Controversial Treatments for Autism Spectrum Disorders

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Autism spectrum disorders (ASD) are a group of neurodevelopmental disorders that share overlapping diagnostic criteria ranging in symptom severity. Currently, diagnoses of ASD are based on a triad of observable behaviors including impairments in communication, impairments in socialization, and repetitive behaviors and restricted interests. And, while the prevalence of the disorders comprising the spectrum continues to be on the rise (Rice et al. 2010; Sun and Allison 2010), the etiology of ASD remains relatively unknown. More concerning for parents of children diagnosed with an ASD is that there is no known cure. As a result, parents are desperate to implement any treatments that have reported effectiveness (Elder et al. 2006), even if reports are anecdotal.

What is agreed upon by researchers, clinicians, and parents alike is that early intervention is imperative for children diagnosed with an ASD. And, research has provided support for early intervention (Hayward et al. 2009). However, what is not yet consistently practiced across professionals is the promotion of only treatments that have empirical support. Unfortunately, alternative treatments that lack evidence of efficacy are being utilized for children diagnosed on the spectrum. For example, researchers have reported that over 30 % of study participants diagnosed with an ASD were being treated with complimentary or alternative methods or medicine (Green et al. 2006; Levy et al. 2003), and these percentages are concerning. The unique and idiosyncratic characteristics associated with ASD, irregular and occasionally advanced skills (e.g., splinter skills or savant abilities), heightened susceptibility of having associated behavioral or psychiatric conditions, increased prevalence of those being diagnosed as having autism, and the permanent (or life-long) nature of the disorder are a few of the factors that have fueled debate about which treatment and intervention choices are most likely to yield favorable outcomes (Pavone and Ruggieri 2005). Due to these reasons, the field of ASD has the distinction of being a boon for numerous popular, but often unsubstantiated, treatment options. Autism is, in essence, a "fad magnet." These highly controversial treatments and intervention strategies are largely invalidated and offer little in the way of empirical data to support the efficacy of the therapy tactics, even when extraordinary and incomparable results are promised.

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Fortunately, empirically supported treatments exist to remediate core and associated symptoms of ASD. However, what constitutes an empirically supported treatment? The Task Force on Promotion and Dissemination of Psychological Procedures (1995) outlined criteria to determine what constitutes a treatment as well established. These criteria include that at a minimum, results from two studies indicate that the treatment under evaluation is superior to an established treatment or superior to a placebo, and the studies need to be conducted by at least two independent researchers. Alternatively, several single-subject design studies that show a treatment is superior to a placebo or other treatment could also ascertain a treatment as well established. Alternative or fad therapies, then, refer to treatments that lack sufficient empirical support to be considered well established. Tuzikow and Holburn (2011) provided the following definition of a fad treatment for ASD: "a technique or approach that is overpromoted in relation to its credibility" (p. 1). The treatments covered in this chapter fall into the latter category because they lack the empirical support required to validate efficacy.

Without scientific support, why then are these alternative treatments being implemented? Tuzikow and Holburn (2011) identified likely groups of promoters of alternative treatments including parents and semiprofessional practitioners. Parents of children with autism are confronted with raising their child who has been identified as having a life-long disability for which there is, at this time, no clear explanation why it manifests nor is there an accepted course of treatment. The stress of having a child with ASD or other developmental disability can lead to frustration and disappointment for the parent (Pavone and Ruggieri 2005; Romanczyk et al. 2003). As a result, parents may seek out many different treatment options out of desperation to help their child, are trusting of professionals promoting alternative treatments, and may lack the knowledge necessary to understand what constitutes a supported treatment (Metz et al. 2004). Thus, it is imperative that professionals promote treatments with supporting empirical evidence and also provide the parents with the knowledge necessary to know what questions to ask when considering a specific treatment for their child. However, even if parents have information regarding which treatments are empirically supported, they may have a sense of urgency to find an effective treatment quickly (Levy and Hyman 2005); therefore, the length of time studies take to be conducted, published, and disseminated may be too far down the road. Parents have also reported trying numerous different strategies at one time to treat symptoms of ASD, which helps to illustrate their need to find an effective treatment quickly. For example, Green et al. (2006) conducted a survey of 111 different treatments used by parents of children with ASD. Results of the survey indicated that on average, parents were presently utilizing seven different treatments for their children (Green et al. 2006). How then would anyone be able to discern which of the seven treatments is responsible for reduced symptomatology, if any at all?

In regards to semiprofessionals, they may not demonstrate the expertise and clinical competence at the same level as professionals who were trained as scientific practitioners. More specifically, they may lack the training necessary to identify research evidence to support or reject the use of a particular treatment (Task Force on Promotion and Dissemination of Psychological Procedures 1995). Furthermore, research has shown that these practitioners value colleague consultation, their own prior experience, how-to-books, and workshops (Blanton 2000). Of concern is that they place a greater value on the aforementioned than on scientific research articles (Blanton 2000). Given this information, it is not surprising then that some semiprofessionals would promote alternative therapies for the treatment of ASD.

So, even though alternative treatments are not empirically supported, what is the harm in using them to treat symptoms of ASD? First, utilizing these unsupported treatments can be a waste of the families' time, money, and may provide families with a sense of false hope (Zane et al. 2009). Secondly, and more problematic is that some adverse side effects have been reported following the utilization of these various fad therapies.

Therefore, the clinical safety of all alternative treatments should be assessed prior to use (Pavone and Ruggieri 2005). And, third, time spent implementing an unsupported treatment could have been better spent implementing a supported treatment with documented success for treating core and associated symptoms of ASD.

Controversial and Alternative Treatments

Gluten-Free and Casein-Free Diet (GFCF)

Gluten is a protein found in foods such as wheat, barley, rye, and oats; and casein is a protein found in dairy products. This dietary intervention involves the total elimination of these proteins in the diet. One of the uses of the GFCF diet is to treat symptoms of ASD, and it was implemented in response to one of the etiological theories of ASD, the opioid excess theory (Shattock and Whiteley 2002). The opioid excess theory, first proposed in 1979 (c.f. Panksepp 1979), postulates that symptoms of ASD result from an overactivity of the opioid system (1979). Panksepp (1979) reported that injecting low doses of morphine into animals produced symptoms similar to those observed in individuals with autism (e.g., no need for social relationships, unusual motor movements). So, what then causes the overactivity of the opioid system? It has been further hypothesized that ASD is caused from peptides derived from incompletely digested proteins (i.e., gluten and casein). These peptides pass through the blood-brain barrier and attach to the opioid receptors (Mulloy et al. 2010). Thus, the brain treats the proteins like opiate-type chemicals.

One of the first studies to address the abnormal production/absorption of peptides in those with ASD was conducted by Cade et al. (1999). Cade and colleagues examined the effects of the GFCF diet on the following symptoms: eye contact, social isolation, mutism, learning skills, hyperactivity, stereotypical activity, hygiene, panic attacks, and self-mutilation. A significant improvement was observed in all areas investigated within 3 months of initiating the diet. Additionally, these gains were maintained through a 12-month follow-up. Although Cade and colleagues reported that the GFCF diet is beneficial in treating symptoms of ASD, the study was not without limitations. Most notable is that ratings of symptoms of ASD were completed by both parents and physicians. While inter-observer agreement between the raters was calculated to be greater than 90 %, the raters were not blind to the treatment. Thus, pre- and posttreatment data were not objectively collected and could have been influenced by opinions and feelings. Also, no control/placebo group was employed for comparison purposes in an effort to rule out threats to internal validity. Furthermore, symptoms of ASD were simply listed and rated on a Likert type scale. The results would have been strengthened if a psychometrically investigated measure was utilized to assess symptoms of ASD.

Since the early studies conducted on the GFCF diet for the treatment of ASD symptomatology, many other studies have been conducted. Fortunately, reviews of these studies have also been completed. For example, Mulloy and colleagues conducted a review of 14 published studies that examined the usefulness of the GFCF diet on symptoms of ASD (2010). Results of the 14 studies were variable in their support for the GFCF diet to treat ASD. However, the results of Mulloy and colleagues review indicated that the diet does not ameliorate symptoms of ASD and that it lacks scientific support. Not only did Malloy and colleagues find a lack of empirical support for the diet, the results of their review also provided evidence against the opioid excess theory as an etiology of ASD. In addition, they identified that the studies reviewed in their research lacked experimental design, did not utilize control groups for comparison, implemented the diet for very short intervals, did not utilize inter-observer agreement, and did not use raters who were blind to treatment (Mulloy et al. 2010).

Studies that have been conducted with scientific rigor have concluded that the GFCF diet is ineffective for the treatment of ASD. For example, Elder et al. (2006) conducted a double-blind investigation of 15 children diagnosed with an ASD who were randomly assigned to a control

group or a GFCF diet group. Symptoms of ASD were assessed through the use of the Childhood Autism Rating Scale (CARS) at baseline, and weeks 6 and 12 of the intervention. No significant differences emerged when assessing symptoms using the CARS. Additionally, the researchers reported nonsignificant differences in the urinary peptide levels of both casein and of gluten. Interestingly, even though no benefits of the GFCF diet were reported, a large percentage of parents decided to keep their child on the diet following the cessation of the study (Elder et al. 2006).

As evident in the literature, the GFCF diet has yielded some promising results, but these results have emerged from studies that lack sound experimental design. Furthermore, the diet is not without risks. First, children who are on the diet have been found to have decreased bone density. For example, Hediger et al. (2008) examined cortical bone thickness (CBT) of male children diagnosed with an ASD. Results indicated that the CBT of boys with ASD increased as the children aged, but the rate of growth was slower over the years compared to typically developing children. The deviation of bone growth was two times greater for boys who were diagnosed with ASD and who were on the GFCF diet compared to boys diagnosed with ASD who were not on the GFCF diet (Hediger et al. 2008). Another negative implication of the diet is protein malnutrition. Arnold et al. (2003) conducted a study to look at nutritional deficiencies in children diagnosed with autism who were on a GFCF diet compared to children diagnosed with ASD who were not on a GFCF diet. The plasma levels of most amino acids were higher for children diagnosed with ASD and not on restricted diets when compared to children diagnosed with ASD and on GFCF diets. Thus, nutritional deficiencies were more evident in children with ASD on the GFCF diet.

At this time, the diet does not have sufficient empirical support to be implemented as a treatment for symptoms of ASD. Furthermore, children may be put at risk in regards to their health following the use of the diet. Therefore, at this time, the diet is only recommended for those who actually have an allergy to gluten or dairy products. With that being said, any children who are on a GFCF diet should be monitored medically.

Secretin

Secretin, a hormone that aids in digestion, has traditionally been used for diagnosing pancreatic disorders by administering a single injection intravenously and analyzing the pancreatic secretions (Metz et al. 2004). Secretin has been approved by the Food and Drug Administration (FDA) for this use only. Gastrointestinal (GI) problems are common in children with autistic disorder, with some estimates of up to half of children with autistic disorder exhibiting problems such as diarrhea, reflux, and/or food selectivity (Kuddo and Nelson 2003). Secretin was first investigated for its effect on symptoms of autistic disorder in 1998 by Horvath and colleagues. Horvath et al. (1998) described three children with autistic disorder who had undergone secretin injections in order to study pancreatic secretions secondary to GI complaints. Compared to the children in the study without autistic disorder, the children diagnosed with autistic disorder exhibited significantly more pancreatic secretions following the secretin injection. As anecdotal data, Horvath and colleagues also reported that at 5-week follow-up, parents of the children with autistic disorder reported decreased GI discomfort in addition to improved eye contact, alertness, and expressive language. Following publication of these findings, there was a dramatic demand for secretin injections by parents of children with autistic disorder resulting in a shortage of the hormone (Levy and Hyman 2005).

Researchers have sought to identify a possible mechanism of action for the reduction in autism symptoms following an injection of secretin. The most common theory has to do with a "brain-gut interaction" (Levy and Hyman 2005). That is, certain hormones produced in the gut are believed to act as neuropeptides, interacting with corresponding hormone receptors in the brain to influence behavior. Animal studies of secretin have demonstrated that secretin is capable of crossing the blood-brain barrier and that secretin receptors

are present in the brain. Secretin has been found to have an activating effect on Purkinje cells, central cerebellar nuclei, the hippocampus, and the amygdala in rats (Koves et al. 2004; Kuntz et al. 2004; Welch et al. 2003). Increases in GABA levels have also been observed as a result of secretin injections (Kuntz et al. 2004; Yung et al. 2001). However, do differences exist in the amount of secretin or secretin receptors in the brains of children with autistic disorder compared to typically developing children? Nelson et al. (2001) found differences in the amount of vasoactive intestinal peptide (VIP), a hormone in the same family of neuropeptides as secretin, in children with autistic disorder. However, no differences in secretin receptors have been identified between children with and without autistic disorder (Martin et al. 2000). Therefore, any potential mechanism of action for secretin improving symptoms of autistic disorder remains unknown.

Following Horvath et al.'s (1998) findings, controlled studies of secretin and its effects on symptoms of autistic disorder rapidly began to appear. Among double-blind placebo controlled studies conducted from 1999–2004 (n=15), no studies found evidence supporting intravenous secretin (in either single or multiple doses and either porcine or human synthetic secretin) as an effective treatment for autistic symptoms (Levy and Hyman 2005). Dependent variables ranged from standardized measures of symptoms of autistic disorder, challenging behaviors, communication and social skills, GI symptoms, sleep, and weight. While some studies reported statistically significant differences on individual dependent variables (Coniglio et al. 2001; Corbett et al. 2001; Roberts et al. 2001; Sandler et al. 1999), overall, a clinically significant effect for secretin was not found. Ratliff-Schaub et al. (2005) investigated the use of a transdermal form of secretin (i.e., secretin cream) used daily over a 4-week period. They found no significant differences between secretin and placebo on behavioral measures of autistic symptoms.

Many studies involving secretin report symptom improvements for both treatment and control groups, suggesting a placebo effect (Carey et al. 2002; Roberts et al. 2001; Sandler et al. 1999; Sponheim et al. 2002). That is, parents reported improvements in symptoms regardless of whether the child received secretin or placebo. To test this effect, several of the researchers asked parents to report whether they believed their child received secretin or placebo (Chez et al. 2000; Coniglio et al. 2001; Coplan et al. 2003; Molloy et al 2002). In every study, parents were no better than chance at predicting their child's group membership. To further demonstrate, in one study 76% of parents whose child received placebo indicated that they would continue the treatment, even after being informed that it had no effect (Sandler et al. 1999). Indeed, the prospect of a "cure" for autistic disorder symptoms in the form of a single injection is appealing to parents desperate for help. It is not uncommon for parents beginning any type of treatment to note improvements (Sandler and Bodfish 2000). Investments in the form of time and money as well as increased attention and reinforcement to positive, adaptive behaviors may be just a few of many factors that contribute to a placebo effect.

To add to the lack of support for the effectiveness of secretin in reducing symptoms of autistic disorder, some studies have actually found adverse effects on behavioral symptoms. Carey et al. (2002) found that children in both the secretin and placebo groups deteriorated on their scores on the Autism Behavior Checklist (ABC; Krug et al. 1993). Specifically, they found that children receiving secretin scored significantly worse on the hyperactivity subscale of the ABC. Similarly, Honomichl et al. (2002) collected data on sleep and found that nighttime awakenings were more frequent for children after receiving secretin. A combination of contradictory findings and little to no evidence of clinical efficacy has led many researchers to conclude that secretin is not an effective treatment for symptoms of autistic disorder.

Given the lack of support for secretin as a treatment for autistic disorder, why is it that many parents continue to consider it as a viable treatment option? Some clinicians continue to suggest that there may be a small subset of children with autistic disorder who respond positively to secretin injections. One study by Kern et al. (2002)

found a decreasing trend in challenging behaviors in a sample of five children who presented with GI upset (i.e., diarrhea). Following administration of secretin, diarrhea symptoms ceased and a subsequent decrease in challenging behaviors was observed. However, it is worth noting that it may have been the decrease in GI upset that led to increased comfort and mood and decreased irritability and challenging behaviors (Metz et al. 2004). Additional studies examining the plausibility of this subgroup effect (i.e., children with GI disturbances and autistic disorder benefit from secretin treatment) have been unable to replicate Kern et al.'s findings (Coniglio et al. 2001; Levy et al. 2003; Roberts et al. 2001).

The continued use of secretin in the treatment of autistic disorder presents several concerns. As with any pharmacological agent, secretin may result in adverse side effects and/or allergic reactions depending on the individual (National Institute of Child Health & Human Development 1998). In addition, while single-dose usage in adults is considered safe, the effects on children have not been studied long term. Furthermore, little is known regarding long-term effects of multiple dose use of secretin or the various forms of administration (i.e., intravenous, transdermal).

Supplements and Vitamins

These were first used to treat individuals diagnosed with schizophrenia, interventions utilizing vitamins began over 60 years ago (Rimland 1964). Since then, a variety of vitamins and supplements have been investigated for individuals diagnosed with an ASD to treat the core and/or associated symptoms of the disorders. In fact, about 30 % of parents of children diagnosed with ASD report using them (Green et al. 2006). These percentages rank supplement and vitamin use amongst the most utilized alternative treatments for ASD. Researchers have sought to identify a possible mechanism of action for the reduction in ASD symptomatology following the use of these vitamins or supplements. Some have reported that supplements and vitamins counteract biomedical errors that have occurred within the body. Reportedly, these errors can lead to the development of psychiatric disorders (Pfeiffer et al. 1995). A review of the most common vitamins/supplements utilized for individuals diagnosed with ASD is outlined below including vitamin B6 with magnesium and omega-3 fatty acid supplements.

Vitamin B6 with magnesium Improvements in ASD symptomatology, more specifically in speech and language, following the use of vitamin B6 was first reported over 3 decades ago (Bönisch 1968; as cited in Nye and Brice 2005). Numerous other studies have been conducted to investigate the potential benefits of this vitamin as a supplement; however most have utilized flawed research methodologies.

The first study conducted that utilized a sound experimental design (i.e., double-blind, placebo-controlled) to evaluate the effectiveness of this vitamin on ASD symptomatology was conducted in 1993. Tolbert et al. (1993) assessed symptoms of ASD grouped into the following domains: social, affective, sensory responses, language, and total scores from the Ritvo-Freeman Real Life Rating Scale for Autism (Freeman et al. 1986). The treatment group received 200 mg/70 kg of pyridoxine and 100 mg/70 kg of magnesium per day. No significant differences emerged from pre- to posttreatment for any of the subscales investigated. A significant reduction emerged on the total score; however, this was observed for both the control and treatment groups. Thus, results suggest that the administration of vitamin B6 and magnesium has no effect on the treatment of ASD symptomatology at these dosage levels. The authors noted that the dose utilized in their study was below than that from previous studies that reported positive findings and the reduced dosage was in an effort to reduce the risk of potential side effects.

Nye and Brice (2005) conducted a review of all randomized trials to examine the efficacy of administering vitamin B6 with magnesium. Their search for published articles prior to 2006 articles yielded only three studies that were double-blind, randomized, placebo controlled, and conducted on individuals diagnosed with an ASD (i.e.,

Findling et al. 1997; Kuriyama et al. 2002; Tolbert et al. 1993). Fifteen other studies were identified; however, they were eliminated from the review due to utilizing non-randomized designs. From the results of their review, Nye and Brice concluded that at this time research conducted yields insufficient support for the use of vitamin B6 with magnesium as a treatment for ASD.

Overall, relatively few adverse side effects have been reported following the use of B6 with magnesium; however, some researchers have reported neurotoxicity (i.e., peripheral neuropathy) following the use of B6 (Schaumburg et al. 1983). Not only do Schaumburg and colleagues report side effects, they further suggest that long-term use is unsafe and also strongly oppose the use due to the lack of studies demonstrating efficacy at this time.

Omega-3 fatty acids Omega-3 fatty acids are essential for normal growth and development. Researchers have reported associations between various neurodevelopmental disorders and fatty acid deficiencies (Richardson 2004). Reportedly and problematic, individuals diagnosed with an ASD have lower levels of these fatty acids (Meguid et al. 2008). At this time, the mechanism of action of omega-3 fatty acid supplements to ameliorate symptoms of ASD is unknown (Bent et al. 2009). Despite an unclear mechanism of action, it is a widely used alternative treatment for ASD. Green et al. (2006) reported that over 25% of children diagnosed with an ASD are being treated with fatty acid supplements.

Many studies have been conducted in an effort to evaluate the effectiveness of omega-3 fatty acid supplements. Bent et al. (2009) conducted a systematic review of these studies. The inclusion criteria for their review consisted of studies conducted between 1966 and 2008 that utilized participants diagnosed with an ASD who were treated with omega-3 fatty acids, and included an outcome measure to evaluate the effectiveness of the supplement. Their review of six studies that met the inclusion criteria indicated that insufficient evidence exists to support this intervention for the treatment of ASD. Furthermore, five of the six studies reviewed lacked experimental control and only one study was conducted that utilized a sound experimental design (i.e., Amminger et al. 2007).

In their study, Amminger et al. (2007) conducted a randomized, double-blind, placebo-controlled study to investigate the effects of supplements for children that met diagnostic criteria for autistic disorder. Children in the treatment group received fish oil supplements and children randomly assigned to the placebo group received coconut oil. Assessments were conducted at baseline and at 6-week follow-up using the ABC (Aman et al. 1985). The ABC assesses symptoms across five subscales including irritability, social withdrawal, stereotypy, hyperactivity, and inappropriate speech. Results indicated that children in the treatment and placebo groups that participated throughout the entirety of the study did not score significantly different from each other at 6-week follow-up (Amminger et al. 2007).

Despite its popularity as a treatment for ASD, empirical evidence does not support the use of omega-3 fatty acids for the treatment of ASD. Fortunately, studies have also reported that no adverse side effects have been observed during trials of omega-3 fatty acids for those diagnosed with an ASD (Bent et al. 2011). However, fatty acids are safe only when they represent less than 10% of dietary intake (Eritsland 2000). Thus, children receiving supplements should be guided by this recommendation or followed by a nutritionist.

Hyperbaric Oxygen Treatment (HBOT)

HBOT is FDA approved for the treatment of carbon monoxide poisoning, severe burn and wound healing, massive blood loss, and diving injuries such as decompression sickness (McDonough et al. 2003). HBOT involves inhaling a mixture of 20–100% oxygen in a pressurized chamber, with atmospheric pressure (atm) typically above 2 (Leach et al. 1998). To begin treatment, the patient enters the chamber and pressure is gradually increased to the target atm. Oxygen is then delivered at the decided upon mixture of room oxygen and pure oxygen, usually for a period of

60 min. However, an individual session of HBOT varies greatly by pressure, oxygen ratio, duration, frequency, and number of sessions depending on the patient and the condition it is targeting (Leach et al. 1998). HBOT has been found to result in increased blood flow to the brain and has the ability to decrease inflammation and repair damaged tissues (McDonough et al. 2003). As a result, HBOT has been utilized in various other disorders as an alternate treatment including stroke, cerebral palsy, fetal alcohol syndrome, and traumatic brain injury. However, controlled research is lacking regarding the effectiveness of HBOT for these conditions.

Several conditions believed to be targeted by HBOT have been identified as possible mechanisms of action for children with autistic disorder, such as cerebral hypoperfusion, oxidative stress, and inflammation (Rossignol 2007). Cerebral hypoperfusion, or reduced blood flow to the brain, has been found in various anatomical locations in children with autistic disorder and appears to correlate with core behavioral symptoms (i.e., language, social, repetitive behaviors; Rossignol and Rossignol 2006). Proponents of HBOT argue that through increased oxygen flow to the brain, cerebral hypoperfusion may be reduced resulting in improvements in symptoms of autistic disorder. However, not all children with autistic disorder exhibit cerebral hypoperfusion and, even among those that do, the areas of the brain that are affected vary from child to child. Some researchers additionally argue that inflammation in the brain may contribute to hypoperfusion (Rossignol 2007). Given that HBOT has been shown to reduce inflammation in general, a reduction in cerebral inflammation may reduce cerebral hypoperfusion and lead to an amelioration of symptoms of autistic disorder.

Children with autistic disorder have been found to have increased oxidative stress, an inability for the body to properly detoxify reactive oxygen species at a sufficient rate (Rossignol 2007). Concerns regarding the effect of HBOT on oxidative stress, specifically whether it would raise it for children with autistic disorder, who already have increased levels, have been raised given that it produces reactive oxygen species. However, studies of HBOT have indicated that oxidative stress is either unaffected or even improved in some cases when pressures less than 2 atm are used for long term (Rossignol 2007). Therefore, HBOT may have the beneficial effect of reducing oxidative stress in children with autistic disorder. How this may affect the behavioral presentation of autistic symptoms, however, has not been studied.

The first preliminary study of HBOT for autistic disorder was conducted by Rossignol et al. (2007) with 18 children receiving 40 sessions of HBOT. Rossignol et al. investigated the safety of HBOT for children with autistic disorder as well as measured the effect of HBOT on oxidative stress, inflammation, and behavioral symptoms of autistic disorder. Rossignol et al. found that at doses of 1.3 and 1.5 atm, only one child was unable to tolerate the pressure, concluding that HBOT appears safe at low doses. Measures of oxidative stress and inflammation yielded minimal improvements for some of the children and no change for others. Finally, parent-report measures of behavioral symptoms of autistic disorder indicated improvements in irritability, social withdrawal, hyperactivity, motivation, speech, and sensory/cognitive awareness. However, the open-label nature of the study and lack of a control group makes it difficult to draw conclusions regarding the efficacy of HBOT for autistic symptoms.

To follow-up the preliminary study, Rossignol et al. (2009) conducted a double-blind placebo controlled study of HBOT for children with autistic disorder. In order to maintain the blind nature of the study, a dose of 1.1 atm was used for the control group so that pressurization in the chamber could mimic that of the treatment group, which received 1.3 atm. Again, 40 sessions of HBOT were administered. Rossignol et al. reported that significant group differences were found. That is, autistic symptoms as measured by standardized parent report measures significantly decreased for children in the HBOT treatment group. They concluded that HBOT was an effective treatment for autistic symptoms. However, Granpeesheh et al. (2010) argue that the authors' conclusions were not supported given

the statistical analyses used. Granpeesheh et al. note that differences between the treatment and control group were not significantly different in the Rossignol et al.'s study. That is, both groups reported improvements in ASD. Where statistical differences between groups were found, they were minimal and unlikely to produce significant clinical differences (Granpeesheh et al. 2010).

Granpeesheh et al. (2010) performed their own double-blind placebo controlled trial consisting of 80 sessions of HBOT at 1.3 atm for the treatment group. A greater number of outcome measures were used than had been in previous studies, including both clinician and parent report standardized measures. Granpeesheh et al. reported improvements in both groups, but no significant differences between groups on any of the dependent measures. They concluded that HBOT is not effective for treating symptoms of autistic disorder, even when delivered twice the previously studied 40 session treatment length.

HBOT has not been shown to be a clinically effective treatment for symptoms of autistic disorder in controlled studies conducted to date. While the side effects of HBOT are rare, they include middle ear barotauma, sinus squeeze, serous otitis, claustrophobia, reversible myopia, and new onset of seizures (Rossignol and Rossignol 2006). In addition, studies have found that patients may drop out due to claustrophobia and/or anxiety related to being in the chamber for an extended period of time (Granpeesheh et al. 2010; Rossignol et al. 2009). The price of HBOT can cost more than US$ 15,000 for one person, with variations depending on the length of the treatment (McDonough et al. 2003). This can be quite a financial undertaking, particularly for a treatment with little empirical support.

Chelation Therapy

Chelation involves the administration of binding agents, typically dimercaptosuccinic acid (DMSA), to bind to heavy metals in the body and facilitate excretion through urine (Akins et al. 2010). Chelation with DMSA is FDA approved for use in adults and children with heavy metal poisoning. Some proponents of chelation therapy argue that by removing heavy metals from the body, recovery of neurocognitive functioning can occur. However, researchers have been unable to demonstrate this effect in controlled studies. In fact, findings suggest no improvements in neurodevelopmental symptoms following chelation (Dietrich et al. 2004; Rogan et al. 2001).

Use of chelation for autism became relevant following a publication by Bernard et al. (2001) comparing symptoms of mercury poisoning to symptoms of autistic disorder. Bernard et al. argued that given similarities between the symptoms of mercury poisoning and autistic disorder, it was plausible that autism was a form of mercury poisoning. They cited symptom onset following vaccinations, a correlation between prevalence of autistic disorder and increases in vaccines, a higher ratio of males to females in both conditions, the heritability of autism and a genetic predisposition to mercury sensitivity, and parent reports of high levels of mercury in children with autistic disorder as evidence for the proposed autistic disorder-mercury relationship. Bernard et al. (2002) specifically targeted thimerosal, a mercury-based additive included in many childhood vaccinations up until 2002.

In 2003, Nelson and Bauman published a review examining the claims made by Bernard et al. (2001). Nelson and Bauman (2003) note that Bernard et al. list several overlapping symptoms between autistic disorder and mercury poisoning; however, they fail to indicate which are the most characteristic versus rare symptoms of each. For example, common motor impairments observed in children with mercury poisoning include ataxia and dysarthria, rarely seen in children with autistic disorder. As such, Nelson and Bauman conclude that there are several distinct core features that differentiate mercury poisoning from autistic disorder.

Regarding a temporal relationship between vaccinations and onset of symptoms, Nelson and Bauman (2003) note several weaknesses in Bernard et al.'s (2001) argument. First, temporal association does not establish causation. Second, retrospective parental report of symptom onset is often poor and may result in erroneously relating

the beginning of the disorder to another recognizable event (e.g., vaccinations). Finally, numerous studies on vaccines and autistic disorder have been conducted and have found no evidence of a relationship (Chen and DeStefano 1998; Dales et al. 2001; Peltola et al. 1998; Taylor et al. 1999). In fact, prevalence studies have found continued increases in autistic disorder diagnoses despite decreases and/or plateaus in vaccination rates and elimination of thimerosal from vaccines (Dales et al. 2001). As such, the official stance of the American Academy of Pediatrics (Halsey and Hyman 2001), Institute of Medicine (Stratton et al. 2001) and the Immunization Safety Review Committee (Williams et al. 2008), is that there is no causal relationship between vaccines and autistic disorder.

Nelson and Bauman (2003) also investigated Bernard et al.'s (2001) argument that children with autistic disorder have higher levels of mercury in their systems. However, research has been unable to confirm this hypothesis. Studies of mercury in hair samples of children with autistic disorder and typically developing children have failed to find significant differences between the two groups (Ip et al. 2004; Wecker et al. 1985; Williams et al. 2008). The difficulty in confirming an excess of mercury in children with autistic disorder leads to additional concerns regarding the safety of chelation in children. A study by Stangle et al. (2007) found that when DMSA was administered to rats without excessive lead in their system, long-term cognitive and emotional problems resulted. As such, the use of chelation without evidence of heavy metal exposure in children may have negative consequences.

The continued use of chelation therapy as a treatment for autistic disorder is alarming given the lack of empirical support for the rationale underlying its use and efficacy. Chelation therapy can result in serious side effects including neutropenia, kidney dysfunction, liver damage, paresthesias, Stevens-Johnson syndrome, and in some cases, cardiac arrest due to hypocalcemia (Akins et al. 2010). In 2006, the CDC reported three deaths (i.e., two children and one adult) following chelation therapy secondary to hypocalcemia. One of the children was being treated for autistic disorder. Based on concerns regarding risk versus benefit, the National Institute of Mental Health (NIMH) canceled plans for the first controlled trial of DMSA in children with autistic disorder (Mitka 2008). Given the lack of evidence for a link between excess mercury and autistic disorder, the use of chelation therapy for these children should not only be considered ineffective, but potentially harmful.

Animal Therapy

Animal therapy is used in the treatment of a variety of disorders for adults and children. Animal therapy for autistic disorder may include the use of dogs as service animals, horse riding, and dolphin-assisted therapy, just to name a few. Advocates for animal therapy argue several benefits including improvements in social skills, decreases in maladaptive behaviors, and increased motor skills (Grandin et al. 2010). However, research regarding the efficacy of animal therapy consists largely of case studies and anecdotal reports. In addition, theories regarding the mechanism of action for animal therapy vary based on the specific therapy and symptoms of the child, and is highly speculative in nature with little empirical support.

The use of a service animal, such as a dog, for children with autistic disorder is commonly for safety purposes (Burrows et al. 2008). That is, a dog may alert parents when their child gets out of bed during the night or prevent the child from running away when outside. However, behavioral improvements have also been reported from the use of a service animal including elevated mood, increased attention, and improved social and communication skills (Martin and Farnum 2002). Explanations for these observed improvements vary from simple reinforcement and positive experiences with the service animal to sensory-based connections between the child with autistic disorder and the animal (Grandin et al. 2010). That is, children with autistic disorder have a difficult time understanding and interpreting verbal and nonverbal aspects of human communication. However, animal communication occurs solely

through nonverbal behavior which may be more easily understood by children with autistic disorder. Perhaps a more parsimonious explanation for perceived improvements in autistic disorder symptoms may be through the inherent increased social opportunities (e.g., others coming up to the child to meet the service animal, family members playing together with the service animal, increased family outings due to an extra "safety net" with the service animal) that coincide with having a service animal (McNulty 2009, as cited in Grandin et al. 2010).

Horses may be used with children with autistic disorder in a variety of ways (Grandin et al. 2010). Recreational riding is a less structured activity often used as reinforcement for other treatment/training techniques. Therapeutic horseback riding targets physical and motor improvements through riding such as posture, balance, and mobility, and is conducted by a certified riding instructor. Hippotherapy incorporates components of therapeutic riding with a more comprehensive treatment plan that uses riding as reinforcement for other training techniques and is conducted by an occupational or physical therapist (Gabriels et al. 2012). All forms of therapy with horses report a social aspect between both trainer and child and horse and child. While there are obvious physical benefits to riding (e.g., balance, posture, muscle tone), possible mechanisms of action for improvements in attention, social, and communication symptoms include enjoyment of the activity, increased social and language exposure with trainers in the presence of a reinforcer (i.e., the horse), and reinforcing vestibular sensory stimulation secondary to rhythmic movements of the horse (Grandin et al. 2010).

There have been few controlled studies examining the effectiveness of therapeutic riding and hippotherapy for children with autistic disorder. Bass et al. (2009) compared children receiving therapeutic riding to a wait-listed control group and found significant improvements on parent report measures of social motivation, sensory integration, and attention. Bass and colleagues acknowledge the potential bias given the non-blind nature of the study and use of parent report alone. More recently, Gabriels et al. (2012) conducted a pilot study of therapeutic riding for children with autistic disorder using both objective and parent report outcome measures. Compared to a wait-list control group, children participating in therapeutic riding exhibited significant improvements in self-regulation, motor control, and communication. Gabriels et al. hypothesized that the sensory experience of riding may induce a sense of calm, resulting in decreased irritability, stereotypic behaviors, and hyperactivity. In addition, communication skills may be fostered through interactions with trainers and horses (e.g., instructing the horse to "walk on"). Gabriels et al. call for more well-controlled studies of hippotherapy to address possible confounding variables such as the increased interaction and attention provided by the trainers, the highly reinforcing nature of the activity, sensory stimulation, and report bias due to the non-blind nature of existing studies.

Dolphin assisted therapy (DAT) involves swimming and interaction with dolphins in captivity or in the wild. In many cases, traditional training takes place and interaction with the dolphin is used as reinforcement for completion of work tasks (Williamson 2008). The extent of interaction with the dolphins varies and may include fin rides, swimming in the tank with the dolphin, or more educational activities regarding training on the care of the dolphin. Proponents of DAT argue that it has several benefits including increasing attention span, motivation, and language and that these results are seen more quickly with DAT than in other traditional forms of therapy (Nathanson 1998; Nathanson et al. 1997). However, as with most animal therapies, research on DAT is scarce, particularly for children with autistic disorder. Of the research that is available, there are many methodological flaws including lack of control groups and procedural integrity that would allow conclusions regarding efficacy to be drawn (Marino and Lilienfeld 2007). At best, research on DAT suggests that observed improvements following treatment are more likely the result of placebo or novelty effects. Marino and Lilienfeld (2007) argue that DAT is a reinforcing experience for some children that likely produces a "temporary feel good effect" (p. 248). However, given the risks

involved with interactions with a wild animal (i.e., risk of injury or infection), the use of DAT as a treatment technique should be considered unethical and irresponsible.

Based on the available research, animal therapy shows some promise in improving symptoms of autistic disorder. However, due to a lack of empirical research, it is unclear whether these therapies act as true treatments on their own or simply provide positive experiences that reinforce skills learned from more traditional therapy techniques. In addition, little is known regarding the maintenance of treatment outcomes once therapy has ended. Parents and consumers should be cautious when exploring these alternative treatments and thoroughly weigh the risks and benefits. If anything, reported benefits from animal therapy provide support that pairing reinforcing experiences with consistent training may lead to symptom improvements for children with autistic disorder.

Facilitated Communication

Facilitated Communication (FC) is an augmentative communication technique that was developed in the late 1970s in Australia and rapidly spread in late 1980s and early 1990s to the USA and other westernized countries, primarily Canada and western Europe. Initially created by Rosemary Crossley to increase the communication of individuals with cerebral palsy (Crossley and McDonald 1980; Crossley 1992), the use of FC has also generalized to people with autism spectrum disorders and other developmental disabilities in the USA (Biklen 1990, 1992, 2005; Biklen et al. 1992; Biklen et al. 1995). The use of FC and its widespread acceptance as a treatment choice for children with ASD has mainly occurred through information being disseminated, supported by, and promoted via training and workshops in other nations and an established network of FC service providers. Likewise, the establishment of the Facilitated Communication Institute at Syracuse University by Biklen in 1993 has further assisted with the expansion of FC to the mainstream audience (Biklen 2005).

FC has been described as a strategy that individuals with limited communication skills can successfully communicate and convey their thoughts by typing or pointing at letters on an alphabet board or by using a typing device (Biklen 1990, 1992, 2005). The premise of FC was based on the belief that with additional support, the user would be able to demonstrate his/her true capacities thereby increasing independency and overall quality of life. In FC, the individual is seated at a keyboard or other letter-displaying instrument. A trained facilitator supports the communicator to communicate by holding and/or physically guiding the individual's hand, arm, and, elbow, or pointer finger to select or point to letters on the keyboard or visual display. According to FC's proponents, the function of the facilitator is to assist the muscular control of the communicator by holding the communicator's arm steady, and yet be noninfluential so that the communicator will "get his or her own words out" and communicate in a way "that had been previously thought impossible" (Biklen 1992; Crossley 1994). The goal of the facilitator is to fade their level of support over time, allowing the individual to communicate without assistance.

FC has been deemed a controversial treatment due to inconsistencies determining the authorship of the individual's message. Out of all of those who cast the first cloud of suspicion on FC, the investigation by Wheeler et al. (1993) is often cited as the classic case whereby researchers were able to demonstrate that some facilitators unknowingly influenced the message of the person they were assisting. In their experiment, researchers selected 12 individuals who were proficient producers of FC. Each pair (communicator and facilitator) was shown a series of pictures of objects (e.g., hat, bread, car, etc.) and were then asked to label the object. The communicator and his/her facilitator were seated side by side, but were separated by a partition so that each person could not see the picture presented to the other. Three different experimental conditions were arranged. In the first, the communicator was presented with a picture, no picture was presented to the facilitator, and the communicator was asked to identify the picture through the use of FC. In the second

condition, the communicator was presented with a picture, no picture was presented to the facilitator, and the communicator was asked to identify the picture without the use of FC. Although the facilitator could not provide physical assistance in the second condition, he/she could use verbal prompts to assist the communicator. In the third condition, both the communicator and facilitator were presented with a card; however in half of the trials the cards were identical and in the other half they were different. Results of multiple presentations of these manipulations by Wheeler and colleagues found that the communicators did not produce accurate labels or descriptions of pictures unless facilitators were shown the same pictures. Furthermore, the communicators were also observed to type out labels or descriptions of the pictures in situations where the pictures were shown only to the facilitators.

Since the investigation by Wheeler and colleagues, a base of literature has amassed on the inconsistencies and inadequacies of FC (ref Bebko et al. 1996; Bomba et al. 1996; Braman and Brady 1995; Cabay 1994; Crews et al 1995; Eberlin et al. 1993; Klewe 1993; Montee et al. 1995; Moore et al. 1993; Myles et al. 1996; Regal et al. 1994; Shane and Kearns 1994; Simpson and Myles 1995; Smith et al. 1994; Szempruch and Jacaobson 1993; Wheeler et al. 1993). These studies have differed substantially in many respects including the kind of tasks involved, the characteristics of the clients and facilitators, the setting of the experiment, and the type of experimental design. Across all of the well-controlled investigations to date, researchers have consistently documented the role of facilitator influence and/or that the message attributed to nonspeaking autistic or developmentally delayed subjects are the exclusive product of facilitator cuing (Mostert 2001; Jacobson et al. 2005). Similarly, the few reports of validated communication under controlled circumstances have been described as occurring erratically amidst extensive cued typing, and as linguistically rudimentary, far below the level of sophistication attributed to subjects.

The research since the mid-1990s dealt a significant blow to the FC movement. Due to the lack of FCs scientific validity, a number of national organizations including the American Psychological Association (APA 2003), along with the American Academy of Pediatrics (AAP 1998), the American Speech-Language-Hearing Association (ASHA 1995), and other scientific and professional organizations have issued official resolutions indicating their failure to support FC. Many of these organizations e.g., Association for Behavior Analysis International have gone so far as to warn professionals of the risks of the technique as well as deem the continued use of FC as unethical (ABAI 1995).

Despite the overwhelming data to disconfirm the use of FC, the strategy still has its proponents and continues to be used in various capacities. In 2008, the parent-based nonprofit organization Autism National Committee (or AutCom) affirmed their belief that FC is "one accepted and valid way in which individuals with autism can exercise their right to say what they have to say" (AutCom 2008). It is reasonable to see how parents would buy into FC. The rationale behind this strategy would be appealing to parents and caregivers because it enables them to believe that their nonverbal child may one day become able to communicate their wants and needs. Families may be told for the first time that by using FC their child will be able to share their thoughts and feelings and therefore, parents may begin to believe that FC will work for their child. This is not to say that the proponents of FC do not acknowledge the controversial nature of the strategy. The Association for Persons with Severe Handicaps (TASH) does state that the topic of authorship with respect to FC has "become particularly controversial when the subject of what has been communicated concerns sensitive issues" (TASH 2000). Advocacy groups claim that the criticism of FC is based upon studies which are "poorly designed and/or whose results are incorrectly extrapolated to the entire population of FC users" (AutCom 2008). Proponents of FC also assert that FC is valid for some persons, and as such it should be continued for those where real user-author communication does occur. Furthermore, TASH advocates that as the FC movement is continued that that "rigorous and ongoing training" is undertaken for facilitators so that they are able

to able to "careful, reflective use" of FC (TASH 2000). As recently as 2009, a bill was introduced to the Massachusetts legislature requesting that teachers be mandated to receive training in FC to use as a treatment for students with disabilities (S. 223 2009). Despite the preponderance of research suggesting otherwise, it appears that due to the unfortunate number of consumers and providers believing that FC is effective this fad will continue to persist.

Sensory Integration Therapy

Sensory Integration Therapy (SIT) is based on theoretical assumptions first developed by Ayres (1972, 1979). This treatment is a form of sensory-motor therapy which has been applied to not only children with autism, but also those with learning disabilities, behavioral problems, intellectual disability, cerebral palsy, and other developmental disabilities (Watling et al. 1999; Case-Smith and Miller 1999; National Board for Certification in Occupational Therapy 2004; Spitzer et al. 1996). Sensory integration is a normal developmental process which involves the ability of the central nervous system to organize sensations from the environment and from within one's body. Ayres posited that children with autism or similar developmental disabilities have deficits in registering and modulating sensory input, and a deficit in the part of the brain that initiates purposeful behavior, which is termed the "I want to do it" system (Schaaf and Miller 2005). SIT, typically delivered in an individual session format, attempts to ameliorate the supposed underlying neurological processing deficits through sensory integration. SIT is most commonly used within occupational therapy programs, although some of the techniques may be used by teachers or other professionals. In a survey of occupational therapists, 82% of respondents reported that they "always" use a sensory integrative approach when working with children with ASD (Watling et al. 1999).

In an attempt to facilitate the integration of sensory information, SIT involves engaging the individual in full body movements designed to provide input in the vestibular, tactile, and proprioceptive systems. The vestibular system, located in the inner ear, integrates sensory input from the vestibular organs, eyes, and muscles, and allows a person to maintain balance and understand where they are in space. The tactile system coordinates sensory input through the sense of touch and disintegration of the tactile system is sometimes evidenced as tactile defensiveness. The proprioceptive system integrates sensory input received through muscles and joints, and is the primary mechanism for motor control and posture. It is believed that sensory difficulties, particularly those in autism, are due to a dysfunction in one or all three of these systems (Ayres 1972, 1979). Stock-Kranowitz (1998, p. 292) states that for children with autism their problem with sensory integration hinders them due to an "inefficient neurological processing of information received through the senses, causing problems with learning, development, and behavior." The purpose of sensory integration, then, is to come to an understanding of how these different types of sensory input have an impact on the child's behavior and learning, and then attempt to change how the brain processes and organizes sensations by providing sensory stimulation allowing the child to effectively begin learning (Bundy 2002).

This method of modifying the child's ability to learn via additional sensory input is often referred to as designing a "sensory diet" for the child. A sensory diet may incorporate environmental modifications, such as reducing unnecessary distractions, changing lighting, modifying classroom tools and materials, and adding specific sensory stimulation techniques. Sensory activities which may be incorporated into SIT include swinging in a hammock, applying brushes to various parts of the body, deep pressure, playing with textured materials, wearing a weighted vest, using a vibrating massager, carrying heavy objects, and engaging in balance activities (Bundy 2002; Schaaf and Miller 2005). According to supporters of the intervention, these sensory experiences are hypothesized to correct the underlying neurological deficits producing the perceptual-motor problems occurring in those

with autism or similar developmental disabilities (Hodgetts and Hodgetts 2007).

A growing literature base has amassed that addresses the outcomes and efficacy of the sensory integration approach. Daems (1994) reviewed the outcomes of 57 studies published between 1972 and 1992 that evaluated interventions based on SIT. More recent reviews (Leong and Carter 2008; Miller 2003; Parham et al. 2007) and meta-analyses (Vargas and Camilli 1999) have indicated that there may be at least 80 published articles that address sensory integration outcomes. Across all of these reviews, it has been demonstrated that for those studies which were well-designed rigorous studies (e.g., included objective measures of behavior, a control group or a second treatment comparison, baseline measures, etc.) results overwhelmingly fail to show that treatments based upon sensory-integration theory are effective in reducing symptoms or ASD and/or providing any clinically-relevant benefit to individuals receiving the intervention.

In a comprehensive review by Leong and Carter (2008) of research on the efficacy of SIT from 1997 to 2007, findings demonstrated a lack of solid evidence to support the use of SIT. The authors went so far as to conclude that the continued use of SIT, given the lack of evidence for its effectiveness, is not justified and may even be contraindicated. For example, Mason and Iwata (1990) compared the effects of SIT and a behavioral intervention within a multiple-baseline across subjects design. During the application of SIT in their study, Mason and Iwata observed that self-injury increased above baseline levels in a 3-year-old participant; however, problem behaviors were later reduced when behavioral interventions were prescribed. Findings similar to that of Mason and Iwata (1990) have also been found by Devlin et al. (2009, 2010) for children with ASD and self-injury. That is, severe problem behaviors did not significantly decrease when SIT was applied and in some cases increased; however, when function-based behavioral interventions were utilized clinically-significant reductions in problem behaviors were observed. These three investigations (Devlin et al. 2009, 2010; Mason and Iwata 1990) raise concerns about the active components of SIT and also call into question the continued and widespread use of SIT for decreasing problem behaviors in children with ASD and other developmental delays.

SIT remains a popular treatment among various consumers despite lack of evidence for its efficacy (National Board for Certification in Occupational Therapy 2004; Schaaf and Miller 2005; Watling et al. 1999). SIT is a resource intensive intervention that is often incorporated with other treatments for autism resulting in an "eclectic" approach. Because of the nature of SIT, it is often proposed as a necessary treatment option for stereotypy or behaviors maintained by automatic/sensory reinforcement. Green (1996) pointed out that although children may find SIT activities enjoyable, this does not provide evidence of any significant, long-lasting benefits in the child's behavior or in any underlying neurological deficits. Furthermore, although applying certain sensory activities (e.g., brushes of increasing firmness to the arms of autistic children) may help to desensitize them to certain stimuli, such benefits are most parsimoniously explained by well-known behavioral principles (e.g., habituation) rather than anything specific to SIT (Smith et al. 2005). Proponents of SIT do acknowledge that there may be some limitations to their approach. However, the vast majority of advocates of this approach indicate that the "supposed drawbacks" are the result of the limited research available which is due to a "lack of funding, paucity of doctorate trained clinicians and researchers in occupational therapy, and the inherent heterogeneity of the population of children affected by sensory integrative dysfunction" (Schaaf and Miller 2005). However, at this time, based on the literature to date, it appears that the actual limitation to SIT is not funding, but rather is the lack of proven effectiveness.

Auditory Integration Training

Auditory integration training (AIT) was developed by Berard (1993, 2006), an ear, nose, and throat doctor. Proponents of AIT claim that the beneficiaries of this treatment suffer from an

inability to organize and process auditory information. Furthermore, this dysfunction not only inhibits the individual's ability to hear but also impairs their ability to learn, comprehend information, and remain focused in/on their environment. How AIT became applied to those with ASD is based upon literature which posits that those with autism show a higher incidence of sensory processing difficulties than the general population (e.g., Baranek et al. 1997; Gillberg et. al. 1990). As a result, practitioners have proposed that AIT is a therapeutic approach aimed at reducing or eliminating auditory sensory processing challenges in those with ASD. The belief is that when individuals with ASD organize their auditory processing abilities, they will become more receptive to other therapies (AIT Institute 2010).

Although there are many variations within AIT (e.g., Berard Method, Somanoas Method, Tomatis Method), the general methodology consists of the recipient listening to music or sounds that have been digitally modified in some way. The actual AIT therapy is applied in an intensive format which involves the individual listening to music/sounds for a total of 10 h, subdivided into 20–30-min sessions across the span of 10 days. The music/sounds are altered in various ways such as dampening or limiting the peak frequencies, randomly varying the high and low frequencies on a random basis, or varying the volume. The auditory sound is modified in particular ways based upon the supposed needs and challenges of the recipient (Berard 2006). The premise is that upon listening to the random variations in sounds the individual's auditory system adjusts to the sounds and thus becomes more normal. The goal of AIT, then, is to "retrain" the acoustical reflex muscle (AIT Institute 2010). In theory, once hearing is retrained persons with ASD will become less sensitive to particular sounds in their environment, and a reduction in sound distortion will be evident. Proponents of AIT claim that benefits include improvement in memory, comprehension, eye contact, articulation, independent living skills, appropriate social behavior, willingness to interact with others, and responsibility in school (Berard 1993; Rimland and Edelson 1994).

Although the advocates of AIT claim that there is scientific evidence to support this therapeutic approach (Edelson et al. 1999; Rimland and Edelson 1994, 1995), the methodological and statistical procedures employed in these studies have been reported to be highly controversial and flawed. As a result, literature supporting the use of AIT has not been widely accepted by the scientific community (Dawson and Watling 2000; Goldstein 2000; Mudford and Cullen 2005; Sinha et al. 2006). Sinha et al. (2006) conducted a recent review of the AIT methods, limiting their review only to those investigations were researchers employed randomized control trials with individuals diagnosed with ASD. Out of the six studies identified, outcomes indicated that AIT was either ineffective to control conditions, or that the reported behavior changes were due to repeated measures on behavior rating scales, not AIT. Sinha and colleagues concluded that there was, at the time, no evidence sufficiently powerful or reliable to support the belief that AIT was empirically proven to be effective. This inability of researchers conducting well-control studies to find supportive evidence for the continued use of AIT has also resulted in public stances against the continued use of this technique by organizations such as the AAP (1998) and the ASHA (2004). ASHA (2004) went so far as to adopt a policy statement indicating that there was no evidence that AIT improves the behavior of persons who use this treatment, and any ASHA member could be found in violation if he/she choose to employ AIT.

Despite the lack of conclusive evidence for AIT with respect to effectiveness in persons with ASD, the use of this therapy continues. It is true that compared to other fads, AIT does offer several perceived advantages including the parent being permitted to remain with their child during the treatment sessions, a clear time commitment, and the use of "fancy" technical equipment. In an internet survey of parents of children with ASD, Green et al. (2006) found that almost half of respondents indicated using a physiological-based treatment with AIT being ranked as the 3rd most used treatment in this category. Given that the proliferation of AIT as well as other fad

treatments may continue to persist, Mudford and Cullen (2005) suggest that parents who are considering purchasing AIT to improve their children's behaviors should reconsider in light of the lack of valid evidence supporting AIT. Romanczyk et al. (2003) also cite reports of negative side effects which they argue raise ethical questions concerning the use of this procedure with people with autism. AIT is one of the more expensive treatment options for people with autism. Furthermore as Romanczyk and colleagues point out, AIT uses equipment capable of producing sounds at decibels that may be harmful to a person's auditory system, and therefore it is important that the intervention only occur under the direction of a trained AIT specialist. However, we would posit that regardless of whether AIT is carried out by a trained specialist or not, the time and money families would waste on this ineffective treatment as opposed to investing it in other empirically-supported treatments renders AIT a useless and, potentially harmful treatment option.

Conclusion

ASDs are a set of neurodevelopmental conditions typified by impairments in social interaction and communication, as well as excesses in restricted interests and/or repetitive behaviors. Symptoms of autism are reported to emerge early in life and persist throughout the individual's lifetime. Although there have been recent advancements in treating and understanding the etiological aspects of ASD, autism-related disabilities continue to remain largely enigmatic. The heterogenous nature of ASD across those diagnosed with the condition further compound the ability to pinpoint effective interventions. The purpose of this chapter was to provide a thorough review of the more popularized controversial and unsupported therapies often used with children diagnosed of having ASD. It is our belief that by reviewing the information contained herein parents and professionals will be able to cast a critical eye on the "latest and greatest" treatment touted by an enthusiastic celebrity, professional, or parent advocate. By being familiar with the literature one is better able to make informed decisions which will be beneficial and in the best interest of the client and his/her family. It is highly plausible that parents may continue certain therapies (e.g., dolphin or equestrian therapy, SIT) not because it provides any significant learning experience or increases the child's ability to function more independently, but because their child genuinely enjoys participating in activity.

We would advocate that, regardless of the child's preferences, evidence-based practices are the central component to any treatment package. In short, evidence-based treatments are those which have amassed a base of research conducted by multiple investigators (other than the main, or central, treatment advocate) that use operationally defined terms, give significant subject/participant details, have reliable measures of behavior change, utilize rigorous experimental designs, and control for multiple sources of bias and other threats to internal validity (Kasari 2002; Newson and Hoanitz 2005; Reichow et al. 2008).

At this time, treatments which have the most empirical support in the literature with respect to effectiveness are those based upon applied behavior analysis (Newson and Hoanitz 2005; Tuzikow and Holburn 2011). Treatments for young children with ASD which can be classified as being based on behavioral principles (i.e., operant learning theory), may vary in their immediate focus; however, they share common features which include: (a) an individualized curriculum focusing on deficit areas (e.g., selective attention, imitation, language, communication, toy play, and social skills); (b) highly supportive teaching environments with explicit attention to the generalization of treatment gains; (c) an emphasis on predictability and routine; (d) a function-based approach to manage challenging behavior; (e) a focus on appropriate educational placements; and (f) parental or caregiver involvement in treatment (Matson and Minshawi 2006; Sturmey and Fitzer 2007). Although treatments grounded in behaviorism have the most support with respect to well-controlled research, it should be stated that, at this time, there is no known "cure" for ASDs. Persons with ASD are not a homogenous group—meaning that not everyone symptomatically pres-

ents exactly the same. For even those treatment modalities with empirical support, the complex nature of the diagnosis of autism has significant implications with respect to prognosis, treatment planning, and treatment outcomes.

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