

HERBS AND NUTRIENTS FOR THE MIND

A Guide to Natural Brain Enhancers

Chris D. Meletis, Jason E. Barke







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Complementary and Alternative Medicine Chris D. Meletis and Margot Longenecker, Series Editors



Westport, Connecticut London

Library of Congress Cataloging-in-Publication Data

Meletis, Chris D.

Herbs and nutrients for the mind : a guide to natural brain enhancers / Chris D. Meletis and Jason E. Barker.

p. cm. — (Complementary and alternative medicine, ISSN 1549–084X) ISBN 0-275-98394-3 (alk. paper)

1. Herbs---Therapeutic use. 2. Dietary supplements. 3. Neurobehavioral disorders-Alternative treatment. I. Barker, Jason E. II. Title. III. Series. RC350.H47M44 2004 616.8'04654—dc22 2004048057

British Library Cataloguing in Publication Data is available.

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Library of Congress Catalog Card Number: 2004048057 ISBN: 0-275-98394-3 ISSN: 1549-084X

First published in 2004

Praeger Publishers, 88 Post Road West, Westport, CT 06881 An imprint of Greenwood Publishing Group, Inc. www.praeger.com

Printed in the United States of America

(∞)

The paper used in this book complies with the Permanent Paper Standard issued by the National Information Standards Organization (Z39.48–1984).

10 9 8 7 6 5 4 3 2 1

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Series Foreword

The world of medicine has evolved with the changing needs and demands of the patient, the third-party payer, and a growing appreciation of the intimate relationship shared between the healthcare provider and patient. The evolution of medicine is not limited to these facets alone. An active reflection of the origins and heritage of medicine leads to a redefinition of medical care. Whether all forms of medicine must be compared to "Western-allopathic" medicine is now being overtly challenged by many patients and some conventional healthcare providers.

Many forms of medicine claim the Hippocratic model as their founding paradigm. However, the question could be raised as to whether Hippocrates would be accepted in the second millennium A.D. by his modern peers. Indeed, would his empirical approach to medicine stand up fully to the current medical model of scientific burden of proof?

Statistics show us that over 70 percent of the world's population uses something other than the Western-allopathic form of medicine as a primary source of medical care. This is not to dismiss the need for modern medicine, but rather to serve as a pivot for reflection of other forms of medicine that have sustained generations prior to our current era. In fact, some 25–33 percent of frequently used conventional prescription medicines originate from natural substances.

It is this new appreciation and objective perspective that has fostered the popularity of what is commonly called Complementary and Alternative Medicine or CAM. It is worthwhile to note that in order to have a complementary and alternative form of care, a single model must proclaim itself the primary form of care.

The goal of this series is to offer valuable insights into medical therapies currently categorized in the realm of CAM by Western-allopathic medicine. As more and more clinical trials are performed leading to scientific validation, current CAM therapies become embraced as mainstream treatment options. The intent of this series is to review healthcare therapies. The criteria for review are that the particular therapy has a foundation of clinical success, partial or full research validation, and/or rich historical use. The reviews of different therapies will provide critical insight into additional adjunctive therapies that might be incorporated in patient care. They will also provide a heighten appreciation of CAM as well as enhance the ability to converse about CAM therapies in an everevolving medical model.

Reflection on the present humbles all disciplines; for as we judge our predecessors, so shall we be judged by future generations for both current brilliance and shortcomings. Medicine is a part of an evolving reality. It is up to each provider to enrich history in the making.

> Chris D. Meletis Margot Longenecker

Introduction

The adage "The mind is a terrible thing to waste" truly lacks a depth of understanding and critical perspective essential to support foundational healing. When addressing brain-centered health promotion, patient and clinician alike must astutely appreciate that the "mind" is inseparable from the "body"; the brain, without question, is a physical structure that in its absence would eliminate any discussion of the concept of mind, psyche, or mental function. As an organ that influences the function of the entire being, the brain is central to both mental and physical health.

A simplistic illustration that compares the heart's function to that of the brain can be made to exemplify an appreciation of the brain and its function relative to mental and physical well-being. The heart beats over 100,000 times a day; it does so prior to our birth to the moment we die without exception, so we hope. This constant and reliable performance, giving blood throughout the body, serves as the cornerstone of the circulatory system and the flow of life. If the heart, which requires a constant source of energy, were to become inadequately fueled without the proper nourishment, it would begin to fail to maintain the very circulatory system that sustains our existence. Classically described conditions such as vitamin B-1 deficiency (known as wet beriberi) as well as recently discovered nutrient (Coenzyme Q10 and carnitine) deficiencies all contribute to the health of the cardiovascular system.

Such is the dilemma faced by the brain. Regardless of the brain's resilience, there is a time and a place in which inadequate intake can begin to yield the signs and symptoms of psychological or organic brain-altered function. Notable is that proper brain functioning is largely individual and can be affected by internal and external variables that may alter the needs of a given person's brain to function within normal expectations. Numerous nutrients must be present in sufficient levels, including B-1 as with the heart, to maintain brain function, yet variables and individual genetic signatures can change the required needs.

Whether it is heart or brain function as the center of discussion, both function as a result of a complex series of biochemical reactions, yielding nerve impulses and chemical reactions that maintain function. Notable in both circumstances is that the difference between the adequate and optimal functions of each can make the difference between surviving and thriving. The presentation of the clinical and germane medical research relative to supporting brain function targets the augmentation of function, with the goal of shifting away from surviving and becoming closer to thriving within the confines of human understanding of the miraculous creation of the central processor, the brain, which governs the human body's entire operating system.

Indeed the concepts of mind, mental and psychological attributes are all dependent upon a very real and physical component of the body, that being the human brain. Often, the terms "mental" or "psychological" are attributed to the mind portion of the mind-body connection.

Throughout this work, it is our hope that the reader does not look at the mind as separate from the body. Realize that the physical presence of a properly functioning brain is the requisite for mental health, accepted psychological presentation, and healthy functioning of the central nervous system. We have thus intentionally included such conditions as Parkinson's and multiple sclerosis in the list of health conditions addressed. Though Parkinson's and multiple sclerosis reflect a well-delineated spectrum of organic brain-altered function, they are in many ways no more organic than the patient with depression.

This book offers a select view of natural medicine interventions, focusing on nutritional and botanical treatments, referred to within the confines of these covers as "Nutra-botanical" therapies, evidenced by clinical practice and research findings. The individual sections of this work are intended as springboards for further investigation by the reader. Often, the human dilemma arises from accepting limits when perceptions of confining factors seem to have been reached even though a new perspective may be just a glimpse away over the walls of our personal reality's boundaries.

It is not the intent of this book to advocate for the replacement of standard drug therapy.

However, individuals suffering from a health condition affecting brain performance should not limit their options to drug therapy when other biochemical interventions could perpetuate heightened function and the ever-important quality-of-life issue. A concerted effort for the integration of natural medicine approaches alongside standard drug therapy should be pursued in every patientdoctor relationship. The sharing of all therapeutic interventions being pursued with all clinical providers is of paramount importance in order to avoid unnecessary potential drug–natural medicine interactions. Tell your medical doctor if you are on any medication and use—or plan to use—natural treatments.

By supporting health through the use of optimal nutrition, people are placed in a decision-making role regarding their health. Previously, health was attributed to nothing more than luck and the use of medicines designed to treat the symptoms of the disease itself. With today's new focus on prevention of disease by fueling the body and its systems correctly, people can now make a decision to pursue health rather than simply react once a disease manifests.

May all that read the following pages do so with an open mind and—equally important—a healthy brain.

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PART I

Brain Ailments and Nutra-Botanical Interventions

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ADD/ADHD

Attention deficit disorder (ADD), formerly known as attention deficit/hyperactivity disorder (ADHD), is one of the most common mental disorders among children today. It is estimated that approximately 3 percent to 5 percent of all children (two to three times as many boys are affected than girls) or nearly 2 million American children (which correlates to one child in each classroom in the United States) have ADHD according to the National Institute of Mental Health.¹ ADHD does not only affect children, as symptoms can progress into adulthood as well.

The specific causes of ADHD are currently unknown, with several factors being responsible in different people. No solitary causative factor has been identified as being responsible for the different behavior patterns observed in ADHD. ADHD is only diagnosed by certain characteristic behavior patterns that are observed over time; no other clear physical signs can be seen. Common behavioral pattern categories in ADHD include inattention, impulsivity, and hyperactivity.

- Inattention: This is marked by difficulty in keeping the mind focused on any one subject and a short attention span. People with ADHD often become bored after only a few minutes at work on a subject, and placing focused attention on new or unfamiliar topics can be challenging.
- Impulsivity: This is marked by an inability to refrain from immediate reactions, making it difficult to wait and first think before speaking or acting.
- Hyperactivity: This is marked by constant perpetual motion; staying in one place and sitting still can be difficult. Adults may feel quite restless and may start several projects and have a difficult time finishing them.

Diagnosis of ADHD is based upon an analysis of the person's behavioral patterns, which are compared to established criteria. These criteria are defined in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*. The manual outlines the three previously mentioned behavior patterns, and people may display varying amounts of each pattern or only one. Because nearly everyone displays some of these symptoms at some time in their life, certain criteria, including age of onset (early in life, before age seven), duration of symptoms (continuous for at least six months), frequency (occurring more often in themselves than others of similar age), and most importantly, behavior(s), must occur in at least two different areas of the person's life, namely, school, home, work, or social settings.

A recent report issued by the Centers for Disease Control and Prevention claimed that nearly 1.6 million elementary school-aged children have a diagnosis of ADHD, and a national survey revealed that the parents of 7 percent of children ages 6–11 years old were told by a healthcare professional that their child had ADHD.² The report also included the following demographic information: boys are nearly three times as likely to have ADHD than girls; white children are twice as likely than Hispanic and black children to have a diagnosis of ADHD; children with health insurance are diagnosed with ADHD more often than children without health insurance; and children with ADHD use more healthcare services, including mental health services, than those without ADHD. This report went on to propose that ADHD is probably overdiagnosed in those with regular access and may be underdiagnosed in those with limited healthcare access.

A common neurodevelopmental disorder, ADHD results in impaired educational processes, social growth, and adaptation that lead to increasing rates of behavioral difficulty, depression, school dropouts, and substance abuse,³ which have lead to the mass prescription of stimulant psychotropic medications in children affected with this disorder. With no fully established biological causes recognized, ADHD does display prominent heritability. Mainstream treatment focuses on the use of mainly stimulant drugs, and because of the perceived relative success of these drugs in alleviating ADHD symptoms, many studies have focused mainly on genes that are responsible for the development and regulation of brain neurotransmitter systems, specifically that of dopamine, wherein the physiologic basis for the action of these drugs exists.

Genetic factors do play a role in the genesis of ADHD; estimates of heritability are greater than those of nearly every other child and adolescent psychiatric disorder and first-degree relatives have increased rates of ADHD, including conduct and affective disorders as well as substance abuse and dependency. Additionally, the subtypes of ADHD (impulsivity, hyperactivity, inattention) do not correlate with that of additional family members, leading researchers to conclude that nongenetic factors are responsible for intrafamialial variability.⁴ Factors other than genetics have been implicated in the development of ADHD prior to birth. Prenatal exposure to nicotine and psychosocial adversity have been identified as risk factors for ADHD; a review of the studies in ADHD literature exploring the relationship between prenatal exposure to these factors and the risk of developing ADHD revealed that smoking (specifically nicotine exposure) and exposure to psychosocial stress during pregnancy indicated greater and modest risk, respectively, in contributing to the development of ADHD.⁵ Other causes/contributors of ADHD that have been implicated in the literature include food sensitivities and allergies, food additive intolerance, imbalance and deficiency of nutrients, environmental toxicity (including heavy metal poisoning, thyroid irregularities, and other toxic pollutants).⁶

NUTRITIONAL FACTORS

The role of vitamins and minerals in brain function is equally important to their contribution to other organ systems. Just as organs and tissue systems may be compromised by inadequate or imbalanced nutrients, the functioning of the brain is easily affected by these imbalances or lack thereof; research points to the benefits of supplementation for nutrient deficiencies that resulted in improved academic and behavioral performance in ADHD children.⁷ One hallmark study followed the effects of vitamin and mineral supplementation in healthy schoolchildren over 18 years old. Researchers found that supplementation with vitamins and minerals resulted in significantly less antisocial behavior and improved cognitive performance in children taking the supplements when compared to those taking a placebo.⁸ However, improvement was not noted unless a frank nutrient deficiency of at least one nutrient (most often folic acid, thiamine, pyridoxine, vitamin C, or niacin) was found in blood testing. Because of findings such as these, the importance of multivitamin and mineral supplementation comes into play. It is interesting to note that these children had actual bloodlevel deficiencies; this comes at quite a nutritional cost with the wide food availability seen in modern times in the United States. Multinutrient dosing provides a backup strategy in the event of inadequate intake, or more commonly, inefficient absorption and utilization of these nutrients. A body that does not receive or is unable to fully utilize the nutrients necessary for optimal functioning will exhibit symptoms of dysfunction at its weakest areas. Studies investigating the use of B vitamins have vielded interesting results as well. One investigator employed varying combinations of B vitamins to successfully treat hyperactive children (more properly known as hyperkinesis) who did not respond to treatment with Feingold's diet (a diet espousing the removal of food additives and salicylate-containing foods).9

One B vitamin in particular, vitamin B-6 (pyridoxine) has been shown to be an effective treatment for hyperactive children; a double-blind study comparing the use of vitamin B-6 to methylphenidate (also known as Ritalin—the most commonly prescribed drug for ADHD) revealed a slightly greater effectiveness of B-6.¹⁰ What is even more interesting in this study is that the researchers based their idea for the study on the observation noted by other doctors that hyperactive children tended to have lower blood levels of a specific neurotransmitter (serotonin) and that supplementing with large amounts of B-6 normalized the levels of serotonin and subsequently improved the children's behavior. B-6 serves as an enzymatic cofactor in the metabolism of several neurotransmitters, including serotonin, dopamine, and histidine.¹¹ The use of B-6 among physicians for the treatment of ADHD symptoms is widespread; it enjoys a positive reputation among clinicians treating these patients.

Phosphatidylserine is a biological molecule known as a "phospholipid." Phospholipids are one of the main components of cellular membranes in the human body and serve to stabilize the other constituents of which the cellular membranes are composed. Phosphatidylserine is the main phospholipid of human brain cells, and it serves to regulate cellular functions such as controlling the internal environment of the cell, communication between cells, signal transduction (communication from outside the cell to within), release of secretory vesicles (another mode of cellular communication), and regulation of cell growth and division.¹² Phosphatidylserine is beneficial to several different brain functions and also contributes to nerve cell synaptic membranes, a key anatomical aspect of nerve signal production and transmission. As a supplement, its benefits include increased neurologic energy via facilitated synaptic communication and increased production, release, and effectiveness of the neurotransmitter dopamine.¹³ One study investigating the use of phosphatidylserine supplementation in ADHD patients resulted in a slightly greater than 90 percent improvement in these cases, with doses of 200 to 300 milligrams per day for up to four months providing the greatest absolution of symptoms.¹⁴ Supplemental administration of phosphatidylserine is thought to normalize brain lipid content, thereby assisting the return of normalized function of neuronal cells.¹⁵

OTHER NUTRIENTS

- Iron: Insufficient iron is one of the most common nutrient deficiencies among children in the United States,¹⁶ and it is known to contribute to decreased attention span, activity, and persistence. Supplementation of nonanemic children with ADHD resulted in fewer symptoms of ADHD (marked by a 30 percent improvement).¹⁷ Interestingly, iron serves as an essential cofactor in the synthesis of the brain neurotransmitters dopamine, norepinephrine, and serotonin, and deficiency in the early years of life can negatively affect neural and behavioral development.¹⁸ It is essential to note that iron poisoning is the leading cause of accidental poisoning, thus guidance by a skilled healthcare provider is essential and close monitoring is a must.
- Magnesium: Magnesium is another commonly deficient nutritional mineral. Magnesium supplementation can be helpful in alleviating some symptoms of ADHD. In one study, one group of children with ADHD was treated for six months with supplemental magnesium and changes

in ADHD symptoms were compared to another group of ADHD children who did not take the supplement. Investigators noted a "significant" decrease in hyperactivity symptoms in the treatment group.¹⁹ Another study demonstrated actual deficiencies of magnesium in 95 percent of ADHD children studied, leading the researchers to conclude that magnesium deficiency in children with ADHD occurs more often than in healthy children without ADHD.²⁰

Zinc: A collection of studies reveal that the level of this mineral is low in people with ADHD.²¹ and lower serum zinc levels are found in children with ADHD in comparison to children without.²² A relationship exists between levels of free fatty acids in the blood and zinc in children with ADHD; these children, when compared to controls without ADHD, were found to have low blood levels of zinc and free fatty acids.²³ This indicates that a deficiency of zinc may contribute to the development of ADHD; the study hypothesized that the low levels of free fatty acids may be a result of the decreased zinc levels. Another interesting study revealed a relationship between the responsiveness to standard stimulant pharmacotherapy and zinc levels in the body: low zinc levels equated to poor treatment response from the medication.²⁴ Zinc serves as a cofactor in the synthesis of neurotransmitters and indirectly affects dopamine metabolism, a neurotransmitter that is believed to be involved in ADHD (low levels of dopamine are associated with ADHD, and supplementation of dopamine has alleviated some ADHD symptoms).²⁵ As the principal investigator of the ZAD (zinc-attention deficit) study, one of the authors of this book, Chris D. Meletis, has noted, through the course of reviewing the scientific literature and clinical findings over the last decade, a clear relationship between low levels of zinc in relation to copper stores and notable signs and symptoms of attention dysfunctions and cognitive deficits.

Because solitary nutrient deficiencies have been implicated in ADHD, it stands to reason that inadequate doses of the earlier mentioned nutrients in combination may act synergistically to cause ADHD. A group of researchers determined that the most common nutrient deficiencies among children with ADHD were magnesium, copper, zinc, calcium, and iron, and these deficiencies occur more often among hyperactive children than healthy children; these deficiencies were determined by measuring their levels in blood serum, red blood cells, and in the hair.²⁶ Of the implicated nutrients, magnesium was the most frequently deficient. Additionally, when the researchers supplemented the ADHD children with magnesium, zinc, and calcium, hyperactivity was decreased; and when a group of these children was treated with standard therapy minus magnesium, symptoms of hyperactivity actually increased.

These studies underscore an important revelation in ADHD in that the cause of these symptoms seems to be related to one or more types of suboptimal nutrient levels. A common theme carried throughout this book and noted in medicinal literature is that each individual has a unique weakness that becomes more manifest when certain environmental influences are exerted (in this case inadequate levels of micronutrients). When adequately supplied with correct nutrition, symptoms are often diminished and can be attenuated with time. In the treatment of the person with ADHD, it may not be as important to discover the exact nutrient or nutrients that are lacking in order to alleviate symptoms. By prescribing a full-spectrum nutritional plan (that may or may not include dietary alterations and supplements), people with ADHD receive the nutritional factors that are needed to avert the manifestation of their symptoms. Patients' individual nutritional needs should be taken into account prior to prescribing a nutritional treatment plan. An individualized approach is important; each person with ADHD may react differently to various nutritional factors. Both current and past dietary practices and habits are important to consider; the development of physiologic systems depend on varying levels and types of nutrients at different periods during the course of development.

Nutritional factors other than deficiency play a large role in the symptomology of ADHD. Food additives, refined sugars, food sensitivities, and food allergies have been linked to ADHD; mounting evidence has shown that children with ADHD will react to more than one food and or its components, leading to negative behaviors.²⁷ Dietary modification plays an equally important role to ensuring adequate nutritional supplementation; both treatments should be part of the ADHD treatment regimen. One of the most influential dietary approaches in the treatment of ADHD is the Feingold diet. Starting in the1970s, Feingold claimed that the cause of up to 50 percent of hyperactivity in children was attributable to food additives, including artificial colorings, flavors, and preservatives as well as naturally occurring salicylates.²⁸ Feingold arrived at this conclusion by investigating 1,200 cases of food additive-linked behavioral and learning disorders in patients; he implicated more than 3,000 different food additives in these cases. A large body of research is dedicated to negating this relationship, however Murray and Pizzorno, in reviewing the outcome data from theses studies, report that 50 percent of the children in these studies actually improved (experienced less hyperactivity) when on the Feingold diet.¹⁷

There are approximately 5,000 food additives in use today, most of which are used to preserve and enhance the appearance of food; Americans consume nearly 15 grams per day on a per capita basis (nearly 100 million pounds of food coloring alone is ingested on a yearly basis in the United States).¹⁴ A study of 78 children with hyperactivity were placed on an elimination diet designed to remove offending foods that may cause hyperactive symptoms and 59 of these children experienced less hyperactivity while on the diet.²⁹ In a crossover portion of the study, the researchers were able to disguise previously established offending foods by mixing them in foods that were tolerated. This resulted in worsened behavior and impaired psychological test performance, demonstrating that observable changes in behavior associated with diet are reproducible using

double-blind methods. The investigators used this point to emphasize the ability of the parent/teacher/caretaker's ability to note the relationship between food ingestion and behavior outcomes and to consider these observations as valid when they present this association to the family physician.

A review of 23 double-blind studies investigating the roles of food dyes versus ordinary foods as the cause of worsened ADHD behavior revealed a worsening of symptoms following dye consumption in eight of nine studies using ADHD children. There was improvement when a food additive-free diet was consumed. In 10 of the other 14 studies, children with ADHD and asthma, food allergies, and/or eczema saw their symptoms improve when additive-free foods were consumed.³⁰ Other subjects in these studies experienced a worsening of symptoms when they consumed food dyes, corn, wheat, dairy products, soy, oranges, and chocolate. Another study demonstrated that 73 percent of ADHD children responded favorably to a food additive elimination diet and also worsened when certain foods, dyes, and additives were reintroduced into their diet.³¹ Another study employed the use of a diet consisting of rice, turkey, pear, and lettuce in the treatment of ADHD symptoms. Of the children studied, 62 percent demonstrated an improvement of symptoms of 50 percent or greater on the Connors list and the ADHD Rating Scale at the end of the study period, leading the researchers to conclude that ADHD children can experience statistically significant symptom improvement when placed on an elimination diet.³² These studies demonstrate the benefits of removing certain foods that may be suspect in causing ADHD symptoms; foods containing additives and those that may be allergenic should be among the first to eliminate from the diets of patients with ADHD—they more than likely play a significant role in the etiology of ADHD. Dietary modification should be attempted prior to treating symptoms with pharmaceuticals.

CONCLUSION

With no specific etiology, ADHD is a perplexing condition that continues to increase in incidence. This chapter includes only a sampling of the studies demonstrating various links between ADHD and nutritional factors. In addition to micronutrient deficiencies, food additives and allergenic foods play a large role in the treatment of ADHD. Although it is difficult to imply that these are the causes of ADHD, research does show a causal relationship: when such foods are removed, patients have fewer symptoms, and when patients are supplemented with the correct amount of nutrients, negative symptoms decrease. Readers of this chapter are strongly encouraged to investigate the pharmaceuticals that are prescribed for ADHD; a large amount of data regarding their toxicity and effects on the developing nervous system are a must-know.

The question that must be posed is whether all the millions of children diagnosed with ADD/ADHD were born drug deficient or whether a deeper fundamental cause needs to be addressed. An analogy may be drawn between the performance and potential of a large eight-cylinder Corvette sports car and the health of a newborn: regardless of the inherent potential of such a highperformance vehicle, if it is operated with suboptimal fuel, then the full horsepower and drivability of such an amazing creation will not be realized, just as the human body's complex fuel needs are essential for peak performance.

NUTRIENTS

- Diet A diet that includes carbohydrates, proteins, and fats in a ratio of 40:30:30 percent three times per day
- Multivitamin/mineral A suitable age-specific supplement should be taken twice per day with meals
- B vitamin complex One capsule of B complex twice per day with meals.
- Phosphatidylserine 200–300 milligrams per day
- Iron Must establish the presence of a deficiency prior to supplementing this nutrient*
- Magnesium
 5 milligrams par kilogram hadu weight par
 - 5 milligrams per kilogram body weight per day
- Zinc
 - 25 milligrams per day

*Testing for food allergies and food sensitivities is imperative.

Alcoholism (Alcohol Abuse/ Dependence)

DEFINITION

Although alcoholism, or alcohol abuse or dependence, is not necessarily caused by deficiencies of nutritional factors, it may be propagated by and definitively leads to frank nutrient deficiencies with startling health effects. Alcoholism, or alcohol dependence, is by definition a disease. Alcohol dependence has a chronic, progressive course, follows a predictable course, and has symptoms; and the risk of developing alcohol dependence is influenced by a person's genes and lifestyle. Cravings can be a strong as the need for food or water, and a person who is alcohol dependent will continue to drink despite its negative effect on family, career, and health. The four most common symptoms of alcohol dependence are:

- Cravings
- Loss of control (unable to stop drinking once drinking has begun)
- Physical dependence manifested by nausea, sweating, tremors, and anxiety when alcohol is withdrawn
- Tolerance, manifested by the need for increasing amounts of alcohol in order to feel the effects of alcohol

More expansive definitions of alcohol dependence and abuse have been developed for clinical and research purposes; this criteria is included in volumes such as *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, published by the American Psychiatric Association, as well as in the *International Classification Diseases*, published by the World Health Organization.

STATISTICS (PREVALENCE AND ETIOLOGY)

The most recent statistics surrounding alcoholism places the lifetime prevalence of the disease in the United States at 20 percent (9.8 million) in men and 8 percent (3.9 million) in women, with a heritability (attribute to both genetic and lifestyle influences) for both sexes at 50–60 percent.¹ Afflicting more than 14 million Americans (1 out of every 13), alcoholism is often associated with several other predisposing disorders such as antisocial personality, depression, anxiety, and tobacco addiction (nearly 80 percent of alcoholics are cigarette smokers). Recent evidence classifies alcoholics into two broad categories: Type 1 alcoholics begin drinking later in life in response to feelings of anxiety, guilt, and avoidance of harm. Type 2 alcoholics are more often men with decreased levels of the neurotransmitter serotonin in their brain and who act impulsively and antisocially.

Recent statistics reveal nearly 20,000 people died from alcohol-induced deaths, excluding motor vehicle fatalities in one year in the United States, 62 percent of 18-year-old and older Americans drank alcohol in the past year, 32 percent of drinkers had five or more drinks on one occasion at least once in the past year, and 61 percent of men 18–24 years and 42 percent of women had five or more drinks on the same occasion.² The toll of alcoholism and drunk driving has been well publicized in the last two decades, with social and judicial tolerance decreasing substantially. The disease of alcoholism itself is lesser appreciated; as like many people with varying types of chronic disease, alcoholics are at some point along their disease continuum able to maintain outward appearances of normalcy.

Causes of alcoholism vary between genetic and lifestyle influences. As stated earlier, a large percentage (50–60 percent) of children of alcoholics will be alcohol dependent themselves. Additionally, researchers have been searching for a definitive genetic link that explains the origins of alcoholic behavior. A recent ongoing study, "The Collaborative Study on the Genetics of Alcoholism (COGA)," is searching for the genes that may contribute to alcoholism and some of its related traits (phenotypes) that include depression. The study so far has revealed a positive link between depressive syndrome (depression that may or may not occur in concert with increased alcohol intake) and alcoholic subjects. Further, this study has linked alcohol dependency and depression to specific chromosomal regions, namely on chromosome #1, suggesting that a solitary gene or genes on chromosome #1 may predispose people to depression and/or alcoholism, which may be induced by depression.³

Specific nutritional deficiencies have not yet been elucidated in the cause of alcoholism itself. However, the progression of the disease is definitively marked by specific conditions resulting from specific nutrient deficiencies and their effects on the human body. As alcoholism progresses, the brain, liver, gastrointestinal tract, and pancreas are severely affected. Nutrient deficiency in alcoholism is attributed by decreased intake (chronic progressive alcoholics derive more and more calories from alcohol rather than food), reduced storage as a result of decreased food intake and nutrient replacement, and impaired utilization due to the effects of alcohol on the gastrointestinal tract.

BOTANICAL MEDICINES IN THE TREATMENT OF ALCOHOLISM

The effects of botanical medicines on alcoholism are providing interesting results. Several herbs demonstrate a reducing effect on voluntary alcohol intake in animal models of alcoholism, suggesting interesting new forms of therapy for alcoholism, and therefore may also demonstrate a preventative effect in individuals prone to this disease. Among the herbs with these effects are Hypericum perforatum (St. John's Wort), Peuraria lobata (kudzu), *Salvia miltiorrhiza* (Dan Shen), and Tabernanthe iboga (Iboga). Additionally, these plants demonstrate an ability to reduce alcohol absorption from the gastro-intestinal tract.⁴

Salvia miltiorrhiza

The use of this herb in reducing alcohol intake in laboratory animals has been demonstrated in several recent studies. Salvia is a botanical medicine with a long history of use in China. Administration of a standardized extract of Salvia dependently delayed alcohol drinking in ethanol-preferring dose animals and was compensated by increased water intake.⁵ Another study demonstrated the ability of the standardized extract of the herb to reduce alcohol intake by 40 percent in animals that were conditioned to prefer alcohol; this effect is attributed to the ability of the extract to alter ethanol absorption from the gastrointestinal tract: 200 milligrams per kilogram of Salvia miltiorrhiza decreased blood alcohol levels by up to 60 percent compared to control animals. Furthermore, alcohol-dependent animals dosed with Salvia extract were less able to discern the effects of alcohol-laden water from plain water than other animals trained to do so; the authors of the study conclude that the reducing effect of Salvia miltiorrhiza extract on ethanol absorption in animals may have caused a decreased perception of the psychoactive effects of ethanol.6

Additionally, *Salvia* also demonstrates antirelapse effects.⁷ Alcohol-dependent animals demonstrate a transient increased rate of alcohol consumption in comparison to previous levels after a period of deprivation. Considered to model alcohol relapse in human alcoholics, alcohol-dependent animals treated with *Salvia* extract exhibited a complete suppression of extra alcohol consumed following deprivation. Because of these findings, *Salvia* may possess antirelapse properties in addition to its alcohol-curbing properties and may constitute a novel strategy for reducing and controlling alcohol consumption in human alcoholics.

Hypericum Perforatum

Hypericum, also known as St. John's Wort, has been established as an effective treatment for mild to moderate depression. Both depression and alcoholism share similar nuerochemical weaknesses, such as decreased brain serotonin levels. In one study, a standardized extract of St. John's Wort was shown to be significantly effective in decreasing alcohol intake, and these effects did not decrease due to tolerance after consecutive doses.⁸ In another experiment. alcohol-preferring animals were given a dry extract of Hypericum and a 30-40 percent reduction in alcohol intake was noted.⁹ It was noted in this study that the effects of Hypericum were selective in that food or water intake was unmodified, and further examination revealed that the decreased alcohol-ingesting effects were not attributable to the antidepressant effects of the herb (decreased alcohol consumption was noted after a single administration of the medicine. whereas antidepressant effects were only noted after repeated doses), and the effects were not related to altered pharmacokinetics of alcohol either. Hypericum has been demonstrated repeatedly to inhibit alcohol intake in alcohol-dependent animals, vet a clear mechanism has not been established. Further studies are needed to identify the exact mechanism for St. John's Wort on alcohol intake; however, the existing findings demonstrate St. John's Wort as a potential therapuetic agent in the treatment of alcoholism.

Pueraria Lobata

Kudzu (Pueraria lobata) exerts several profound pharmacological actions including antidipsotropic (antialcohol abuse) activity. Pueraria has a history of use in treating the symptoms of alcohol overdose (hangover), including stomach upset, headache, nausea, vomiting, and dizziness. In traditional Chinese medicine, kudzu was used for managing alcoholism and drunkenness and other disease conditions. An extract of kudzu, known as daidzein, can decrease alcohol consumption and blood-alcohol levels, as well as decrease the duration of alcohol-induced sleep in animal models; and the ability of kudzu to lower bloodalcohol levels is attributed to its ability to delay gastric emptying, slowing the entrance of alcohol into the bloodstream.¹⁰ Other affects of kudzu on the body, which may contribute to its use in treating excessive alcohol intake, include its ability to decrease platelt aggregation; its antioxidant ability; the ability to dilate heart and brain blood vessels, increasing flow to these areas; and increased blood oxygen levels.¹¹

An extract of the plant was shown to suppress the alcohol intake of alcoholdependent animals when given a choice between water and alcohol.¹² Researchers attribute two isoflavone constituents of the plant, daidzin and daidzein, for this action. Additionally, other studies have repeated and confirmed the suppressant effect of this plant on both genetically alcohol-dependent animals and on animals that were trained to crave large amounts of alcohol. Earlier research suspected that daidzin was capable of inhibiting an enzyme known to detoxify alcohol known as aldehyde dehydrogenase. Disulfiram, otherwise known as antabuse, is a pharmaceutical medication used by some patients incapable of stopping alcohol intake that operates on this mechanism. When a person consumes alcohol while taking this medication, only small amounts of alcohol will cause tremendous nausea and physical suffering, acting as a deterrent to continued alcohol intake. However, newer research reveals that inhibition of aldehyde dehydrogenase is not the mechanism that inhibits drinking behavior, and it is suspected that daidzin operates in a different biochemical pathway. Newer research has revealed that daidzin inhibits a second step of a pathway known as MAO/ALDH-2 (monoamine oxidase/aldehyde dehydogenase-2), a pathway of alcohol detoxification in the body, leading to its suppressive effect on alcoholcraving animals.¹³

Tabernanthe Iboga (Iboga)

Native to Africa, Iboga has been used ceremonially as a hallucinogen. A powerful medicinal plant, Iboga has several pharmacological effects that have led it to be employed in the use of breaking addictive cycles, including tobacco and alcohol addiction. An extract of this plant (Ibogaine) can cause stimulation of the brain (central nervous system) ranging from mild excitation to euphoria and hallucinations.¹⁴ Additionally, iboaine exerts serotonergic effects, meaning that it can mimic the effects of this neurotransmitter, which is often found in low amounts in alcoholics. Animal studies have shown that these effects may exert some value in the treatment of human addiction, including alcoholism.¹⁵ Ibogaine administered to animals exerts short-lived decreases in alcohol intake, and it is suspected that longer term effects may be mediated over long-term treatment periods with this plant extract as the extract is stored in fatty tissues, allowing for a sort of time release effect.¹⁶ Iboga and its constituent iboaine have been used successfully in breaking cycles of addiction; further studies of the plant medicine may reveal greater understanding of its use in breaking alcohol dependency.

NUTRIENTS AND ALCOHOLISM

Thaimine

The most well-known vitamin deficiency associated with alcoholism is that of thiamine (B-1) deficiency. Classically, long-term deprivation of this vitamin leads to Wernicke-Korsakoff syndrome and features neurologic symptoms such as confusion, memory loss, impaired movements, and peripheral neuropathy. Wernicke-Korsakoff syndrome is actually two disorders that can occur independently or together. Wernicke's disease involves damage to the central and peripheral nervous systems, and can include alcohol withdrawal symptoms. Korsakoff syndrome involves impairment of memory and intellectual skills. The most distinctive symptom is confabulation, or fabrication of facts as the person tries to fill in gaps in memory when recounting experiences. Depending on time of treatment and how long the patient has been deprived of thiamine, this condition may or may not be reversible. Treatment of this condition involves the administration of thiamine intravenously and in repeated doses over a period of days to weeks.

Zinc

Zinc is required for several biological functions including DNA synthesis, cell division, and expression of genes. Additionally, zinc is required for the functioning of numerous enzymes in biologic systems and immune system function. Alcoholism is a predisposing factor for zinc deficiency due to the effects of alcohol on nutrient absorption. Zinc can positively affect the metabolism of alcohol in the body and can reinforce the functioning of both stomach and liver alcohol dehydrogenase enzymes.¹⁷ These effects can lead to increased metabolism of alcohol in the body, thereby negating some of its negative side effects, especially in the liver and brain.

The development of alcohol dependence is accompanied by a decrease in zinc content in an area of the brain known as the hippocampus, and supplementation of zinc may prevent this deficiency.¹⁸ In another interesting study demonstrating the importance of zinc and alcoholism, alcohol-dependent animals were shown to have deficient zinc brain levels, and when supplemented with zinc, alcohol consumption was reduced.¹⁹ These studies demonstrate a link between zinc and healthy brain functioning; supplementation of zinc in chronic alcoholics may serve to improve treatment and prevent some negative long-term effects of deficiency.

Niacin

Deficiency of niacin (vitamin B-3) is known to occur in alcoholics as well. Frank deficiency of niacin leads to a disease known as pellagra. Pellagra leads to the triad: dermatitis, diarrhea, and dementia, eventually followed by death; skin changes are characteristic and define the condition by themselves. Often masked by other alcohol-related nutritional deficiencies, pellagra can coexist with other vitamin deficiency diseases in chronic alcoholics. Because of this, supplementation with a multivitamin and mineral is imperative in the treatment of chronic alcoholic disease, although pellagra by itself is responsive to niacin therapy.

Considered a mainly psychiatric disease, the known familial and genetic influences on this disease have become increasingly well defined. Indeed, the adage that genetics may load the gun, but diet and lifestyle pull the trigger rings true when it comes to alcoholism. Not everyone with a family history of alcoholism becomes an alcoholic. Nutritional, botanical, and lifestyle can all help ward off the consequences of alcoholism. A preemptive strategy for individuals with family histories of alcohol abuse should at a very young age (at least age 12) take a multivitamin that has abundant sources of chromium and zinc in balance with other nutrients. Taking these nutrients in the form of a high-quality multivitamin is better than individual dosing, since these and most nutrients are dependent on the synergy of other vitamins and minerals. Another important consideration is to look for early signs of hypoglycemia that clinically appear to present in some, but not all, alcoholics. Thus the presentation of fluctuations of blood sugar after eating or if a meal is missed may be a warning sign indicating that working with a nutritionally oriented physician could be helpful.

BOTANICALS*

- Hypericum perforatum (St. John's Wort) 300 milligrams (standardized to 0.3 percent hypericin or 4 percent hyperforin content), three times daily
- Pueraria lobata (Kudzu) 1,500 milligrams root extract, twice daily
- Salvia miltiorrhiza (Dan Shen) 2,000–3,000 milligrams, twice daily
- Tabernanthe iboga (Iboga)

The standardized extract Ibogaine is efficacious at 200–300 milligrams twice daily. However, this herb is hallucinogenic and should only be used under close medical supervision.

NUTRIENTS**

- B-1 (Thiamine) Doses of 5 to 300 milligrams have been used depending on state of deficiency. A good starting dose is 10 milligrams twice per day.
- B-3 (Niacin) 100 milligrams three times per day
- Chromium 200 micrograms twice per day
- Zinc 40 milligrams per day, divided doses with food

*It is important to consume these doses with a multivitamin/mineral supplement, as many nutrients are dependent on each other for proper assimilation and physiologic synergy.

**All herbs should be taken in capsule or tablet form or as a tea; liquid extracts should be avoided due to potential alcohol content that can be as high as 50 percent (100 proof).

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