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Nutrition in the Treatment of Attention-Deficit Hyperactivity Disorder: A Neglected but Important Aspect

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Attention-deficit hyperactivity disorder (ADHD) is multidetermined and complex, requiring a multifaceted treatment approach. Nutritional management is one aspect that has been relatively neglected to date. Nutritional factors such as food additives, refined sugars, food sensitivities/allergies, and fatty acid deficiencies have all been linked to ADHD. There is increasing evidence that many children with behavioral problems are sensitive to one or more food components that can negatively impact their behavior. Individual response is an important factor for determining the proper approach in treating children with ADHD. In general, diet modification plays a major role in the management of ADHD and should be considered as part of the treatment protocol.

KEY WORDS: ADHD; diet; nutrition.

Attention-deficit hyperactivity disorder (ADHD) is characterized by difficulty in delaying gratification, inattentiveness, distractibility, impulsive behavior, anxiety, and excessive motor activity (Barkley, 1981; Kanarek & Marks-Kaufman, 1991). It is often accompanied by emotional immaturity, aggressiveness, and poor academic performance (Barkley, 1981). It has been estimated that approximately 3% of children may suffer from ADHD, with the disorder six to nine times more common in boys than in girls (Johnson, 1988). There has been much speculation about the etiology of ADHD and both genetic and environmental factors have been implicated.

ADHD has been investigated extensively over the last 30 years. At this point most researchers agree that ADHD is a problem of complex etiology that can be investigated as a function of multiple interactions. The fields of psychology, education, and conventional and complementary medicine have attempted to find solutions to this disorder. Although

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clinicians and researchers alike acknowledge the complex etiology of ADHD, most treatment studies have focused on a limited number of modalities, often investigating just a single modality. This has been especially true with respect to neurofeedback treatments, with few exceptions (Lubar & Lubar, 1984, who combined neurofeedback with academic training; Thompson & Thompson, 1998, who combined neurofeedback training with metacognitive strategies, which included teaching strategies to succeed in math, reading, and listening, to treat ADHD). To devise more comprehensive treatments and conduct more meaningful research, clinicians and researchers will have to cooperate with other specialists or learn additional new treatment modalities.

It is also certain that different strategies will work for different patients diagnosed with ADHD and researchers are beginning to attempt to identify variables associated with treatment success. However, one of the components affecting ADHD has been relatively ignored.

The effect of diet and nutrition on ADHD is an issue that merits greater recognition by practitioners in the field. For example, Uhlig, Merkenschlager, Brandmaier, and Egger (1979) investigated the relationship between diet and brain electrical activity in children with ADHD. They found that certain food sensitivities not only influence ADHD symptomology, but may also alter brain electrical activity. This research underscores the need for practitioners to understand the role of nutrition in ADHD.

In the past two decades there has been increasing interest in various aspects of diet that may adversely affect hyperactive children. Particular attention has focused on the role of food additives, refined sugars, food allergies, and fatty acid metabolism.

FOOD ADDITIVES

The food additives hypothesis was introduced by Feingold, who proposed that food additives, specifically synthetic food colors and flavors, and naturally occurring salicylates were responsible for hyperactive behavior in some children (Feingold, 1975). The diet, which Feingold labeled the Kaiser-Permanente (K-P) diet, named after the medical center where he worked, required children to eliminate all artificial colors and flavors as well as all foods containing salicylates. Feingold reported that as many as 50% of hyperactive children who carefully followed his regimen responded favorably. In the scientific community, however, controversy ensued surrounding the validity of Feingold's claims. Feingold's conclusions were based on his own clinical observations rather than on rigorous experimental evidence. Critics also suggested that the success of his diet may be related to the overwhelming attention parents gave to their children by focusing on dietary compliance, rather than to the specific effects of the diet. In response to the controversies, a number of agencies established guidelines for further research to investigate the relationship between diet and hyperactivity. Subsequent studies employing the guidelines recommended by these agencies fell into two groups: those that evaluated the behaviors of hyperactive children while on the Feingold diet as compared to a placebo diet (dietary crossover designs), and those that investigated responses to specific food dye challenges (Lipton & Mayo, 1983).

In the dietary crossover studies, hyperactive children were randomly assigned to either the Feingold K-P elimination diet or to a control diet and then crossed over to the other

treatment condition. Researchers working in this area found that it was difficult to insure compliance with the Feingold diet because of the severe dietary restrictions. They also found it difficult to develop an equivalent placebo diet. So it is not surprising that only two comprehensive studies of the Feingold diet were conducted.

Harley et al. (1978) selected 45 hyperactive boys for their study; 36 participants were between the ages of 6 and 12, and 10 participants were between the ages of 3 and 5. Each child was maintained on the Feingold diet for a period of 3 or 4 weeks and on a control diet for the same amount of time. The study team provided all foods, and dietitians made weekly home visits to ensure adherence to the diet. The order of the diets was counterbalanced across the children, and all persons working directly with the children and their families were blind as to the experimental conditions. Parents and teachers rated the children. Parental ratings indicated that some of the boys responded favorably when following the Feingold diet. However, teacher ratings, observation measures, tests of achievement, and psychophysiological performance did not confirm these findings. In contrast, much more pronounced behavioral differences were found for the preschool boys. All of the 10 mothers found their children to be more manageable when following the experimental diet. However, teacher ratings and classroom observation were not available for this group.

Another double-blind crossover study was conducted by Conners, Goyette, Southwick, Lees, and Andrulonis (1976), which included 15 hyperactive children, 6–12 years of age, who were maintained on the Feingold diet for 4 weeks and on a placebo diet for 4 weeks. Order of the diets was counterbalanced across the children. Improvement in behavior was noted in 4 out of the 15 children, but only by the teachers when the control diet was given first followed by the elimination diet. These improvements were not demonstrated when the order was reversed.

Most other researchers evaluating Feingold's claims have limited their investigations to studying the effects of food dyes on behavior. In these challenge designs, children were maintained on Feingold's elimination diet throughout the studies. Periodically, the children were challenged with foods that contained the suspected offending chemical (e.g., artificial food colors). Measures were taken to note changes in behavior.

The data from these studies suggested that there is a small group of children with behavioral disturbances who respond to some aspects of the Feingold diet. However, in general, the specific elimination of synthetic food colors from the diet did not appear to be a major factor in the reduction of hyperactive behavior in the majority of children (Silver, 1986).

One exception to the overall findings was a food challenge study performed by Swanson and Kinsbourne (1980). They improved on the previous studies in three ways. First, the researchers admitted the children into the hospital so that the elimination diet could be administered in a controlled manner, avoiding problems with dietary noncompliance. Second, they adjusted the amount of food coloring in the challenge to better reflect the average daily consumption of children. Rimland (1983) found that the majority of the food dye researchers were using challenge doses of 1.6–26 mg of coloring per day, after the FDA had identified children's consumption of colorings to be between 59 and 300 mg per day. This might explain why previous studies found no effect when challenging children with food dyes. Swanson and Kinsbourne tested two challenge doses, 100 and 150 mg. The third improvement over the previous studies concerned the measurement procedures. Previous studies relied primarily on rating scales that Swanson and Kinsbourne maintained were not

sensitive enough to monitor the effect of diet on hyperactive behavior, particularly if the focus of change is cognitive performance more than conduct. To rectify this problem, they used a laboratory test of cognitive functioning, called the paired-associate learning task, in their double-blind crossover design. They found that either the 100- or the 150-mg food dye challenge adversely affected all the 20 hyperactive children in their study. Performance on the paired-associate learning task was significantly worse after the dye challenge than after the placebo challenge.

Examination of the data from these studies indicates that the effects of the Feingold diet are less dramatic and predictable than would be expected on the basis of Feingold's claims. However, these studies suggest that there is a small subset of children who do demonstrate a dramatic reduction in hyperactive symptoms when following the diet. Preschool children, as compared to school-age children, may be more sensitive to food dyes, and may respond more favorably to the Feingold diet (Harley et al., 1978; Lipton & Mayo, 1983).

It is important to note that Feingold eliminates many other substances in addition to food dyes. Food dyes account for only 10 out of over 3,000 additives, that are eliminated from the diet (Rimland, 1983). How researchers can claim that they have tested the Feingold diet, which eliminates 3,000 additives by conducting experiments on fewer than 10 food dyes, is questionable. Another problem with the food dye challenges is that the researchers used trivially small doses of coloring, trying to provoke hyperactivity in the children. When Swanson and Kinsbourne (1980) used colorings at the higher level, the effects were supportive of the Feingold hypothesis. Also, because Feingold eliminates almost all processed foods, there are other dietary variables that may be responsible for the positive effects demonstrated by the diet. For example, children on the Feingold diet often consume lower quantities of refined sugars (Prinz, Roberts, & Hantman, 1980). So, in evaluating the efficacy of Feingold's diet, other dietary factors must be taken into consideration.

REFINED SUGARS

The speculation that sugar consumption may cause or aggravate hyperactivity is especially relevant, given the increasing amount of sugar children consume. It is estimated that, on the average, each child in the United States consumes about 2 lb of sugar per week (Goldman, Lerman, Contois, & Udall, 1986). Langseth and Dowd (cited in Prinz & Riddle 1986), administered a 5-h glucose tolerance test to 261 hyperactive children (ages 7–9). Seventy-four percent of the children had abnormal glucose tolerance curves. Half of the abnormal curves were low and flat, similar to those seen in individuals with hypoglycemia. According to Langseth and Dowd, hypoglycemia is associated with an increased production of epinephrine, which in turn can stimulate a nervous or restless reaction. In susceptible individuals, the ingestion of large amounts of sugar may result in reactive hypoglycemia, which can set off this type of reaction. Whether the suspected behavioral effects of sucrose are due to reactive hypoglycemia or to a direct effect of sucrose (allergic reaction) remains unclear (Varley, 1984).

Prinz et al. (1980) obtained 7-day food diaries for 28 hyperactive and 26 normal 4–7-year-old children. Trained observers, unaware of the nature of the experiment, rated the children for a variety of behaviors during play. These included destructive–aggressive acts (attempts to damage, strike, kick, or throw objects in the room), restlessness (repetitive

arm, leg, hand, or head movements), and overall movement. Hyperactive and normal children consumed equivalent amounts of sugar rich foods. For the hyperactive children, the amount of sugar consumed was positively correlated with destructive–aggressive and restless behaviors. In contrast, sugar intake was not correlated with destructive–aggressive behavior in the normal group, but was correlated to total body movements. Their findings also suggested that younger children might be more susceptible to the adverse effects of sugar. The authors were careful in interpreting their results as being only suggestive evidence for the role of sugar in hyperactive behavior, because causality and directionality cannot be deduced from a correlational study.

In a subsequent study, Prinz and Riddle (1986) selected a group of nonhyperactive children and measured their ability to sustain attention after evaluating their diet for sugar consumption. On the basis of entries in a 1-week food diary, those consuming above the 75th percentile for sugar intake (5.47 g/kg of body weight) were less able to sustain attention than those below the 25th percentile (3.23 g/kg).

One limitation of these studies is that it is impossible to determine a cause and effect relationship. Despite the fact that these studies support the possibility of a sugar–behavior relationship, we cannot determine causality. It is likely that a third variable, such as lack of parental discipline (Prinz & Riddle, 1986), may be the causal factor in the sucrose/hyperactivity relationship making it appear as though they are related.

Other problems with these correlational studies are that they are based on retrospective assessments of the children's food intake, and provide data only on what children had been eating a week or two before the study. Sugar's effect on behavior may be more immediate. Most reports of sugar adversely affecting behavior are noted within .5–1 hr after consumption. In order to discern direction of causality between sugar consumption and hyperactive behaviors, an experimental manipulation of sugar consumption is required. The dietary challenge method is commonly used along with attempts to approximate real-life situations reported by parents to demonstrate the adverse effects of refined sugar on their children's behavior, which include hyperactivity, aggression, irritability, and decreased attention span. In these studies, children's behavior is rated for several hours after they have consumed either a sugar-containing food or beverage, or a placebo containing an artificial sweetener (aspartame or saccharine). Double-blind procedures are used to prevent expectations from influencing the results. Crossover procedures are also performed in which children are given the sugar-containing item on one day and a placebo on another, with the order of presentation varying among participants.

Behar, Rappaport, Adams, Berg, and Cornblath (1984) investigated the effects of sucrose on the behavior of 21 boys, 6–14 years old. All of the participants were reported by their parents to have adverse behavioral reactions to sugar. After an overnight fast, the participants were given a lemon-flavored beverage containing either sucrose (at 1.75 g/kg of body weight), glucose, or saccharin of equivalent sweetness. The amount of sucrose used in this study was about 52 g taking a child of 30 kg body weight as a reference. This is approximately equal to 13 teaspoons of sugar, the amount usually found in a 12-oz can of *Mountain Dew* (colas contain approximately 10 teaspoons of sugar per 12-oz can). Motor activity, spontaneous behavior, and performance on psychological tests were assessed for 5 hr after the boys drank the beverages. The only significant finding was that the boys were less active 3 hr after ingesting the sugar-containing beverages compared to the controls. They found no change in cognitive performance or behavior.

Similar negative findings were reported by Wolraich, Milich, Stumbo, and Schultz (1985). They conducted two carefully controlled challenge studies assessing the effects of sucrose ingestion on the behavior and learning abilities of 32 hyperactive boys (ages 7–12). In both studies, the participants were admitted to a clinical research center, where they were given a sucrose-free diet for 3 consecutive days. On Day 1, baseline measures of behavior and cognitive performance were taken. On Days 2 and 3, the participants drank a fruit-flavored drink containing either sucrose, at 1.75 g/kg of body weight, or aspartame of equivalent sweetness. In the first study, the drink was given 1 hr after lunch and in the second study, the drink was given in the morning after an overnight fast. Sugar intake did not affect behavior or performance on a number of cognitive tasks designed to measure attention, memory, and learning abilities.

A limitation of the preceding studies is that they were conducted in a laboratory environment. This rather unnatural environment may mask the effect of sugar on behavior. To address this issue, Milich and Pelham (1986) evaluated 16 hyperactive boys after a sucrose (1.75 g/kg) or placebo (aspartame) challenge in a naturalistic setting. Actual classroom measures of academic productivity and accuracy, and on-task behavior were examined, as well as direct observations of social interactions, noncompliance with adult requests, and positive and negative peer interactions in a playground setting. Sugar intake had no adverse effects on either behavior or academic performance.

Ferguson, Stoddart, and Simeon (1986) examined the behavioral and cognitive effects of a range of doses of sucrose. Eight children from 5 to 13 years of age, who were reported by their parents to have adverse reactions to sugar, were selected for the study. Five out of the eight participants were diagnosed as having attention-deficit disorder with hyperactivity. They were fed a low sucrose diet for several weeks, and then challenged with either high (1.5 g/kg), medium (1.0 g/kg), or low levels (0.5 g/kg) of sucrose or aspartame (placebo). Their performance on a task requiring sustained attention and on a series of tests examining learning, memory, and problem-solving abilities was then evaluated. The authors found no deleterious effects of sucrose on any of the measured variables.

Goldman et al. (1986) investigated the effect of sucrose consumption on the behavior of eight normal, healthy preschool children. Each child was observed for 90 min following a sugared drink (2 g/kg) and for 90 min following a placebo. During these observations the child alternated between 15-min sessions of work on structured tasks and 15-min sessions of free-play. Following the sucrose drink the children showed a decrement in performance in the structured testing situation and they were more active and less task-oriented during the free-play sessions. Goldman et al.'s findings suggest that younger preschool children may be more sensitive to sucrose than are older children (Goldman et al., 1986).

Recently there have been some reports about possible adverse behavioral effects of aspartame (nutrasweet or equal). If indeed some children do react negatively to aspartame, then all the prior studies conducted comparing sucrose ingestion to aspartame ingestion would be invalid. Wolraich et al. (1994) attempted to address this problem in a double-blind controlled trial with two groups of children. One group consisted of 23 school-age children (6–10 years old) who were described by their parents as being sensitive to sugar, and the other group consisted of 25 normal preschool children (3–5 years old). The participants and their families were placed on a different diet for each of three consecutive 3-week periods. One of the three diets was high in sucrose (4.5 g/kg) with no artificial sweeteners, another was low in sucrose and contained aspartame, and the third was low in sucrose and contained

saccharin (the placebo). No challenge was administered in this study. All the diets were free of additives, artificial food coloring, and preservatives. The children, their families, and the research staff were blind as to the sequence of the diets. The children's behavior and cognitive performance were evaluated weekly. None of the three diets produced any significant cognitive or behavioral changes in either normal preschool children or in school-age children believed to be sensitive to sugar.

A problem with this study is that participants were selected solely on the basis of parental reports describing their children as being sugar sensitive. What were the criteria? Only five of the presumably sugar-sensitive children met the criteria for attention-deficit disorder with hyperactivity. Other preceding studies (Behar et al., 1984; Ferguson et al., 1986) also used the same protocols, while Goldman et al. (1986) studied normal children without any history of adverse reactions to sugar. Studying the reaction of a normal population to sugar ingestion is hardly generalizable to the ADHD population.

Another controversial issue concerned the timing of the behavioral and cognitive assessments. Goldman et al. (1986) proposed that behavioral reactions to sucrose may vary depending on the amount of time elapsed between sucrose ingestion and the behavioral assessment. Researchers testing cognitive and behavioral effects of sucrose do so soon after a sucrose challenge. Maximum reactions generally occur between 30 min and 2 hr following ingestion of a sucrose challenge. Wolraich et al. (1994) did not consider the timing of the ingestion of sucrose, aspartame, or saccharin when studying behavioral and cognitive variables.

In general, the results of these studies do not support the contention that sugar consumption "causes" hyperactivity. Many limitations in the study designs must be addressed to properly evaluate their results. One point already addressed concerns participant selection. Do children who are identified as sugar sensitive by their parents accurately reflect the population we are interested in studying (Behar et al., 1984; Ferguson et al., 1986; Wolraich et al., 1994)? Many of the studies suffered from a small sample size; Goldman et al. (1986) and Fergueson et al. (1986) both selected only 8 children for their studies, Milich and Pelham (1986) studied 16 boys, and Behar et al. (1984) looked at 21 participants. These studies with small sample size have limited power to detect significant effects. It is possible that larger samples would have produced larger and more significant differences in behavioral and cognitive variables.

Another potential limitation of these studies concerns the dosage level of sucrose employed. Dosages used in the challenge studies were probably too small to have any significant effect when compared to children's normal daily intake. Goldman et al. (1986) contend that children consume around 2 lb of sugar per week. That is about 4.6 oz or 128 g or 32 teaspoons of sugar per day. It is now 15 years since this statistic was published and children presently consume between 40 and 50 teaspoons of sugar per day. This is surprisingly easy to obtain in the U.S. diet. Children who consume approximately 3–4 cans of soda per day can easily reach 40 teaspoon per day, especially when consuming the typical U.S. diet of sugar-coated cereals, cookies, ice-cream, and other snack foods. Most of the studies tested 1.75–2.0 g/kg of body weight, which in a 30 kg child adds up to only 52.5–60 g or 13–15 teaspoons of sugar (Behar et al., 1984; Goldman et al., 1986; Milich & Pelham, 1986; Wolraich et al., 1985). In the only study investigating different dosage levels (Ferguson et al., 1986), the dosage ranged from 0.5 to 1.5 g/kg. All dosages were lower than those examined in the previous studies.

The previous studies are similar in that they challenged participants with one or two substances. What if these children are sensitive to a variety of food items? If multiple sensitivities are a problem in this population, than eliminating only one of the offending substances (e.g., sugar or food dyes) may not make a significant difference in behavior.

FOOD ALLERGIES/SENSITIVITIES

It is well recognized that approximately 15–20% of the population have respiratory or cutaneous allergies. There is approximately a 70% incidence of allergy in the hyperactive population. This strongly suggests a relationship between allergy and hyperactivity (Rapp, 1978). When compared to controls, participants with ADHD had a statistically higher incidence of asthma, stomachaches, and ear infections. A greater percentage of participants with ADHD had tubes placed in their ears because of repeated ear infections (Stevens et al., 1995).

Allergy can manifest in various ways: as skin rashes (eczema), respiratory problems (rhinitis, asthma, and ear infections), urinary disorders, headaches, stomachaches, and muscle and joint disorders. Some physicians and environmental medicine specialists (Crook, 1980; Rapp, 1979, 1991) have demonstrated that behavioral and learning disorders can be caused by specific allergies. Several methods have been used to test the food allergy hypothesis. To determine if diet is provoking behavioral symptoms, a child can eliminate the suspected food from the diet for 2–3 weeks to see if the condition improves. If improvement occurs the suspected food can then be reintroduced to the diet to see if the symptoms recur. Some children with food allergies are often addicted to the very food item that is causing their symptoms. Often symptoms can temporarily intensify when the item is eliminated from the diet (Crook, 1980).

Another method to diagnose food allergies is to follow a chemically defined diet, where all meals are replaced with an allergy-free liquid supplement. Hughes, Weinstein, Gott, Binggeli, and Whitaker (1982) gave 10 participants a chemically defined diet, which provided 1,800 calories and total nutritional support. All 10 participants showed improvement on objective measures for ADHD. The researchers concluded that by replacing the diet with a hypoallergenic drink, all potential allergenic foods were eliminated from the diet and participants' behavior improved. Although they did not take the study further, the next step would have included testing foods one by one to identify any food sensitivities.

Most of the previous studies (Behar et al., 1984; Conners et al., 1976; Ferguson et al., 1986; Goldman et al., 1986; Harley et al., 1978; Wolraich et al., 1985) have investigated either the role of food colors or sugar consumption on hyperactivity. They have paid no attention to milk, wheat, corn, eggs, chocolate, and other foods that commonly cause adverse or allergic reactions in children. Even if the children were sensitive to the substances being tested, if they were also sensitive to other foods that were not eliminated from their diet, no significant improvement could be demonstrated. "If you have ten pebbles in your shoe and you remove one, you will still limp" (Rapp, 1991, p. 166).

Researchers who study food allergies do not focus on a specific food substance. They work individually with children to try to identify the foods or substances to which the child might be sensitive. Researchers note that children are allergic to some common and nutritious foods including milk, wheat, eggs, corn, as well as food additives and colorings (Crook, 1980).

Egger, Carter, Graham, Gumley, and Soothill (1985) selected 76 hyperactive children for their study. A high proportion of their sample had allergic conditions; 32 children had either eczema, asthma, or hay fever, and 52 children had close family members with these conditions. Twenty-four participants reported a history of ADHD in the immediate family. In the first phase of the study, the participants were placed for 4 weeks on an individually designed elimination diet consisting typically of two meats (e.g., lamb and chicken), two carbohydrate sources (e.g., potatoes and rice), two fruits (e.g. banana and apple), vegetables, water, calcium, and vitamins. In addition, participants were asked to avoid any foods suspected of provoking behavioral or allergic reactions and those for which the child had a particular craving or dislike. Sixty-two of the children (82%) showed improved behavior after following the elimination diets.

In the second phase of the study, foods that were suspected of causing reactions were reintroduced weekly, one at a time. If a reaction occurred the food was discontinued. If no reaction occurred the food was integrated into the diet. In the third phase, children who reacted to various food items were asked to enter a double blind, crossover, placebo-controlled trial of reintroduction of one incriminated food. Similar to the food dyes and sugar studies, the placebo and challenge foods were prepared so that it was not possible to distinguish between them. Children were assessed by their parents and by members of the research team. Ratings indicated that the children's behavior deteriorated when the offending food item was ingested. In addition to documenting a relationship between food sensitivities and hyperactive behavior, Eggers et al. (1985) also illustrated the wide variety of foods to which individual children may be sensitive. Forty-seven provocative foods were identified and most children reacted to more than one food. The most common foods found to cause allergic reactions were benzoic acid (preservative) and tartrazine (food coloring); 79% of the children tested for these reacted negatively. Other offending foods were cow's milk (67%), chocolate (59%), wheat (49%), oranges (45%), and eggs (39%). Interestingly, sugar elicited a negative reaction in only 16% of the children tested.

Carter et al. (1993) confirmed the results of the Egger et al.'s study (Egger et al., 1995). Seventy-eight participants took part in the elimination diet, food reintroduction, and double-blind challenge. The participants had outcomes similar to the participants in the previous study. Food additives caused reactions in 70% of the participants; other food sensitivities included cow's milk (64%), chocolate (64%), oranges (57%), wheat (45%), tomatoes (22%), and eggs (18%).

Breaky (1997) and Boris and Mandell (1994) found peanuts, corn, fish, and soy to elicit negative reactions in sensitive children. Boris and Mandell (1994) additionally found that 73% of the hyperactive children in their study responded favorably to an elimination diet. The participants were then placed on a double-blind placebo controlled food challenge where they were given capsules of the foods they responded to negatively. There was a statistically significant improvement on the placebo days as compared with the challenge days. Children with allergies such as eczema, asthma, rhinitis, and urticaria had a significantly higher response rate than the nonallergic group demonstrating a connection between typical allergies and ADHD.

Rapp (1978) demonstrated that as long as the children avoided the offending food substances they remained symptom-free. Compliance, however, with individualized diets, which omit several frequently consumed foods, can be a major challenge for both parents and children. An attempt was made to treat these patients with food extracts, which would

eliminate the need to omit the allergenic food (Rapp, 1979). Eleven participants from her original study were given extracts either sublingually or subcutaneously of the foods they identified as causing allergic reactions. After receiving the extracts they were allowed to ingest the offending food and they remained asymptomatic. Five of these patients completed a double-blind evaluation of the food extract and successfully differentiated the food extract from two placebo solutions.

Rapp (1991) is strongly in favor of the provocative/neutralization test (P/N). She argues that it is a reliable, sensitive, and accurate method of dietary challenge, which can confirm a suspected allergy. In the P/N test, allergies are determined by "provoking" children with a small dose (0.05 ml/2 drops) of the suspected food allergen. A positive test is determined by behavioral symptoms after the intradermal or sublingual dose is administered. Neutralizing doses are achieved by sublingually giving the participants 0.05 ml of the substance of succeedingly weaker strengths until normal behavior response is obtained. Children can then be treated with three drops of this allergy extract either under the tongue three times a day, or via an injection once or twice a week.

O'Shea and Porter (1981) conducted a double-blind study testing 14 children for allergies with the use of the provocative intradermal and sublingual method. An optimal neutralizing dose was then developed and administered sublingually. The study was conducted over a 6-week period—3 weeks of treatment with the allergen extract and 3 weeks with a placebo. Parents, teachers, and a psychologist monitored each child's behavior weekly. Out of 14 participants, 11 reported significant improvement during the treatment period as compared to the placebo.

Results of the Egger et al. (1985) study along with the results of related investigations (Boris & Mandell, 1994; Carter et al., 1993; Crook, 1980; O'Shea & Porter, 1981; Rapp, 1978, 1979, 1991) effectively demonstrate that food sensitivities or allergies can be involved in provoking behavior problems. It is clear that more research is needed, but investigators working in this area are faced with a number of problems. Food sensitivities are highly individualized in terms of the offending food and in the behavioral responses that are provoked. Many of the scientific designs that are considered acceptable are difficult to perform in this population. For example, the gold standard of experimental design includes random selection of participants, random assignment of participants to groups, and identical treatment procedures. These procedures cannot be applied in research with these highly individualized participants. More innovative methods must be developed where "*N*" of one and case studies are acceptable .

FATTY ACID RESEARCH

A new and promising area of study involves essential fatty acid (EFA) research. The omega-6 (ω-6) and the omega-3 (ω-3) fatty acids are considered to be essential fats. The body cannot synthesize them and they must be obtained from the diet. Through the processes of elongation and desaturation, the omega-6 fatty acid, linoleic acid (LA: 18:2ω-6), forms longer and more unsaturated fatty acids, including gamma-linolenic acid (GLA: 18:3ω-6), dihomogamma-linolenic acid (DGLA: 20:3ω-6), and arachidonic acid (AA: 20:4ω-6). The omega-3 fatty acid, alpha linolenic acid (ALA:18:3ω-3), is a precursor to eicosapenaenoic acid (EPA: 20:5ω3) and docosahexaenoic acid (DHA: 22:6ω3). These long chain fatty acids are known as eicosanoids (contain 20 or more carbons) and are necessary for the formation

of prostaglandins, leukotrienes, and thromboxanes, which are local hormones responsible for regulating many functions in the body.

Recent studies have found that many children with ADHD have altered fatty acid metabolism. Stevens, Zentall, Abate, Kuczek, and Burgess (1996) found a greater number of behavior problems in participants with lower omega-3 fatty acid concentrations. Many of these children also manifested other symptoms of fatty acid deficiency such as thirst, frequent urination, dandruff, dry skin, and dry hair. Mitchell, Aman, Turbott, and Manku (1987) found levels of DHA, an omega-3 fatty acid, DGLA, and AA omega-6 fatty acids to be significantly lower in hyperactive children than in controls.

Colquhoun and Bunday (1981) hypothesized that children who have a deficiency in EFAs are either unable to absorb or metabolize fats, or have a higher than normal requirement for EFAs. They observed the following: many of the foods the children are allergic to inhibit the conversion of EFA to eicosanoids; ADHD is much more common in boys than in girls, and males are known to have much higher EFA requirements than females do; many children with ADHD have abnormal thirst, which is a sign of EFA deficiency; many hyperactive children have eczema, allergies, and asthma, which may be alleviated by EFAs; and many children are deficient in zinc, which is required for conversion of LA to GLA.

Kane (1999) analyzed the fatty acid content of the red cell membrane and found imbalances in EFAs in children with ADHD. These included the omega-3 fatty acids, EPA and DHA, and the omega-6 fatty acids, GLA and AA. Children who were supplemented with the specific fatty acids they were deficient in showed noticeable improvements in their behavior.

SUMMARY

Recent research has investigated the link between diet and ADHD. Although there is a great deal of controversy surrounding the effects of specific food components (e.g. food colorings and sucrose) on the behavior of hyperactive children, there is increasing evidence that there is a subset of children with behavioral problems who are sensitive to one or more food components that may precipitate or contribute to their hyperactive behavior. Research indicates that it is futile to try to identify a specific food or substance that will precipitate negative behavior in all hyperactive children. Studies clearly demonstrate that not all children respond to the same offending substance. Future research should include investigating simple methods to identify individual allergens and sensitivities, determining who is susceptible, understanding the mechanisms behind these reactions, and devising effective methods for treating food sensitivities.

Recently it has been proposed that by correcting imbalances in EFA metabolism, hyperactive behavior may be resolved along with food and chemical sensitivities (Kane, 1999). This is an exciting area that needs further investigation and may prove to be a significant step toward managing ADHD.

Another exciting area for future research is the use of topographic EEG mapping of brain electrical activity (Uhlig et al., 1979) as a diagnostic tool for identifying food sensitivities. Currently identification of food sensitivities is based solely upon participants' behavioral and cognitive performance after consuming a provoking food substance. By identifying changes in brain electrical activity, EEG mapping can provide a strong objective measure. Larger studies using this technique are warranted.

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