Attention-Deficit Hyperactivity Disorder

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Attention-deficit hyperactivity disorder (ADHD) is the most common neurologic condition in children and comprises a range of behavioral problems, including inattention, hyperactivity, and impulsivity. In most children with ADHD, the cause is unknown, but is thought to be multifactorial. According to the Centers for Disease Control and Prevention (CDC), 7.4% of children ages 3–17 in the United States have been diagnosed with ADHD, totaling 4.7 million children. Boys are diagnosed much more frequently with ADHD than girls, with 9.5% of boys ages 3–17 diagnosed with ADHD, compared with 5.9% of girls of the same age range.[1] Additional research has shown that ADHD is 2.3 times more common in boys than girls. Comorbid conditions, such as mood disorders and anxiety, are also common.[2]

ADHD often persists into adulthood, with a prevalence greater than 4% among adults.[3] According to the CDC 2003 data, 2.5 million youths ages 4–17 are currently receiving medication treatment for ADHD.[4] In a survey of children with ADHD, 67.6% of families reported current or past use of complementary and alternative medicine (CAM) to manage this condition including modified diet, vitamins and/or minerals, dietary supplements, aromatherapy, and chiropractic.[5]

Diagnosis

ADHD is a chronic condition characterized by inattention, impulsive hyperactivity, or both, such that daily functioning is compromised. The symptoms of the disorder must be present at levels that are higher than expected for a person's developmental stage and must interfere with the person's ability to function in different settings. Additional criteria from the *DSM-IV** include symptoms causing impairment present before age 7; some impairment from the symptoms present in two or more settings (e.g., at school/work and at home); clear evidence of significant impairment in social, school, or work functioning; symptoms that do not occur only during the course of a pervasive developmental disorder, schizophrenia, or other psychotic disorder; and symptoms that are not accounted for better by another mental disorder. Three types of ADHD have been established based on which symptoms are strongest in a given patient.

Predominantly Inattentive Type

According to *DSM-IV* criteria, six or more of the following symptoms of inattention should be present for at least 6 months to a point that is disruptive and inappropriate for developmental level:

(1) Often does not give close attention to details or makes careless mistakes in schoolwork, work, or other activities
(2) Often has trouble keeping attention on tasks or play activities
(3) Often does not seem to listen when spoken to directly
(4) Often does not follow instructions and fails to finish school work, chores, or duties in the workplace (not due to oppositional behavior or lack of understanding instructions)
(4) Often has trouble organizing activities
(5) Often avoids, dislikes, or does not want to do things that take a lot of mental effort for a long period of time
(6) Often loses things needed for tasks and activities (e.g., toys, school assignments, pencils, books, or tools)
(7) Is often easily distracted
(8) Is often forgetful in daily activities.

Predominantly Hyperactive-Impulsive Type

According to the *DSM-IV*, six or more of the following symptoms of hyperactivity-impulsivity should be present for at

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least 6 months to an extent that is disruptive and inappropriate for developmental level:

(1) Often fidgets with hands or feet or squirms in seat
(2) Often gets up from seat when remaining in seat is expected
(3) Often runs about or climbs when and where it is not appropriate (adolescents or adults may feel very restless)
(4) Often has trouble playing or enjoying leisure activities quietly
(5) Is often "on the go" or often acts as if "driven by a motor"
(6) Often talks excessively
(7) Often blurts out answers before questions have been finished
(8) Often has trouble waiting one's turn
(9) Often interrupts or intrudes on others.

Combined Type

Symptoms of the above two types are equally predominant in the person.[6]

Comorbid Conditions

Other conditions are common with ADHD. In fact, half of people with ADHD also have other mental disorders.[7] These conditions commonly include anxiety disorders and mood disorders. Sleep problems also occur frequently. According to one survey of children with ADHD, 28.5% had mild sleep problems and 44.8% had moderate or severe sleep problems. Moderate or severe sleep problems were associated with poorer psychosocial quality of life and poor daily functioning in the affected child as well as poorer family functioning, and caregiver mental health.[8]

In addition, children with ADHD may have more frequent and severe injuries, and treatment may incur significantly higher medical costs, compared with such costs for children without ADHD. Health care costs for children with ADHD may be more than twice as high as such costs for children without this condition.[9] Research also suggests that ADHD is a risk factor for subsequent substance abuse-disorders and that ADHD pharmacotherapy in childhood reduces the risk for substance abuse disorders.[10]

Etiology and Risk Factors for ADHD

The etiology of ADHD is not known. However, research indicates that causes of ADHD are multifactorial, including both genetic predisposition and environmental and perinatal influences. Many biologic and environmental factors have also been proposed as risk factors for ADHD, such as food additives, diet, lead contamination, cigarette and alcohol exposure, maternal smoking during pregnancy, and low birth weight.

Genetics

Numerous twin studies indicate that there is a strong genetic influence for ADHD.[11] Twin studies consistently indicate that heritability ranges from 60% to 90%.[12] In addition, studies have shown that approximately 25% of close relatives in the families of children with ADHD also have ADHD, compared with a rate of about 5% in the general population.[13] In fact, family studies have demonstrated a two- to eightfold increase in the risk for ADHD in parents and siblings of children with ADHD.[14]

Research suggests multifactorial, causes of ADHD, such as genetic predisposition and environmental and perinatal influences.

Due to the high heritability of this disorder, various genes are currently being studied. The majority of the candidate genes studied involve the dopamine, norepinephrine, and serotonin neurotransmitter systems. Some of these genes include the norepinephrine transporter (NET), dopamine transporter (DAT1) gene, the catechol-O-methyltransferase (COMT), the serotonin transporter promoter polymorphism (SERTPR) and the A/G variant within SERTPR, and the variable number tandem repeat (VNTR) variant in the dopamine D4 gene.[15–17]

Brain Abnormalities

Researchers have also found brain abnormalities in children and adults with ADHD. In one study, it was found that children with ADHD had 3%–4% smaller brain volumes in all regions including the frontal lobes, temporal gray matter, caudate nucleus, and cerebellum. In addition, children with ADHD who were on medication had a white-matter volume that was the same as the non-ADHD controls, while the never-medicated patients with ADHD had an abnormally small volume of white matter.[18]

It is believed that the dopaminergic system is involved in ADHD. There is additional evidence that the glutamatergic system is also abnormal, and it is theorized that there is a possible abnormal interaction between the two systems.[19] In one study, short echo proton magnetic resonance spectroscopy was performed to examine glutamate in the prefrontal cortex, left striatum, and the occipital lobe as a control area in male treatment-naïve children with ADHD and non-ADHD control subjects between ages 6 and 11. Striatal glutamate, glutamate/glutamine, and creatine concentrations were greater in the subjects with ADHD at baseline compared with controls, and only striatal creatine was reduced after stimulant treatment in the patients who had ADHD, providing evidence of striatal creatine/glutamatergic dysregulation in ADHD.[20]

Dietary Factors

Nutritional factors, such as food additives, refined sugars, food allergies, and essential fatty-acid (EFA) deficiencies have all been associated with ADHD.[21] The National Institutes of Health (NIH) held a scientific consensus conference in 1982 to discuss this issue, and it was established at that time that

diet restrictions helped about 5% of children with ADHD, mostly young children who had food allergies.[22] However, more recent research indicates that reactions to foods and food additives may be more pronounced.[23,24]

In the first phase of one study, 200 children with suspected hyperactivity were evaluated in a 6-week open trial using a diet free of synthetic food coloring. The parents of 150 children reported improvement in behavior with the dietary change, and deterioration of behavior with the introduction of foods containing synthetic coloring. The second phase of the study involved a double-blind, placebo-controlled, repeated-measures trial using 34 children that were suspected or uncertain reactors and 20 control subjects for 21 days. Placebo or one of six dosage levels of the food coloring agent tartrazine (1 mg, 2 mg, 5 mg, 10 mg, 20 mg, 50 mg), was administered randomly each morning, and behavioral ratings were recorded by parents. The researchers found that 24 children were clear reactors, who had irritability, restlessness, and sleep disturbances. Significant reactions were observed at all six dosage levels, and a dose–response effect was observed. In addition, the researchers noted that, with a dose increase greater than 10 mg, the duration of effect was prolonged.[23]

In another study, 55 children with suspected hyperactivity underwent a 6-week trial of consuming the Feingold diet.† At the end of the trial, 72.7% of the children had improved behavior and 47.3% retained the improvement following a modified diet over an additional 3–6 months. A second phase of the study was conducted with 8 children who were suspected reactors. The children were given a diet free from synthetic additives and were challenged daily for 18 weeks with a placebo or with 50 mg of either tartrazine or carmosine, each for 2 separate weeks. Two of the 8 children were identified as significant reactors whose behavior involved extreme irritability, restlessness, and sleep disturbances when the children were exposed to food additives.[24]

Environmental and Perinatal Factors

Numerous environmental factors have been associated with ADHD. In one study, environmental and perinatal factors were evaluated in a randomly selected sample of children ages 6–11, including 200 children with ADHD and 286 healthy controls. The diagnosis of ADHD was associated with various environmental and other factors, such as moderate-to-severe physical illness in the mothers during gestation, prenatal cigarette and alcohol exposures, miscarriage symptoms, premature delivery symptoms, maternal respiratory viral infections, neonatal seizures, asphyxia or anoxia, severe neonatal illness, mild speech retardation, moderate brain injuries, and febrile seizures.[25] Additional research indicates that higher blood lead concentrations are significantly associated with ADHD.[26]

Decreased levels of the EFA docosahexaenoic acid (DHA) may also be a factor. Research using animal models has demonstrated that reductions in perinatal brain DHA accrual results in deficits in serotonin and dopamine neurotransmission; neurocognitive deficits; and increased behaviors, such as anxiety, aggression, and depression.[27]

Studies have also correlated a shortened duration of breastfeeding with childhood ADHD. According to one study, 60% of children with ADHD were breastfed less than 3 months, compared with 32.5% non-ADHD controls who were breastfed less than 3 months.[28] Studies with twins indicate that low birth weight is also a risk factor for symptoms of ADHD. Lighter twins in birth weight–discordant pairs had approximately 13% higher ADHD symptom scores at ages 8–9 and 12% higher ADHD scores at ages 13–14 compared with the scores of heavier twins in the pairs. These results suggest that fetal growth restriction may represent a modest environmental influence on the development of ADHD symptoms.[29]

Numerous environmental factors have been associated with ADHD.

Neonatal exposure to high levels of manganese has also been associated with ADHD, due to the similarity of dysfunction in brain dopamine systems seen with manganese toxicity and ADHD. In one study, newborn rats were given manganese in their water for postnatal days 1–21. There was a statistically significant relationship between dietary manganese exposure and behavioral variability and striatal dopamine levels, which supports the hypothesis that neonatal manganese exposure is related to brain dopamine levels and behavior.[30]

Another study examined manganese exposure in public water systems and hyperactivity in humans. Children from homes with water supplied from wells with differing levels of manganese were evaluated. Children whose houses were supplied by the well with elevated manganese had higher hair manganese levels than those supplied by the well with low levels of manganese. Elevated hair manganese was significantly associated with the Revised Conners' Rating Scale for oppositional and hyperactivity subscales. All of the children with elevated oppositional and hyperactivity scores (> 65) had elevated hair manganese levels (> 3.0 μg/g).[31]

Thyroid Dysfunction

Some researchers suggest that abnormal thyroid function may play a role in ADHD pathology. This is based on the fact that thyroid hormones are essential to normal brain development and influence behavior and cognitive function. Researchers are currently examining potential thyroid-hormone disruptors, such as polyhalogenated aromatic hydrocarbons in relation to ADHD.[32] Studies using animal models have shown that rats who had experienced perinatal hypothyroidism are hyperactive and restless, have a shortened attention span, and panic easily.[33]

One study followed the neuropsychologic development of the offspring of women from a moderately iodine-defi-

†The Feingold Diet was developed by pediatrician Benjamin F. Feingold, M.D. (1899–1982) to identify and remove food additives such as artificial coloring as well as salicylates from the diets of children with hyperactivity (ADHD). www.feingold.org

cient area and of control women from a marginally iodine-sufficient area whose thyroid function had been monitored during early gestation. ADHD was diagnosed in 68.7% of the children from the moderately iodine-deficient area, compared with 0% of the children from the marginally iodine-sufficient area. The study also showed that total intelligence quotient scores were lower in the children from the moderately iodine-deficient area, compared with those from the marginally iodine-sufficient area. In addition, 63.6% of the children with ADHD were born to the mothers in the moderately iodine-deficient area and had become hypothyroxinemic at early gestation, whereas, of 5 children who did not have ADHD, only 1 child was born to a woman who was hypothyroxinemic during gestation. These findings suggest that ADHD may be related to maternal hypothyroxinemia possibly due to iodine deficiency, resulting in a reduction of intracellular T3 available to the developing fetal brain.[34]

Conventional Treatment

Conventional therapy is often multimodal including both behavioral therapies and medication. Many of the approved drugs for ADHD are stimulants, which work by increasing dopamine levels. Stimulant medications approved by the U.S. Food and Drug Administration (FDA) include methylphenidate (Ritalin, Concerta, Metadate), amphetamine (Adderall), dextroamphetamine (Dexedrine, Dextrostat), dexmethylphenidate (Focalin), and pemoline (Cylert).[35] In addition, the FDA has recently approved atomoxetine (Strattera), which is not a stimulant and which acts as a selective norepinephrine reuptake inhibitor.[35]

A large, long-term study was performed with 579 children, 7.0–9.9 years old, who were diagnosed with combined type ADHD. Each child was assigned to one of 4 groups: (1) medication management; (2) intensive behavioral treatment; (3) medication plus intensive behavior treatment; or (4) or standard commu nity care. In all 4 groups, there was a reduction in symptoms over time. For the majority of ADHD symptoms, children in the combined treatment and medication management groups had significantly greater improvement than children who were given intensive behavioral treatment only or given community care only. The combined treatment was superior for non-ADHD symptom and positive functioning outcomes such as oppositional/aggressive symptoms, internalizing symptoms, teacher-rated social skills, parent–child relationships, and reading achievement.[36]

Although studies indicate a correlation between stimulant medication use and substance abuse, a review of long-term studies on stimulant medication and substance abuse showed that teenagers with ADHD who remained on their medications during adolescence had a lower likelihood of substance use or abuse than did ADHD adolescents who were not taking medications.[37]

Natural Therapies

Essential Fatty Acids

The principal omega-3 fatty acid in the brain, DHA, is highly accumulated in nervous-tissue membranes and is important for neural function. Several studies have identified abnormalities in membrane fatty acids in some subjects with ADHD. It has been suggested that ADHD is associated with an imbalance in polyunsaturated fatty acid (PUFA) composition, with abnormal low levels of DHA. Animal models have shown that hyperactive individuals had less omega-3 PUFA in the frontal cortex than hypoactive individuals.[38]

Several studies have identified abnormalities in membrane fatty acids in some subjects with ADHD.

According to one study, adolescents with ADHD consumed more energy and fat than controls. Children with ADHD consumed equal amounts of omega-3 and omega-6 fatty acids, compared with control children. However, children with ADHD had significantly lower levels of DHA and total omega-3 fatty acids, higher omega-6 fatty acids, and lower ratios of omega-3:omega-6 fatty acids, compared with control children. This study also showed that lower omega-3 status correlated with higher scores on several Conners' behavioral scales. These results suggest that adolescents with ADHD have abnormal EFA profiles, which are not explained by differences in intake.[39]

To investigate the abnormal fatty-acid metabolism further, another study measured exhalant ethane levels to evaluate oxidative damage to omega-3 fatty acids in patients with ADHD.[40] Patients with ADHD had higher levels of ethane in exhalant than did healthy volunteers. Approximately 50% of the patients with ADHD had ethane levels above the control range, and these subjects' levels of butane, a marker of protein oxidation, were normal. These results suggest that there is an increase in oxidative breakdown of omega-3 PUFAs in patients with ADHD.[40]

Another clinical trial evaluated the effects of high-dose supplementation with the EFAs eicosapentaenoic acid (EPA) and DHA on the behavior of children with ADHD. The children were initially given 16.2 g of EPA/DHA per day, followed by adjusted dosing that depended on the ratio of arachidonic acid (AA) to EPA in their isolated plasma phospholipids at 4 weeks. After 8 weeks, the children who were given EPA/DHA had significant increases in EPA and DHA, as well as significant reductions in AA:EPA ratios. A psychiatrist, who was blinded to supplement compliance and dosage modifications, reported significant reductions in inattention, hyperactivity, oppositional/defiant behaviors, and conduct disorder–related behavior. There was also a significant correlation between the reduction in AA:EPA ratio and global severity of illness scores.[41]

In a similar study, adults with ADHD were found to also have significantly lower levels of total PUFA, total omega-3 fatty acids, and DHA; and significantly higher levels of total saturated fatty acids measured in erythrocyte-membrane phospholipids.[42]

In a randomized, placebo-controlled, double-blind study, children with ADHD ages 7–12 were given PUFAs alone, PUFAs plus micronutrients, or placebo for 15 weeks. Significant medium-to-strong positive treatment effects were found on parent ratings of the ADHD symptoms of inattention and hyperactivity/impulsivity based on the Conners' Parent Rating Scale in both PUFA treatment groups compared with the placebo group. After a one-way crossover to active supplements in all groups for an additional 15 weeks, these positive results were also found in the placebo group, and the treatment group continued to have significant improvements according to the Conners' Parent Rating Scale.[43]

Dietary Interventions

Dietary factors may play a significant role in the etiology in many children with ADHD. Nutritional factors, such as food additives, refined sugars, food sensitivities, food allergies, and fatty-acid deficiencies have all been linked to ADHD. Several studies have evaluated the efficacy of elimination diets on symptoms of ADHD.

In one study, children ages 3–7 who met the *DSM-IV* criteria for ADHD were given an elimination diet consisting of rice, turkey, pear, and lettuce after 2 weeks of their usual diets. At the end of the study, 62% of the children had improvements in behavior of at least 50% on both the Conners' list and the ADHD Rating Scale, according to the parent ratings.[44]

In the first phase of another study, 19 of 26 children diagnosed with ADHD responded positively to an elimination diet. In addition, all 19 children reacted to many foods, dyes, and/or preservatives during an open challenge. In the second phase of this study, 16 children underwent a double-blind placebo-controlled food challenge. There was a significant improvement on placebo days, compared with challenge days. In addition, atopic children with ADHD had a significantly higher response rate than nonatopic children.[45]

The first phase of a third study showed that more than 75% of hyperactive children placed on a "few foods" elimination diet had improvements in behavior. In the second phase of this study, 19 of the children underwent a placebo-controlled double-blind challenge protocol. The results of a crossover trial with these 19 children showed a significant worsening of behavior and impaired psychologic test performance with the provoking foods.[46]

A similar study showed that 73% of the children with ADHD improved according to their parents and 70% improved according to the children's teachers in an elimination-diet group, compared with controls. The number of ADHD criteria noted on the ADHD Rating Scale indicated a scale reduction of 69.4%, and comorbid symptoms of oppositional defiant disorder (ODD) also were decreased more significantly in the intervention group than in the control group.[47]

In a double-blind, randomized, crossover study, hyperactive children were given drinks containing food additives or placebos, and physiologic measures, including heart rate and electroencephalographic (EEG) measures, were obtained prior to and following ingestion. The hyperactive children had a greater degree of physiologic activity following both the drink containing food additives and the placebo drink than did the children in the control group. However, the hyperactive children had a greater magnitude of physiologic changes in response to ingestion of the additives than to placebo, indicating that some hyperactive children are affected adversely by food additives.[48]

Nutrients

Magnesium

A research study with 116 children with ADHD ages 9–12 showed that magnesium deficiency was evident in 95% of those examined, most frequently in hair (77.6%), red blood cells (58.6%), and in serum (33.6 %).[49] A following study evaluated the effect of magnesium supplementation in 50 hyperactive children diagnosed according to the *DSM-IV* criteria for ADHD with recognized magnesium deficiency in the blood and hair, using atomic absorption spectroscopy. Magnesium was administered at a dose of 200 mg per day for 6 months. The control group consisted of 25 children with ADHD and magnesium deficiency, who were treated with standard therapy only. The children given the magnesium had an increase in hair magnesium levels and a significant decrease in hyperactivity based on the Conners' Rating Scale for Parents and Teachers, Wender's Scale of Behavior, and the Quotient of Development to Freedom from Distractibility, compared with baseline and compared with the control group.[50]

In another study, children with ADHD were evaluated during a magnesium–vitamin B_6 (pyridoxine) regimen (6 mg/kg/day of magnesium and 0.6 mg/kg/day of vitamin B_6) for 8 weeks. The children with ADHD had significantly lower intraerythrocyte magnesium values than the control children. In almost all of the children with ADHD, the magnesium and vitamin B_6 supplementation for 2 months significantly modified the ADHD symptoms. In particular, hyperactivity and hyperemotivity/aggressiveness were reduced, and school attention was increased. In addition, the magnesium and vitamin B_6 supplementation significantly increased intraerythrocyte magnesium levels. After the supplementation was stopped, the clinical symptoms of ADHD returned and intraerythrocyte magnesium values were decreased.[51]

Vitamin B_6

Vitamin B_6 is an important cofactor in numerous metabolic reactions, including the metabolism of serotonin, gamma aminobutyric acid (GABA), norepinephrine, and dopamine. A small, double-blind clinical study with children diagnosed with hyperkinetic syndrome compared the administration of vitamin B_6, methylphenidate, and placebo on hyperkinetic symp-

toms. Both vitamin B_6 and methylphenidate were more effective than placebo for suppressing hyperkinesis symptoms.[52]

Another small study evaluated the levels of serotonin in hyperactive children. The researchers found that there was a significant decrease in serotonin levels in the blood samples from hyperactive patients compared with controls. Taking vitamin B_6 orally resulted in a significant increase in serotonin levels and a very large increase in the pyridoxal phosphate levels in the blood of the hyperactive patients.[53]

Zinc

Zinc is an important cofactor for several physiologic reactions, including those involving neurotransmitters and fatty acids, and is believed to be involved in ADHD. Several studies have reported evidence of lower zinc tissue levels in children with ADHD, compared with normal controls.[54] A clinical trial was performed to evaluate a middle-class American sample of children with ADHD in relation to zinc status. Serum zinc levels correlated with parent–teacher-rated inattention, even after controlling for gender, age, income, and diagnostic subtype.[55]

A double-blind study with 400 children diagnosed with ADHD was performed to evaluate the efficacy of zinc sulfate supplementation for children with this condition. For 12 weeks, the patients were given zinc sulfate at a dose of 150 mg per day or placebo. Zinc sulfate supplementation was statistically superior to placebo in reducing hyperactive, impulsive, and impaired socialization symptoms, but not in reducing attention deficiency symptoms in children with ADHD.[56]

Iron

In one study, 23 nonanemic children with ADHD ages 5–8 with serum ferritin levels < 30 ng/mL were given, for 12 weeks, either oral ferrous sulfate at a dose of 80 mg per day or placebo. There was a progressive and significant decrease in the ADHD Rating Scale after 12 weeks with iron supplementation, compared with placebo. In addition, the mean Clinical Global Impression (CGI)-Severity significantly decreased with iron supplementation at 12 weeks, with no change in the placebo group.[57]

Lead exposure has been correlated with ADHD symptoms. In the central nervous system (CNS), lead may contribute to dopaminergic dysfunction and may disrupt the structure of the blood–brain barrier, which is essential for brain integrity. Iron supplementation protects the integrity of the blood–brain barrier against lead insults and iron deficiency could increase the toxic effect of lead. This suggests a neuroprotective effect of iron supplementation on dopaminergic dysfunction due to lead exposure. Iron deficiency was correlated to ADHD symptoms' severity; thus, researchers propose that iron supplementation may reduce symptoms of ADHD in patients with low ferritin levels.[58] Chelation has been proposed as a potential therapy for heavy metal toxicity in children with ADHD. One small pilot study showed that 7 of 11 children with hyperkinetic syndrome improved with chelation therapy.[59]

Carnitine

In a randomized, double-blind, placebo-controlled, double-crossover trial, carnitine (dosage was not stated) was given to boys with ADHD. In 54% of the boys receiving carnitine, home behavior improved as assessed with the Child Behavior Checklist total score and school behavior improved as assessed with the Conners' Teacher-Rating score. Responders showed a significant improvement of the Child Behavior Checklist total scores, compared with baseline measurements. The responders to carnitine treatment showed a decrease in attention problems of 20%–65% according to the Child Behavior Checklist total problem rating scale, compared with baseline. These results showed that carnitine supplementation significantly decreased the attention problems and aggressive behavior in some boys with ADHD.[60]

Nutrient Levels

One study evaluated the dietary patterns in children with ADHD compared to control children. No differences were found in dietary patterns of these children with ADHD, compared with a control group except for the increased intake of iron and vitamin C. However, the fatty-acid composition of red blood cell membrane phospholipid in the children with ADHD differed from that of the control children. Differences in the fatty acids in the phospholipids isolated from red blood cell membranes included significantly higher oleic acid, and significantly lower nervonic acid, linoleic acid, AA, and DHA acid in the children with ADHD. Plasma gamma-linolenic acid (GLA) in children with ADHD was also higher than that in control children. In addition, the blood total protein content in subjects with ADHD was significantly lower than that in control subjects.[61]

Conclusion

ADHD is an increasingly common neurologic and behavioral condition and has a detrimental impact on children affected and their families. As the etiology is still unknown, research continues to identify factors that play roles in this condition. Natural therapies such as dietary modification, including elimination diets and nutritional supplementation, have shown to be efficacious in many cases of ADHD. ■

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