Autism Spectrum Disorders and the Search for Answers

Autism spectrum disorders (ASD) are pervasive neurodevelopmental disorders that are characterized by delayed or abnormal ability to communicate and interact with others, as well as patterns of repetitive, restricted or stereotypical behavior. These problems occur early in childhood (before age 3) and are more frequent in boys than girls (4 boys have ASD to every girl who has it). The occurrence of this disorder has increased dramatically over the past 30 years. According to the CDC in the past decade alone, the incidence of ASD has increased 78%. In 2000 and 2002 it is estimated 1 in 150 children had the disorder, by 2006 the number had increased to 1 in 110 and current data suggests that 1 in 88 children have autism (CDC, 2012). While there is no doubt that greater awareness and early detection have had some influence on this, it is also clear that neither of these factors is a major component to the fast paced increase of ASD cases.

In the DSM-IV, ASD was divided into several types. These include autism, Asperger's syndrome, and pervasive developmental disorder not otherwise specified (PDD-NOS). Two other conditions are upon occasion included in this list, Rett's syndrome and childhood disintegrative disorder. Both are pervasive developmental disorders, but are thought to be unrelated to ASD. In the recently published DSM-V, the differentiated conditions, autism, Asperger's syndrome and PDD-NOS have been replaced by the umbrella term Autism Spectrum Disorders and will now be rated by severity (level 1, 2 or 3).

The new DSM-V criteria for ASD include the following, and a person must fit all 4 criteria:

A. Persistent deficit in social communication and social interaction across contexts, not accounted for by general developmental delays, and manifest by all 3 of the following:
    1. Deficits in social-emotional reciprocity: ranging from abnormal social approach and failure of normal back and forth conversation through reduced sharing of interests, emotions, and affect and response to total lack of initiation of social interaction.
    2. Deficits in nonverbal communicative behaviors used for social interaction; ranging from poorly integrated-verbal and nonverbal communication, through abnormalities in eye contact and body-language, or deficits in understanding and use of nonverbal communication, the total lack of facial expression or gestures.
    3. Deficits in developing and maintaining relationships, appropriate to developmental level (beyond those with caregivers); ranging from difficulties adjusting behavior to suite different social contexts through difficulties in sharing imaginative play and in making friends to an apparent absence of interest in people.

B. Restricted, repetitive patterns of behavior, interests, or activities as manifested by at least two of the following:
    1. Stereotyped or repetitive speech, motor movements, or use of objects (such as simple motor stereotypes, repetitive use of objects or idiosyncratic phrases).
    2. Excessive adherence to routines, ritualized patterns of verbal or nonverbal behavior, or excessive resistance to change (such as insistence on same route or food, repetitive questioning or extreme distress at small changes).
    3. Highly restricted, fixated interests that are abnormal in intensity or focus (such as strong attachment to or preoccupation with unusual objects, excessively circumscribed or perseverative interests).
    4. Hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of environment (such as apparent indifference to pain/heat/cold, adverse response to specific sounds or textures, excessive smelling or touching of objects, fascination with lights or spinning objects).

C. Symptoms must be present in early childhood (but may not become fully manifest until social demands exceed limited capacities),

D. Symptoms together limit and impair everyday functioning.

The Causes of ASD

Currently there is no single known cause for ASD, but numerous risk factors and possible causes have been identified. Research suggests that ASD most likely results from a complex combination of causes including genetic, environmental, immunological and neurological factors. Genetics certainly can be involved, and at least 15 possible gene interactions have been identified as being involved with ASD (Cook, 2001). The siblings of people with ASD (or the children of parents with this condition) have a 15-30 times greater risk of developing this disorder. Whether this is due to innate genetic factors, a similar environment or a combination of both is not known. In ASD with concomitant tuberous sclerosis, Fragile X syndrome, neurofibromatosis or chromosomal abnormalities there is evidence of a strong genetic component (Ashwood, et al, 2006). Other risk factors include increased parental age (>35 for women and 40 for men), gestational diabetes or maternal obesity, the use of some prescription drugs during pregnancy, low birth weight, exposure to a number of environmental toxins, immune dysfunction, abnormal folate and methionine metabolism, excessive glutamate and decreased oxytocin production, impaired cellular and systemic detoxification with reduced endogenous antioxidant levels, increased neuro-inflammation, phenylketonuria, low vitamin B-12, D and folate levels, pre and postnatal viral infections (measles, rubella, varicella, CMV, herpes simplex, mumps), and mitochondrial dysfunction.

Poor parenting and vaccines, both of which were blamed as causes of ASD, have been shown to not be causative factors (Uno, et al, 2012). Further discussion of some of these potential causes is warranted.

Immune dysfunction - over the past 20 years, research has revealed the incredible complexity and interconnectedness of immune and neurological function. Children (and adults) with ASD have been found to have abnormal Th-1 and Th-2 T lymphocyte ratios, decreased peripheral lymphocytes, inhibited T cell mitogen response, increased autoimmunity, and elevated monocytes and interferon-Υ. Immune imbalances such as these can modulate brain function, impair learning and emotional processing and cause inflammation with resultant damage to neurological tissue (Ashwood, et al, 2006). In a fascinating article in the journal, The Scientist, a father used worm therapy, which alters Th-1/Th-2 T-lymphocyte balance and has been used to treat autoimmune disease, with his autistic son. With a regular dose of porcine whipworm eggs (Trichuris suis), the boy's extreme behavior (smashing his head into a wall dozens of times per day, biting himself until he bled, gouging his eyes and face, screaming and kicking tantrums) disappeared and the father stated that he had his son back "or in many ways, it was like giving me a son that I didn't ever have" (Grant, 2011). A healthy Th-1/Th-2 T lymphocyte balance is also dependent on a healthy gut flora (Critchfield, et al, 2011). Many people with ASD have abnormally high levels of Clostridium and Desulfovibrio bacteria, some of which can produce neurotoxins.

In addition, immune response to dietary proteins and peptides (casein, caseomorphins, gluten and gluteomorphins) can stimulate T-cells and cytokine production and induce peptide-specific T-cell response, all of which can cause autoimmune reactions, disrupt neuroimmune communications and promote inflammation (Vojdani, et al, 2004). Children with ASD often have impaired digestion, with increased gut permeability and gluten or casein peptides can be easily absorbed. Numerous studies show that the levels of urinary peptides are much higher in people with ASD than in normal subjects (Nelson, et al, 2001). This has led to many parents of children with ASD to adopt a gluten-free, casein-free (GFCF) diet.

Research also shows that increased neuro-inflammation, as evidenced by elevated levels of cytokines [including interleukins, interferon-γ, tumor necrosing factor-alpha (TNF-α), heatshock proteins (HSP70), caspase 7 and transforming growth factor) probably plays a significant role in autism. It is also postulated that testing for these markers and changes in their levels could be an objective method of determining efficacy and safety of ASD treatments (El-Ansary & Al-Ayadhi, 2012).

Exposure to environmental toxins – laboratory, animal and human studies clearly indicate that the prenatal/fetal period is very susceptible to epigenomic dysregulation that can cause neuro-developmental deficits as well as many diseases (Perera & Herbstman, 2011). Many environmental chemicals, as well as pharmaceuticals and a lack of essential nutrients, have been shown in preliminary research to affect neurological development. Air pollution, especially polycyclic aromatic hydrocarbons (PAHs) such as benzo[a]pyrene can induce DNA damage, cause mutations, they are neurotoxic and act as endocrine disruptors. They are fat-soluble and can cross the placenta and fetal blood brain barrier (Perera & Herbtsman, 2011). Exposure to these chemicals is associated with developmental delays, reduced IQ, reduced birth weight, slow development and increased behavioral disorders. Bisphenol A (BPA) is commonly used in plastics and to line cans containing food. It is also fat soluble and recent epidemiological studies suggest that low dose prenatal exposure to this ubiquitous compound interferes with sexual dimorphism in brain structure and behavior, while negatively affecting social behavior and cognitive function. BPA is also linked to increased inflammation, it interfered with thyroid hormone, and in animal studies it increased hyperactive behavior and disrupted neocortical patterning (Nakamura, et al, 2007). In addition to these two compounds, prenatal exposure to pharmaceutical medications (Valproate, SSRIs, Misoprostol and possibly acetaminophen), phthalates, PCBs and organophosphate pesticides, have all been found to have negative effects on neurodevelopment and may possibly be a factor in the development of ASD. Studies have also found that exposure to environmental pollutants can cause mitochondrial dysfunction, which is significantly more common in children with ASD than in normal children (Napoli, et al, 2013; Rossginol & Bradstreet, 2005). Mitochondrial disorders can cause seizures, ataxia, and cardiac conduction problems and have been associated with developmental regression and growth retardation in children with ASD (Poling, et al, 2008).

Impaired cellular and systemic detoxification and endogenous antioxidant systems. - people with ASD have increased levels of oxidative stress and a reduced ability to eliminate metabolic wastes via methylation. They also have reduced levels of endogenous antioxidants such as sulfates, glutathione, superoxide dismutase and catalase (Ghanizadeh, et al, 2012). This means that environmental toxins (see above) and heavy metals are more difficult to eliminate, and the cellular antioxidant systems needed to mediate their effects are less active. Studies have repeatedly shown that children with ASD have higher levels of heavy metals including aluminum, arsenic, cadmium, mercury, antimony and lead (Blaurock-Busch, et al, 2012). In addition, some people with ASD have a dysfunctional folate-methionine metabolism. Methionine is needed to synthesize SAMe, which is essential for proper methylation and it is converted into S-adenosyl-homocysteine (SAH), which provides cysteine for glutathione production (Main, et al, 2010). In multiple human studies nutrients that enhanced methylation, sulphation and act as antioxidants have also shown the ability to improve some ASD behaviors. Recent research indicates that prenatal inflammation is a significant risk factor for development of ASD (Napoli, et al, 2013). Mothers who had elevated C-reactive protein (Top 20th percentile) had a 43% greater chance of having a child with autism (Brown, et al, 2013).

To further complicate this already challenging problem, many people with ASD have other disorders including epilepsy (11-39%), mental retardation (40-69%, more likely in females than males), learning disabilities (25-75%), chronic anxiety (7-84%), sensory processing disorder (42-88%), chronic gastrointestinal problems (67.6%), allergies (62.2%), sleep disturbances and depression (4-58%). Studies have also found significant overlap with people with high functioning ASD and ADHD (Rommelse, et al, 2010). Research clearly shows that ASD is a complex multi-system disorder and thinking of it as one condition with one cause or one treatment is a flawed concept. In order to adequately understand, prevent and treat this disorder we need to think beyond the brain and look at each person, subtypes of disorders and family. Prevention may have more to do with the environment and maternal health/nutrition or inflammation than genetics. Treatment of someone with level 1(high functioning) ASD, who has GI problems and allergies may differ significantly from another person who is also level 1 but has seizures, depression and sleep issues.

Orthodox Treatment and Management of Autism Spectrum Disorders

Standard treatments of this challenging condition aim to reduce the behavioral/emotional or intellectual deficits and increase the person's quality of life and independence. Early behavioral therapy, social skills therapy, specially tailored education, as well as speech and occupational therapy are often implemented. Two programs, Early Intensive Behavioral Intervention (EIBI) and Applied Behavior Analysis (ABA) are well studied and can help improve communication skills, behavior and learning in children with ASD. The drawback to this type of therapy is that they are time and staff intensive, requiring each child to undergo as many as 30-40 hours per week of therapy for several years with highly trained therapists (Lofthouse, et al, 2012). Other programs have found that autistic children respond well to interactions with a human therapist and specially engineered teaching robots. Children with ASD are often fascinated with objects and technology and robots are easier to understand as they have no complicated facial expressions, body language or tonal changes in their voices (Woolston, 2011).

A number of SSRIs, antipsychotic, psychostimulants, and other pharmaceutical medications have been used to reduce ASD behaviors such as irritability, hyperactivity, aggression and repetitive actions. Only Risperidone and Aripiprazole (Abilify) are FDA-approved for treating ASD-associated irritability (in 5-16 year olds). Many other medications are used "off label". Some work best for children, other for adolescents or adults and all can have significant adverse effects. Research suggests that 47% of children and adults with ASD are taking these medications (Lofthouse, et al, 2012).

Some Commonly Used Pharmaceutical Medications for ASD Symptoms

Medication-type	Benefits	Adverse Effects	Used for	
			Children	Adults
Clomipramine-Tricyclic antidepressant	Reduces repetitive behavior and stereotypies, may reduce aggressive behaviors and hyperactivity	Sleep disturbances, constipation, fatigue, depression, dystonia, seizures, behavioral problems		√
Fluxoxamine-SSRI	Reduces repetitive behaviors, maladaptive behaviors and aggression	Well tolerated in adults. In children, increased insomnia, aggression, ritual behavior, anorexia, anxiety and irritability		√
Fluoxetine (Prozac™)-SSRI	Reduces stereotypy, irritability, inappropriate speech and angry outbursts	Adverse effects more common in children and adolescents. Hypomania, anxiety, agitation		√
Sertraline-SSRI	Moderately reduced repetitive behaviors and aggression	Increased anxiety, agitation, skin picking, weight gain	√	√
Citalopram-SSRI	None-no better than placebo in large study	Increased impulsiveness, hyperactivity, stereotypy, diarrhea, insomnia and pruritus	√	
Haloperidol-antipsychotic	Reduced stereotypies and social withdrawal alleviated aggression, irritability	Increased sedation, dystonia, rare dyskinesias	√	
Risperidone-atypical antipsychotic	Reduces irritability, stereotypy, hyperactivity and non-compliance behavior	Increased appetite, weight gain, fatigue, anxiety, rhinitis, upper respiratory tract infection and dyskinesias	√	√
Methyphenidate-psychostimulant	Moderately reduces hyperactivity	Increased irritability. Is more effective for ADHD alone	√	√
Aripiprazole-atypical antipsychotic	Reduces irritability, hyperactivity and stereotypes	Increased weight gain, sleepiness, drooling and tremors	√	

CAM Treatments for ASD

Dietary Therapies

A significant number of people with ASD also have major GI problems, including chronic diarrhea, GERD, constipation, flatulence, and abdominal pain. Studies of autistic children reveal that they often have a distinct GI pathology with duodenitis, abnormal bowel flora, increased gut permeability and decreased small intestine enzymes (Geraghty, et al, 2010a).

For this large sub-group of ASD patients the gluten-free, casein-free (GFCF) diet has been shown to not only alleviate many of their digestive issues, but also reduce ASD behaviors and improve social behaviors (Pennesi & Klein, 2012;Harris & Card, 2012). This may be explained by the effect of the gluten and casein peptides known as gluteomorphins and caseomorphins. These compounds have been shown to cross the blood-brain barrier, negatively effect neurotransmission and they may inhibit CNS maturation (Geraghty, et al, 2010b). The challenge of implementing the GFCF diet is two-fold. First sources of gluten and casein are ubiquitous in the western diet. Eliminating dairy and wheat is not adequate. Gluten is also found in rye, spelt, triticale and in some oats. Gluten and gluten-products are often added to processed foods and this is also true of casein. According to some sources, the GFCF diet must be adhered to strictly for at least 7-9 months, before results are evident and maximal improvement may take two years to achieve (Kidd, 2003). The second issue is that many children with ASD are picky eaters and resistant to dietary changes. Other foods which may act as excitotoxins include refined sugars, artificial additives (aspartame, artificial food colorings and preservatives) and foods which the person has an intolerance to. Food sensitivities are especially problematic with ASD, as increased gut permeability, abnormal gut flora and impaired eliminatory abilities are all co-factors for creating food intolerances. After wheat/gluten and dairy, the most common problem foods are soy, corn, eggs and citrus fruits.

Herbal Therapies

There is little research into the use of herbs for ASD. Ginkgo, in a case study (3 patients), helped to reduce ASD symptoms (Niederhofer, 2009). In a RCT Ginkgo given with Risperidone did not improve treatment outcomes (Hasanzadeh, et al, 2012). In a 12 week, prospective, open label study with 40 children. a Kampo formula, Yokukansan was shown to improve ASD symptoms. This formula contains Atractylodes lancea, Poria, Cnidium, Gambir spines, Japanese Angelica root, Bupleurum and Licorice and it reduced irritability, stereotypic behavior, hyperactivity and inappropriate speech (Miyoka, et al, 2012). The herbs in this formula enhance digestion, inhibit inflammation, reduce hyperactivity and help re-regulate immune and endocrine function. This may explain some of its benefits exhibited in the study.

Based on what we know about ASD and its probable underlying mechanisms there are several herbal approaches that, while untested, would seem to make sense and offer little in the way of risk (especially compared to pharmaceutical medications).

A Theoretical Protocol for Treating ASD Symptoms and Metabolic Abnormalities

People with ASD often have allergies and abnormalities in their immune and inflammatory response system (IRS). Studies have found elevated levels of inflammatory cytokines including tumor necrosis factor-α (TNF-α), interferon γ, heat shock proteins (HSP 70), transforming growth factor (TGF-β_2), caspase 7 and interleukins 1, 6, 8, 10, 12 and 1β (El-Ansary & Al-Ayadhi, 2012;Lee & Kong, 2012). If pro-inflammatory cytokines are chronically activated, it can not only cause systemic and neurological inflammation but GI symptoms as well.

There are several categories of herbs that can be effective for re-regulating a disordered immune/endocrine system. They include immune amphotericks, immuno-regulators, some adaptogens, and antioxidant/ antiinflammatory herbs. Immune amphotericks are “foods for the immune system”. They strengthen and nourish immune function, allowing a disordered immune system to regain its normal regulatory capacity. Maitake, Cat’s Claw or Astragalus are effective immune amphotericks. Other herbs such as Licorice, Reishi, Eleuthero, Asian and American Ginseng and Schisandra berry are both immune amphotericks and adaptogens. Adaptogens help to re-regulate the immune, nervous and endocrine systems while enhancing an organism’s ability to tolerate and deal with acute and chronic stress. The term immuno-regulator is one I coined to describe antiinflammatory herbs which reduce pro-inflammatory cytokine levels and help to moderate an abnormal Th-1 and Th-2 T lymphocyte balance. Herbs in this category include Baikal Scullcap, Sarsaparilla, Gotu Kola, Unprocessed Rehmannia, Dan Shen/Salvia miltiorrhiza, Madder root, Turmeric, Boswellia and Bupleurum.

Studies have shown that people with ASD also have decreased ability to excrete metabolic wastes (⇓methylation) and ineffective endogenous antioxidant systems which allows increased damage and inflammation from these wastes. Antioxidant/antiinflammatory herbs can help to reduce oxidative stress and the resultant inflammation. Herbs in this category include Amla fruit, Turmeric, Blueberry/Bilberry, Green tea, Rosemary, Hibiscus, Rose hips, Lycium fruit, Beets, Hawthorn, Grape Seed extract, Pine bark extract, and Triphala.

In addition, several herbs and supplements have been shown to enhance hepatic glutathione levels allowing for more effective excretion of wastes by the liver ((Phase II detoxification). Herbs that increase hepatic glutathione levels include Turmeric, Schisandra, Milk Thistle and Picrorhiza. Nutritional supplements which can enhance hepatic glutathione levels include alpha lipoic acid, n-acetylcysteine and SAMe.

Herbs that can enhance systemic elimination are known as alteratives. These herbs gently promote kidney, liver, lymph, skin and bowel function. Many mild alterative herbs could be useful as a part of an ASD protocol including Cleavers, Dandelion root, Yellow Dock root, Sarsaparilla, Oregon Grape Root, Burdock root, Violet leaf and Red Clover.

Botanicals that help reduce anxiety, hyperactivity, insomnia and irritability are also indicated. Nervines are calming herbs that help to reduce irritability, agitation and hyperactivity. Useful herbs might include Linden flower, Scullcap, Chamomile, Passion Flower and Blue Vervain. Anxiolytics relieve anxiety and Bacopa, Motherwort, Blue Vervain and Chinese Polygala are good choices. Nootropics enhance cerebral circulation, improving memory, focus and concentration. Effective nootropics include Bacopa, Ginkgo, Gotu Kola, Rosemary, Eclipta and White Peony. Many of the herbs mentioned as nervines, anxiolytics or nootropics also are effective for treating insomnia, seizures and depression, which are common co-morbidities in ASD.

A significant number of people with autism also have digestive issues which are clearly linked to increased agitation, irritability, hyperactivity and anxiety. Carminatives and GI antispasmodics (along with probiotics-see under Supplements) would be a useful addition to their protocols. Effective carminatives include Ginger, Chamomile, Fennel seed and Angelica. Gastrointestinal antispasmodics/analgesics include Xiang Fu/Cyperus, Wild Yam, Hops, Mu Xiang/Saussurea root and Catnip.

Nutritional Therapies

A number of nutritional supplements have been studied for treating ASD symptoms. The number of studies and the quality of them makes it difficult to accurately access the true benefits of many of these nutrients. With the scarcity of significant adverse effects in supplement studies and the lack of effective pharmaceuticals with their propensity for side effects, the benefit to risk ratio is in favor of supplement use.

B-6 and magnesium - in several clinical trials high dose B-6 (600-1,125 mg) combined with magnesium (400-500 mg per day), as well as magnesium alone have shown the ability to improve alertness and reduce outbursts, self-mutilation and stereotyped behaviors (Mousain-Bosc, et al, 2006; Martineau, et al, 1985). Other studies have found either negligible or no benefits. Some research suggests that people with ASD have elevated serum B-6 levels. This may indicate an inability to convert pyridoxal to pyridoxal-5-phosphate (P-5-P), the active metabolite of B-6.

Folic acid – in children with early onset low-functioning autism and Rett's Syndrome, folate receptor (FR) autoimmunity has been found to be a common problem. This inhibits folate binding to the choroid epithelial cells and produces cerebral folate deficiency (Ramaekers, et al, 2012). Oral supplementation of folinic acid improved cerebral folic acid levels and reduced ASD behaviors (Ramaekers, et al, 2007). The use of pre-natal folic acid supplements has also been found to significantly reduce the risk of having children with ASD (Surén, et al, 2012).

B-12 (methylcobalamin) - both children and adults with ASD have impaired metabolic detoxification pathways (transmethylation and transsulfuration) and reduced cellular antioxidant activity (glutathione, methionine, SAMe, homocysteine and cysteine). Some researchers believe this may be a contributing factor to developing ASD and its clinical manifestations. Several studies have looked at supplementing methylcobalamin, which is needed for production of SAMe used for methyltransferase reactions. Folate is also essential to these processes, and in two studies supplementing methylcobalamin (75 mcg/kg 2 x per week) and folinic acid (400 mcg BID) improved these metabolic imbalances and may have also improved speech and cognitive function (James, et al, 2009 & 2004).

Essential Fatty Acids – many studies of children with ASD have found that a large percentage have low levels of Omega-3 fatty acids. These compounds are essential for brain development and healthy neurological functioning. There have been four open trials of EPA/DHA as well as two small RCTs. There is some evidence of modest benefits but the results are not conclusive (Lofthouse, et al, 2012). In a double blind, placebo-controlled randomized trial, teenagers with ASD were given arachidonic acid (ARA) and docosahexaenoic acid (DHA). ARA is essential for signal transduction needed for neuronal maturation. In this study the fatty acids improved plasma antioxidant levels, social responsiveness and social interactions (Yui, et al, 2012).

Probiotics – a large number of people (67.7%) with ASD have GI dysfunction (constipation, diarrhea, IBS, GERD, dyspepsia, flatulence, etc.), which has been linked to increased irritability, tantrums, aggressive behavior and sleep disturbances (Critchfield, et al, 2011). Children with ASD also have abnormalities of the gut flora. Studies have found 10-fold increases in Clostridium spp., some of which produce neurotoxins. A small study of ASD patients with chronic diarrhea found that vancomycin (8 weeks) significantly improved behavior and communication skills (Sandler, et al, 2000). Probiotics offer a healthier and more sustainable method of promoting a healthy GI microbiome. In addition many people with ASD also have increased gut permeability and GI-based immune dysfunction. Probiotics have been shown to have antiinflammatory, anxiolytic and immune regulatory effects and may offer significant benefits for improving not only GI problems associated with ASD, but behavioral problems as well.

Vitamin/mineral combinations – nutrient deficiencies are common in ASD. While some may be due to picky eating habits, others may be due to impaired digestive function, reduced methylation and gut dysbiosis. In a RCT 141 children and adults with ASD were given a vitamin/mineral supplement containing a broad range of nutrients. Not only did nutritional and metabolic status improve (including methylation, sulfation, glutathione levels and reduced oxidative stress), there were also reductions in hyperactivity and tantrums (Adams, et al, 2011).

L-carnitine – was given to 30 children with ASD. The dose was 50 mg L-carnitine/kg bodyweight per day for three months. In this RCT, significant improvements in cognitive skills and other measures of autism severity were observed (Geier, et al, 2011). The researchers looked at using this amino acid because it has been shown to enhance mitochondrial function, fatty acid metabolism and it acts as a cellular antioxidant.

Melatonin – has been found to help alleviate sleep problems in children with autism and fragile X syndrome. In a small preliminary RCT, 3 mg of melatonin per day reduced sleep latency and prolonged sleep duration (Wirojanan, et al, 2009).

L-carnosine – is a dipeptide made up of two amino acids, beta-alanine and histadine. It is abundant in brain tissue and muscles. It has antioxidant and neuroprotective activity and has been shown in laboratory studies to protect against shortening of the telomeres. In a small RCT, autistic children were given 800 mg per day of carnosine. The study found that it improved behavior, communication and socialization (Chez, et al, 2002),

Other Therapies for ASD

Animal assisted therapy – people with autism often find interacting with animals easier than with people. Anecdotal reports suggested that equine-assisted therapy may be of benefit for children with ASD. A six-month long study of children with ASD aged 3-12 found that therapeutic horseback riding reduced the severity of autism symptoms as determined by the Childhood Autism Rating Scale (CARS) (Kern, et al, 2011).

Music therapy – has been found to improve language skills (verbal and body language), compliant behavior, social engagement and eye contact in children with ASD (Akins, et al, 2010).

Exercise – has been found in preliminary studies to reduce self-stimulatory behavior and it improved academic performance in autistic children (Lofthouse, et al, 2012).

Massage – in several small studies daily massage has been found to modestly improve attention span, decrease stereotypic behaviors and enhance sleep in autistic children (Akins, et al, 2010).

Neurofeedback (NF) – is a technique which enhances self-regulation of the brain by providing real-time video/audio information about EEG function. In several studies NF improved attention span, sensory/ cognitive awareness, communication skills and social interactions in people with ASD (Lofthouse, et al, 2012).

Ineffective Therapies for ASD

Many pharmaceutical medications used to treat ASD symptoms have been found to have a higher risk to benefit ratio for children (and adults) and so are not effective as a treatment. This includes Ritalin, Citalopram and Fluoxamine (in children). Porcine Secretin has also failed to show benefits (Krishnaswami, et al, 2011). Chelation therapy has not been shown to be effective and there are safety concerns with this practice (Akins, et al, 2010). In addition, EEG biofeedback (Kouijzer, et al, 2012) and hyperbaric oxygen therapy (Sampanthavivat, et al, 2012) have not proven to be useful treatments.