Role of Nutrition in Treatment of Attention Deficit Hyperactivity Disorder

The Role of Nutrition in the Treatment of Attention Deficit Hyperactivity Disorder (ADHD)

Attention Deficit Hyperactivity Disorder (ADHD) is a neurological syndrome which is characterized by distractibility, impulsivity and restlessness. Theories vary as to whether it has a hereditary basis, if it is a psychological disorder caused by parenting styles or environmental factors, or whether it is caused by societal factors such as teaching styles in schools and the impact of television. The role of nutrition in ADHD has been examined and debated. Significant findings show a link between sugar consumption, food additives, food allergies, vitamin and mineral status and heavy metal toxicity and hyperactive/attention deficit behaviors. These findings have been largely discredited by the orthodox medical profession, who predominantly support the view that the cause is a biochemical imbalance in the brain which requires medication to rectify. The result of this is a large number of people destined for a life of medication, who claim to ‘feel’ so much better on the medication, but who are not really treating the underlying mechanisms, nor learning new skills to adapt to their disorder.

In my opinion, the role of nutritional factors has been largely overlooked by the both the medical profession and the general public. While many advocates of nutritional therapy have conducted large numbers of studies supporting their claims, the counter studies and criticisms by the medical profession have led to the discrediting of these theories in many books that have been written about ADHD. These are being widely read by laypersons, who are often the sufferers of the disorder or the parents of sufferers. Nutritional therapy is still being seen as an ‘alternative’ approach, despite the increasing evidence of nutritional factors in the etiology of many diseases and disorders.

Diagnosis of ADHD is largely made by studying the history of the patient – there is no definitive ‘test’ for ADHD. The Diagnostic and Statistical Manual, Fourth Edition (DSM-IV), published by the American Psychiatric Association, has published a behavioral diagnostic criteria. Other testing for ADHD may include blood tests to rule out thyroid disorders and electroencephalograms for other mental disorders. Psychological tests include the Rorschach inkblot test, Weschler intelligence tests and the TOVA (Test of Variability of Attention).

The etiology of ADHD is unclear, however theories abound. Neurological explanations claim that ADHD is due to some dysfunction in the brain. Indirect drug response research has been used to conclude that ADHD may be due to insufficient quantities of the neurotransmitters dopamine and norepinephrine, known as catecholamines. Biochemical studies and neurological testing of neurotransmitter metabolites in the urine have not, however, been able to document the specific role of catecholamines in ADHD.
Other researchers claim that there is reduced blood flow in the frontal lobe area of the brain of ADHD patients, causing lowered activity in this area (1). The frontal lobe is responsible for functions such as planning, initiative and ability to regulate behavior, and so reduced functioning may cause disruptions to these behaviors similar to ADHD characteristics. This was tested by Dr Alan Zametkin at the National Institute of Mental Health, who used positron emission tomography or PET scans to record the radioactivity of the brain following glucose ingestion. The deficit in glucose uptake and hence energy to the brain following a specific task in ADD subjects (8% lower than the control group) indicates a link between diminished frontal lobe activity and ADHD. ADHD behavior is described as responding to this situation by seeking out higher levels of stimulation to sufficiently engage and satisfy the brain and sensory centers.

Some theorists argue for the role of the Reticular Activating System of the brain stem in ADHD. The RAS regulates the level of arousal of a person, from waking consciousness to deep sleep. Impairment in this system may cause the ADHD problems with alertness. Larry Silver likens the problem to a 'faulty filter system', which cannot screen out irrelevant information or sensory stimuli as well as it should, leading to an overload of messages. Other experts believe that a malfunction in the communication feedback loop, which links the reticular activating system and frontal lobe, may be the cause of the disordered, inconsistent and erratic behavior typical of ADHD (2).

While a definite correlation between brain dysfunction and ADHD has not been confirmed, evidence suggests that frontal lobe dysfunction or catecholamine response may be factors in the etiology.

The heredity of ADHD has also been examined, and studies show that there may be an element of genetics in the disorder. Joseph Biederman's studies showed that approximately 30% of all parents with ADHD children have ADHD themselves. One large study showed that 51% sets of identical twins both had ADHD, while only 33% of fraternal twins both had it. In 1991, a controversial study published in the Journal of the American Medical Association considered the role played by a particular dopamine receptor, called the D2 receptor, which is made by a particular gene that appears more commonly in patients with ADHD.

Behavioral theories have also been put forward. Parenting styles, family environments, the 'Theory of Blame' and dysfunctional families, put forward by child rearing experts, all point to family dynamics and their role in the syndrome.

There is also a broad range of societal theories implicated with ADHD, including the 'psychological hazards' of our increasingly complex society, and the risks of people becoming overwhelmed by the pace of Western culture. School systems and learning techniques; effects of television and advertising; computers and electronic games; lack of exercise, reading and sunlight; and the profit motives of pharmaceutical companies have all arisen. While all these factors may certainly contribute to ADHD behavior in our society, evidence is lacking as to their level of significance in clinical etiology.

The role of nutrition in the etiology of ADHD is controversial in orthodox medical circles. Various nutritional theories have been proposed, and will now be examined individually.

Refined sugars, especially sucrose, and aspartame, have been linked with ADHD and other behavioral problems in children. The presumed reaction to sucrose has been attributed to a variety of causes, including a rise in blood sugar shortly after ingestion, reactive hypoglycemia several hours after ingestion, and allergic response. Hypoglycemia eventually results in hyperactivity as adrenaline and other stimulants are released by the adrenal glands in response to the low blood sugar level. The presumed reaction to aspartame has been attributed to the possibility that its metabolism results in elevated plasma phenylalanine concentrations, which in turn may alter the transport of essential amino acids to the brain.
Several studies have been done to point to sugar as a behavioral factor. The Langsdeth and Dowd findings (1978) were based on a five hour oral glucose tolerance test on 261 hyperactive children, in which 74% showed abnormal glucose tolerance curves.

Girardi et al (1995) reported on their studies a blunted catecholamine response after glucose ingestion in children with ADHD. 17 ADHD children and 11 control children were challenged with a dose of glucose. Three and five hours after ingestion, epinephrine and norepinephrine levels were tested. It was found that epinephrine levels of the ADHD children rose at a “significantly greater” rate in the ADHD children than the control group, while the norepinephrine levels fell in the ADHD children, but not the control group. Thus this test gives greater credibility to the sugar hypothesis, as it explains the mechanisms behind the phenomena.

DA Gans criticizes such studies, claiming that many theories have been based on anecdotal reports, misinterpretations of scientific literature, or flawed interpretation of questionable data. Gans goes further to claim that simple carbohydrates may even elicit calming responses in subjects showing signs of antisocial/ hyperactive behavior.

Hoover and Milich (1994) associate the behavioral differences following sugar ingestion on the mother's expectancy of behavior and subsequent methods of interaction, as opposed to the metabolic effects of the sugar itself. A study was conducted and showed that mothers rated their children as significantly more hyperactive when they believed the child had consumed sugar (all the children had, in fact, taken a placebo), and as such responded by differing behavior, such as exercising more control by maintaining physical closeness, criticizing, looking at, and talking to their sons more than control mothers.

Other studies by Kanarek (1994), Wolraich et al. (1994) and Behar et al. (1984) deny any association between sugar and behavioral tendencies when given a short term abstinence and challenge situation. However such short term tests do not take into account the longer term affects of high sucrose diets (sucrose ranging from 25-60% of total caloric intake) on total mineral, vitamin and amino acid status, which can affect brain function. This may explain why most controlled, low-dose, short duration, double blind trials fail to find a relation between sucrose and behavior, despite widespread public belief to the contrary.

Clifton Kurukawa MD (1994) also writes in support of the overriding evidence of the sugar link despite scientific studies. He claims that parents of children who participated in a double blind challenge test which showed no positive link between sugar and behavior, were followed up several months later. Most parents continued to believe that sugar caused aggressive, loud, non compliant, or overly active behavior in their children, and continued to restrict sugar in the diet where possible.

Despite the lack of consistent objective data regarding the link between ADHD and sugar ingestion, subjective reports of such a link continue to be widespread. Di Battista and Shepherd (1993) report that in a questionnaire sent out to Canadian primary school teachers, over 80% of the 389 responses pointed to a belief that sugar consumption contributes to overactivity in normal children and behavioral problems in hyperactive children. 55% of these teachers had already counseled parents in their views regarding reducing the child's sugar consumption, with parents frequently doing so.

The persistence of such reports on sugar consumption and its behavioral implications is significant enough to warrant a trial period of low simple sugars in the diet of an ADHD patient. In this case a lack of scientific evidence is not valid enough a reason to discount the theory, which has been repeatedly hypothesized.

Another widely speculated and highly controversial view is the role of food additives in ADHD. ‘Food additives’ covers a wide range of chemicals – 5000 are used in the USA, such as anti-caking agents (e.g. calcium silicate), antioxidants (e.g. BHT, BHA), bleaching agents (e.g.
Much of the research supporting the link between food additives and ADHD was done by allergist Benjamin Feingold, hence the term 'Feingold Hypothesis'. Feingold claims that 40-50% of hyperactive children are sensitive to artificial food colors, flavors and preservatives and to naturally occurring salicylates and phenolic compounds. This is based on his experience with over 1,200 cases in which food additives were linked to learning and behavior disorders. Feingold presented his theory to the American Medical Association in 1973, opening the door to years of widely debated support and criticism of his work.

Feingold does not believe that the mechanism behind his food additive theory is immunological, but instead is a toxic effect. Children who are affected by food additives may differ biochemically from those who are unaffected (Brenner 1979). Therefore, children who are susceptible to such reactions may be genetically predisposed to it. This is backed up by reports that low concentrations of a food dye, called Red No 3 or erythrosin B, which is widely used in candy, powdered desserts and beverages, partially prevents the brain from taking in dopamine, which then has profound effects on motor activity.

The first major test done to test the Feingold diet (or Kaiser-Permanente (K-P) diet) was undertaken by psychologists Dr Connors and J Harley in 1976. They each carried out a double blind study, placing a group of hyperactive patients on one month trials of a control diet, and a Feingold diet. Behavior was studied before the test was carried out, and then during and after the two month testing period. In Dr Connors study both parents and teachers reported a reduction in activity with the Feingold diet, but not on the control diet. Parents were less likely to be able to differentiate. Interestingly the group who showed the most marked improvement received the Feingold diet after the placebo diet. This may indicate that when the Feingold diet was given first, its benefits extended into the placebo diet month. The end result of this study, however, is that the results were inconclusive.

There have been many advocates of Feingold's treatment approach. Australian Drs Cook and Woodhill found that 66% of 15 children tested responded favorably to the diet, and relapsed when it was discontinued. Dr Salzman found that 58% of 31 hyperactive patients reacted positively when tested with sublingual food dye, and that 93% showed significant improvement in behavior within four weeks when placed on the Feingold diet.

Rowe and Rowe (1994) reported on their study of synthetic food coloring and behavior of 'hyperactive' children. 200 children were put on a five week open trial of a diet free of synthetic food coloring. Parents of 150 children reported behavioral improvement with the diet, and deterioration on the reintroduction of such foods.

Another study by Boris and Mandel (1994), conducted in New York, USA, showed that 73% of 26 ADHD children reacted favorably to treatment with a multiple food elimination diet (restricting food dyes, preservatives and artificial flavorings), and that on open challenge, all 19 subjects reacted to the reintroduction of those substances.

Unfortunately, the medical profession has largely discredited Feingold's findings. Jeffrey A Mattes, MD, in the New England Journal of Medicine worked through several tests that showed supportive results for the Feingold diet, and systematically discredited them. Curiously, it appears that most of the tests conducted in the US show negative results, the majority of these being carried out by the Nutrition Foundation, an organization supported by the major food manufacturers. Tests in Australia and Canada show more positive results.

Bernard Rimland PhD responds to such criticisms, arguing that, firstly, most of the studies performed on food additives in response to Feingold's evaluations were conducted on approximately ten dyes, despite Feingold's assessment of 3000 additives in our foods. Rimland...
points out that the dosages used in testing were too small to produce significant results when challenged. 1.6-26mg of additives were given per day, with no significant results. However it is commonly believed that the average daily intake of additives is 76mg. Studies conducted using colorings at the 90th percentile bracket, however, show conclusive and clearly supportive results.

Rimland also highlights the failure to recognize the role of the subject's prior nutritional status, and claims that the differences in baseline health between the Feingold subjects and the normally poorly nourished hyperactive children may have led to confusing and inconclusive results. He describes other aberrant factors that were not taken into consideration, such as the copper levels of subjects, which were not tested, but may have led to differences in the way various factors were metabolized. Rimland suggests that anti-Feingold bias may have played a role in the negative conclusions, such as the vested interests of the food manufacturers. Finally, Rimland argues that insignificant attention was paid to animal and in vitro studies which supported Feingold's theory.

While not every patient will find a complete reversal of symptoms on the Feingold diet, there is certainly enough evidence to implicate food additives such as colorings and preservatives in the cognitive and behavioral functions of some patients. The Feingold diet is nutritionally sound and follows desirable nutritional principles such as eating more natural foods, and less refined, processed foods with synthetic additives. For many ADHD patients, it provides an alternative to drug therapy, and is certainly more beneficial for the long term health of the patient. This treatment also addresses the cause of the problem, instead of merely controlling the symptoms as do many stimulant medications and other treatment protocols. The Feingold hypothesis is therefore, a credible theory, and one that deserves serious consideration in the clinical setting.

Allergies and their role in ADHD and hyperactivity have also received much speculation. While this argument crosses over the barriers of the food additive theory, the major differentiation is that Feingold hypothesized that the mechanisms behind the affects of food additives were toxic, not immunological. Food additives may be allergens, but they are not necessarily so.

Allergy is the term used to denote an individual's increased reactivity to a specific substance as a result of prior exposure of the individual to that substance or to a chemically related substance (3). Food allergies affect 2-3% of the adult population and 10-15% of all children, with another 30-40% showing minor manifestations.

Results linking food allergies and hyperactivity in double blind studies appear to be far more conclusive and consistent. One large controlled study (Eggar et al, 1975) treated 76 subjects with an oligoantigenic diet (lamb, chicken, potatoes, rice, bananas, apple, brassica family vegetable, calcium gluconate 3g/ day and a multivitamin). After a four week trial, 82% improved and a normal range of behavior was achieved in 21 of these. Other symptoms, such as headaches, abdominal pains and fits also improved. Reintroduction of foods to which the child was sensitive led to reappearance of symptoms and hyperactive behavior.

Egger, Stolla and McEwen (1992) agree with the link between allergies and hyperactivity, however, they state that restricted diets are expensive, socially disruptive and often nutritionally inadequate. They propose the use of enzyme-potentiated desensitization (EPD) as an alternative treatment protocol. In a test conducted in Munich, Germany, 116 out of 185 subjects whose behavior responded had provoking foods identified by reintroduction. Of these 40 patients participated in hypersensitization trials. Half the group received three doses of EPD (beta-glucuronidase and small quantities of food antigens) intradermally at two month intervals, the other half received placebo. Of the 20 treated patients, 16 became tolerant towards provoking foods compared with 4 of the patients who received placebo. This test then shows that EPD allows children to eat foods that previously produced symptoms of food induced hyperkinetic syndrome.
According to tests conducted by Trites, Tryphonas and Ferguson (1980), 47% of all hyperactive children tested showed a food allergy (according to RAST testing of 43 food antigens). Of hyperactive, emotionally inattentive and learning disabled children, the average rate of allergies was highest in the hyperactive group. It does show, however, that the highest rate of hyperactivity and allergy association occurred when learning disordered or emotionally-inattentive behaviors were also present.

McGee, Stanton and Sears (1993) argue that the relationship between ADHD and allergic disorders is questionable, and that in a large number of subjects sampled (including parent, teacher, and self-reports of ADHD behaviors) very little correlation was found between the ADHD and a history of allergic disorders (asthma, eczema, rhinitis and urticaria) in the 9-13 age group. This report further argues that reports of ADHD behavior at 13 years were not related to atopic responsiveness by skin test or serum IgE levels.

Once again, the role of allergies in ADHD has received mixed reports. There are tests in support of and tests arguing against any association. Clearly a history of atopic symptoms in a child displaying ADHD behaviors suggests a high probability that there is a link between the two. However, clinical studies carried out on non-atopic sufferers highlight food allergies as a significant factor in the etiology of ADHD, and so food allergies should be thoroughly investigated in all ADHD cases.

Although sugar ingestion, food additives and chemicals, and food allergies are the three main nutritional factors argued to be causative factors of ADHD, there are other nutritional aspects that may come into play in its etiology, and these should be examined.

Mineral toxicities and deficiencies are seen to play a significant role in ADHD behaviors. Hair mineral analysis has been receiving increased attention as a research tool and clinical diagnostic technique, as it can measure both levels of essential minerals, and the body's burden of toxic heavy metals.

An analysis by Rimland and Larson of 51 studies on the effects of minerals and heavy metals on various learning disorders and behavioral syndromes showed high levels of lead, cadmium, mercury and copper in hyperactive disorders.

Tuthill (1996) undertook a study of 277 first grade pupils with ADHD, with hair lead levels ranging from 1 to 11.3 mcg/g. Results showed a striking dose response between levels of lead and negative teacher ratings, and an even stronger relationship between hair lead levels and physician-diagnosed ADHD in the same children.

David, Clark and Voeller (1972) tested hyperactive children against non-hyperactive children for blood-lead levels and urine levels after challenge by a single oral dose of the chelating agent penacilamine. Hyperactive children were seen to have significantly higher values on both measures than did the controls. More than half the hyperactive children had blood-lead levels in the range considered to be raised but not 'toxic', and 60% of post-penacilamine urine levels were in the toxic range. This test concluded that high body lead levels may exert previously unrealized consequences on children's behavior and hyperactivity levels.

Since lead exposure occurs mainly from inhalation of petrol fumes, burning of coal, leaded paints, drinking water delivered through lead pipes and most canned foods, these environmental and nutritional factors must be closely examined.

Iron deficiency is a common nutrient deficiency in Western societies, and in fact is the most prevalent nutrient disorder in the US. It is associated with markedly decreased attentiveness, less complex or purposeful, narrower attention span, decreased persistence and decreased voluntary activity, all key symptoms of ADHD. Several investigators have demonstrated that correction of even subtle
nutritional variables may exert a substantial influence on learning and behavior, without necessarily presenting a diagnosis of anemia. Iron is important in biochemical pathways involving electron transport, catecholamine catabolism, and porphyrin synthesis. Given the link between ADHD and catecholamines, this may be a causative factor. This finding has relevant application to ADHD patients, and iron levels should be tested and corrected immediately.

Zinc deficiencies have also been linked with hyperactive behavior and ADHD symptoms. Zinc deficiency suppresses melatonin secretion, which in turn affects serotonin production, thus causing the symptoms of ADHD. It has also been suggested that maternal zinc deficiency in pregnancy may contribute to the development of hyperactivity syndrome, and that the risk of this is heightened if pre-eclampsia is present also.

Several other minerals have been implicated in behavioral disorders and hyperactivity. Marlowe (1986) found that elevated hair selenium correlated significantly with increased scores on the Walker Problem Behavior Identification Checklist (WPBIC). Studies show that children suffering from learning disorders or hyperactivity are more likely to have elevated serum aluminum levels. Three separate studies show an association between high manganese levels and behavioral and learning disorders in children. This may be due to higher manganese levels causing decreased dopamine turnover leading to increased motor activity. Mercury toxicity, occurring largely from eating contaminated fish, or from leaching from amalgam dental fillings, has also been implicated.

There has been a link made between symptoms of ADHD and thyroid dysfunction. Generalized resistance to thyroid hormone (GRTH) is a genetic predisposition that has been linked to a defective human thyroid-receptor B gene on chromosome 3. In tests conducted of 22 adults with GRTH, 50% met the criteria for ADHD; of 27 children tested, 70% met the ADHD criteria. This led to the conclusion by the authors that ADHD is strongly associated with GRTH.

Elia et al. undertook a similar study which led them to the conclusion hyperthyroidism may be part of the ADHD etiology, however this is not necessarily a genetic function. Hyperthyroidism can produce impairment in attention, cognition and performance, as can thyroid hormone replacement following hypothyroidism. The possibility that ADHD may be secondary to hyperthyroidism warrants investigation in the clinical setting.

It has also been suggested that hypothyroidism may be behind an ADHD diagnosis. This has partly been attributed to a diet low in iodine, resulting from iodine depleted soil, and modern farming methods. Synthetic fertilizers replace only a fraction of the nutrients depleted from the soil, therefore the foods found in supermarkets today may be dramatically lower in iodine than those produced before the advent of chemical fertilizers. Hence iodine supplementation, or a diet rich in iodine containing foods such as kelp and seafood, may help a number of ADHD sufferers.

Research has also been carried out into the role of essential fatty acids in ADHD. Stevens et al (1995) reported that in a study of 96 subjects, the 53 subjects with ADHD had significantly lower concentrations of key fatty acids in the plasma polar lipids and in red blood cell total lipids than did the 43 control subjects. While this study appears to show a definite correlation, the mechanisms behind the link are not clear.

Vitamins and mega-vitamin therapy have been studied in relation to ADHD, both in terms of its etiology and treatment. While it is very difficult to pinpoint the role of vitamin deficiencies and their isolated role in ADHD, many ADHD patients benefit from a vitamin and mineral regime that includes the following amounts daily (4):

<table>
<thead>
<tr>
<th>Under 35 lbs</th>
<th>Over 45 lbs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin B1 (Thiamin) 50-100mg 100-1000mg</td>
<td>Vitamin C 500-1000mg 1000-3000mg</td>
</tr>
</tbody>
</table>
Vitamin B3 (Niacin, Nicotinamide or Nicotinic Acid) 500-1000mg 1000-3000mg
Vitamin B6 (Pyridoxine) 100-200mg 200-400mg
Calcium Pantothenate 200-300mg 400-600mg
Vitamin E 50-100mg 1500mg
Manganese 3-5mg (this often enhances response to B6 and Zn) Zinc 10-15mg
Magnesium 100-350mg Calcium 250-750mg
Lecithin granules 8-15mg

While vitamin and mineral supplementation may help improve mental functioning and behavior, it is unclear whether mega-vitamin therapy may produce more profound results. Several experts advise against its use on the basis of potential toxicity.

Mega-vitamin therapy is based upon the dual belief that many learning and behavioral disorders are based on biochemical imbalances in the brain, and that such disturbances can be ameliorated by consuming large amounts of vitamins, ten times or more the recommended daily allowance.

Use of megavitamin therapy in hyperactivity was first suggested by Cott in 1969, following his success with such treatment in schizophrenic patients. In 1971 the concept was introduced that children with learning disabilities improved with megadoses of vitamins. Cott claimed that between 1969 and 1971 he treated 500 children successfully with this regime.

Robert Haslam, a Canadian neurologist, states “During the past decade, with the increased emphasis on holistic medicine and a growing reluctance to accept traditional and scientifically proven methods of medical care, there has been a movement to unorthodox methods of treatment. Megavitamin therapy is no exception!” It is attitudes like this on behalf of the medical profession that keep nutritional therapies such as megavitamin therapy on the “fringe” of contemporary medicine. Unfortunately, it is these beliefs that are translated to patients by their doctors.

Haslam then goes on to claim that there is no scientific efficacy for the use of megavitamins in disorders which are not caused by gross vitamin deficiencies (he concedes the use of the therapy for vitamin-related diseases such as rickets and pyridoxine-dependent seizures). He conducted a study of 41 children with ADHD, using large doses of ascorbic acid (3g), niacinamide (3g), calcium pantothenate (1.2g), and pyridoxine (0.6g). These doses were given for 3 months (Stage 1). Stage 2 consisted of four, six week, double blind, repeated crossover periods. The 29% of subjects who showed improvement in behavior in stage 1 were used for the cross over phase of the study, however, in this period no significant differences between subjects using vitamins and those on placebo. There was no significant serum pyridoxine and ascorbic acid levels between subjects and control subjects. 42% of subjects were reported as reaching upper limits of serum transaminase levels while receiving the vitamin therapy. Some patients also experienced nausea, vomiting and abdominal cramping during the study. It was therefore concluded that megavitamin therapy was ineffective in bringing about any significant lasting change, and that due to its potential toxicity and risk of hepatic injury, it should not be used in the treatment of ADHD.

Dr Rimland counters Haslams findings in a letter to the editor in Pediatrics (1986), stating that Haslam used an incomplete version of megavitamin therapy, and that “practitioners of megavitamin therapy have known for a decade that the entire B complex and magnesium must be used when megavitamin amounts of any of the B vitamins are used.”

Eugene Dembicki points out in the Journal of Psychiatric Nursing that part of the skepticism towards megavitamin treatments may be due to the ‘non-prescription’ status of the treatment. This may reflect pressure on the orthodox medical profession from pharmaceutical companies,
who would prefer to see a stimulant medication or anti-depressant prescribed, and partly because of concern over 'shotgun' approaches to treatment, where self-medication may lead to lack of results, or in fact, adverse side effects.

Carter (1973), however, did find that megavitamins reduced hyperactivity, leading to improved concentration and increased attention span.

Other studies conducted by Rimland, Callaway and Dreyfus (1978) also show support for the use of megavitamin therapy. Rimland et al. treated 200 children with autistic symptoms with large doses of Vitamins C, B6, niacinamide and pantothenic acid. Rimland found Vitamin B6 to have the most profound effect on behavior, and conducted a double blind study to confirm this. While autism will have differing clinical etiology and features to ADHD, there are several features of this test that make it relevant to this discussion. Firstly, the findings showed B6 to elevate serotonin levels, through its role in converting dietary L-tryptophan to serotonin, leading to the conclusion that B6 may correct, or partially correct, tryptophan related metabolic disorders. Secondly, there was no evidence from the testing that massive doses of B6 have been or can be harmful. Wilcken and Turner reported a decrease in level of blood folate in children receiving massive doses of B6 for an extended period of time but found no anemia, morphological changes in blood cells, or other problems. They recommended a small supplement of folate with the B6 to offset this. Rimland (1974) noted that some children experienced increased irritability, sound sensitivity and enuresis when megadoses of B6 were given, but these problems disappeared when magnesium was added to the children's dietary intake.

The role of megavitamins in the treatment protocol for ADHD remains unclear. While several orthodox practitioners claim that megavitamin therapy may produce toxicity, those same practitioners most readily choose lifelong drug therapy as the alternative. It appears that the rift between orthodox and alternative practitioners on this subject may obscure the true findings. Scientific evidence produced by the orthodox medical profession should not be accepted as the definitive answer. Such findings may mask a financial or political bias, or a lack of understanding as to the correct treatment protocol. Further research needs to be undertaken as to the efficacy of a mega-vitamin regime, however, for many patients, it is an option worth consideration, particularly as an alternative to stimulant medication and other drug therapy.

While it is difficult to draw conclusive findings from the barrage of scientific testing and counter testing done on hyperactive children, it is clear that nutrition plays a large role in ADHD. Food additives, sucrose, food allergens, heavy metals and vitamin and mineral deficiencies may all play a role. Unfortunately the role of each one specifically may be hard to determine in some situations, due to the interrelation of all factors.

In my opinion the most prudent approach to nutrition in ADHD is a multifactorial approach. Studies such as those carried out by Colgan and Colgan (1984), show that a diet containing sufficient levels of vitamins and minerals, being low in refined sugars, focusing on natural, unprocessed and chemical free foods where possible, without heavy metal contamination, produces positive changes in hyperactive children. These dietary guidelines represent the basic recommendations that naturopathic doctors support in all situations. Some patients may, however, need more intensive nutritional control, and while this may produce greater effort in food management, it is still a preferable alternative to a life of drug therapy. If it helps the child's behavior and attention, there is likely to be reasonably high motivation to continue with the dietary regime. Specific treatment protocols may include avoidance of known or suspected allergens following food elimination and challenge tests; following the Feingold diet; professionally administered megavitamin therapy; exclusion of all sugar and aspartame from the diet; and detoxification therapy to eliminate heavy metals from the body. The particular treatment must be chosen
according to the history and clinical status of the individual.

Non-nutritional treatment protocols may also be included, such as psychotherapy, homeopathy, traditional Chinese medicine, meditation and mind control techniques, biofeedback and support in learning life skills to help people become more functional with ADD. These have been very useful to many sufferers.

A nutritional approach to treatment of ADHD, coupled with counseling and support in learning how to adapt to the disorder, is a safe, long term and effective treatment protocol. This approach avoids long term drug therapy, and will add to the general health of the patient by providing a well balanced, natural diet. It is my conclusion that nutrition as part of a natural therapies regime can produce a profound change in the quality of life of ADHD sufferers, and that natural practitioners must work towards educating the public to this effect. Already in America, the use of drugs such as Ritalin and Prozac in the treatment of ADHD is widespread. This does not serve to treat the cause of the problem, but it is a reflection of the society that people would prefer to take a pill than really work towards a long term solution. At the same time, consumption of high-sugar, high fat, heavily processed foods are being eaten at the highest rate in the world; produce is being grown in soil that is nutrient poor, and then treated, either genetically or chemically, to appeal to the market. In Australia, the prognosis is not so grim, however, we must urge the community as a whole, not only the ADHD community, to embrace these principles, to try the nutritional approach before they reach for the prescriptions, and to be open to the significant improvements that dietary modification can bring to sufferers of ADHD.

EXAMPLES OF FOODS RESTRICTED ON THE FEINGOLD DIET

Group 1 – Foods Containing Natural Salicylates
Fruits/ nuts – Almonds, apples, apricots, berries, currants, grapes & raisins, nectarines, oranges, peaches, plums and prunes.
Vegetables – Tomatoes and all tomato products, cucumbers and pickles.

Group 2 – Foods Containing Artificial Flavor or Color
Cereal/ grain – All manufactured cakes, cookies, pastries, sweet rolls, doughnuts etc.
Frozen baked goods, many packaged baking mixes.
Dairy – Manufactured ice creams, sherbets, puddings, gelatins, colored butters and cheeses, flavored yoghurt products, unless label specifies no synthetic colorings or flavorings.
Beverages – Prepared chocolate milk, cider, wine, beer, diet drinks, soft drinks, all instant breakfast drinks, all quick mix powdered drinks, tea.
Protein – Luncheon meats, barbecued poultry, all turkeys with prepared basting, stuffing etc, frozen fish fillets or sticks.
Misc – All manufactured candies, all powdered puddings and dessert mixes, oleomargarine, mustard, all mint flavored items, soy sauce (if flavored or colored), cider vinegar, wine vinegar, barbecued flavor chips, cloves, ketchup, chili sauce.

ENDNOTES

REFERENCES


Rimland, Callaway & Dreyfus, “Effect of high doses of Vitamin B6 on


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Naturopathic Approaches to Lyme Disease Treatment
NATUROPATHIC APPROACHES TO LYME DISEASE TREATMENT By Nicola McFadzean, N.D. Naturopathic medicine is a system of medicine that utilizes natural therapies such as herbal medicine and homeopathy, along with diet and lifestyle changes. The philosophy underlying naturopathic medicine is to treat the underlying cause of disease, to treat the person holistically, and to start treatment using least invasive therapies first, working up the therapeutic order as necessary. Naturopathic medicine is well placed to assist […] [Continue Reading]
Attention deficit hyperactivity disorder (ADHD) is the most common psychiatric disorder in childhood. Several interventions are effective in treating children with ADHD, including medications and behavior therapy. To examine how intensive treatment with medications compares with intensive behavior therapy, or with the combination of the two, NIMH sponsored the Multimodal Treatment of ADHD (MTA) study. The main findings from this study were published in December 1999, and are discussed below.

Q. What is the MTA?
A. The MTA was a multisite study designed to evaluate the leading treatments for ADHD. It aimed to examine how intensive treatment with medications compares with intensive behavior therapy, or with the combination of the two.

Attention-deficit/hyperactivity disorder (ADHD), a behavioral syndrome characterized by inattention and distractibility, restlessness, inability to sit still, and difficulty concentrating on one thing for any period of time. ADHD most commonly occurs in children, though an increasing number of adults also suffer from it. Another form of treatment, often used in conjunction with drug therapy, is cognitive behavioral therapy, which focuses on teaching affected individuals to learn to monitor and control their emotions.