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

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

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Instructor: Bill Kleber, DC DABCI

October 6 - 7, 2012 Session #4 (Kansas City, MO)
Gastrointestinal Disorders - Upper and Lower (Old 24 & 25)
Instructor: Delilah Anderson, DC DABCI

October 13-14, 2012 Session #18 (Chicago, IL)
Allergy Part 2, Management of the Hypertensive Patient
Instructor: Cindy Howard, DC DABCI

October 20-21, 2012 DOT Medical Exam (Omaha, NE)
DOT Medical Examiner NRCME Training 12 Hours
Instructor: Christopher Murray, DC DABCI

October 27-28, 2012 DOT Medical Exam (Wichita, KS)
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Instructor: Darren Kirchner, DC DABCI

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November 3-4, 2012 Session #5 (Kansas City, MO)
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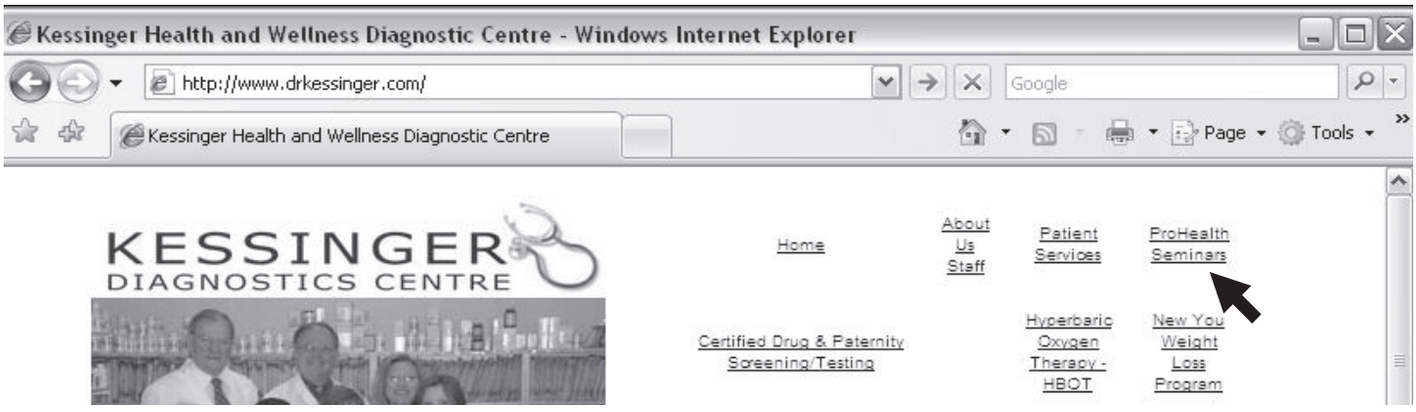

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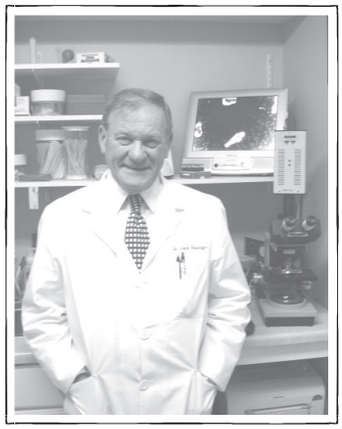

When Treating Human Illness, Think Like a Gardener

When treating human illness, think like a gardener. Growing great organic vegetables takes rich, humid top soil, proper sunlight, oxygen, and adequate water. To insure the proper nutrients and water are available, it is necessary to analyze the soil periodically. Keeping weeds from multiplying and robbing the soil of its precious nutrients is also an important step. As with cancer, it is most beneficial to identify and destroy weeds while in the earliest stages of development.

Life is an ever-evolving scheme of predation, involving both animals and plants. Plants rely on other predators for nutrition and oxygen. Plants get their nutrients from the soil, which are provided by microbes who in turn are predators. With adequate oxygen and water, microbes leach the minerals out of rock and decaying matter, and their waste products become humus which is eventually used by growing plants.

Human tissue and body organs have much in common with the ecology of garden plants. Good health begins at the cellular level. Just as healthy garden plants require adequate nutrition, minerals, water, air and sunlight, so does human tissue.

Historically our bodies have matured to withstand constant attacks, chiefly from viruses, bacteria and fungi. In order to keep up with the monumental task of staying healthy (avoiding infections while maintaining overall health), it is necessary for all cells to have a proper nerve supply, receive optimal nourishment, get an ample supply of oxygen and utilize it efficiently, have a slightly alkaline state of blood, and properly detoxify and eliminate cellular and bowel debris. However, during the past few decades, our immune system has been additionally challenged by the accumulation of combinations of tens of thousands of hazardous chemicals, pollutants, pesticides, fungicides, herbicides, food preservatives, free radicals, and more. It is impossible to avoid these pollutants since they are found in the air we breath, the water we drink and the food we eat.

So is it any mystery that degenerative conditions, like cardiovascular disease, cancer, arthritis, kidney failure, chronic infections, AIDS, diabetes mellitus, fibromyalgia, chronic fatigue syndrome, etc., abound when the human body exists in such a hostile environment? The damage begins with an overload of the organs of detoxification (primarily the liver), and the organs of elimination (kidneys [50%], lungs [28%], skin [20%] and bowel [2%]). Refined (nutritionally depleted) sugars and flour, food preservatives, tobacco smoke, hazardous environmental materials, industrial pollution and solvents, pesticides, most medicines (including antibiotics), synthetic hormones, etc., in addition to bacteria, parasites and fungi, add extra work to our detoxification and elimination systems, thus weakening our cellular ecosystem. The organ that eventually shows up as a *disease process* is often a matter of individual genetic make-up.

In our modern world, children begin overloading their bodies early in life with sugar and caffeine (from chocolate, soft drinks, many over-the-counter medications, etc.) and antibiotics. This persistent bombardment causes them to suffer from constant infections (usually ear or throat) and other maladies. As a result microbes become predators, and the host's own cells become the prey.

With the rare exception of conditions such as homocysteinemia and neural tube birth defects, traditional medicine seldom concerns itself with identifying the underlying cause of a condition. It was only acknowledged that homocysteinemia indeed leads to arterial plaquing in adults after years of denial by traditional medicine. It has only recently been accepted that homocysteinemia can always be reversed by supplementing orthomolecular doses of B12, pyridoxine, folic acid and vitamin C. The recent conclusion that adequate amounts of folic acid reduce the risk of neural tube defects has caused the food processing industry to begin adding this essential nutrient to processed flour. Usually the probability of improper nerve supply, malnutrition, food sensitivities, and toxicities (including heavy metals, chemical or other environmental exposures) is not considered by traditional medical practitioners as an underlying cause of a disease process.

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Our vision is limited by our ideas which, in turn, are dictated by our experiences. Doctors who have never been exposed to the possibilities of natural health care will naturally (innately) be limited in their experiences, and therefore their perceptions. Greed must also be recognized as a large part of worsening health problems. Pharmaceutical companies cannot patent nautral substances, such as air, water, etc., and, therefore, they cannot charge exorbitant prices for them. It is in their best financial interest to not promote natural therapeutic health care strategies.

It is important to appreciate that a non-drug approach to health requires careful attention to a number of interacting factors which may either enhance or weaken the body and its defense systems. It is also important to remember that no two individuals are exactly alike. Supportive treatment strategies and therapies should always be customized to the individual needs by employing a detailed history, followed by a good physical examination and proper laboratory procedures.

Optimal health begins (and ends) at the cellular level. Each and every cell in the body depends on blood for nourishment. Blood also acts as the most important vehicle for the defense system. The health of the vascular system is also dependent on proper blood supply. A simple venipuncture can easily produce the human tissue essential for biopsy which can provide information about the patient that cannot be obtained from any other source.

Other than a detailed history, a multichannel blood test, with a CBC with a differential, a thyroid screen, and ferritin can provide more information about the individuality of the person than any other single test or laboratory procedure. It is often necessary to consider allergies, which are best evaluated by blood tests.

The first step for any gardener attempting to grow a prize winning vegetable or flower is to have the soil tested to insure that the proper nutrition is available for the particular plant. Likewise, the first step any doctor should take when evaluating a health and wellness patient is to order a complete blood examination to help evaluate any nutritional needs.

Editors note:
This article was originally printed in The Original Internist, March 2000, Vol. 7, No.1. It continues to amaze me at the strong visionary Dr. Kessinger was. In our lives we are fortunate to know a handful of people who can see the "big picture." What a privilege to have been a part of his life's journey.◆

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The Legacy Continues

Dr. Jay Kessinger

by: A. Jay Kessinger IV, DC, ND, DABCI
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At Kessinger Health & Wellness Diagnostic Centre, in Rolla, MO, we learned first hand, although accidentally, the extraordinary benefits of intravenous nutritional supplementation. It was an eye opening clinical revelation witnessing the power of healing and recuperation at hand for even the sickest of all our patients through the intravenous application of nutritional supplementation. In 1995, the same year I joined Dad's clinic, a crisis occurred that forever changed the course of our family's history, and, potentially, the destiny of chiropractic in the state of Missouri.

My late brother, Clint, who had become HIV positive following a whole blood infusion in 1986 during a surgical appendectomy, presented with full blown AIDS in February of 1995. Clint was given two weeks to live and a strong suggestion to quickly get his affairs in order. Because of Dad's (Dr Jack's) intervention, later that year, Clint was actually well enough to develop Clint Publications, the company that continues to produce and circulate *the Original Internist,* and *The Missouri Chiropractor,* the official MSCA journal. He passed away December 31, 1995, only spending his last week with obvious disability; i.e., fatigue, lethargy, and loss of acute hearing (*he was not involved with direct care of patients; however, he was publicly visible daily with no signs of ill health*).

HIV positive becomes AIDS when the clinical signs elevate to a certain point. Dad initially diagnosed Clint's condition and was actually able to reverse his CD4:CD8 ratio utilizing oral supplementation with chiropractics natural philosophy; however, over the course of time Clint's stick-to-it-tiveness waned and the inevitable was seemingly too soon a reality. Clint came to Rolla in acute distress in February of 1995. Dad's fast paced "ace in the hole" plan of action was intravenous nutrition which had been approved, accepted, and applied by qualified chiropractors in Oklahoma. This added course of treatment, without a doubt, extended the quality of life for my brother and afforded us, as a family, precious time to rally around Clint and enjoy an extended quality of life for another year.

In March, 1996 we were approached by an osteopath asking to utilize our clinic for intravenous therapy for a patient with multiple myeloma. The patient, a man in his mid-fifties, had undergone two bone marrow transplants and was being treated with steroids. His demeanor paralleled his level of physical well being. He had one of the worst attitudes I've ever experienced in clinical practice. There was no promise of cure given to him, but after receiving a few weeks of intravenous nutritional supplementation he had a clearing of his mental fog. We experienced a drastic improvement in his demeanor and a return of the ability to do the things he loved; i.e., fly fishing with his wife. From the first time we saw him until his death in June, a mere 3 months from the first time we treated him nutritionally, the quality of life for him and his wife was immensely improved.

Dad said he had always accepted patients with cancer, but he treated the person, not the cancer. He said that he treated a whole lot of bald headed men too but never expected them to grow a whole lot of hair. A terminal diagnosis is just that, terminal; however, there is still room to improve the quality of life and the potential to extend the quantity, also. In my opinion, quality of life is a much greater gift than quantity.

At the same time that we were treating the man with multiple myeloma with intravenously nutritional supplementation, we were able to retrieve and treat a patient with colon cancer. Earlier we had referred him to Oklahoma to receive intravenous nutrition. He was in remission following the removal of the cancerous colon tissue. As a result, he had a very productive retirement following the initial diagnosis of colon cancer, and lived another 15 years from the time of his original diagnosis. He died of carcinoid syndrome in the latter part of the fall, 2010.

In both of these cases, the quality of life was definitely improved and the quantity of life was also positively affected through the natural intervention of intravenously supplied nutritional support. You can't trust people who are really sick to digest their nutrients normally. Intravenous nutrition bypasses the gut and is placed in the system for immediate use. Mononucleosis

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TOXINS AND THE GENETIC CONNECTION

by: E. Blaurock-Busch, PhD

The human body is exposed to a wide array of xenobiotics in one's lifetime, and complex enzymatic mechanisms are genetically available to detoxify these substances. A variety of mechanisms support or impair the body's natural ability to detoxify and scientific literature suggests an association between impaired detoxification and certain diseases.

Missing or nonfunctioning enzyme systems impair biotransformation systems, consequently increasing the need for lifestyle changes, including the avoidance of certain toxins.

An individual's ability to tolerate toxins depends on how quickly the body can eliminate the toxic burden, and this important biological detoxification mechanism depends on enzyme functions. The human body contains multiple enzyme systems involved in the detoxification process, but when one or more important enzymes are missing or are functioning improperly, the body's ability to eliminate the excess burden is affected. Normal detoxification is impaired.

To put it simply: if a person misses one or two enzyme systems, even a moderate exposure to metals can overwhelm the system, because the elimination process does not function properly.

In the United States, case-control studies have reported that an important detoxification enzyme is missing in 23%-41% for individuals of African descent; 32%-53% for those of Asian descent, 40% -53% for those of Hispanic descent, and 35%-62% for those of European descent. Several population studies have reported the deletion polymorphism among U.S. Caucasians as ranging from 48%-57%. Other countries have reported varying frequencies of the deletion polymorphism, and an Iranian study showed that in 31% to 38% of the population the GSTM1 enzyme was missing. Groups such as Pacific Islanders and Malaysians have a reported frequency of 62%-100%. Other Asian populations have high-reported frequencies of the deletion genotype ranging from 48%-50% for Japanese and 35%-63% for Chinese. A population-based study conducted among Chinese reported a frequency of 51% for the GSTM1 deletion genotype. Two Korean case-control studies found frequencies of 53% and 56% for the GSTM1 deletion genotype.

The above statistics demonstrate that missing enzyme systems are playing a large role in most populations. Genetic testing is relatively inexpensive and, in most cases, it has to be done only once in a lifetime.

The Detoxification Pathway Phase I

While much is known about the role of Phase I enzymes in the metabolism of pharmaceuticals as well as their activation by environmental toxins, the role of Phase I detoxification in clinical practice has received less consideration than the Phase II enzyme systems.
Enzymes involved in the Phase I metabolism are Cytochrome P450, and the SOD Enzymes.

The Cytochrome P450, Specifically the CYT 4501A1

These enzymes are involved in the metabolism of drugs or exogenous toxins such as chemical solvents or drugs, including steroids. The amount of the CYP enzymes present in the liver reflects their importance in the detoxification process.

The Superoxide Dismutase (SOD) Enzymes are present in practically all cells and in extracellular fluids. The SODs are considered free radical scavengers, preventing oxidative damage and thus are considered important to delay the aging process. Genetic polymorphism in SOD enzymes and their altered expressions and activities are associated with oxidative DNA damage and an increased cancer risk. *(Khan MA, Tania M, Zhang D, Chen H. Antioxidant enzymes and cancer. Chin J Cancer Res 22(2);87-92. 2010).*

SOD enzymes contain metal cofactors which can be copper, zinc, manganese or iron. While all people have an abundance of SOD enzymes, deficiency in any of these metals will lower certain SOD levels and function. In other words, a nutritional deficiency in any of these trace elements potentially impairs SOD enzyme function, leading to a disruption in the detoxification pathway.

(Continued on next page)

Superoxide Dismutase 1 (SOD1)

SOD1 is also called the copper/zinc superoxide dismutase or CuZnSOD. It is present in the cytosol, the nucleus and the mitochondria. Its primary function is to act as an antioxidant enzyme, lowering the steady-state concentration of superoxide. High concentrations are found in liver, brain and testes; but also in red blood cells, pancreas and the lung. Inactivity of an SOD enzyme disturbs the cell metabolism.

A copper or zinc deficiency reduces the function and activity of the SOD1 enzyme.

Superoxide Dismutase 2 (SOD2)

This gene, also called MnSOD, is a member of the iron/ manganese superoxide dismutase family. Mutations in this gene have been associated with idiopathic cardiomyopathy, premature aging, (IDC) sporadic motor neuron disease, and cancer. *(NCBI Report. SOD2 superoxide dismutase 2. upd May2011.)*

SOD-Gene defects have been associated with diseases such as Amyotrophic lateral sclerosis (ALS) *(Banzi et al. SOD1 und amyotrophic lateral sclerosis: mutation and oligomerization. PLoS 3/-/2008. NCBI; Furukawa Y et al. Complete loss of post-translational modifications triggers fibrillar aggregation of SOD1 in familial form of ALS. J. Biol.Chem.283/35/2008).*

A reduced Phase I Metabolism reduces the detoxification ability of a variety of xenotoxins including the potentially toxic metals.

Phase II Enzymes

Phase II reactions follow Phase I reactions. Also known as conjugation reactions (e.g. with gluthation or amino acids or sulfonates), the Phase II system is an important defense mechanism against intake of toxins. The Gluthation Transferases and N-Acetyltransferase 2 (NAT2) belong to the group of Phase II Enzymes.

A reduced Phase II detoxification leads to the accumulation of toxins. Gene variants in the glutathione S-transferases (GST) may lead to poor management of the extremely radical intermediates from the Phase I responses and thereby transmit a predisposition for diseases associated with oxidative stress.

The glutathione S-transferases (GSTM1, GSTT1, etc) are one family of enzymes responsible for the detoxification process, particularly mercury and other toxic metal compounds. These enzymes are also known to play a role in the detoxification of polycyclic aromatic hydrocarbons found in tobacco smoke.

Glutathion-S-Transferase M1 (GSTM1)

GSTM1 is produced in the liver. Through conjugation with glutathion, it functions in the detoxification of environmental toxins and products of oxidative stress, electrophilic compounds, including carcinogens and therapeutic drugs.

Individuals with the GSTM1 */0 Genotype do not have this functioning enzyme and are at greater risk to develop carcinomas.

Glutathion-S-Transferase T1 (GSTT1)

GSTT1 is found in lymphocytes and the liver, and is involved in the detoxification process of a variety of environmental chemicals, such as the ones used in polymer productions. Like all GST Enzymes, GSTT1 detoxifies cancer-causing chemicals as found in cigarette smoke. Approximately 38% of Kaukasions show a complete lack of GTT1 activity. This group with the GSTT1 */0 Genotype shows a high risk for carcinoma of the lung, breast and larynx.

Glutathion S-Transferase P1 (GSTP1)

GSTP1 is built in blood lymphozytes and tissues such as prostate, lung, breast and brain. It plays an important role in detoxification by catalyzing the conjugation of many drophobic and electrophilic compounds with reduced glutathion.

About 50% of the caucasion population shows complete loss of function, which aids the accumulation of reactive products and thus increases the risk of cancer and neurological diseases.

N-Acetyltransferase 2 (NAT2)

The NAT2 functions to both activate and deactivate arylamine and hydrazine drugs and carcinogens. Polymorphisms in this gene are responsible for the N-acetylation process in which humans are segregated into rapid, intermediate or slow acetylator phenotypes.

Lack of NAT2 function is associated with higher incidences of cancer and drug toxicity. Rapid acetylators have a higher risk for colorectal cancer.

(Continued on next page)

(Osian G., Procopciuc L, Vlad L. Nat2 gene polymorphism and sporadic colorectal cancer. Prevalence, tumor stage and prognosis. J.Gastrointestin Liver Dis. 2006; 15(4):357-53).

Apolipopotein E (ApoE)

Apolipoprotein E is a apolipoprotein essential for the metabolism of triglyceride-rich lipoprotein constituents. It has been recognized for its importance in lipoprotein metabolism and cardiovascular disease.

ApoE genotyping may help guide lipid treatment when cardiovascular risks are high. It is used as an adjunct test to aid in the diagnosis of dementia and Alzheimer Disease, but an association has not been confirmed.

Abnormalities in the ApoE gene have been found in neonates with brain injuries and/or defects, and may increase the risk for Cerebral Palsy. (Kuroda MM, Weck ME, Sarwark JF, Hamidulla A, Wainwrigt MS. *Association of apolipoprotein E genotype and cerebral palsy in children. Pediatrics 2007;119(2):306-313).*

ApoE Research

The persistent CNS (central nervous system) effect of lead may be more toxic in individuals who have at least one ApoE-Epsilon4 allele. This study suggests that individuals with ApoE polymorphisms may vary in susceptibility to the long-term effects of lead on the central nervous system.

(Stewart WF, Schwartz BS, Simon D, Kelsley K, Todd AC. *ApoE genotype, past adult lead exposure, and neurobehavioral function*. Environm Health Perspect. 2002; 110(5):5401-505).

A reduction of ApoE gene type that contains two cysteines, decreases detoxification capabilities and the removal of mercury and other thiol-reactive toxicants. (Haley B., *The relationship of the toxic effects of mercury to exacerbation of the medical condition classified as Alzheimer's disease.* Medical Veritas 4 (2007) 1484-1498).

Summary: Why genetic testing?

- When we know genetic 'disabilities', we are in a better position to protect and support our child's system
- We can use care in preventing toxic overexposure
- Since we have numerous enzyme systems involved in the detoxification process, we can strengthen our detoxification ability by supporting and strengthening other enzyme systems.
- SOD testing may not be needed, since SOD enzymes are present in everybody's system. We can test if these enzyme systems function properly, but it would make more sense to first pay attention to potential deficiencies, particularly copper, zinc and manganese before SODs are tested.
- Zinc and manganese deficiencies are not uncommon. Boys are susceptible to zinc deficiency. Blood or hair analyses are an option. While blood tests reveal acute and immediate deficiencies, hair detects if the body has been chronically undersupplied. I would hesitate to have blood drawn from an infant or young child. I consider hair analysis a suitable alternative in locating the problem. If hair analysis results indicate a need for supplementation, it would be logical to supplement for a few weeks before SOD testing is attempted.
- Even in the presence of metabolic or digestive disorders, sufficient nutrients would be absorbed to restore SOD enzyme function to near normal.
- Phase II Enzymes are either present or not. When we know that Phase II Enzyme systems are missing or non-functional, we also know that we need to support the body's detoxification potential—because the system cannot detoxify properly on its own. Nearly 50% of the world population misses the GSTM1 enzyme. These people accumulate toxins readily, simply because their body cannot properly detoxify.
- When we know specifics about our individual detoxification potential, we will know how much outside support in the form of natural or synthetic chelation treatment is needed to prevent our body from continuously accumulating the toxins we are exposed to on a daily basis. The sooner we act, the better.

How to test?

Genetic testing for detoxification enzymes is simple and inexpensive. The material needed for testing is 1ml of whole blood drawn into an EDTA tube, or 10 drops of whole blood on filter paper, or a gum swap. Test kits are available on request.

For more information: www.microtraceminerals.com

More Literature

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ADDRESSING THE NUTRITIONAL NEEDS OF CHILDREN

by: Rachel Olivier, MS, ND, PhD

Children have specific nutritional needs, and like adults they require a minimum daily intake of essential vitamins, minerals, and fatty acids. Their need is more critical than adults, however, since they are laying down basic core pathways, which will affect them the rest of their lives. Consequently, it is essential to ensure that they consume those essential components, as required for the growing body of a child.

It is well recognized that the Standard American Diet is high in processed foods, and lacking in essential vitamins, minerals, and fatty acids. Accordingly, judicious dietary choices that include unprocessed foods containing a varied supply of these essential nutrients are of utmost importance. Given the known fact that many foods are lacking in nutritional value, equally important is daily supplementation with at minimum a multivitamin, essential fatty acids, and vitamins D and K. Additional supplementation, such as vitamin C or other essential nutrients may be added to the daily regimen for specific deficiencies, or as deemed necessary.

Children's diets can be particularly high in fat and calories, too often the wrong kinds of fats, thus it is not surprising that an estimated thirty-two percent (32%) of children in the US are overweight or obese. In recent decades increased weight has resulted in a triple-fold increase in the incidence of obesity-linked diseases.[1] In addition to affecting the quality of life, obesity contributes to many health conditions. In children with increased adiposity, an association with increased blood pressure has also been observed.[2] Besides hypertension and diabetes, other health conditions linked to obesity include liver and gallbladder disease, sleep apnea, respiratory problems and mental health conditions.[3]

The measure of body weight relative to height is referred to as the body mass index (BMI), and is a tool often used to determine if a person is at a healthy weight, overweight, or obese. For children in addition to weight and height, age must also be utilized to calculate the BMI. Four different categories are utilized to classify BMI values, as indicated in the table below, with the percentile indicating the relative position of a child's BMI number among children of the same sex and age group.

Weight Status	Percentile
Underweight	$<5^{th}$
Healthy Weight	5^{th} to $<85^{th}$
Overweight	85^{th} - $<95^{th}$
Obese	$\geq 95^{th}$

To calculate the BMI, the link http://www.nhlbisupport.com/bmi/ may be used. For children's BMI charts, see http://www.bcm.edu/cnrc/images/pdfs_nyc/cdc_bmi_boys.pdf, and http://www.bcm.edu/cnrc/images/pdfs_nyc/cdc_bmi_girls.pdf.

Regardless of the weight status, empty calories should not be a major part of the diet. For most people, it is recommended that no more than 15% of calories come from solid fats and added sugars. However, according to the USDA, about 35% of the calories Americans typically consume (eat and drink) are empty calories, which means that foods and drinks with too high a content of solid fats and added sugars are typically chosen.[4]

Harvard's School of Public Health[5] has published ten top nutritional tips for eating right, which include:

1) Selecting good (complex) carbohydrates, as opposed to no carbohydrates
2) Selecting high-quality protein sources, such as fish, lean meats, and nuts
3) Selecting foods with beneficial fats, while limiting foods high in saturated fats, and avoiding trans fats
4) Consuming a fiber-filled diet, rich in whole grains, fruits, and vegetables; especially green vegetables for their vitamin K content. Vitamin K is necessary for optimal calcium (Ca) utilization, as it functions to activate the protein carrying Ca to the bone
5) Consuming a high quantity of vegetables and fruits
6) Including a source of calcium in the diet
7) Including plenty of water, and avoiding sugary drinks and juices
8) Choosing fresh, in season foods, as opposed to processed foods
9) Taking a daily multivitamin as a "nutrition insurance policy"
10) Adding vitamin D and vitamin K, for an "extra health boost."

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In addition to the above dietary choices, select groups of children may need specific, additional supplementation, tailored to their individual needs. In all age groups, food assessments can serve as an essential tool in determining sufficient intake of essential nutrients. By recognizing those nutrients that are deficient, specific supplementation may be implemented to address individual deficiencies. For example both dietary surveys and the Children's Nutrition Research Center (CNRC) suggest that adolescent girls tend to be significantly lacking in their dietary intake of both calcium and iron, as recommended for their age group.[6]

As a consequence of the growing weight issue in young children and teens in America, the United States Department of Agriculture (USDA) has developed a program called **W**ays to **E**nhance **C**hildren's **A**ctivity & **N**utrition *(We Can!*). This nationally recognized program is designed for parents, caregivers and communities to facilitate the weight management of children 8 to 13 years old.[7] As part of this program, foods are designated into three categories, "GO, SLOW and WHOA foods." GO foods are nutritious and low in both fat and added sugar. These foods can be eaten anytime. SLOW and WHOA foods are higher in fat, added sugar and calories, and should be eaten less frequently. By identifying the good foods, and eliminating the foods that are not beneficial, a healthy lifestyle can be attained, which ultimately results in weight control. To assist with caloric intake, the information on calories per serving, serving size and the grams of fat and sugar located on packages are essential. Additionally, many of the fast-food restaurants voluntarily provide nutritional information about the foods they serve upon request, or on their website, allowing an awareness of the caloric content of the foods consumed. The caloric content is important to recognize, so that wise food choices can be made, such that over consumption does not result.

As mentioned above, equally important is daily supplementation with at minimum a multi-vitamin, essential fatty acids, and vitamin D, in an effort to alleviate dietary insufficiencies.

Daily Multivitamin. To supplement the deficiencies in the daily diet, the primary consideration is the addition of a daily multivitamin. Use of a daily multivitamin offers numerous benefits, particularly if the child doesn't eat a well balanced meal, or has eating difficulties. One primary consideration for the type of multivitamin is the availability of the individual components. Liquid multivitamins are one choice for children, as this eliminates the need to swallow a tablet or capsule, and secondly these vitamin forms have high bioavailability. With liquids, however, it is important to recognize that folic acid and iron are often missing in aqueous multi products. This is because folic acid solubility is very pH dependent, and requires a basic medium (pH) for solubility, while iron does not readily dissolve in aqueous solutions. Iron can also react with vitamin C, negating any value. Subsequently, a judicious choice is to also supplement with these essential nutrients. For older children, tablet or capsule forms of a multi may be suitable. Good choices include those that contain essential B-vitamins, trace minerals, including zinc, selenium and copper, and macronutrients like calcium, iron, and potassium. Also a consideration is the availability of these nutrients, as some tablets and capsules are high in fillers or other inert ingredients, thus offer limited benefits.

Vitamins D & K. It is estimated that children spend an average of 93% of their time indoors. As a consequence of these lifestyle changes, and along with an increased usage of sunscreen, a diminished exposure to sunlight has resulted. Consequently, a high percentage of the population is presenting with vitamin D deficiency, as determined by decreased serum levels of vitamin D (<50nmol/L). Since vitamin D is a critical factor in optimal health, an adequate daily intake is also recommended for children and adolescents. According to Dr. Carol Wagner, MD, FAAP, professor of pediatrics at the Medical University of South Carolina, in addition to deficiency in adolescents and adults, evidence of vitamin D deficiency also exists in infants and children of all ages.[8]

In adults, vitamin D deficiency is defined as a serum 25-OH-vitamin D of <50nmol/L, while in children and infants, a consensus with regard to the concentration of 25-OH-vitamin D necessary to define insufficiency has yet to be determined.[9,10,11,12] However, the American Academy of Pediatrics (AAP) recently recommended doubling the amount of vitamin D for babies and children from 200 International Units (IU) per day to 400 IU per day,[13] since 200IU/day does not maintain 25-OH vitamin D concentrations above 50nmol/L.

Vitamin D has multiple actions, including the role it plays in an immunomodulatory fashion. It is a potent stimulator of the innate immune system, acting through toll-like receptors on monocytes and macrophages. Low levels of vitamin D leads to increased release of parathyroid hormone (PTH),[14] which increases bone resorption, and in turn results in a decrease of bone

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mass. Due to its effect on calcium absorption, vitamin D insufficiency is also associated with bone loss and an increased fracture risk. When vitamin D status declines, absorption of dietary calcium also declines, to about 10% to 15%; however, with sufficient vitamin D status absorption of dietary calcium is approximately 30% to 40%.[15]

There are a number of variables that contribute to the ability to absorb vitamin D, with the obvious being the amount of time spent in the sun. Other factors that contribute include the amount of skin pigmentation, the degree of latitude, season, cloud cover, air pollution, amount of skin exposed, and UVB protection (e.g., clothing, sunscreen). In addition to these, body mass can also be a factor in vitamin D absorption. In fact an elevated BMI (>30) is associated with decreased circulating 25(OH)D, as fat serves as a vitamin D reservoir.[16] Thus, in overweight children and adolescents a greater need for supplementation may exist. For this group, testing for vitamin D deficiency may be especially pertinent.

Two forms of vitamin D are available for supplementation, vitamin D2 (ergocalciferol) and vitamin D3 (cholecalciferol). Of these two forms vitamin D3 has demonstrated greater efficacy in raising circulating 25-OH-D concentrations under certain physiological situations,[17] and, therefore, is the preferential form of supplementation.

Vitamin K is equally important as vitamin D, as it works in conjunction with vitamin D to aide in proper bone formation and in maintaining bone density.[18,19,20,21] Unlike other fat-soluble vitamins, vitamin K is not stored in the body, thus a daily intake is necessary. It is well recognized that supplementation or injection is necessary for the newborn infant, as the fetus has low levels of vitamin K, and adequate levels of vitamin K are required for proper blood clotting.22 In the fetus tight regulation of vitamin K levels helps to control the rate of rapid cell division during fetal development. However, without supplementation, deficiency in this group can result in just a few days,[23] predisposing the infant to complications associate with hemorrhaging, specifically of the brain. Beyond its role in blood clotting, recent research has revealed that vitamin K also plays a vital role in maintaining healthy bones and arteries by keeping calcium in the bones and out of the arteries.[24]

Essential Fatty Acids. Detailed surveys by The Hyperactive Children's Support Group (HCSG) have concluded that many children are deficient in essential fatty acids (EFAs).[25] These deficiencies have been correlated to either metabolism or absorption issues, or a combination of these. Brain cells are especially rich in certain long chain polyunsaturated fatty acids (PUFAs), and their dietary status has an influence on both cognitive function and behavior. In addition to the conclusion noted above by the HCSG, this group has also sited specific evidential components that point to EFA insufficiency and/or deficiency in children. These criteria include the following: a) many of the foods that "cause trouble in children are weak inhibitors of the conversion of EFAs to prostaglandins (PGs)"; b) boys are affected to a greater degree than girls and "males are known to have much higher requirements for EFAs than females"; c) abnormal thirst is one of the cardinal signs of EFA deficiency; many children suffer with this symptom; d) "many children have eczema, allergies and asthma", all of which can benefit, or be alleviated by EFA supplementation; e) many children are zinc deficient, and zinc is required for conversion of EFAs to PGs; and f) allergies affect many children, especially wheat and milk, "which are known to give rise to exorphins in the gut," which in turn may block conversion of EFAs to PGEl.[25] Besides their importance in learning, behavior, and attention disorders, EFAs are also extremely important for the growing brain. The presence of good oils in the diet has decreased dramatically as a consequence of the high quantity of hydrogenated oil, subsequently, a daily supply of EFAs can provide good fats, and in turn alleviate the above noted issues. In addition, both DHA and EPA are necessary for vital brain function, including memory, speech, and specific motor skills.

As stated by President John F. Kennedy, "Children are the world's most valuable resource and its best hope for the future", thus we must supply them with the opportunity to thrive and be healthy. Their individual nutritional needs, although dependent upon family dynamics, are vitally important in an effort to facilitate optimal health. It can't be stressed enough how the early stages of development are a critical time as foundational core pathways and structures are being built. Many children have become accustomed to diets high in simple carbohydrates and fatty, fast foods. However, a practical strategy for busy families would begin by incorporating four basic components into the daily diet, a multivitamin, essential fatty acids, and vitamins D and K. This strategy, in conjunction with simple lifestyle changes, like drinking water in place of sugary drinks and juices, and getting involved in organized sports or classes to build their physical core, can assist

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FEMALE SEX HORMONE BALANCE: KEY TO BREAST CANCER PREVENTION

by: Dawn Cutillo Hiestand, B.S. Health & Nutrition; Author, *The Hormone "Shift"*

As October approaches, another "breast cancer awareness" month will come and go. We have to ask ourselves, "*Are we really raising awareness in our clients/ patients?"* A leading national women's magazine revealed last October that Americans have collectively raised $6 billion for breast cancer awareness. Yet the average woman is "not aware" that an imbalance in sex hormones is key for the most common type of estrogen-sensitive breast cancer. I tell women daily in my health center that balancing sex hormones will not only prevent this deadly disease but it is also key to weight management, sleep, mood, and prevention of virtually all PMS and menopausal symptoms while also keeping other female health issues (ovarian fibroids/cysts, endometriosis, heavy periods) at bay. Let's face it; a woman doesn't just wake up one day with breast cancer. This deadly disease is a culmination of physiological and psychological stressors that is now being understood as having a foundation in adrenal fatigue/exhaustion and a female sex hormone imbalance. Genetics, toxicity levels, and immune system capacity all come into play but the fact remains that no doctor can show you a woman with breast cancer who has balanced sex hormones. It is also an obvious fact that women who are genetically predisposed to breast cancer do not always get the disease and women who have no genetic predisposition often do get the disease. So, we must look beyond genetics and also look at a woman's lifestyle and her total stress load factors that result in adrenal fatigue/exhaustion and an imbalance between estrogen and progesterone.

I had the experience of working with hormone specialist, Mr. Joeseph Beldonza C.C.N, ND, who had done over 20,000 saliva hormone tests while starting a hormone testing lab years ago. He used this experience to educate doctors in interpreting saliva hormone profiles. With all this lab experience, Mr. Beldonza saw obvious trends in saliva profiles of women with female-related cancers. I knew this information would be more credible if a small pilot study was done to validate it. I facilitated the study at my health center, The Rejuvenation Center, in Lancaster, Pa. Bio Health Lab from California evaluated the saliva test and **Dr. Lorraine Bernotsky, research director, West Chester University, (West Chester, Pa.) tabulated the study results**.

Study:
A small sample of 25 local women, who had breast cancer in the last 5 years, was compared to 20 women who never had breast cancer. The parameters of the study required that the breast cancer survivors were not currently on chemotherapy, radiation or any type of medication such as the commonly used estrogen blocker, *Tamoxifen*.

The goal of the study was to show trends in saliva hormonal profiles of women who have had breast cancer. These trends could then serve to aid women who want to prevent breast cancer by allowing them to compare their saliva test results to these in an effort to detect at the earliest time the possible manifestation of the disease.

The growing national trend is to remove the breast even before the woman is diagnosed with breast cancer as a way of prevention of the disease if breast cancer runs in her family. This is obviously extremely invasive and does not change the underlying hormonal imbalance that contributed to her cancer. We decided to do this pilot study in hopes that our research will someday allow women to test their hormones with a simple saliva test as an early detection tool in order to avoid this type of surgery. Looking ahead at the hormonal trend towards breast cancer is *true early detection* as opposed to a mammogram that is considered early detection of an already existing tumor in the beginning stages of development!

The main findings of the study:

- Low levels of progesterone are linked to breast cancer
- High levels of estrogen (specifically estrodiol) in relation to progesterone (an imbalance), is linked to breast cancer as shown in specific estrogen to progesterone ratios.
- All women with breast cancer have a sex hormonal imbalance
- More research is warranted to see specific ratios of the three main estrogens and breast cancer occurrences

These are interesting findings not often brought to women's attention via their doctor, the media or even more holistic health practitioners because these facts are not commonly known.

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This is mainly due to the fact that although some biomedical literature recognizes the importance of the relationship between progesterone levels and the incidence of breast cancer, mainstream medical practice does not. However, some medical professionals have investigated and tracked this relationship in their own patients (see for example, Dr. John R. Lee's work) and have suggested that the use of estrogen therapy to address symptoms of menopause puts patients at risk for female-related cancers with respect to decreased levels of progesterone. In fact, the American Cancer Society's webpage does not differentiate between estrogen and progesterone when citing hormones that could be related to the incidence of breast cancer. In this manner, it would seem that traditional medical opinions do not recognize or emphasize the potentially important role of progesterone in women's health, including breast cancer among various other health concerns.

Details of the Study Results:
There appears to be a relationship between the incidence of breast cancer and low progesterone levels when comparing the 25 women who had breast cancer to 20 similar aged women who never had breast cancer. All of the women with breast cancer had low progesterone levels based on "normal/healthy" levels as well as poor estrogen to progesterone ratios needed for health.
When comparing the cross-sample of 45 women, over 69% of women with extremely low levels of progesterone (under 100 pg/ml) had breast cancer, while only 42.9% of those with slightly higher levels of progesterone (over 100 pg/ml) had breast cancer. It is important to note that those with levels over 100 pg/ml still were low overall as far as healthy levels; however, the lower the progesterone levels were, the risk of breast cancer was increased.

This is compounded by the relationship between the incidence of breast cancer and the ratio of progesterone to estrodiol (an estrogen) when comparing women with ratios under 50 to those with ratios over 50. The study showed that 73.1% of women with progesterone to estrodiol ratios lower than 50 had breast cancer, while only 31.3% of those with ratios over 50 had breast cancer.

More interesting, were the trends that require further research in the specific levels and ratios of the three estrogens tested. It was found that 64% of these women had an estrodiol level lower than their estrone level. Almost all of the women (96%) had an estriol level that was lower than their estrodiol level, while 76% had an estrodiol level that was different by less than 1 point from their estriol level. "*While not being statistically significant at this point, this finding is worth further research"* reports Dr. Bernotsky as she tabulated the results.

More research is obviously needed into the exact estrogen profiles but a strong link exists between progesterone levels and breast cancer as well as the ratios of high estrodiol to progesterone levels as an indicator of breast cancer.

Conclusion and Education on Hormones:
Naturally balancing estrogen to progesterone levels before breast cancer can develop would be the most preventative advice at this point for women who fear developing the disease. Please note that estrodiol is 12 times more potent than estrone, and in the presence of adequate progesterone, estrodiol will often be converted to estrone sulfate, an essentially inactive form that is stored mainly in the fat cells for later use as needed. Progesterone also induces the production of an enzyme used to convert estrodiol to estrone. This seems to be a natural checks-and-balances system to ensure natural estrogen does not get too high, which could eventually cause diseases like breast cancer. A study in the *American Journal of Epidemiology* found that women with a progesterone deficiency had 5.4 times the risk of premenopausal breast cancer; while a Mayo Clinic study showed that women with a history of progesterone deficiency had 3.6 times the risk of postmenopausal cancer. This further establishes that there are trends to be observed with women who get breast cancer. Dr. Reiss, *Natural Hormone Balance for Women*, agrees with this same concept of the "progesterone protection factor" by showing the conversion of estrodiol into estrone can only take place in the presence of progesterone, coupled with a specific enzyme. This shows that progesterone is involved in converting excess estrogen (estrodiol) into a safer, weaker form (estrone).

It is important for women to raise their progesterone levels to balance out estrogen and relieve a condition dubbed by Dr. Lee, (foremost researcher on progesterone therapy) as "estrogen dominance". Some symptoms of "estrogen dominance" are PMS, depression, anxiety, irritability, insomnia, headaches, hot flashes, night sweats, mood swings, food cravings, fluid retention, heavy periods, fibroids, endometriosis, etc. Now we can add increased incidence of breast cancer to this list.

The main reason for depleting progesterone levels in American women is the conversion of progesterone to cortisol when women are under chronic psychological

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or physiological stress. What Mr. Beldonza has seen as a trend in his analysis of over 20,000 hormone saliva tests and what I have seen in the thousands of women I have worked with over the years, is that initial adrenal fatigue due to chronic stress, demanding high cortisol output (shown by elevated cortisol on saliva tests), will be associated with lower progesterone levels and hence "estrogen dominance". Therefore, it is ineffective to try to build a woman's progesterone levels without addressing the main factor of adrenal fatigue/exhaustion. I find that pure-sourced adrenal glandulars are essential to repair and rebuild the adrenal glands to allow for full endocrine balance. The only caution here is to be sure that the animal source from where the glandular is derived has not been tainted with hormones, steroids, and antibiotics. I have found the purest sources come from New Zealand. This supplementation combined with the fast acting affects of a trans-dermal* adrenal support cream, that goes directly into the bloodstream (bypassing the digestive system), will provide fast relief of almost all PMS and menopausal symptoms as well as long-term healing benefits. Proper ingredients in a trans-dermal adrenal support cream will give the body the "building blocks" to make the cortisol it needs while under chronic stress. This prevents the body from converting progesterone to make more cortisol when the demand for it is so high. Over time, progesterone levels can be raised, allowing the given supplemental progesterone to reach adequate "normal" levels to balance out "estrogen dominance". It is this foundation that is needed for a trans-dermal progesterone cream to be effective in quickly relieving symptoms of PMS and menopause while providing the underlying hormone profile that is healthy and balanced to prevent female related cancers.

It is important to note that even after ovarian production of estrodiol has stopped (around menopause), keeping cortisol levels stable and progesterone levels adequate provides the basis for the body to produce estrone as the primary postmenopausal estrogen which will keep postmenopausal symptoms (bone thinning, thinning vaginal walls, low libido, etc.) at bay.

Standard progesterone hormone creams that have an alcohol delivery system and that recommend rotating points of application can have the tendency of building up in the fatty tissue and reaching saturation points, allowing excess progesterone to build up in the body. I have seen this happen with health food store brands as well as doctor compounded bio-identical mixtures. Excess progesterone will give the same returning symptoms as the original condition of "estrogen dominance". It is vital that if you choose to use progesterone in a cream form that it is a true trans-dermal delivery system. Oral forms of progesterone tend to need higher dosages due to some of their potency being lost through the digestive system.

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Other areas to advise clients/patients in are to avoid excess processed foods, sugars (to stabilize insulin), foods they are sensitive to (mainly wheat, dairy, soy and peanuts), caffeine and alcohol. Increased consumption of cruciferous vegetables is advised as they are full of nutrients and have been proven to convert the more potent estrogen (estrodiol) into the weaker less active form of estrogen (estrone).

Less intense exercise such as yoga, walking, and light resistance training is preferred over more intense cardiovascular exercise such as running, which will elevate cortisol levels and exacerbate the existing hormonal imbalance. A final word of advice for all women is to take time daily to break the sympathetic "fight or flight" stress cycle. Dr. Herbert Benson, The Relaxation Response, advocates taking 20 minutes per day to do this. I have found amongst my clients that a simple relaxation CD that uses "sound wave therapy" can relax brain wave patterns from the highly active beta waves down through the slower alpha wave and theta wave patterns to reach even the slowest Delta wave patterns, where healing and regeneration begin. This relieves mental and emotional stress in a short time with all the physiological and psychological benefits connected to it, including lowering the demand for cortisol (aiding female sex hormone balance).

As holistic healthcare practitioners, we can provide testing and supplementation for subclinical issues which provides a true foundation for the prevention of even deadly diseases like breast cancer. This, coupled with basic hormone education for the client/patient, can inspire lifestyle changes that will synergistically support the body in the prevention of diseases–even through the aging process. We must strive to educate our female clients/patients in the simple fact that chronic stress (and the adrenal fatigue/exhaustion effect) once again seems to be a key factor in a deadly disease that affects so many American women.

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Her main area of interest is the connection between stress and weight gain, allowed her to be the "Stress and Weight Gain" expert for Ediets*. She was published internationally in* Aromatherapy and Wellbeing magazine *for her original work in developing the "*Conditioned Response*" program which utilizes meditative visualization, aromatherapy and sound-wave therapy to aid in stress hormone balance and behavior modification for weight management. Her interest and years of work with hormones has culminated in her new book,* The Hormone "Shift' *(Balboa Press).*

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LEGACY *(Cont. from page 87)*

can be cleared in a matter of weeks rather than months. Diabetic neuropathies have been reversed utilizing intravenous nutritional therapies. There are protocols of intravenous nutritional supplementation we have utilized to help addictive behaviors, shorten inflammatory conditions, improve immunity, and enhance cardiac vitality; however, none of therapies we apply can be credited with curing any condition. A true cure can only come from within. Each person has to make the decision to take the raw materials necessary for them to have health and put them into use. Without exercise, without avoidance of external toxins, without choosing only the best food sources and avoiding the pro-inflammatory and otherwise poor food choices, and without avoiding the emotional feelings of hopelessness, even the best nutritional supplements and adjustments will be all for naught.

Nutritional supplementation, regardless of the route of administration, is to supplement a good diet rather than to be taken instead of a good diet. Dad told the story in the chelation (IV) room about the guy a lot of people envied in the 1950's. The guy had an air-conditioned Cadillac who pulled into a filling station, leaving his car running. He barely cracked open his window to tell the attendant, "Fill it up! With Ethyl.", before rolling his window back up. After some time the attendant came back knocking on the driver's window. He said, "Would you mind shutting it off, Sir? I think you're gaining on us!" This same analogy applies to the patient who changes none of their bad habits, but expects results from nutritional supplementation alone.

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OVERVIEW OF THE REFINED FOOD ADDICTION HYPOTHESIS

Running Title: Refined Food Addiction

by: Jessica Tsuei MS[1], Joan Ifland[2] and Harry G. Preuss MD[1]

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Introduction

Obesity, an accumulation of excess body fat due to energy intake exceeding energy expenditure, is among the most ubiquitous unhealthful conditions in the United States.[18] Not only does it diminish quality of life on an emotional level, it also leads to major risk factors in a variety of health situations.[18] Despite knowledge of its detrimental consequences, obesity has become more prevalent during the past 20 years; and the rate is still increasing. Today, more than one-third of U.S. adults (35.7%) and 17% of children are obese.[18] The steady increase has been ascribed to failure in self-management and ineffective long-term treatments. This paper examines the possibility that some obese patients have refined food addiction as a primary cause behind excess fat accumulation. This point is important, because the absence of classic addiction treatment may explain, at least in part, the failure to manage obesity successfully. Accordingly, knowledge that food addiction exists becomes a vital question to answer in order to develop the best strategies to overcome the obesity epidemic and its dangerous associate – type 2 diabetes mellitus.

The Symptoms of Food Addiction

Substance addiction (as opposed to behavioral addictions) is scientifically referred to as substance dependence and popularly seen as the state of being enslaved to a habit.[19] The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) defines substance dependence as three or more of the following seven criteria occurring within 1 year: (i) tolerance, (ii) withdrawal symptoms, (iii) difficulty in controlling usage, (iv) attempts to cut down, (v) excessive time spent obtaining, using, or recovering from use, (vi) neglecting or postponing activities, and (vii) continued use despite adverse consequences.[8]

At this time, there is no accepted blood test to definitively diagnose refined food addiction.[14] The Yale Food Addiction scale (YFAS) is a tool to operationalize food addiction.[11] It is partially based on the DSM-IV criteria for addiction and is designed to increase the methodological strength of food addiction research.[11] It is scaled toward detecting whether a person is addicted to food specifically.[12] Craving is also a common symptom of addiction and is strongly associated with binge eating.[6] Even though craving is not part of the DSM-IV substance dependence criteria, it is proposed as criteria for the DSM-V.[8]

History

Approximately 60 years ago, Randolph proposed the concept of "food addiction" as a specific adaptation to consume highly sensitive foods on a regular basis.[21] He stressed two important characteristics of food addiction--size and frequency of consumption. He further believed that people are addicted to high caloric and high carbohydrate foods.[21] Unfortunately, he was generally ignored and criticized by the medical community.[14] The term "food addiction" is often used interchangeably with "carbohydrate addiction," which was coined in 1963 by Robert Kemp, a biochemist at Yale University. Kemp noticed that many obese people prefer palatable carbohydrate-rich foods and "exhibit the vicious cycles of negative behaviors and physiological imbalances that defy the individual's willpower."[14]

In 1994, Doctors Richard and Rachael Heller, both recovered from carbohydrate addiction, hypothesized the link between hyper-insulinemic obesity and carbohydrate addiction.[14] They postulated that hyperinsulinemic obesity is caused by carbohydrate addiction, which sustains the chronic high circulating insulin levels and insulin resistance seen in many obese people.[14] Since then, there has been a worldwide shift in perspective of addict-ion to carbohydrate-rich foods.[7] Today, carbohydrate addiction is defined as a recurring compelling hunger,

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craving, or desire for carbohydrate-rich foods, such as breads, cakes, cereal, chocolate, potatoes, ice cream, rice, sugar-sweetened beverages etc.[14,19]

Many researchers have shown that obsessive behaviors in carbohydrate-addicted individuals parallel the behaviors shown in those with other addictive habits. Like other kinds of addicts, carbohydrate-addicted individuals experience denial and fail to realize that they are "hooked." They are completely focused on attaining a satisfying feeling from consuming carbohydrate-rich foods.[14]

The Refined Carbohydates Model of Food Addiction

Two important models for food addiction have been described via the research route. The first model is the "salted food hypothesis," which states that salt can stimulate opiate and dopamine receptors in the brain's reward centers. This, in turn, promotes overeating and weight gain.[6] While the second model encompasses the salted food model, the refined food addiction model focuses on other food items like refined carbohydrates, fats, and caffeine in addition to salt that are readily available in large quantities in our food markets today.[16] The second model looks specifically at refined foods, which are substances that have been industrially processed such as sugar-sweeteners, flour, salt, and certain fats. These are foods that are not found in refined form in nature.[15] The hypothesis suggests that these substances can be addictive and act similar to other drugs of abuse when consumed frequently at high doses.[6,15] We will be focusing on the refined food addiction model in this review.

Some have linked carbohydrate addiction to evolution.[7] They argue that people are adapted to seek and consume energy-producing food without a lot of effort. Industrialized societies encourage a sedentary lifestyle and low-priced refined foods. Accordingly, people are thought to prefer foods that are high in fat, sugar, and salt substances even though these foods were only found in small amounts in the diets of our evolutionary ancestors.[7] The hypothesis is that repeated exposure to and consumption of refined food eventually leads to food dependence.[7] Although this may manifest as binge eating in some obese people, others exhibit grazing patterns and volume eating that does not meet the criteria for the concentrated, intense eating characteristic of binge eating. Ifland et al, have advanced the refined food addiction model to explain the behavior of overeating seen in many overweight and obese individuals.[15] They believe overeating can be characterized as an addiction to refined foods because of the presence of loss of control in regulation of food intake.[15] This characterization aligns with the concepts of addictive behavior defined in the DSM-IV and Kemp's categorization of uncontrolled carbohydrate consumption as an addictive behavior.

More support for the concept of refined food addiction derives from trends in the per capita consumption of refined foods that corresponds to the dramatic increase of obesity in the United States.[15] Also, Davis et al. have shown that "Food Addiction" is a classifiable condition with clinical symptomatology and a psycho-behavioral profile similar to that of conventional drug abuse disorders.[7] They showed that a sugar-enhanced diet increases daily food intake over time and that removal of sugar causes mice to show aggression, anxiety, teeth chattering, and head shaking – symptoms consistent with withdrawal symptoms from drugs like heroin.[7] The refined food addiction model claims that these processed foods are similar to tobacco in that they are not addictive until extracted and concentrated.[15]

Avena et al. have found that it may not be a single refined substance itself that promotes addiction, but rather a combination of substances.[1] By looking at the history of food agriculture and lifestyle changes, we can see the logic behind this proposal. The discovery of high fructose corn syrup, heavily added to many processed foods, is an example frequently used to illustrate that combined industrialized foods are highly addictive and harmful to our bodies.[10] Through qualitative and quantitative research, Ifland et al. have demonstrated that the pathology behind the behavior prevalent in refined food addiction does comply with the DSM-IV's addictive behavior criteria and thus can be accounted as addiction.[14] Nevertheless, the refined food addiction model has yet to be validated by the broader research community.

The Mechanisms behind Food Addition

The powerful urges that compel substance-seeking individuals to lose their better judgment are common features of addiction disorders. These cravings are different from hunger in that the desires are more intense and specific.[19] Like drug cravings, food cravings can easily be triggered by exposure to the sight, smell, or image of the craved food. As refined foods become more readily available, they have become our main source of nourishment and energy.

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Previous research has shown that food cravings can be induced by repeatedly eating a specific refined food when hungry or craving. Cravings increase over time due to sensitization while the desired effect, i.e. the reduction of dysphoria, depression, or anxiety, lessens. This is the process of tolerance--one of the DSM-IV substance dependence criteria.[23] The similarities between refined food addiction and other addictive processes strongly suggest that refined carbohydrates, salt, and caffeine indeed have abuse potential for people who crave them.[23]

Depression, ADHD, and the addicted brain are all known to be mesolimbic dopamine-dyregulated conditions.[7] Human PET and fMRI studies have shown that palatable food also activates the same mesolimbic dopamine reward system, which positively reinforces the preference for high-energy food and addictive eating behavior.[27] The mesolimbic dopamine system projects from the ventral tegmental area (VTA) to the nucleus accumbens (Nacc), where reward processing such as food seeking behavior and feeling of satiety takes place. Several other areas of the brain including the hippocampus, caudate putamen, amygdala, medial prefrontal cortex, hypothalamus are all regulatory regions that are activated in food craving.[27] For example, the hippocampus is likely to sustain and retrieve memories of desired food while the amygdala is activated in response to food-related stimuli, tastes and odor, and produces greater motivational incentive value.[27]

Both central neuronal circuits and peripheral signaling systems help to regulate food consumption. Ghrelin is released from the stomach and acts on the hypothalamus and the dopaminergic reward pathway to stimulate feeding while leptin relays information to the brain about the body's fat reserves to decrease food intake by inhibiting the reward circuit.[4,12,27] Thus, leptin and insulin resistance have been linked to obesity, as both become resistant in overeaters.[20]

A great deal of evidence from PET and fMRI studies have indicated that the obese brain shows changes in ways which not only reinforce food consumption but also blunt their ability to derive pleasure from other activities besides eating.[4,27] Extensive access to palatable food causes changes in the responsiveness of the reward system promoting food-seeking behavior, while the effects of these foods become less evident.[16] So, it is important to note that obese individuals exhibit different brain activity during the consumption and anticipation of palatable food when compared to lean individuals. Through the use of fMRI, Stoeckel et al. showed that obese women exhibited significantly greater activation in the brain regions mentioned above when shown pictures of high-calorie foods as compared to normal weight women.[23] This suggests that the limbic system in these obese women may be hyper responsive to food rewards. Gearhardt et al. further suggested that even though obese and substance-dependent individuals show heightened responsiveness in their reward system to food and substance cues respectively, their actual intake of food and drugs is associated with reduced reward circuitry activation.[12] Food addiction scores correlated positively with activation in the Nacc, medial orbitofrontal cortex, and amygdala in response to anticipated intake of palatable food but were not significantly related to activation in response to palatable food intake.

This evidence is consistent with research showing that activation in the nucleus accumbens and medial OFC is associated with craving in substance use disorders. This suggests that food addiction is similar to other addictive processes. In summary, the intensive craving for refined foods increases even as the satisfactory effect that it brings decreases over time causing one to overeat. Recent research also found that there is very little difference in the reward circuitry activation between highly food-addicted individuals and less severely food-addicted individuals.[12] The only difference was that participants with high food addiction scores showed neural activation patterns associated with reduced inhibitory control.[12] Gearhardt et al. proposed that increased frequency and dosage of palatable food consumption in highly addicted individuals might override one's desire to limit food intake, causing the disinhibition pattern seen in their altered neural circuitry.[12]

Overview of Hormonal and Genetic Background Involved in Refined Food Addiction

Dopamine

Normally, dopamine works with different hormones and acts on the prefrontal area, ventral medial hypothalamus and the arcade nucleus to prevent hyperphagia.[3] There is evidence that even though drugs of abuse like cocaine and heroin act differently physiologically, they both cause an increase in dopamine turnover in the brain. This abnormal increase in dopamine levels will produce the overloaded pleasure sensation known as a "high" and increased state of anxiety. Depression is a symptom of drug withdrawal. Likewise, carbohydrates such as sugar substitutes can prime the release of

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dopamine and may trigger recurring carbohydrate cravings or weight gain.[27] PET studies have shown that similar to drug-dependent individuals, obese individuals have lower dopamine-2 (DA D2) receptors in the striatum showing an inverse relationship between D2 receptor density and body mass index (BMI).[24]

The DA D2 receptor is associated with pleasure; with a decrease in DA D2 receptors, obese individuals tend to exhibit decreased sensitivity and blunted dopaminergic release during refined food consumption.[24] Research has shown that DA D2 receptor knock out mice developed addiction-like reward deficits and the onset of compulsive-like palatable food seeking behavior rapidly.[5] Thus, obesity is sometimes referred as having the "reward deficiency syndrome". Avena et al., showed that rats continuously consuming copious amounts of sucrose solution have significantly more dopamine-1 receptor binding, allowing the down regulation of dopamine-2 receptors.[1] Since dopamine is highly involved in our brain's reward center, the euphoric feeling produced by dopamine has an addictive effect reinforcing food-addicted individuals to have cravings for carbohydrate-rich foods. Davis et al. have indeed demonstrated that food addicts reported prominent impulsivity and stronger food cravings than the non-food addicted group.[7] Furthermore, long-term abuse of palatable food in obese individuals causes them to rely on consumption of these foods to increase dopamine levels through positive reinforcement.[23]

Serotonin

Kaye et al. have proposed that instability of the serotonin system may explain why bulimic individuals binge eat.[17] The same may be applied to explaining the mechanism of food addiction. Carbohydrate consumption leads to increased central production of serotonin, a neurotransmitter known for its calming and mood elevating effect.[9] Thus, carbohydrate cravings are also described as a craving for serotonin or tryptophan, a precursor for serotonin production in the brain.[9] For example, women with bulimia nervosa who also suffer from overeating experience a greater desire to binge when given a tryptophan-deficient amino acid mixture to drink compared to when they were given an amino acid-balanced mixture.[17]

Endorphins

In addition to their effect on dopamine and serotonin, refined foods also have an effect on the endorphin system, increasing endogenous beta-endorphin levels and producing an analgesic effect, leading to reinforcement of food cravings.[9] Pelchat et al. have shown that infants getting a heel stick for blood collection cry less when fed sugar water instead of plain water.[19] This endogenous opiate-mediated analgesic is repeatedly seen in numerous human models.[19,22,25]

Genetics

Besides refined foods reinforcing properties of the different major neurotransmitters in the human body, the craving for such foods is also hypothesized to have a direct genetic link. Fortuna found that people with paternal alcohol dependence history are three times more likely to prefer sweets than individuals who do not have this paternal history.[9] Moreover, it was observed that alcoholics and cocaine users are more likely to prefer sweet foods compared to non-alcoholics and non-cocaine users.[9]

The dopamine-2 receptor gene, also known as the reward gene, is crucial for experiencing pleasure in normal life. A deficiency in the number of dopamine 2 receptors is a common marker for increased risk of abuse alcohol and other drugs.[21] The A1 allele of the dopamine 2 receptors has been implicated in a wide variety of addictive disorders including alcohol abuse, cocaine dependence, bulimia, binge eating disorder and obesity. The presence of the TaqI A1 allele is strongly correlated with reduced dopamine-2 receptor density and the reduced sensitivity in the reward system.[4] This is evidence that food addiction could be the result of a mixture of both genetic and environmental factors. However, even though the presence of the TaqI A1 allele is correlated with overeating, extended access to palatable food can also cause the decrease in dopaminergic release in obese individuals even in the absence of the Taq1 A1 allele.[4]

Congenital leptin deficiency, which is a deficient leptin gene, has also been discovered to result in hyperphagia and acute weight gain.[24] For example, Volkow et al, found that subjects showed activation of the dopaminergic mesolimbic system to visual food stimuli even after they had just ate.[24] However, the mesolimbic system did not activate after 1 week of leptin treatment, which suggests that leptin diminished the rewarding responses to food.[24] Another fMRI study done on congenital leptin deficiency subjects also showed that leptin could diminish food reward and enhance the response to satiety signals generated during food consumption through modulation of neuronal activity.[27] Similarly, leptin-deficient mice also presented increased DA activity in reward-related brain regions.[20] Pfaffy et al. have shown that leptin increases DA D2 receptor binding in leptin-deficient obese, leading to weight lost in obese mice.[20]

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Intervention For Food Addiction

Obesity has been studied for decades, but previous preventions and therapies targeting a change in lifestyle and weight lost have only had minimal effects on a long-term reduction of body fat. Over the long term, many regimens have proved to be completely ineffective due to the high relapse of overeating and the lack of self-control. For example, in 1998, Bernstein has proposed of using an anorexient-like prescription or non-prescription medications, amino acids, and herbal preparations to reduce or eliminate carbohydrate craving.[2] However, he noted that this form of therapy would mostly be a life-long treatment.[2] Recent re-investigation for effective prevention and therapy models has looked to include modulation of environment risk factors such as socioeconomic status as well as media consumption.[13] An increasing number of research findings regarding the similarities between food addiction in obesity and drug addiction have led researchers to look into new treatment possibilities similar to addiction withdrawal models for treating obesity.[13] A number of pharmacological treatments derived from addiction models for obesity are either under development or in clinical trials, but all possess significant mood disturbances and other side effects.[4]

In an attempt to avoid unwanted side effects, Blum et al. have proposed a paradigm for amino acid therapy using a nutrigenomic formula.[3] It involves genetic testing to identify risk alleles and D2 receptor stimulation using neuroadaptogen amino acid precursor enkephlinase-catecholamine-methltransferase (COMT) inhibition therapy.[3] Since more recent studies support an association of the A1 allelic form of the DA D2 gene with the reduction of DA D2 receptors seen in obesity, substance abuse vulnerability and other compulsive behaviors, Blum et al. hypothesized that the proliferation of D2 receptors will attenuate drug-craving behavior.[3]

Classic addiction treatment protocol call for the understanding that addicts cannot trust their minds or bodies because the addiction has already unconsciously took over.[22] It encourages addicts to join group recovery programs to get guidance and accurate information from older members to achieve abstinence.[22] Just like alcoholics anonymous, food addicts in recovery anonymous based their program from the Twelve Steps of alcoholism.[26] Following through with the twelve steps, food addicts will have the opportunity to face themselves, stand up to the disease, and work towards recovery through spiritual awakening.[26] However, it is impossible to achieve long-term abstinence with food. Thus, recovery programs aim to focus on strategies that help food addicts to avoid refined foods, the primer that triggers uncontrollable eating.[25]

The development of evidence-based psychotherapy has motivated patients to develop coping strategies to handle high-risk relapse situations. Cognitive behavioral interventions such as community reinforcement approach, self-change, self-control training, and cue exposure treatment are among the interventions with the highest efficacy.[13] All of these interventions focus on the mesolimic reward system by teaching the patient to focus less on the supposed rewarding stimuli and focus on another reinforcing alternative.[13] These interventions not only help strengthen the patients' motivation to lose weight, they also provide patients with educational information about automatic responses to food-associated cues and urges, thus helping them to self-reflect on the positives and negatives of their actions.[13] It is noteworthy to recognize that cognitive factors such as social environment, emotional state, or intentional efforts to control consumption can also influence food intake.

Conclusions

Obesity is a chronic condition just like addiction and requires immediate attention from the medical community to advance prevention and treatment protocols. Current pharmaceutical and behavioral intervention guidelines are very similar between food addiction and drug addiction.[25] The validation of the refined food addiction model could help to manage the obesity epidemic. There are some psychotherapies such as the cognitive behavioral therapies that are being scrutinized, but further obesity treatments or interventions should be considered in the context of food addiction.

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EFFECTS OF STATIN DRUGS ON ATHEROSCLEROSIS AND CARDIOVASCULAR DISEASE DUE TO REDUCTASE INHIBITION AND NUTRIENT DEPLETIONS

by: Brett R. Martin MsAc, DC, and
Daniel L. Richardson Ms, PhD, DAANC

Older medications originally were derived from plant and animal sources to treat a wide range of medical conditions[1]. However, an increase in the production of synthetic pharmaceuticals has shifted products for the treatment of health-related conditions from organic sources to chemical; both synthetic and semi-synthetic products. Today, even drugs derived from animal and plant sources are made in pharmaceutical laboratories using specific chemical formulas.[1] The formula gives the medication its chemical and biological properties. These properties alter cellular functions, which determine the effects that the medication will have on the body.[1] Typically, more than one effect is generated by any given medication. The desired effect produced is known as the therapeutic effect, while other undesirable effects are known as side, or untoward effects.[1] The undesirable effects range from mild discomfort to potentially harmful complications. These undesirable effects may be due to the depletion of vitamins and nutrients.

One of the groups of medications commonly prescribed today are the 3-hydroxy-3-methylglutaryl-CoA (HMG-CoA) reductase inhibitors or "statin" drugs. These drugs are prescribed to patients who present with high levels of LDL cholesterol. Although statins have many beneficial effects that help to maintain the efficiency of the cardiovascular and circulatory system, these agents may reduce myocardial function[2] and according to Dr. Joseph Mercola, may contribute to congestive heart failure.[3] A possible mechanism to explain this potentially untoward result is the effect of statins in reducing the concentration of ubiquinone, also known as coenzyme Q-10 (CoQ10), β-carotene and vitamin E, resulting in a deficiency or depletion of these nutrients.[4] Deficiencies of CoQ10, β-carotene and vitamin E can be predisposing factors for the development of cardiovascular disease (CVD). The purpose of this review is to examine the potential consequences associated with the deficiency of CoQ10, β-carotene and vitamin E due to the administration of statin medications.

Background

The liver synthesizes high density lipoproteins (HDL) and very low density lipoproteins (VLDL) for cholesterol transport.[5] VLDLs are composed mainly of triacylglycerol and apolipoprotien B-100. VLDLs are released from the liver into the vasculature. Within the vasculature, VLDLs receive apolipoproteins C and E from HDLs. Apolipoprotein C activates lipoprotein lipase in the tissues, which catalyzes the cleavage of triacylglycerol molecules into free fatty acids to be used by the tissue and glycerol to be transported back to the liver.[5] Triacylglycerols are also transferred to HDLs. The abstraction of these compounds causes VLDLs to decrease in size and increase in density becoming intermediate density lipoproteins (IDL). The IDLs are converted into low density lipoproteins (LDL) by the removal of apolipoprotein C and E. Upon conversion, cholesterol and apolipoprotein B-100 become the predominant components of LDLs and apolipoprotein B-100 is the biological marker that allows LDLs to bind to cellular receptors.[5] Cells utilize LDLs for various physiological processes. If more cholesterol is required by a cell to maintain adequate function, synthesis of LDL receptors is initiated. This increases the cells capacity to bind to LDLs and absorb them through endocytosis.[5] Within the cell, LDLs are stored as unesterified cholesterol. Unesterified cholesterol has the ability to attach to areas within the intima lining of arteries at the sites of free radical damage or endothelial dysfunction resulting in the progression of tissue damage and a predisposition to arteriosclerosis.[5]

HDLs function as a natural mechanism of the body to

(Continued on next page)

minimize the amount of damage due to LDLs in the vasculature. After HDL cholesterol is produced in the liver, it is secreted into the vasculature. Within the peripheral circulation, HDL cholesterol functions as a reverse transport system by attracting and extracting triacylglycerol from low density lipoproteins (LDL), VLDL and incorporating these compounds into its structure.[5] HDL is then converted into HDL3. HDL3 delivers the triacylglycerol to the liver for degradation and excretion from the body and/or reuse in the biosynthesis of cholesterol.[5]

There are many different methods of controlling the ratio of HDLs and LDLs. These methods include diet, exercise, prescription drugs or natural supplementation with vitamins and botanicals. Each method of treatment has its benefits and can be effective to some degree. However, a very effective method of regulating cholesterol is statin drugs.[1] Statins are the most commonly prescribed cholesterol controlling agents.[1] These drugs are designed to inhibit the enzyme HMG-CoA reductase.[1] HMG-CoA reductase is the rate limiting step in the synthesis of cholesterol in the liver[1]. Therefore, it determines if conditions are adequate for cholesterol synthesis to occur. Inhibition of this enzyme forces the liver to produce more HMG-CoA reductase, but at the same time it increases the number of LDL receptors in the liver.[1] This allows the liver to filter out more LDLs carrying cholesterol from the vascular circulation for degradation resulting in a reduction in the concentration of LDLs.

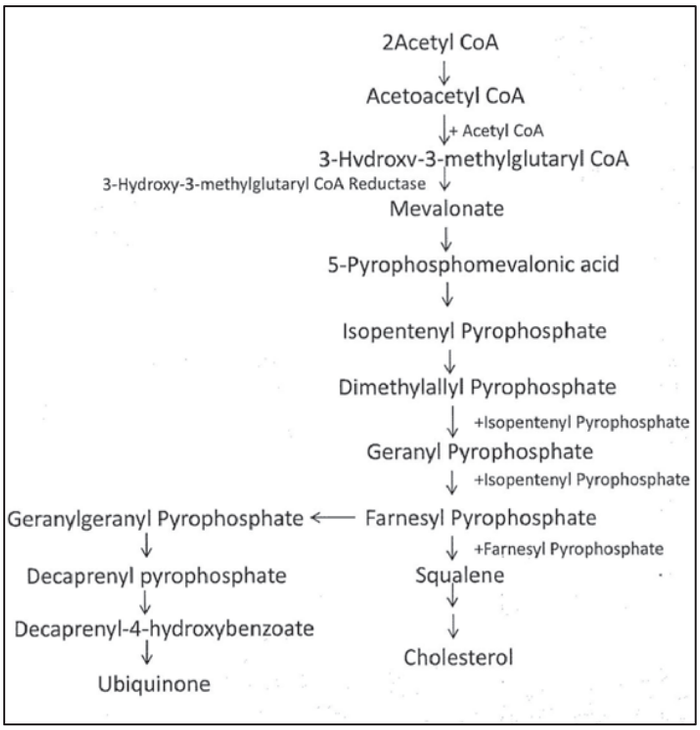
In addition to the aforementioned mechanism of action, statin drugs are capable of inhibiting the generation of apolipoprotein B-100.[1] As previously discussed, apolipoprotein B-100 is predominately found in VLDLs and LDLs. A reduction in the production of apolipoprotein B-100 prevents the synthesis of VLDLs drastically decreasing the concentration available for conversion to LDLs in circulation.

The last mechanism of statin drugs is the elevation of HDLs.[1] It should be noted that not all cholesterol lowering medications are capable of raising HDL levels.[1] However, most can raise HDL levels to some degree. On average statin drugs increase HDL levels by 5-16%.[1]

Discussion

Although the action of statin drugs is to reduce cholesterol synthesis, it also inhibits the production of CoQ10 (ubiquinone). CoQ10 and cholesterol share a common synthetic pathway.[6] HMG CoA reductase is the rate limiting enzyme for both of these products. Therefore, statin drugs ability to inhibit cholesterol synthesis will ultimately reduce the production of CoQ10. Figure 1 illustrates cholesterol and ubiquinone synthetic pathways.[6]

Figure: 1



Statin drugs may also lower plasma levels of other fat soluble vitamins such as β-carotene and vitamin E.[7] Currently, the mechanism of lowering plasma levels of these nutrients is unknown. However, the reduction in VLDL and LDL levels may reduce the transport of both β-carotene and vitamin E thereby lowering the plasma levels. About 58-73% of β-carotene is incorporated into LDLs for transport.[8] Another consideration is that vitamin E prevents β-carotene from being oxidized during digestion and absorption.[8] Therefore, reduction of vitamin E levels impairs digestion and absorption of β-carotene.

CoQ10 is an important amphipathic chemical that functions in the electron transport chain (ETC).[5] The ETC is located within the inner mitochondrial membrane. The ETC is involved with adenosine triphosphate (ATP) production. ATP is cellular energy. The ETC consists of a series of four complexes and cytochrome proteins. Electrons are donated and accepted in a sequence of oxidation-reduction reactions between the complexes and cytochrome proteins. The ETC utilizes the reduced forms of nicotinamide

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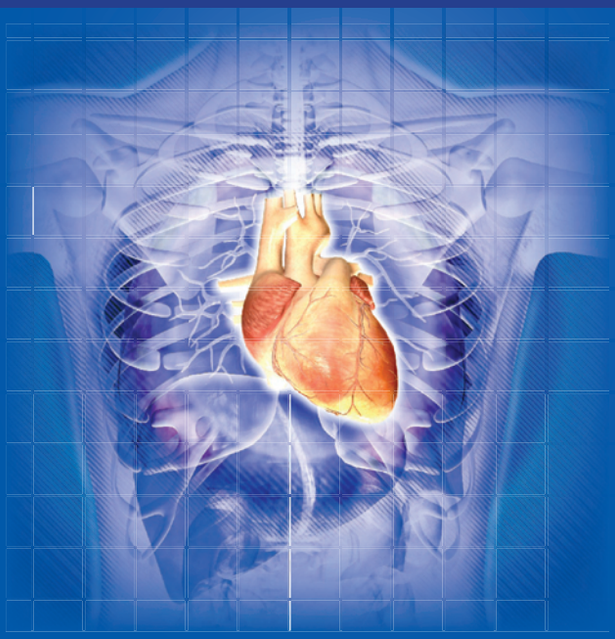

- Inflammation and Cholesterol are Key Components of ***Cardiovascular Disease.***
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adenine dinucleotide (NADH) and flavin adenine dinucleotide ($FADH_2$) produced from glycolysis and the tricarboxylic acid cycle (TCA), to initiate the transfer of electrons[5]. CoQ10 accepts electrons from either complex I (NADH dehydrogenase) or complex II (Succinate dehydrogenase) and shuttles the electrons to complex III (Cytochrome c dehydrogenase).[5] From complex III, cytochrome c carries the electrons to complex IV. The chain terminates at complex IV and the electrons are accepted by an oxygen molecule to form water.

The transfer of electrons along the chain within the inner mitochondrial membrane produces free energy in an electrochemical gradient. The energy produced by the electrochemical gradient is conserved and is sufficient to pump hydrogen ions from the inner mitochondrial matrix into the intermembrane space.[5] Pumping hydrogen ions into the intermembrane space decreases the pH thus producing a pH gradient as well as an electrochemical gradient. Complex V (ATP synthase) also uses the energy produced by the ETC in conjunction with the pH gradient to transport hydrogen ions from the intermembrane space back into the mitochondrial matrix. As hydrogen ions are transported through complex V, adenosine diphosphate (ADP) is phosphorylated, producing an ATP molecule.[5]

The energy produced by the ETC is essential for tissues that are highly metabolically active such as cardiac tissue. The cardiac musculature continuously contracts all day. Therefore, the cardiac musculature requires an abundant amount of energy to maintain its function. The energy demands of the cardiac musculature are satisfied by fatty acids.[5] Fatty acid oxidation produces acetyl CoA, $FADH_2$ and NADH. Each acetyl coA molecule is capable of entering the TCA generating 3 NADH, 2 $FADH_2$ and 1 GTP.[5] Each $FADH_2$ molecule can yield 2 ATP while each NADH molecule can create ~3 ATP and each GTP can produce 1 ATP.[5] Oxidation of a 16 carbon fatty acid would generate 7 NADH, 7 $FADH_2$ and 8 acetyl coA, which can yield 131 ATP.[5]

Although a large amount of ATP can be produced from fatty acid oxidation, the process is dependent upon the ETC. The NADH and $FADH_2$ molecules produced cannot be utilized unless CoQ10 is available within the ETC to accept their electrons to create the electrochemical gradient. Dissipation of the electrochemical gradient results in the inability to pump hydrogen ions into the intermembrane space. Thus, hydrogen ions will not be available for complex V to transfer back into the matrix for phosphorylation of ATP. Lower amounts of ATP generated due to a CoQ10 deficiency can interfere with that ability of the cardiac muscular to contract.

A cascade of events must occur to initiate a muscular contraction. First an action potential needs to be generated. Within the cardiac musculature two separate channels are opened. The first are the fast sodium channels and the second are the slow opening calcium channels.[9] The influx of both sodium and calcium into the cardiac muscle fiber propagates and maintains a prolonged depolarized state. The prolonged depolarization results in a plateau in the action potential. Calcium ions continue to enter the muscle fiber during the plateau period stimulating the contractile process and reducing the permeability of potassium efflux[9]. Efflux of potassium ions initiates repolarization of the cell terminating the action potential.

The action potential diffuses along the transverse tubules (T tubules) to the interior of the muscle fiber interacting with the longitudinal sarcoplasmic tubules.[9] This potentiates the release of calcium ions from the sarcoplasmic reticulum into the sarcoplasm. In addition, calcium channels within the T tubular membrane are opened increasing the concentration of calcium within the sarcoplasm. These calcium ions interact with ryanodine receptor channels. Ryanodine receptor channels are located in the membrane of the sarcoplasmic reticulum and stimulate the release of calcium from the sarcoplasmic reticulum.[9] These calcium ions interact with troponin to initiate the chemical reactions necessary to promote the cross-bridge between actin and myosin filaments. The myosin head contains an ATPase enzyme that is required for the degradation of the high energy phosphate bonds of ATP producing ADP.[9]

A CoQ10 deficiency can interfere with the cardiac function by reducing the amount of ATP available to sustain the cross bringing between the myosin and actin filaments generated during a muscle contraction. Reducing the contractile nature of the cardiac musculature may reduce its ability to continually transport blood to the other tissues. This may result in a compensatory increase in the rate of myocardial muscle contraction and induce vasoconstriction and thus elevating blood pressure. Elevating the blood pressure increases peripheral resistance requiring a stronger contractile force to pump the blood throughout the body. A combination of the greater contractile demands and increased rate for prolonged periods of time can contribute to left ventricular hypertrophy.

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This sequence of events for a prolonged period of time can result in a variety of conditions such as atherosclerosis, hypertension and congestive heart failure (CHF).

A study was performed evaluating the effects of statin drugs on left ventricular diastolic function. During the study, 14 asymptomatic patients prescribed statin drugs were examined using a Doppler echocardiograph to determine the left ventricular diastolic function[10]. Evidence from the echocardiograph demonstrated that 10 out of the 14 patients evaluated expressed more than one marker indicating a regression of the left ventricular function.[10] The study also showed that CoQ10 supplementation reversed dysfunction.[10]

A correlation between CHF and CoQ10 deficiency has been observed in several studies. In these studies, CoQ10 deficient patients typically demonstrate a reduction in left ventricular ejection fraction. A case study involving 23 patients evaluated the effects of CoQ10 supplementation versus placebo.[11] Each patient was assigned to one of four groups: 100 mg CoQ10, 100 mg CoQ10 plus exercise, placebo or placebo plus exercise. The investigators found that the subjects administered CoQ10 had a four-fold increase in plasma antioxidant activity and that exercise attenuated the effects[11]. There was also a 3% increase in HDL levels in the groups receiving CoQ10.[11]

An increase in oxidative stress was also noted in the above study. Oxidative stress is caused by free radical damage. Free radicals are generated by a wide variety of normal biochemical processes within the body as well as from exposure to smog, ozone, chemicals, drugs, radiation and high oxygen levels. The activity of the mitochondria largly contributes to the production of free radicals. As previously discussed, the ETC functions by exchanging electrons through a series of oxidation-reduction reactions. However, during this process ubiquinone (CoQ10) is converted to semiquinone, which is an intermediate free radical. Reduction of semiquinone results in the release of a superoxide radical.[8] Within the ETC, superoxide radicals can also be generated by electrons leaking from the autoxidation reactions and associated with an oxygen molecule. Oxygen molecules can interact with catecholamines such as epinephrine and dopamine to produce superoxide.[8] It is important to note that superoxide radicals are well contained due to their poor ability to diffuse great distances and they are relatively weak species.

The superoxide radicals generated are converted into hydrogen peroxide by superoxide dismutase. Hydrogen peroxide is a more potent reactive oxygen species (ROS) with a higher tendency to diffuse farther away from its origin.[8] Glutathione peroxidase is capable of converting hydrogen peroxide into two water molecules while catalase converts it into an oxygen molecule and two water molecules. Failure of these enzymes to catalyze the conversion allows hydrogen peroxide to diffuse and interact with other organic compounds found within the cell causing damage and producing hydroxyl radicals.[8] Hydroxyl radicals are capable of migrating the farthest and are highly reactive.[8]

Hydroxyl radicals interact with organelles within the cell, the nucleus and the plasma membrane. The first step of the process is initiation. Initiation is characterized by the hydroxyl radical reacting with an organic molecule. Polyunsaturated fatty acids (PUFA) within the plasma membrane are particularly at risk due to the double bonds between carbon atoms attached to a methylene group, which contains reactive hydrogen atoms.[8] The hydroxyl radical oxidizes the methylene group generating a lipid carbon-centered radical.

The next step is propagation. In this phase, the carbon-centered radical combines with molecular oxygen to form a lipid peroxyl radical.[8] The peroxyl radical can extract a hydrogen atom from another PUFA, perpetuating the oxidation process and thus destabilizing the integrity of the cell.[8]

Oxidative damage occurring within the vasculature produces two separate mechanisms that contribute to atherosclerosis and the progression to cardiovascular disease (CVD). The first process is associated with oxidative damage resulting in endothelial cell dysfunction. Endothelial cell dysfunction impairs the ability of the vasculature to release nitric oxide (NO).[12] NO is a potent vasodilator. A reduction in NO activity prompts an imbalance between vasoconstrictive and vasodilatory compounds resulting in an increased peripheral resistance and the potential to develop CVD. Endothelial cell dysfunction produces procoagulant activity, an increased expression of adhesion molecules and the generation of ROS and inflammatory mediators.[12] All of these are positively correlated with the development or exacerbation of atherosclerosis.[12]

The second mechanism contributing to atherosclerosis and CVD due to oxidative damage is the attraction of unesterified LDLs to damaged endothelial tissue. Unesterified LDLs are capable of adhering to the site of tissue damage in the intima lining of the arteries.[8]

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Within the intima lining, apoprotein B-100 has the potential to become oxidized.[8] Oxidized LDLs and ROS activate monocytes causing them to attach to the intima. Within the intima, monocytes are converted into macrophages. Macrophages possess receptors that are specific for oxidized LDLs. The macrophages phagocytize the oxidized LDLs and are transformed into foam cells.[8] Foam cells release growth factors and cytokines, promoting cellular proliferation. Foam cells become trapped in the intima causing them to accumulate thus perpetuating endothelial cell damage and resulting in the formation of fatty streaks. Platelets are recruited to the site of tissue damage. They bind and accumulate, forming soft atheromatous plaques.[8] The plaque formation narrows the lumen and contributes to sclerosis of the vasculature. Eventually this process can lead to the restricted and turbulent blood flow that is characteristic of atherosclerosis.[5]

It should be noted that the severity of damage incurred by the second mechanism in patients with hypercholesterolemia will be more prominent than in patients on statin drugs due to the ability of statin drugs to reduce cholesterol levels. However, not all statin drugs are capable of alleviating endothelial cell dysfunction and oxidative damage even at normal LDL cholesterol levels.[13] Therefore, although normal cholesterol levels are being maintained in patients prescribed statin drugs, there is still the potential for adherence of unesterified LDL cholesterol to the intima due to endothelial cell dysfunction and oxidative damage.

β-carotene, vitamin E and CoQ10 all have potent antioxidant effects and implement a protective function within the cell membranes. Vitamin E and β-carotene work synergistically within the cell membrane to reduce the amount of oxidative damage caused by peroxyl, hydroperoxyl and carbon-centered radicals,[8] while CoQ10 is capable of reducing hydroxyl radicals preventing oxidative damage.[8]

A deficiency of these agents not only hinders their ability to perform their functions and increases damage caused by ROS, but it also impairs their potential to regenerate other antioxidants. CoQ10 reduces vitamin E from its oxidized state allowing it to function as an antioxidant again.[8] Failure of antioxidants to be regenerated creates an imbalance that can alter the oxidative status.

Several studies support the cardioprotective effects of vitamin E and β-carotene and a few denote the association between vitamin E deficiency and atherosclerosis. Studies have demonstrated the ability of β-carotene and vitamin E to reduce LDL oxidation[8]. Supplementation with β-carotene and vitamin E has been positively correlated with a decreased risk of developing CVD.[14] Supplementation was also found to reduce the occurrence of both nonfatal and fatal myocardial infarctions.[15] An epidemiological survey conducted in Thai adults found that there was an association between atherosclerosis and vitamin E deficiency.[16]

Studies have also shown the cardioprotective function of CoQ10. One study in particular evaluated the antioxidant potential of CoQ10 on diabetic rats. The rats were treated with 15 mg/kg of CoQ10 daily for 8 weeks and then compared to a placebo group. Upon evaluation of the treatment, it was found that CoQ10 significantly reduced LDL oxidation and inhibited leukocyte infiltration and glomerulosclerosis compared to the placebo group.[17] It was also noted that CoQ10 significantly increased the serum levels of glutathione, catalase and superoxide dismutase.[17] All of these effects will benefit the cardiovascular system.

Conclusion

Statin drugs are very useful pharmaceutical agents for the lowering od LDL cholesterol and potentially raising HLD and therefore help in the prevention of cardiovascular disease. However, these drugs may contribute to the deficiency or depletion of vitamins and nutrients that are essential for the maintenance of cardiovascular health. CoQ10, vitamin E and β-carotene all exhibit cardioprotective effects. Impairing the function of these agents has been correlated with an increased risk of atherosclerosis. Although statin drugs reduce LDL cholesterol which reduces the risk of atherosclerosis, they have no effect on oxidative status or endothelial cell dysfunction. Therefore, we suggest patients taking Statin drugs be supplemented with these nutrients during and following administration of the statins in order to promote optimum health and prevent the consequences of potential depletion of nutrients.

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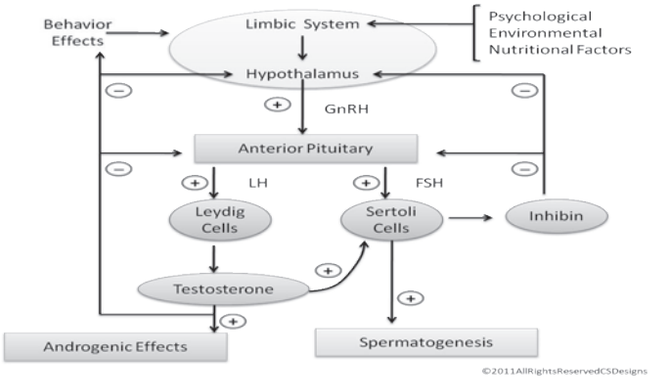
HORMONAL CONTROL OF MALE SEXUAL FUNCTION

by: Wayne Sodano, DC, DABCI, DACBN



A majority of the control of sexual functions in the male (and the female) begins with secretions of gonadotropin-releasing hormone (GnRH) by the hypothalamus.[1] You may remember that the hypothalamus is driven by the limbic system, and therefore many psychological factors can influence the release of GnRH. GnRH stimulates the release of two other hormones, luteinizing hormone (LH) and follicle stimulating hormone (FSH) from the anterior pituitary.

Both LH and FSH are glycoproteins that exert their effect on the testes, which in turn activate specific enzyme systems in the testes. LH stimulates the interstitial cells of Leydig to synthesize and secrete testosterone. The majority of the circulating testosterone is made by the Leydig cells. FSH binds to the receptors on the Sertoli cells in the seminiferous tubules where it causes the Sertoli cells to grow and secrete spermatogenic substances. Testosterone and dihydroxytestosterone (DHT) enter into the interstitial spaces of the seminiferous tubules where they have a strong effect on spermatogenesis. Therefore, FSH and testosterone are the regulators of spermatogenesis. The Sertoli cells also secrete a glycoprotein hormone called inhibin in respond to spermatogenesis occurring to rapidly. This hormone decreases the secretion of FSH and GnRH (inhibins are also secreted by the ovary).

There is a symbiotic relationship between the brain and testosterone. Many of the central nervous system functions are regulated by testosterone, among them is behavior and cognition. The inputs to the central nervous system, such as psychological stress, can lower the release of GnRH, and therefore decrease serum testosterone levels. Low serum testosterone is also a component of insulin resistance. A research study in 2009 concluded that low serum testosterone was independently associated with insulin resistance in non-diabetic older men.[2]

From a functional medicine perspective, it's important to assess for environmental factors that can influence testosterone level. Since over 90% of the testosterone is produced in the testis by the Leydig cells, it important to assess for factors that might inhibit Leydig cell production of testosterone. Disruption of androgen biosynthesis and actions by environmental endocrine disrupting compounds can inhibit critical cellular processes controlling steroidogenesis in the Leydig cells. Disruption can occur with the transport and delivery of cholesterol to the mitochondria, interference with the enzymatic activity along the steroidogenesis pathway, or by interfering with the androgen receptor.

Table 1: Natural and synthetic chemicals affecting Leydig cell function.

Chemical	Proposed target	Application or source
Procymidone	Androgen receptor antagonist	Fungicide, control of plant diseases
Linuron	Androgen receptor antagonist	Herbicide, postemergence control of weeds in crops
Vinclozolin	Androgen receptor antagonist	Fungicide
p, p 'DDT	Androgen receptor antagonist	Pesticide
Dioxins	Aryl hydrocarbon receptor agonist	By-product of chlorinated hydrocarbons
Phthalates	Peroxisome proliferator-activated receptors (PPARs)?	Plasticizers
Genistein	ERs stimulator	Soy-derived food
Resveratrol	ERs stimulator	Red wine, red grape
Bisphenol A	ERs stimulator	Synthesis of polycarbonate plastics

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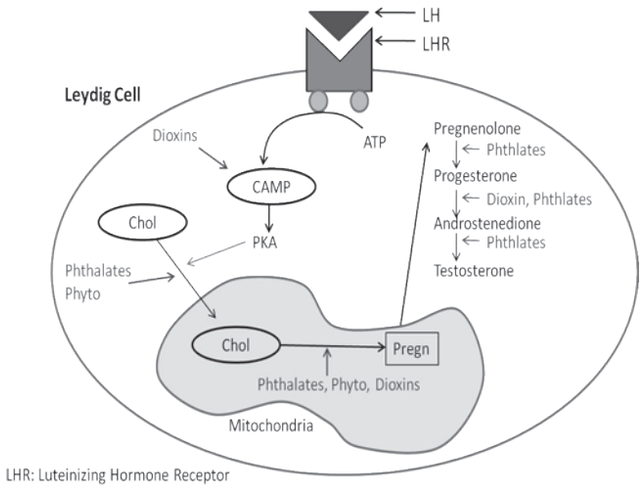
On the previous table you will notice that compounds that stimulate estrogen receptors affect Leydig cell function. The inhibitory effect of estrogens on male reproduce-tive function appears to be mediated by suppression of LH. A direct effect of estrogen on Leydig cell steroidogenesis has also been demonstrated.[3]

The Potential Sites of Action of Endocrine Disrupting Compounds



LHR: Luteinizing Hormone Receptor
PKA: Protein Kinase A (cholesterol transport to mitochondria)
Chol: Cholesterol
Phyto: Phytoestrogens
Pregn: Pregnenolone

'The action of endocrine disrupting compounds on Leydig cell function and the reproductive potential is a complex process that depends on the exposure route, dose, the developmental stage of the target organism and many other factors. Together, these factors determine the potential risk for adverse consequences with long-lasting effects on the male reproductive function'.[3]

'Together with Leydig cells, adult rat germ cells are able to express P450arom mRNA, which is translated as a biologically active enzyme involved in estrogen production. Consequently, germ cells not only produce estrogens but contain estrogen receptors as well, which would explain part of the role (autocrine and/or paracrine) of estrogens in male germ cell development. The mechanism of action of estrogens in the reproductive organs of the male remains to be clarified, as well as the regulation of aromatase gene expression, especially in germ cells during testicular development. Nevertheless, we have begun to understand the physiological roles (as well as the pathological effects) of these female hormones in males, and, obviously, their involvement in several steps of sperm production and maturation. Thus it is anticipated that parts of male gonadal function are not only androgen regulated but also estrogen controlled in mammals.' [4]

Male Infertility

Research studies between the late 1930's and the mid-1990's have found a substantial decline in sperm concentration and sperm quality among men living in industrialized countries. Observational studies and animal research suggest that exposure to various environmental pollutants may be contributing to this decline in male fertility, possibly by exerting estrogenic or other endocrine-disrupting effects. [5]

Suspected Contributing Factors to Male Infertility[6]

- Toxins from cigarette smoke
- Alcohol and drugs that lower sperm count (cocaine, marijuana, [certain antifungals, antihypertensives, and antibiotics] cimetidine, methotrexate, sulfasalazine and others)
- Heavy metal – lead mercury and cadmium
- Obesity – excess body fat converting testosterone to estrogen
- Environmental toxins – bisphenol a, phthalates, vincolozolin (fungicide), pesticides, dry-cleaning agents, and others.
- High soy intake
- Oxidative stress – spermatozoa are highly sensitive to oxidative stress because of their high concentration of polyunsaturated fatty acids and inability to repair damages membranes.
- Hypothyroidism
- Celiac disease and food allergy
- Poor diet

Advanced Lab Testing Considerations for Male Infertility

Aside from primary lab testing, advanced functional medicine test should be considered as indicated by the patient history, physical examination and primary lab testing. The functional/advanced tests of consideration include:

- Organic acid test
- Toxicity Profile test
- RBC Nutrient and Toxic Elements
- Urinary Porphyrin test
- Celiac test/Food allergy
- ASI test
- RBC fatty acid test

(Continued on page 120)

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Treatment and Nutritional Considerations

The main treatment include: optimizing liver function, far-infrared sauna treatment, optimal fatty acid balance and the use of antioxidants.

- Zinc
- L-Arginine
- L-carnitine and acetyl-L-carnitine
- Vitamin E
- Selenium
- Vitamin C
- Lycopene
- B vitamins
- Essential fatty acids

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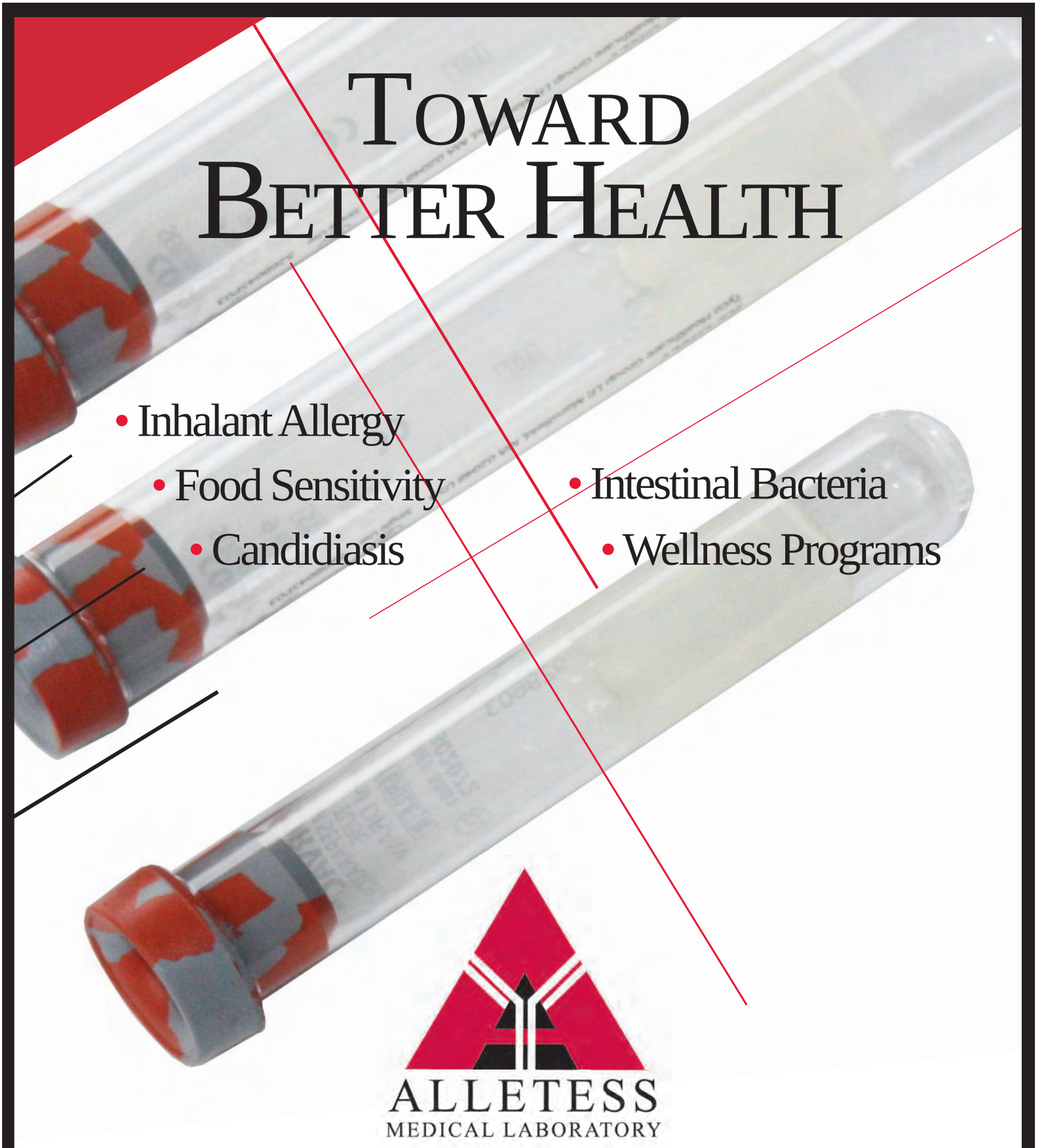
Dr. Wayne Sodano has over 26 years of combined private practice and teaching experience in functional medicine under the paradigm of natural internal medicine. He is a diplomate of the American Board of Chiropractic Internists and the American Clinical Board of Nutrition and is a former instructor for the Diplomate program for the American Board of Chiropractic Internists. Dr. Sodano is noted for his past radio appearances and current lectures on various functional medicine topics that include Celiac Disease, malignant diseases, AIDS, gastrointestinal disorders, and nutrient and toxic elements. Dr. Sodano's lecture topics are continually expanding and may be found on www.functionalmedicineuniversity.com. ◆



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CHILDREN NUTRITIONAL NEEDS
(Cont. from page 96)

in offsetting multiple disease processes, and in turn provide the nutritional capacity for optimal development and health.

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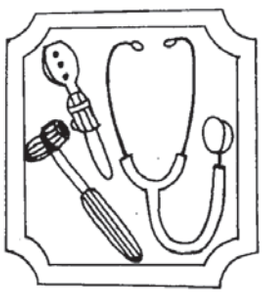

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