



## Primary Care Approaches

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# Diet and Child Behavior Problems: Fact or Fiction?

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Dietary treatment of children with behavioral disorders has had wide public appeal and been a source of controversy since the 1920's. Yet, to date, there is little empirical evidence supporting the effectiveness of dietary restrictions in treating child psychiatric disorders, in particular, autism and attention deficit hyperactivity disorder (ADHD). Thus, the purpose of this article is (a) to provide historical background information regarding dietary treatment in children with behavioral disorders, (b) review the evidence-based literature for common dietary interventions, (c) discuss limitations in the research, including challenges inherent in conducting well-controlled dietary studies, and (d) provide recommendations regarding how nurses in primary care settings can assist families in making informed decisions.

### Case #1

Adam, a 10-year-old boy, was diagnosed at 30 months with autism and hyperactivity. Adam has been treated with a variety of medications including mood stabilizers, psychostimulants, and anti-anxiety medications, but continues to display severe tantrums when frustrated and other behaviors his mother, Ann, describes as "unpredictable" and "almost impossible to manage, particularly in public." Along with these troubling behaviors, Adam exhibits classic autistic traits such as impaired social relatedness, delayed language development, apparent disinterest in other children, lack of imaginative play, and difficulty with changes in his environment and routine.

Like many families of children with autism, Adam's parents spend much time on the Internet searching for possible solutions and reading testimonials of individuals who have made "miraculous recoveries." Adam's mother has recently become intrigued by reports indicating positive results from diets restricted of gluten (found in wheat products) and casein (present in dairy foods). During a routine visit, Ann asked the nurse for her opinion, "What do you know about it? Should I try the diet?"

### Case #2

Jimmy, a 6-year-old boy, was recently diagnosed with attention deficit hyperactivity disorder (ADHD) by a child psychiatrist and prescribed amphetamine sulphate (Adderall). His mother, Lillian, tells the psychiatric nurse

*practitioner, who is monitoring Jimmy's medication, that her son is lethargic, complains of stomachaches, and refuses to eat most of the day. He is also irritable in the evening and has difficulty getting to sleep. Lillian says that she has been exploring alternatives to medication for Jimmy and wants the nurse practitioner's opinion about dietary treatment for ADHD. She has with her information on dietary modifications she has downloaded from web sites on ADHD. "I really feel guilty about making Jimmy take this medication when it makes him sick. I've read that cutting down on food dyes and refined sugars can help kids with ADHD calm down and focus better at school. Why hasn't anyone mentioned this to me before?"*

### Introduction

For many parents and health care providers of young children with behavior or developmental problems, choosing an effective and acceptable treatment is difficult. As is often the case with chronic childhood disorders such as autism and ADHD, a broad array of treatments have been tried and continue to be used, yet only a select few have been scientifically validated as safe and effective. Despite their questionable efficacy, the use of complementary and alternative therapies, in particular dietary interventions, has become widespread in primary care settings (Chan, Rappaport, & Kemper, 2005). Concerned parents frequently approach clinicians in primary care settings with questions about the potential benefit of dietary restrictions on child behavior, and health care providers are accountable for supplying relevant, empirically sound, and helpful information. This article will review the evidence for the most common dietary modifications employed in the treatment of autism and ADHD, including historical background information regarding dietary treatment in children with behavioral disorders, the evidence-based literature published over the past two decades, limitations in the research, including challenges inherent in conducting well-controlled dietary studies, and recommendations regarding how nurses in primary care settings can assist families in making informed decisions.

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The *Primary Care Approaches* section focuses on physical and developmental assessment and other topics specific to children and their families. If you are interested in author guidelines and/or assistance, contact Patricia L. Jackson Allen at pat.jacksonallen@yale.edu.

## Historical Background and Significance

Dietary treatment of behavioral problems in children is not new. According to clinical reports and lay media, however, it appears to be gaining in popularity. Beginning in the 1920s (Shannon, 1922), literature has included reports on restricted diets and their effect on child behavior. Most famous is Feingold's work in the 1970s, in which he noted that at least 50% of hyperactive and learning disabled children improved when placed on diets free of salicylate and additives (Feingold, 1975). Subsequent controlled studies failed to support Feingold's claims but did identify a small subset of children with true sensitivities to food additives (Connors, Goyette, Southwick, Lees, & Andrulonis, 1976; Harley, Ray, Tomasi, Eichman, Matthews, et al., 1978). In the early 1980s other researchers reported adverse effects of sugar on hyperactive and aggressive behavior (Prinz, Roberts, & Hantman, 1980; Wolraich, Stumbo, Milich, Chenard, & Shultz, 1986). Over time, most papers advocating dietary treatment have combined the recommendations of Feingold and later investigators to restrict not only food additives, preservatives and sugars, but also dairy products, wheat, corn, yeast, soy, citrus, eggs, chocolate, and nuts—foods commonly associated with allergic reactions in children (Boris & Mandel, 1994; Carter, Urbanowicz, Hemsley, Mantila, Strobel, Graham, et al., 1993; Crook, 1980; Egger, Carter, Graham, Gumley, & Soothill, 1985; Kaplan, McNicol, Conte, & Moghadam, 1989; Rapp, 1991). Others have proposed that correcting imbalances in fatty acid metabolism may resolve child behavior problems along with food and chemical sensitivities (Colquhoun & Bunday, 1981; Stevens, Zentall, Abate, Kuczek, & Burgess, 1996; Kane, 1999).

Currently, with mounting concern about the efficacy and safety of medications, health care providers and families are seeking alternative approaches to treatment for child behavior and/or developmental problems associated with autism and ADHD. Diet modification holds considerable appeal for parents, as it augments their sense of control and efficacy and aligns well with the desire to promote a healthy lifestyle for their child. However, controversy continues concerning the effects of nutritional factors on the behavior of children. Proponents of dietary treatments cite evidence of a subset of children with behavioral problems who are sensitive to one or more food components that may precipitate or contribute to behavior problems (Schnoll, Burshteyn, & Cara-Aravena, 2003). Critics emphasize shortcomings in study designs that raise doubts about the validity of these research findings (Rojas & Chan, 2005). In general, dietary interventions have been studied by placing the child on a restricted diet for a period of time and then challenging them with the offending food component versus placebo, or placing different groups of children on diets that differ only in respect to the offending agent (e.g. sucrose versus artificial sweetener). Crossover designs are often employed in which the child experiences the challenge agent and placebo or the active diet in sequence, thus acting as his or her own control.

## Common Dietary Interventions

**Additive-free diet.** Originally introduced in 1975, Feingold hypothesized that food additives (in particular, artificial flavors and colors, and naturally occurring salicylates) were associated with learning disabilities and hyperactive behavior in some children. Feingold's claims gave rise to an avalanche of case reports and media attention, but controversy ensued in the scientific community about the validity of his findings, which were based on clinical observations rather than rigorous experimental evidence (Wender, 1986). Subsequent controlled studies either evaluated the behav-

iors of hyperactive children while on the Feingold diet, as compared to a placebo diet (dietary crossover designs), or investigated responses to specific food dye challenges (Lipton & Mayo, 1983).

In the early crossover studies of the Feingold diet, hyperactive children were randomly assigned to either the elimination diet or to a control diet, then crossed over to the other treatment condition. These studies generally used behavior ratings by parents and teachers and occasionally psychological tests of attention and impulsivity to measure outcomes, as opposed to physiological indicators. Several reviews (Mattes, 1983; Wender, 1986; Williams & Cram, 1978) and one meta-analysis (Kavale & Forness, 1983) have concluded that the Feingold diet is not an effective treatment for hyperactivity, highlighting a range of methodological challenges and flaws such as defining the study population, obtaining an adequate sample size, specifying a consistent diet, ensuring dietary compliance, developing an equivocal placebo control, using an adequate dose of artificial additive or coloring, and measuring appropriate outcomes in a standardized and precise manner. Even studies employing a double-blind, placebo-controlled design with a clearly defined study population, adequate sample size, and outcome measures demonstrated negative or at least ambiguous results (Connors, Goyette, Southwick, Lees, & Andrulonis, 1976; Harley, Ray, Tomasi, Eichman, Matthews, et al., 1978). Researchers who limited their investigations to evaluating child behavior responses to specific food dye challenges found only a small group of children who responded to some aspects of the Feingold diet (Silver, 1986).

The most recent studies of the effects of specific food additives and/or preservatives on child behavior have been conducted using double-blind, placebo-controlled, challenge crossover designs with children who have a history of atopy or parent-reported adverse reactions to food additives, but do not necessarily meet diagnostic criteria for ADHD. Selection bias continued to be a problem in these studies due to attrition, with possible overrepresentation by families interested in hyperactivity. Children did, however, demonstrate increased behavioral symptoms when challenged with artificial flavors, most notably tartrazine and preservatives such as calcium propionate (Bateman, Warner, Hutchison, Dean, Rowlandson, et al., 2004; Dengate & Ruben, 2002; Rowe & Rowe, 1994).

The recent meta-analysis conducted by Schab and Trinh (2004) also warrants mention. This analysis focused specifically on the effect of artificial food colors on hyperactivity rather than the Feingold Diet as a whole. Compared to previous meta-analysis by Kavale and Forness (1983), these authors employed hypotheses that were more explicit and rigorous, consisting of only double-blind placebo-controlled trials, including 6 trials conducted subsequent to the earlier analysis, and used statistical techniques that more richly exploit the advantages of crossover trials. The results of this meta-analysis support the hypothesis that artificial food colors can contribute to symptoms of childhood hyperactivity in some children.

**Sugar elimination diet.** The idea that foods containing sugar, mainly sucrose, might have an adverse effect on behavior was first hypothesized by Shannon (1922) and revisited by Randolph (1947) in his description of tension fatigue syndrome. Sucrose later appeared as a suspected offending substance in the 1970s as a result of coverage in the lay literature on the condition called functional reactive hypoglycemia (Deutsch, 1977). Using food diaries and observations of behavior, two cross-sectional studies found varying correlations between sugar intake and hyperactivity

(Prinz, Roberts, & Hantman, 1980; Wolraich, 1996). These early studies were limited by the use of correlational designs in which it is impossible to determine causality or directionality. In short, it was as equally possible that the children's adverse behavior caused the increase in sucrose intake as it was that the increased sucrose intake caused the behavior. Other variables such as lack of parental discipline may have also been causal factors (Prinz & Riddle, 1986).

Wolraich, Wilson, and White (1995) conducted an extensive and thorough review of 16 double-blind, placebo-controlled studies evaluating the effects of sugar on child behavior. Participants included normal children, children identified by parents as behaving poorly after sugar ingestion, children with diagnosed hyperactivity or ADHD, and aggressive, delinquent children. Measures focused primarily on the behavior of children with ADHD and used behavior-rating scales completed by parents and teachers (along with neuropsychological measures) to assess vigilance, impulsivity, memory, and motor skills. In spite of considerable variation in subjects, challenge agents, and dependent measures, the results were remarkably consistent. Findings did not support the hypothesis that refined sugar affects hyperactivity, attention span, or cognitive performance of children, although the possibility of an effect on a subset of children could not be ruled out.

It is interesting to note that despite presentation of clinical evidence to the contrary, many participating parents remained convinced of an association between sugar and adverse behavior. White and Wolraich (1995) suggest that parental expectations may lead to mistaken interpretations about context-driven behavior variations (e.g., parties or holidays), associating them with sugar consumption. Hoover and Milich (1994) found that parents who believed their child was receiving a challenge dose of sugar, when it was actually artificial sweetener, rated the child's behavior as significantly worse and more demanding than parents who rightly expected their child to receive an artificial sweetener.

**Food allergies and sensitivities.** The idea that hyperactivity in children can result from sensitivity to specific provocative foods overlaps with existing conceptions of food allergies (Marshall, 1989). Several investigators have broadened Feingold's original hypothesis to restrict not only food additives and dyes, but also sugars, dairy products, wheat, corn, nuts, eggs, chocolate, and other foods that commonly cause allergic reactions in children (Boris & Mandel, 1994; Kaplan, McNicol, Conte, & Moghadam, 1989; Rapp, 1979). These studies have reported improvements in behavior symptoms associated with ADHD after 2-3 weeks on the experimental diet.

Other investigators have utilized more controlled research designs to assess the effects of the highly restrictive oligoantigenic diet (OAD), devoid of known food allergens (Carter et al., 1993; Egger, Carter, Graham, Gumley, & Soothill, 1985; Egger, Stolla, & McEwen, 1992; Schmidt, Mocks, Lay, Eisert, Fojkar, et al., 1997). OAD studies typically consist of three or four phases. In Phase 1, generally 4 weeks, subjects receive the OAD, with an alternative OAD available if they do not show improvement with the first version. Those subjects whose symptoms resolve by the last 2 weeks of Phase 1 move on to Phase 2, when excluded foods are gradually reintroduced. If symptoms do not recur, these foods are added to the subject's diet. Those who are eventually able to tolerate a satisfactory diet in terms of nutrition and preference, and who previously reacted adversely to a food for which both a test version and a placebo version are available, then enter Phase 3. This consists of the placebo-controlled, crossover food challenge trial, to assess whether

symptoms are reproducible. The trial reintroduces one or more provoking foods or placebos, given for approximately 1 week, followed by a washout period. The studies cited above all reported improved behavior ratings both during the OAD period (Phase 1) as well as during the placebo-controlled food challenge (Phase 3).

As a whole, the OAD studies effectively demonstrate that food sensitivities or allergies can be involved in provoking behavior problems in certain children. They also suggest that children who do have identifiable sensitivities to certain foods might benefit from an elimination diet such as the OAD. However, evaluation of the relationship between food allergies and child behavior is complicated by the methodological problems in this line of research. These problems include (a) the questionable reliability and validity of various forms of allergy tests; (b) the small sample sizes, precluding the possibility of demonstrating significant results (only a small percentage of children enrolled in the OAD portion of the study eventually qualify for the food challenge trial phase); (c) the use of subjective rating scales from informants (usually parents) who may be biased; and (d) difficulties in determining appropriate challenge doses and timing of testing to accommodate the idiosyncrasies of different individuals' allergic reactions (Rojas & Chan, 2005; Marshall, 1989).

**Fatty acid supplementation.** A recent area of study involves essential fatty acids (EFA), in particular arachidonic acid (AA), eicosapentaenoic acid (EPA), and docosahexanoic acid (DHA). It is well established that these fatty acids are important in the structural and functional development and maintenance of neuronal membranes (Horrocks & Farooqui, 2004; Wainwright, 2002). Deficiencies in essential fatty acids have been implicated in the pathogenesis of a range of developmental and behavioral disorders, including ADHD (Burgess, Stevens, Zhang, & Peck, 2000; Richardson, 2004). Further, studies identifying lower plasma concentration levels of certain essential fatty acids among children with ADHD have led researchers to postulate that deficiencies are responsible for key features of ADHD (Colquhoun & Bunday, 1981; Stevens, Zentall, Abate, Kuczek, & Burgess, 1996; Yehudi, Rabinovitz, & Mostofsky, 1998).

To date, however, published trials of fatty acid supplementation in children with ADHD have failed to demonstrate improvements in symptoms of ADHD (Aman, Mitchell, & Turbott, 1987; Arnold, Kleylamp, & Votolato et al., 1989; Arnold, Kleykamp, Votolato, Gibson, & Horrocks, 1994; Richardson & Puri, 2002; Stevens et al., 2003; Voigt, Llorent, Jensen, Frayley, Berretta, et al., 2001). Again, a range of methodological problems may have limited the researchers' ability to identify true effects. Most of the studies had small sample sizes, did not confirm a diagnosis of ADHD, included subjects with coexisting psychiatric and developmental conditions, and addressed the issue of concurrent stimulant medication differently (Rojas & Chan, 2005). Refinements in study design are indicated before firm conclusions can be drawn about the efficacy of EFA supplementation with children who have developmental or behavioral disorders.

**Gluten-free, casein-free (GF/CF) diet.** In recent years there has been mounting interest in the use of a GF/CF diet for individuals with autism. Unfortunately, there is a paucity of empirical data to support claims that even include "miraculous cures." Cade and colleagues (1999), some of the first to study this diet in autistic populations, expanded initial laboratory work to applied settings and conducted a study of 270 individuals. One hundred and twenty of these participants were diagnosed with schizophrenia, and 149

met the DSM III criteria for a diagnosis of autism. All children with autism were treated with a GFCE diet, a synthesis of the *Milk Free Kitchen* by Kidder (1991) and the *Gluten-Free Gourmet: Living Well Without Wheat* by Hagman (1990). During the study, parents, physicians and some teachers independently assessed the children for the presence and severity of the diagnostic manifestations of autism using a 4-point Likert scale. These ratings were done initially and repeated after 1 month of treatment and then every 3 months for 1 year. Parent and physician reports were averaged, with variability of individual observer scores reported as less than 10%. Blood samples were examined to measure the absorption of peptides contained in wheat products (gluten) and dairy (casein) and the associated antibodies, immunoglobulin G (IgG) and transindolylacryloylglycine (IgA) for each of these food products. The study found that 87% of the children with autism had high titer IgG antibodies to gliadin, and 30% had high titer IgA antibodies to gluten or casein prior to initiation of the diet. Treatment with a GFCE diet was accompanied by reports of improvement in 81% of children within 3 months. A strength of this work was the combined use of physiological and behavioral measures. The behavioral results are limited, however, by the heavy reliance on reports from parents and teachers who knew that the children were on the GFCE diet.

In a comparative study, Arnold, Hyman, Mooney, and Kirby (2003) evaluated amino acid patterns of 26 children with autism on a regular diet, 10 on a gluten-casein free diet, and 26 with developmental delays who served as controls. The children with autism had higher deficiencies in essential amino acids compared to the control group. These findings suggest that children with autism are at high risk for amino acid deficiencies and may benefit from a structured diet. Clearly, this is an area that warrants further investigation. The authors note that a major limitation in the study was the small sample. An additional concern is the lack of strict dietary control for children on gluten-casein free diets, a commonly encountered problem in conducting dietary research in children.

Knivsberg, Reichelt, Høien, and Nodland (2002) and others conducted a randomized single blind study with 20 subjects to assess the effect of a gluten-casein free diet on children with autistic syndrome and urinary peptide abnormalities. The children in the control and experimental groups were matched according to severity of autistic symptoms, age, and cognitive level. The experimental group showed more significant changes than did the control, and demonstrated improvement in autistic behavior, non-verbal cognitive level, and motor problems. Conversely, Elder and colleagues (2006) recently conducted a randomized clinical trial of the GFCE diet with 15 children diagnosed with autism and no statistically significant findings were noted on any of the objective measures, although several parents reported perceived improvement. Clearly, there is a need for replicating this work with larger samples and rigorous controlled clinical trials that evaluate both physiological and behavioral effects.

### The Question of Parental Placebo Effect

Several of the studies reviewed in this paper introduce an important question regarding the possible influence of parental perceptions and/or expectations. Wolraich (1996) urges clinicians to use caution when considering dietary restrictions, noting that even though some parents firmly believe that diet greatly affects their children's behavior or learning, the power of suggestion on the part of parents can be significant. In addition to the influence of expectations, critics have also suggested that the success of dietary inter-

ventions may be related to the overwhelming attention parents give to their children by focusing on dietary compliance, rather than specific effects of the diet (Schnoll et al., 2003). Studies using correlational designs make it impossible to deduce causality and directionality of diet components and behavior because other factors, such as changes in parental attention and discipline practices, may be responsible for improved behavior (Prinz & Riddle, 1986).

### Resolution of Cases

**Case #1.** *The pediatric nurse practitioner carefully listened to Adam's mothers' inquiries regarding the GFCE diet and provided her with empirically sound literature that was kept updated and on-file in her office. She also gave the mother Internet links to resources needed to purchase the special dietary products, including price lists. Ann was encouraged to examine the pros and cons of implementing the diet before deciding. After learning that the mother also had concerns about her son's limited food repertoire, a referral was made to a nutritionist who was also well-informed about dietary treatments in children.*

*After examining the literature and resources and speaking with the nutritionist, the mother decided to try the diet for 3 months. She was urged to keep careful records of the child's intake, weight, and behavior exhibited at home and school. While some decrease in hyperactivity was noted during the 3-month period, Adam continued to exhibit autistic features. The mother also found it difficult to maintain the dietary restrictions, particularly when Adam was around other children. At the end of the trial period the mother made an informed decision to discontinue the GFCE diet and focus more on implementing behavioral strategies that she and her husband had been taught as part of an in-home parent training program.*

**Case #2.** *The nurse practitioner acknowledged Lillian's concerns regarding the unpleasant side effects Jimmy was experiencing on the Adderall and her interest in alternative treatments. She reviewed the material Lillian had with her on elimination diets (primarily refined sugars, food additives and/or dyes) and supplemented this with current, evidence-based literature on these dietary interventions for ADHD. She also provided a list of Internet resources containing practical guidelines for eliminating specific food components from a child's diet. She encouraged Lillian to consider the scientific support before pursuing a dietary modification requiring considerable time, energy, and financial expense. The nurse practitioner also indicated there were alternatives to Adderall should Lillian decide to reconsider stimulant medication at any time in the future.*

*Lillian's review of resource material motivated her to try eliminating artificial flavors, dyes, and preservatives from Jimmy's diet. The nurse practitioner provided assistance in identifying target behaviors to monitor once she implemented the diet. After a month on the diet, Lillian noted a significant reduction in hyperactivity at home and more cooperation with homework, but Jimmy's teacher reported little change in behavior at school. Lillian decided to continue the diet at home but also elected to give another stimulant medication a try during the school day.*

### Discussion

Both of the case studies presented in this paper illustrate a number of points that have implications for clinical practice and future research. First, it is important to evaluate not only what parents "know" about their child's disorder but also what they "believe" and who they consider to be credible sources. Second, because disorders such as autism and ADHD have no known cure, parents often feel powerless,

wanting desperately to help their children. They often look for the “magic” intervention rather than recognizing that successful treatment involves a variety of educational, behavioral, and parental interventions. Thus, astute clinicians must listen carefully to caregivers but also consider the possibility of treatment placebo effects. Discussion of nutritional status in general and the possible effect that might have on behaviors provides a valuable opportunity to learn about a family’s values and attitudes toward treatment and facilitates mutual exchange of information that reinforces the therapeutic alliance.

### Pediatric Nursing Role in Primary Care

The literature and case studies that have been reviewed suggest that primary health care providers have important roles in helping families make informed decisions before embarking on very costly and/or labor intensive interventions. The pediatric nurse must be open and accepting of parents’ ideas regarding alternative therapies that involve dietary modifications and prepared to share relevant information. A sound knowledge of the current evidence-based research regarding dietary interventions is essential. If parents express an interest in a particular dietary treatment, the nurse can provide them with pertinent literature, credible Internet resources, and practical information that can help them evaluate the pros and cons of pursuing the proposed dietary change.

Adherence to a highly restricted diet for even a short period requires tremendous commitment on the part of the family. Parents need to be aware of the difficulties and limitations these diets impose on the family as well as possible nutritional deficits. They also should be informed of the potential for parental bias and dietary placebo effects. The pediatric nurse plays a key supportive and educational role in helping families comprehend these realities, arrange consultation with a nutritionist if indicated, and provide objective evaluation tools to measure change if the diet is attempted.

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### References

Aman, M.G., Mitchell, E.A., & Turbott, S.H. (1987). The effects of essential fatty acid supplementation by Efamol in hyperactive children. *Journal of Abnormal Child Psychology*, 15, 75-90.

Arnold, G.L., Hyman, S.L., Mooney, & R., Kirby, R.S., (2003). Plasma amino acids profiles in children with autism: Potential risk for nutritional deficiencies. *Journal of Autism and Developmental Disorders*, 33, 449-454.

Arnold, L.E., Kleykamp, D., Votolato, N.A., Taylor, W.A., Kontras, S.B., & Tobin, K. (1989). Gamma-linoleic acid for attention-deficit hyperactivity disorder: Placebo-controlled comparison to D-amphetamine. *Biological Psychiatry*, 25, 222-228.

Arnold, L.E., Kleykamp, D., Votolato, N., Gibson, R.A., & Horrocks, L. (1994). Potential link between dietary intake of fatty acids and behavior: Pilot exploration of serum lipids in ADHD. *Journal of Child & Adolescent Psychopharmacology*, 4, 171-180, 1994.

Bateman, B., Warner, J.O., Hutchison, E., Dean, T., Rowlandson, P., Gant, C., et al.(2004). The effects of a double blind, placebo controlled, artificial food colorings and benzoate preservative challenge on hyperactivity in a general population sample of preschool children. *Archives of Disease in Childhood*, 89, 506-511.

Boris, M., & Mandel, F.S. (1994). Food and additives are common causes of the attention-deficit hyperactivity disorder in children. *Annals of Allergy*, 72, 462-468.

Burgess, J.R., Stevens, L, Zhang, W., & Peck, L. (2000). Long-chain polyunsaturated fatty acids in children with attention-deficit hyperactivity disorder. *American Journal of Clinical Nutrition*,

71(Suppl. 1), 327S-330S.

Cade, R., Privette, R.M., Fregly, M., Rowland, N., Sun, Z., Zele, V., et al. (1999). Autism and schizophrenia: Intestinal disorders. *Nutritional Neuroscience*, 2, 57-72.

Carter, C.M., Urbanowicz, M., Hemsley, R., Mantila, L., Strobel, S., Graham, P.J., et al. (1993). Effects of few food diet in attention deficit disorder. *Archives of Disease in Childhood*, 69, 564-568.

Chan, E., Rappaport, L.A., & Kemper, K. (2005). Complementary and alternative medicine in the treatment of attention deficit hyperactivity disorder. *Journal of Pediatrics and Child Health*, 41, 23-26.

Colquhoun, I., & Bunday, S. (1981). A lack of essential fatty acids as a possible cause of hyperactivity in children. *Medical Hypothesis*, 7, 673-679.

Connors, C.K., Goyette, C.H., Southwick, D.S., Lees, J. M., & Androlonis, P.A. (1976). Food additives and hyperkinesia: A controlled-double blind experiment. *Pediatrics*, 58, 154-166.

Crook, W.G. (1980). Can what a child eats make him dull, stupid, or hyperactive? *Journal of Learning Disabilities*, 13, 53-58.

Dengate, S., & Ruben, A. (2002). Controlled trial of cumulative behavioral effects of a common bread preservative. *Journal of Pediatric Child Health*, 38, 373-376.

Deutsch, R.M. (1977). *The new nuts among the berries*. Palo Alto, CA: Bull Publishing.

Egger, J., Carter, C.M., Graham, P.J., Gumley, D., & Soothill, J.F. (1985). Controlled trial of oligoantigenic treatment in the hyperkinetic syndrome. *Lancet*, 325, 540-545.

Egger, J., Stolla, A., & McEwen, L.M. (1992). Controlled trial of hyposensitization in children with food-induced hyperkinetic syndrome. *Lancet*, 339, 1150-1153.

Elder, J.H., Shankar, M, Shuster, J., Theriaque, D., Burns, S. & Sherrill, L. (2006). The Gluten-Free, Casein-Free Diet In Autism: Results Of A Preliminary Double Blind Clinical Trial. *Journal of Autism and Related Disorders*, 36, 413-420.

Feingold, B.F. (1975). *Why is your child hyperactive?* New York: Random House.

Hagman B., (1990). *The gluten-free gourmet: Living well without wheat*. New York: Henry Holt & Co.

Harley, J.P., Ray, R.S., Tomasi, L., Eichman, P. L., Matthews, C., Chun, R., et al. (1978). Hyperkinesia and food additives: Testing the Feingold hypothesis. *Pediatrics*, 61, 818-828.

Horricks, L.A., & Farooqui, A.A. (2004). Docosahexaenoic acid in the diet: Its importance in maintenance and restoration of neural membrane function. *Prostaglandins, Leukotrienes and Essential Fatty Acids*, 70, 361-372.

Hoover, D.W., & Milich, R. (1994). Effects of sugar ingestion expectancies on mother-child interactions. *Journal of Abnormal Child Psychology*, 22, 501-515.

Kane, P. (1999, March). *Metabolic assessment and nutritional biochemistry. Clinical and research aspects*. Symposium conducted at the Great Lakes College of Clinical Medicine, XXXI International Congress, Asheville, NC.

Kaplan, B.J., McNichol, J., Conte, R.A., & Moghadam, H.K. (1989). Dietary replacement in preschool-aged hyperactive boys. *Pediatrics*, 83, 7-17.

Kavale, K.A., & Forness, S.R. (1983). Hyperactivity and diet treatment: A meta-analysis of the Feingold hypothesis. *Journal of Learning Disabilities*, 16, 324-330.

Kidder, B. (1991). *Milk-free kitchen: Living well without dairy products*. New York: Owl Books.

Knivsberg, A.M., Reichelt, K.L., Høien, T., & Nodland M. (2002). A randomized, controlled study of dietary intervention in autistic syndromes. *Nutritional Neuroscience*, 5, 251-261.

Lipton, M.A., & Mayo, J.P. (1983). Diet and hyperkinesia – An update. *Journal of the American Dietetic Association*, 83, 132-134.

Marshall, P. (1989). Attention deficit disorder and allergy: A neurochemical model of the relation between the illnesses. *Psychological Bulletin*, 19, 434-446.

Mattes, J.A. (1983). The Feingold diet: A current reappraisal. *Journal of Learning Disabilities*, 16, 319-323.

Prinz, R.J., & Riddle, D.B. (1986). Associations between nutrition and behavior in 5-year-old children. *Nutrition Reviews*, 44, 151-157.

Prinz, R.J., Roberts, W.A., & Hantman, E. (1980). Dietary correlates

of hyperactive behavior in children. *Journal of Consulting and Clinical Psychology*, 48, 760-769.

Randolph, T.G. (1947). Allergy as a causative factor in fatigue, irritability, and behavior problems in children. *Journal of Pediatrics*, 31, 560-72.

Rapp, D.J. (1978). Does diet affect hyperactivity? *Journal of Learning Disabilities*, 11, 56-62.

Rapp, D.J. (1979). Food allergy treatment for hyperkinesis. *Journal of Learning Disabilities*, 12, 42-50.

Rapp, D.J. (1991). *Is this your child? Discovering and treating unrecognized allergies in children and adults*. New York: William Morrow.

Richardson, A.J. (2004). Clinical trials of fatty acid treatment in ADHD, dyslexia, dyspraxia, and the autistic spectrum. *Prostaglandins, Leukotrienes and Essential Fatty Acids*, 70, 383-90.

Richardson, A.J. & Puri, B.K. (2002). A randomized double-blind, placebo-controlled study of the effects of supplementation with highly saturated fatty acids on ADHD-related symptoms in children with specific learning disabilities. *Progress in Neuropharmacology and Biological Psychiatry*, 26, 233-239.

Rojas, N.L., & Chan, E. (2005). Old and new controversies in the alternative treatment of attention-deficit hyperactivity disorder. *Mental Retardation and Developmental Disabilities Research Reviews*, 11, 116-130.

Rowe, K.S., & Rowe, K.J. (1994). Synthetic food coloring and behavior: A dose response effect in a double-blind, placebo-controlled, repeated-measures study. *Journal of Pediatrics*, 125 (5 Pt 1), 691-698.

Schab, D.W. & Trinh, N.T. (2004). Do artificial food colors promote hyperactivity in children with hyperactive syndromes? A meta-analysis of double-blind placebo-controlled trials. *Developmental and Behavioral Pediatrics*, 25(6), 423-434.

Schmidt, M., Mocks, P., Lay, B., Eisert, H., Fojkar, R., Fritz-Sigmund, D., et al. (1997). Does oligoantigenic diet influence hyperactive/conduct-disordered children - A controlled trial. *European Child and Adolescent Psychiatry*, 6, 88-95.

Schnoll, R., Burshteyn, D., & Cea-Aravena, J. (2003). Nutrition in the treatment of attention-deficit hyperactivity disorder: A neglected but important aspect. *Applied Psychobiology and Biofeedback*, 28(1), 63-72.

Shannon, W.R. (1922). Neuropathologic manifestations in infants and children as a result of anaphylactic reaction to foods contained in their dietary. *American Journal of Disabled Children*, 24, 89-94.

Silver, L.B. (1986). The "magic cure": A review of the current controversial approaches for treating learning disabilities. *American Journal of Disease*, 140, 1045-1052.

Stevens, L.J., Zentall, S.S., Abate, M.L., Kuczek, T., & Burgess, J.R. (1996). Omega-3 fatty acids in boys with behavior, learning, and health problems. *Physiology and Behavior*, 59, 915-920.

Stevens, L., Zhang, W., Peck, L., Kuczek, T., Grevstad, N., Mahon, A., et al. (2003). EFA supplementation in children with inattention, hyperactivity, and other disruptive behaviours. *Lipids*, 38, 1007-21.

Voigt, R.G., Llorent, A.M., Jensen, C.L., Fraley, J.K., Berretta, M.C., & Heird, W.C. (2001). A randomized, double-blind, placebo-controlled trial of docosahexaenoic acid supplementation in children with attention-deficit/hyperactivity disorder. *Journal of Pediatrics*, 139, 189-196.

Wainwright, P.E. (2002). Dietary essential fatty acids and brain function: A developmental perspective on mechanisms. *Proceedings of the Nutritional Society of Australia*, 61, 61-69.

Wender, E.H. (1986). The food additive-free diet in the treatment of behavior disorders: A review. *Journal of Developmental Pediatrics and Behavioral Pediatrics*, 7, 35-42.

White, J.W., & Wolraich, M. (1995). Effect of sugar on behavior and performance. *Journal of Clinical Nutrition*, 62, 242S-247S.

Williams, J.I., & Cram, D.M. (1978). Diet in the management of hyperkinesis: A review of the tests of the Feingold hypotheses. *Canadian Psychiatric Association Journal*, 23, 241-248.

Wolraich, M.L. (1996). Diet and behavior: What the research shows. *Contemporary Pediatrics*, 13, 29-39.


Wolraich, M. L., Stumbo, P., Milich, R., Chenard, C., & Shultz, F. (1986). Dietary characteristics of hyperactive and control boys. *Journal of the American Dietetic Association*, 86, 500-504.

Wolraich, M.L., Wilson, D.B., & White, J.W. (1995). The effect of sugar on behavior or cognition in children: A meta-analysis. *Journal of the American Medical Association*, 274, 1617-1621.


Yehudi, S., Rabinovitz, S., & Mostofsky, D.I. (1998). Essential fatty acids and sleep: Mini-review and hypothesis. *Medical Hypotheses*, 50, 139-145.

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