliminary support. In one such trial, patients took 200 mg of bitter melon two times per day in combination with their conventional anti-diabetic medication leading to a significant drop in blood sugar, even with half the dose of the conventional medication.16 This result led the authors to conclude that bitter melon acts synergistically with the anti-diabetic drugs. Another study consisted of patients drinking 200 mL dried fruit tea after meals for 12 weeks. Results indicated that fasting blood sugar and glycosylated hemoglobin were both reduced.17

Cinnamon bark contains cinnamaldehyde and polyphenolic polymers that have been found to have glycemic and antioxidant effects that should be considered in MS and T2DM. Favorable effects on serum glucose have been observed in several studies.18,19,20,21,22,23,24 In (Continued on page 198)
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one study of 60 individuals with T2DM, cinnamon at three different doses of 1.3 and 6 grams per day for 40 days reduced fasting glucose 18% to 29%, triglycerides by 23% to 30%, LDL-C 7-27% and total cholesterol 12 to 16%.\textsuperscript{20}

The blueberry leaf contains phenolic compounds, such as chlorogenic acid and caffeic acids that are involved in optimizing glucose absorption and glucose metabolisms. Blueberries themselves, have also been studied for their impact on insulin sensitivity. In a recent double-blinded, placebo-controlled study of 32 obese, middle aged, nondiabetic, and insulin-resistant women and men, participants were randomized to consume either a smoothie twice daily for 6 weeks containing 22.5 mg of two varieties of blueberries that were freeze dried and powdered, or a smoothie without the blueberry. The dose of freeze dried powder was equivalent to 2 cups of fresh whole blueberries. Using hyperinsulinemic-euglycemic clamps after a 10 hour fast, the daily doses of blueberries resulted in improved insulin sensitivity approximately four times greater than the placebo group.

Numerous other botanicals should be considered and have evidence and potential in lowering glucose in humans, improving insulin sensitivity and increasing glucose uptake including curcumin, Jambul seed, fenugreek, ginger root and green tea. In addition, botanicals should be considered to treat specific dyslipidemia, hypertension and cardiovascular/endothelial inflammation and dysfunction.

**Summary**

T2DM is serious disorder affecting more than 9 million women in the US. Assertive intervention is needed to achieve improved glycemic control, to delay or prevent the progression of the microvascular and macrovascular complications. Comprehensive lifestyle interventions are paramount including significant dietary modifications, daily exercise and weight reduction strategies. Additional management with evidence based and traditional botanicals and evidence based nutraceuticals will likely be necessary, with further management utilizing medications when needed. In the perimenopausal woman, who is often plagued with swings in blood sugar and more variability in insulin sensitivity, more frequent blood glucose monitoring and treatment plan adjustments may be necessary. In the T2DM postmenopausal woman, the increased risk of cardiovascular risk should be appreciated by the practitioner such that optimizing the management of her glycemic control and diabetes with a mind towards reducing her cardiovascular risk is a focus of treatment.

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