

Can Attention Deficit-Hyperactivity Disorder Result from Nutritional Deficiency?

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The national consensus is that the etiology of attention deficit-hyperactivity disorder (ADHD) is unknown; yet a cohesive body of evidence has accumulated in recent years suggesting that ADHD may well be a manifestation of nutritional deficiency. A growing body of evidence suggests that (ADHD) may be an unanticipated manifestation of the new American diet.

Major and unprecedented dietary changes have occurred over the last century in the U.S. Per-capita use of sugar has doubled. Starch consumption from grain and potatoes has risen 40 percent. Egg use has dropped significantly. Use of saturated fats such as lard, beef fat, dairy fat, and coconut oil has decreased, while consumption of fats and oils derived from vegetable seeds such as soy, safflower, and canola have grown from essentially zero to about 66 pounds per person per year. This nationwide changeover to vegetable fats has seriously unbalanced the essential fatty acid intake of the population.¹

During the same period, ADHD, which was essentially unheard of 50 years ago, increased to the point that it is now diagnosed in 5 percent of school children and many adults.²

Historical Perspective

History shows that when diets change, nutritional diseases can appear.

Beriberi appeared after a new grain milling process removed the oily, vitamin-containing germ from grains, to prevent the stored grain from becoming rancid. The so-called "white rice" produced by this process was responsible for untold deaths in Asia during the 18th and 19th centuries. A deficiency of the B-vitamin thiamine was found to be the specific cause of beriberi, and subsequent laws requiring the addition of this vitamin to refined grain products have virtually eliminated the disease.

Pellagra appeared about 300 years ago when corn became a major food source. Because of its low price, corn largely replaced meat, eggs, and milk as the primary source of calories in large population groups. The cause of pellagra, a deficiency of the B-vitamin niacin, was not discovered until the early part of the 20th century. Addition of niacin to refined grains generally ended the problem, but pellagra is also easily cured by increasing the amount of meat, eggs, and milk in the diet.

Birth defects can also be caused by nutritional deficiencies. Cretinism, a condition characterized by stunted growth and mental retardation, was common in the areas of the world where the diet was deficient in iodine. Spina bifida, a neural-tube defect, was recently recognized to result from a dietary deficiency of the B-vitamin folic acid during pregnancy.

The Biochemical Effects of Recent Dietary Changes

Dietary changes of the last century have had two major nutritional impacts. First, for the average person, increased consumption of sugar and starch has required more and faster secretion of insulin to control the rapid rises in blood glucose caused by these high-glycemic carbohydrates. Chronic use of high-glycemic carbohydrates is a major cause of the hypoglycemia, insulin resistance, and type-2 diabetes now prevalent.¹

Second, the vegetable fats and oils that today dominate in the mass market are, for all practical purposes, devoid of lipids from the omega-3 essential fatty acid family, including alpha linolenic acid, EPA (eicosapentaenoic acid), and DHA (docosahexaenoic acid).

This problem is worsened by the fact that the vegetable fats and oils contain unusually large amounts of omega-6 linoleic acid, the precursor of the omega-6 family of essential fatty acids, and its important metabolite arachidonic acid (AA).

In short, the traditional fats and oils contained a healthy ratio of omega-6 to omega-3 essential fatty acids of four to one or less, plus an ample amount of omega-3 fatty acids. The new vegetable fats and oils, on the other hand, are deficient in omega-3 fatty acids and, at the same time, contain an extremely high and unhealthy omega-6 to omega-3 ratio of approximately 20 to 1. Moreover, many of these vegetable products contain trans-fatty acids, which are known to interfere with the metabolism of the essential fatty acids.¹

Products made from the vegetable fats and oils include salad and frying oils, vegetable shortening, and margarine. All are derived from corn, cottonseed, peanuts, soybeans, canola, safflower, or sunflower.

Clinical Considerations in ADHD

According to the National Institute of Mental Health (NIMH), the federal agency responsible for research on the brain, mental illnesses, and mental health, ADHD affects approximately 2 million American children—3 to 5 percent of the school-age population—and is about four times more common in boys than in girls.³ Over the past several decades this condition has grown to become the most commonly diagnosed behavioral disorder of childhood.

New findings, published in February 2002, suggest that the prevalence of ADHD may be considerably higher than the numbers NIMH published.⁴ A University of North Carolina School of Medicine/National Institute of Environmental Health Sciences study of 6,099 children in 17 North Carolina rural county elementary schools found that more than 15 percent of the boys and 5 percent of the girls had been diagnosed with ADHD, and that about two-thirds of those diagnosed were taking medication for it. About 9 percent of all fourth and fifth-grade children in the study were taking medication to treat ADHD.

ADHD-affected children present difficult behavioral problems and perform poorly in school because of their inability to focus on school tasks or to sit still during the school day. Symptoms typically begin at about age 3 and include inattention, inability to concentrate, failure to listen when spoken to, hyperactivity, squirming, talking out of turn, impulsiveness, disruptive behavior, sleep problems, and poor learning ability.³

ADHD persists into adulthood, although adults in many cases are more able to control their behavior and mask their difficulties. Even so, statistics show that there is an increased incidence of juvenile delinquency and adult encounters with the law among people who had ADHD as a child. Worst-case outcomes of ADHD are school failure and dropout, delinquency, and criminal behavior.³

According to the 44-page NIMH booklet on ADHD,³ the cause of the condition, once called hyperkinesis or minimal brain dysfunction, is not known. It states that ADHD is not usually caused by minor head injuries, infections in early childhood, too much TV, poor home life, poor schools, excess sugar, food additives, or food allergies. Possible links to the mother's use of cigarettes, alcohol, and other drugs, or to toxins such as lead in the environment have been suggested but not proven. Other research suggests a genetic link, based on the tendency of ADHD to run in families.⁵

Many treatment methods have been tried, with the most common approach being psychiatric intervention and use of

prescription drugs such as methylphenidate (Ritalin). These drugs reduce the severity of symptoms and help the affected children control their impulses and participate in classroom routine.

According to the NIMH, health professionals stress that because no one knows what causes ADHD, it does not help parents to look backward for possible reasons. They point out that there are too many possibilities to pin down the cause with certainty and that it is far more important for the family to move forward in finding ways to obtain the right help.^{3,5}

The Role of DHA and AA

Despite official preoccupation with treating ADHD with drugs, there is compelling scientific evidence that ADHD is a birth defect caused by deficiencies of maternal DHA during pregnancy and while nursing, and the virtual absence of DHA and AA in infant formula. AA is a long-chain omega-6 fatty acid found primarily in meat and eggs, whereas EPA and DHA are found primarily in the fat of cold-water fish.

It is known that DHA and AA are required building blocks for the brain and eyes of newborns.⁶ The brain begins growing very rapidly in the third trimester of pregnancy and continues to grow for 24 months after birth. The required DHA and AA are normally supplied directly from the mother to the child during pregnancy and by the mother's milk during the nursing period. When the mother's and/or the child's diets are deficient in DHA and/or AA, the brain and the eyes cannot develop properly.

A number of studies have found that brain volume is 3 to 4 percent smaller in individuals with ADHD.^{7,8} The most recently published study on the subject compared the brain volumes of 152 ADHD children and adolescents between the ages of 5 and 18 years against a control group of 139 age and sex-matched controls. In addition to shrinkage in total brain volume, this study found that shrinkage also occurred in brain components, such as the cerebrum and cerebellum, and persisted across all ages and sexes in both the medicated and unmedicated children with ADHD. The authors of this study suggested that genetic and/or early environmental influences on brain development in ADHD are permanent, not progressive, and unrelated to the drugs commonly used for ADHD treatment.⁹

Studies also show that the head circumference of newborns is a good predictor of brain weight in preterm infants, and that low DHA levels in umbilical arteries are associated with smaller head circumferences in these infants.⁶ Other studies indicate that the high maternal dietary consumption of linoleic acid (an omega-6 fatty acid that is overabundant in vegetable fats and oils) is related to low blood levels of DHA observed in both mothers and their newborns.⁶ This effect is only one example of a number of adverse health effects caused by high dietary levels of omega-6 linoleic acid that result from the wide use of vegetable fats and oils.¹

At least one study has found a correlation between DHA in blood and DHA in brain tissue of term infants.¹⁰ This means that the tests showing DHA levels in blood referred to later in this article can be used to infer DHA levels in the brain. This same study also examined both breast-fed and formula-fed infants, and found that the breast-fed infants had greater proportions of DHA in their blood and brain tissue than did the formula-fed infants. In order to avoid repeated blood collections to determine fatty acid status in infants, researchers developed a non-invasive method for analysis of buccal mucosal cells collected on cotton swabs that correlates closely with fatty acids in blood samples.¹¹⁻¹³

Breast milk normally contains both DHA and AA, but the infant formula used in this study (and also used throughout the US at the time) contained neither DHA nor AA. Following a recent favorable ruling by the Food and Drug Administration, infant formula containing DHA and AA in concentrations similar that found in mothers' milk began entering the American market in early 2002.¹⁴

A worldwide consensus for fortification of infant formula has been growing for a number of years: In 1978 the Food and Agriculture Organization and the World Health Organization recommended that formula mimic breast milk. In 1991 the European Society for Pediatric Gastroenterology, and in 1992 the British Nutrition Foundation recommended that preterm infant

formulas include DHA and AA. In 1993 a World Health Organization and Food and Agriculture Organization joint committee of experts recommended that preterm and term infant formulas include DHA and AA.¹⁴

Justification for these recommendations is that the preponderance of evidence shows that infants fed unfortified formula have poorer vision and lower IQs than infants fed with formula fortified with DHA and AA.^{6,15}

Human breast milk tends to have adequate levels of AA for infant nutrition. The diet of the mother does not ordinarily require any supplementation with AA because it is readily available from common foods such as meat and eggs. On the other hand, the American diet is seriously deficient in omega-3 fatty acids, including DHA. As a result, childbearing women are very often deficient in DHA. According to Sears, average DHA levels in nursing mothers' milk in the US are among the lowest in the world. He states that this DHA shortage represents a real risk to the mental capacity of future Americans.¹⁶

According to Hornstra, lower levels of DHA in blood plasma of low-birth-weight infants at age one year correlate significantly with psychomotor and mental developmental indices; that breast-fed infants have a higher IQ than those fed with standard unfortified baby formula; and the longer the period of breast feeding, the better the mental performance of the child.⁶

Studies with school-age children and young adults are also consistent with the proposition that ADHD results from nutritional deficiency. Stevens et al. reported that a group of 56 individuals with ADHD had significantly lower concentrations of both DHA and AA in red blood cell lipids than did a control group of 43 individuals.¹⁷ The authors noted that the children in the study group with the worst symptoms had the lowest plasma levels of DHA and AA and indicated that the precise reason for the lower fatty acid levels in some children with ADHD is not clear, although altered fatty acid metabolism was a possibility.

Mitchell et al. compared 48 hyperactive children with 49 age- and sex-matched controls.¹⁸ Serum DHA and AA levels were significantly lower in the hyperactive children than in the control children. The hyperactive children had auditory, visual, language, reading and learning difficulties, and lower average birth weight than the controls. The hyperactive children also were more likely to suffer coughs, colds, serious illness, or accident in the year before the study.

Studies designed to determine whether ADHD can be helped by dietary supplementation with DHA have shown mixed results. For example, Voigt et al., using a randomly assigned group of 63 children of ages 6-12 with ADHD, all receiving maintenance therapy with stimulant medication, reported that supplementation with 345 milligrams per day of DHA or a placebo for four months showed no benefit.¹⁹ On the other hand, Richardson and Puri, using a group of 41 ADHD children randomly assigned to supplementation or placebo for 12 weeks, reported a significant reduction in ADHD symptoms in the treated group.²⁰

Sears¹⁶ has written that such contradictory results may be expected because some studies tend not to control dietary intake of high-glycemic carbohydrates (sugar and starch), and do not provide their study groups with sufficiently high doses of DHA and/or EPA. His reasoning is that the American diet is not only low in DHA and other omega-3 fatty acids, but also too high in sugar and starch. Dietary sugar and starch raise insulin levels that, in turn, disrupt brain chemistry by interfering with essential fatty acid metabolism.

In a study of ADHD children, Sears reported uniformly good results. Within weeks the children's ability to concentrate increased dramatically and their behavior at home and school improved. In this study Sears controlled dietary sugar and starches and used pharmaceutical grade fish oil to provide doses of 10-15 grams per day of DHA and EPA. EPA is the omega-3 metabolic precursor of DHA, and occurs in fish oil as well.

The brain chemistry involved is complex and not completely understood, but high levels of omega-6 fatty acids coupled with deficiencies of both EPA and DHA, and/or high and unstable insulin levels have been found to stimulate the enzyme delta-5

desaturase. This results in increased production of inflammatory eicosanoids (messenger biochemicals present in every cell) that cause increased biosynthesis of corticosteroids, including cortisol, and reduced serotonin and dopamine levels in the brain.^{2,16}

High cortisol levels, among other negative effects, cause impaired short-term memory and feelings of stress. Serotonin and dopamine are important neurotransmitters. Low levels of both are common in ADHD patients. Low serotonin is associated with depression, impulsive behavior and violence, and low dopamine is associated with Parkinson's disease, violent behavior, and inability to concentrate or focus on a task.^{2,16} The latter references provide a good overview of what is known about the impact of omega-3 fatty acids on mental disorders.

Conclusions

The evidence summarized here strongly suggests that ADHD may be a birth defect caused by a nutritional deficiency of DHA and perhaps other omega-3 essential fatty acids. The studies cited above provide a scientifically documented and logical connection between the extremely low levels of DHA in the American diet and the DHA-deficient brains and blood of ADHD children. However, nutritionally induced learning and behavioral problems among children may not be a new phenomenon.

Powerful evidence that ADHD or something similar was occurring in the 1930s came from a landmark book by Price.²¹ His extensive and well-documented studies compared the health of isolated racial groups still living on their native diets with matched groups of the same racial stocks living on modern foods.

The modern diets consisted chiefly of foods made from flour, sugar, polished rice, vegetable fats, and canned goods. The primitive diets varied according to local customs but were quite consistent in the regular inclusion of protein and fat from fish, shellfish, animals, insects, and whole dairy products. Sugar and refined starches were absent from this diet and carbohydrate was always in the form of fruit, fresh vegetables, and small amounts of unprocessed grain.

Children in the groups eating the modern foods showed symptoms of physical and mental degeneration after only one generation on this new diet. These symptoms included narrow nostrils, faces, dental arches, and hips; crooked and overlapping teeth; high rates of dental caries; high susceptibility to tuberculosis; and learning and behavioral problems in children that often progressed to delinquency. These physical and mental symptoms did not occur in the groups of people living in the same areas who had continued eating their primitive diets.

Price commented near the end of his book that such physical and mental degeneration among the young seemed never to occur where seafood was used liberally. He attributed the degeneration to malnutrition of the parents, particularly the mothers of these children.

The choice seems clear. We can either continue to depend on prescription drugs to mask the symptoms of ADHD, or consider preventing ADHD by modifying the American diet, particularly for childbearing women and their children. Should we decide to continue to depend on prescription drugs, which do not remedy the underlying causes of nutritional deficiency diseases, we can look forward to a country in which there will be more and more children with undersized brains who cannot learn, use costly prescription drugs, drop out of school, commit crimes, and cause anguish for their parents.

The appropriate course of action is aptly summed up by Dr. Artemis Simopoulos, a physician who has spent her professional lifetime studying and writing about the essential fatty acids:

The scientific basis for the development of a public policy to develop dietary recommendations for essential fatty acids, including a balanced omega-6/omega-3 ratio, is robust. What is needed is a scientific consensus, education of professionals and the public, the establishment of an agency on nutrition and food policy at the national level, and willingness of governments to institute changes.²²

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Attention-deficit hyperactivity disorder is an early-onset, clinically heterogenous disorder of inattention, hyperactivity, and impulsiveness. The diagnosis and treatment of attention-deficit hyperactivity disorder continues to raise controversy, and, there is also an increase in treatment options. In this 6-week double blind, placebo controlled-trial, we assessed the effects of zinc plus methylphenidate in the treatment of children with attention deficit hyperactivity disorder.Â These results suggest that low iron stores contribute to ADHD and that ADHD children may benefit from iron supplementation. View. Show abstract.