Separating Fact from Fiction in the Etiology and Treatment of Autism

A Scientific Review of the Evidence

James D. Herbert, Ian R. Sharp, and Brandon A. Gaudiano
MCP Hahnemann University

Autistic-spectrum disorders are among the most enigmatic forms of developmental disability. Although the cause of autism is largely unknown, recent advances point to the importance of genetic factors and early environmental insults, and several promising behavioral, educational, and psychopharmacologic interventions have been developed. Nevertheless, several factors render autism especially vulnerable to pseudoscientific theories of etiology and to intervention approaches with grossly exaggerated claims of effectiveness. Despite scientific data to the contrary, popular theories of etiology focus on maternal rejection, candida infections, and childhood vaccinations. Likewise, a variety of popular treatments are promoted as producing dramatic results, despite scientific evidence suggesting that they are of little benefit and in some cases may actually be harmful. Even the most promising treatments for autism rest on an insufficient research base, and are sometimes inappropriately and irresponsibly promoted as "cures." We argue for the importance of healthy skepticism in considering etiological theories and treatments for autism.

Autism is a pervasive developmental disorder marked by profound deficits in social, language, and cognitive abilities. Prevalence rates range from 7 to 13 cases per 10,000 (Bryson, 1997; Bryson, Clark, & Smith, 1988; Steffenberg & Gillberg, 1986; Sugiyama & Abe, 1989). It is not clear if the actual prevalence of autism is increasing, or if the increased frequency of diagnosis has resulted from wider recognition of the disorder and especially recognition of the full range of pervasive developmental disorders, often referred to as "autistic-spectrum disorders."[1] Either way, autism is no longer considered rare, occurring more commonly than Down's syndrome, cystic fibrosis, and several childhood cancers (Fombonne, 1998; Gillberg, 1996).

The degree of impairment associated with autism varies widely, with approximately 75% of autistic individuals also meeting criteria for mental retardation (American Psychiatric Association [APA], 1994). Autism occurs three to four times more frequently in males than females (Bryson et al., 1988; Steffenberg & Gillberg, 1986; Volkmar, Szatmari, & Sparrow, 1993). Although recent advances have been made with respect to possible causal factors (Rodier, 2000), the exact etiology of autism remains unknown. Moreover, although certain behavioral, educational, and pharmacological interventions have been demonstrated to be helpful for many individuals with autism, there is currently no cure for the disorder.

Why Autism Is Fertile Ground for Pseudoscience

Several factors render autism especially vulnerable to etiological ideas and intervention approaches that make bold claims, yet are inconsistent with established scientific theories and unsupported by research (Herbert & Sharp, 2001). Despite their absence of grounding in sci-

1. We use the term "autism" throughout this paper to refer not only to classic autistic disorder (American Psychiatric Association, 1994), but in some cases to the full range of autistic-spectrum disorders. The vast majority of the research reviewed in this paper does not distinguish among the various subtypes of autistic-spectrum disorders. It is therefore often impossible to judge the degree to which research findings are unique to autistic disorder per se, or are generalizable to other pervasive developmental disorders.

James D. Herbert, Ian R. Sharp, and Brandon A. Gaudiano, Department of Clinical and Health Psychology, MCP Hahnemann University, Philadelphia, Pennsylvania.

Correspondence concerning this article should be addressed to James D. Herbert, Department of Clinical and Health Psychology, MCP Hahnemann University, Mail Stop 988, 245 N. 15th Street, Philadelphia, PA 19102-1192; E-mail: **james.herbert@drexel.edu**.

ence, such theories and techniques are often passionately promoted by their advocates. The diagnosis of autism is typically made during the preschool years and, quite understandably, is often devastating news for parents and families. Unlike most other physical or mental disabilities that affect a limited sphere of functioning while leaving other areas intact, the effects of autism are pervasive, generally affecting most domains of functioning. Parents are typically highly motivated to attempt any promising treatment, rendering them vulnerable to promising "cures." The unremarkable physical appearance of autistic children may contribute to the proliferation of pseudoscientific treatments and theories of etiology. Autistic children typically appear entirely normal; in fact, many of these children are strikingly attractive. This is in stark contrast to most conditions associated with mental retardation (e.g., Down's syndrome), which are typically accompanied by facially dysmorphic features or other superficially evident abnormalities. The normal appearance of autistic children may lead parents, caretakers, and teachers to become convinced that there must be a completely "normal" or "intact" child lurking inside the normal exterior. In addition, as discussed above, autism comprises a heterogeneous spectrum of disorders, and the course can vary considerably among individuals. This fact makes it difficult to identify potentially effective treatments for two reasons. First, there is a great deal of variability in response to treatments. A given psychotropic medication, for example, may improve certain symptoms in one individual, while actually exacerbating those same symptoms in another. Second, as with all other developmental problems and psychopathology, persons with autism sometimes show apparently spontaneous developmental gains or symptom improvement in a particular area for unidentified reasons. If any intervention has recently been implemented, such improvement can be erroneously attributed to the treatment, even when the treatment is actually ineffective. In sum, autism's pervasive impact on development and functioning, heterogeneity with respect to course and treatment response, and current lack of curative treatments render the disorder fertile ground for quackery.

A number of contemporary treatments for autism can be characterized as pseudoscientific. Most scientists agree that there are no hard-and-fast criteria that distinguish science from pseudoscience; the differences are in degree, rather than kind (Bunge, 1994; Herbert et al., 2000; Lilienfeld, 1998). Although a detailed treatment of pseudoscience in mental health is beyond the scope of this paper, a brief discussion of the features that distinguish it from legitimate science is important in order to provide a context for considering currently popular etiological theories and treatments for autism. In general, pseudoscience is characterized by claims presented as being scientifically verified even though in reality they lack empirical support (Shermer, 1997). Pseudoscientific treatments tend to be associated with exaggerated claims of effectiveness that are well outside the range of established procedures. They are often based on implausible theories that cannot be proven false. They tend to rely on anecdotal evidence and testimonials, rather than controlled studies, for support. When quantitative data are considered, they are considered selectively. That is, confirmatory results are highlighted, whereas unsupportive results are either dismissed or ignored. They tend to be promoted through proprietary publications or Internet Web sites rather than refereed scientific journals. Finally, pseudoscientific treatments are often associated with individuals or organizations with a direct and substantial financial stake in the treatments. The more of these features that characterize a given theory or technique, the more scientifically suspect it becomes.

A number of popular etiological theories and treatment approaches to autism are characterized by many of the features of pseudoscience described above (Green, 1996a; Green, 2001; Herbert & Sharp, 2001; Smith, 1996). Still other treatments, although grounded on a sound theoretical basis and supported by some research, are nonetheless subject to exaggerated claims of efficacy. What follows is a review of the most popular dubious theories and questionable intervention approaches for autism. We also review promising etiologic theories and treatments. Some intervention programs are designed specifically for young children, whereas others are applied across a wider age range.

THE ETIOLOGY OF AUTISM: SEPARATING FACT FROM FICTION

Psychoanalytic Explanations

Although modern theories of autism posit the strong influence of biological factors in the etiology of the disorder, psychoanalytic theories have abounded traditionally. Kanner (1946) was the first to describe the parents of children with autism as interpersonally distant. For example, he concluded that the autistic children he observed were "kept neatly in refrigerators which did not defrost" (Kanner, 1973, p. 61). However, Kanner also stressed that the disorder had a considerable biological component that produced disturbances in the formation

of normal emotional contact. It was Bruno Bettelheim who was perhaps the most influential theorist promoting psychoanalytic interpretations of autism. Bettelheim rose to prominence as director of the University of Chicago's Orthogenic School for disturbed children from 1944 to 1978. He rejected Kanner's conclusions positing a biological role in the etiology in autism and was convinced that autism was caused by "refrigerator" mothers. According to Bettelheim, autistic symptoms are viewed as defensive reactions against cold and detached mothers. These unloving mothers were sometimes assumed to be harboring "murderous impulses" toward their children. For example, in his book *The Empty Fortress*, Bettelheim (1967) wrote that one autistic girl's obsession with the weather could be explained by dissecting the word to form "we/eat/her," indicating that she was convinced that her mother, and later others, would "devour her." Based on his conceptualization of autism, Bettelheim promoted a policy of "parentectomy" that entailed separation of children from their parents for extended periods of time (Gardner, 2000). Other psychoanalytic therapists such as Mahler (1968) and Tustin (1981) promoted similar theories positing problems in the mother-child relationship as causing autism (see Rosner, 1996, for a review of psychoanalytic theories of autism).

After his suicide in 1990, stories began to emerge that tarnished Bettelheim's reputation (Darnton, 1990). Several individuals claimed abuse at the hands of the famous doctor when they were at the Orthogenic School. Furthermore, information emerged that Bettelheim often lied about his background and training. For example, although he frequently claimed to have studied under Freud in Vienna, Bettelheim possessed no formal training in psychoanalysis whatsoever, and instead held a degree in philosophy. Also, Bettelheim claimed that 85% of his patients at the Orthorgenic School were cured after treatment; however, most of the children were not autistic and the case reports he presented in his books were often fabrications (Pollak, 1997). Despite the continued acceptance of Bettelheim's theories in some circles, no controlled research has been produced to support the refrigerator mother theory of autism. For example, Allen, DeMeyer, Norton, Pontus, and Yang (1971) did not find differences between parents of autistic and mentally retarded children and matched comparison children on personality measures. Despite the complete absence of controlled evidence, even today some psychoanalytic theorists continue in the tradition of Bettelheim by highlighting the putative role of early mother-child attachment dysfunctions in causing autism (Rosner, 1996).

Candida Infection

Candida albicans is a yeastlike fungus found naturally in humans that aids in the destruction of dangerous bacteria. Candidiasis is an infection caused by an overgrowth of candida in the body. Women often contract yeast infections during their childbearing years. In addition, antibiotic medication can disrupt the natural balance among microorganisms in the body, resulting in an overgrowth of candida (Adams & Conn, 1997). In the 1980s, anecdotal reports began to emerge suggesting that some children with candidiasis later developed symptoms of autism. Supporters of this theory point to animal studies in which candida was shown to produce toxins that disrupted the immune system, leading to the possibility of brain damage (Rimland, 1988). Furthermore, Rimland speculated that perhaps 5 to 10% of autistic children could show improved functioning if treated for candida infection. Proponents often recommend that Nystatin, a medication used to treat women with yeast infections, be given to children whose mothers had candidiasis during pregnancy, whether or not the children show signs of infection. However, there is no evidence that mothers of autistic children have a higher incidence of candidiasis than mothers in the general population and only uncontrolled case reports are presented as evidence for the etiological role of candida infection in autism (Siegel, 1996).

Adams and Conn (1997) presented the case study of a 3-year-old autistic boy who reportedly showed improved functioning following a vitamin treatment for candida infection. However, the boy was never medically diagnosed with candidiasis and was only reported to meet criteria based on questionnaire data. In addition, reports of the child's functioning were mostly based on parental report (especially concerning functioning prior to the course of vitamin treatment) and not on standardized assessment instruments. Although interesting, such presentations provide no probative data on the possible role of candidiasis in causing autism. Without reliable and valid evidence to the contrary, case reports cannot rule out a host of confounding variables, including any natural remission or change in symptoms due to developmental maturation or even merely to the passage of time. It is important to remember that many people, especially women, contract candidia infections at different points in their lives, sometimes without even knowing that they are infected because the symptoms are so mild (Siegel, 1996). However, there is no evidence that even severe candidiasis in humans can produce brain damage that leads to the profound deficits in functioning found in autism.

MMR Vaccination

There has recently been much public concern that the mumps, measles, and rubella (MMR) vaccine is causing an increased incidence of autism. As evidence of the link between the MMR vaccine and autism, proponents point to the fact that reported cases of autism have increased dramatically over the past two decades, which appear to coincide with the widespread use of the MMR vaccine starting in 1979. In fact, Dales, Hammer, and Smith (2001) found in their analyses of California Department of Developmental Services records that the number of autistic disorder caseloads increased approximately 572% from 1980 to 1994. Indicating a similar trend in Europe, Kaye, Melero-Montes, and Jick (2001) reported that the yearly incidence of children diagnosed with autism increased sevenfold from 1988 to 1999 in the United Kingdom. Fears that the MMR vaccine may be responsible for this rise in the increasing incidence of autism have been picked up in the media and some parents have decided to decline vaccinations for their children in an effort to protect them from developing autism (Manning, 1999).

Rimland (2000) saw "medical overexuberance" as producing a tradeoff in which vaccinations protect children against acute diseases while simultaneously increasing their susceptibility to more chronic disorders, including autism, asthma, arthritis, allergies, learning disabilities, Crohn's disease, and attention deficit hyperactivity disorder. Pointing out that the average number of vaccines school-age children receive is now at 33, Rimland blamed the "vaccine industry" for making products that have not been properly tested before their widespread usage. He concluded by stating that research on this problem should be of the "highest priority."

In fact, it was preliminary research findings that initially raised the possibility that the MMR vaccine might be related to the apparent increase in the incidence of autism. The British researcher Andrew Wakefield and colleagues (1998) reported 12 case studies of children who were diagnosed with particular forms of intestinal abnormalities (e.g., ileal-lymphoid-nodular hyperplasia). Eight out of the 12 children demonstrated behavioral disorders diagnosed as representing autism, which reportedly occurred after MMR vaccination. The authors concluded that "the uniformity of the intestinal pathological changes and the fact that previous studies have found intestinal dysfunction in children with autistic-spectrum disorders, suggests that the connection is real and reflects a unique disease process" (p. 639). However, Wakefield et al. made it clear in their report that they did not prove an actual causal connection between the MMR vaccine and autism.

Although the Wakefield et al. (1998) case reports suggested that the MMR vaccine may be associated with autism, recent epidemiological research has provided strong evidence against any such connection. Kaye et al. (2001) conducted a time trend analysis on data taken from the UK general practice research database. As discussed earlier, they found that the yearly incidence of diagnosed autism increased dramatically over the last decade (0.3 per 10,000 persons in 1988 to 2.1 per 10,000 persons in 1999). However, the prevalence of MMR vaccination among children remained virtually constant during the analyzed time period (97% of the sample). If the MMR vaccine were the major cause of the increased reported incidence of autism, then the risk of being diagnosed with autism would be expected to stop rising shortly after the vaccine was instated at its current usage. However, this was clearly not the case in the Kaye study, and therefore no time correlation existed between MMR vaccination and the incidence of autism in each birth order cohort from 1998 to 1993.

In an analogue study in the United States, Dales et al. (2001) found the same results when using California Department of Developmental Services autism caseload data from the period 1980 to 1994. Once again, the time trend analysis did not show a significant correlation between MMR vaccine usage and the number of autism cases. Although MMR vaccine usage remained fairly constant over the observed period, there was a steady increase of autism caseloads over the time studied. It is important to note that the increased incidence of autism found in these two studies most likely reflects an increased awareness of autism-spectrum disorders by professionals and the public in general, along with changes in diagnostic criteria, rather than a true increase in the incidence of the disorder (Kaye et al., 2001). Most recently, the U.S. government's Institute of Medicine, in a comprehensive report cosponsored by the National Institutes of Health and the Centers for Disease Control and Prevention, recently concluded that there exists no good evidence linking the MMR vaccine and autism (Stratton, Gable, Shetty, & McCormick, 2001).

The MMR hypothesis reveals several important lessons for the student of autism. First, parents and professionals alike can easily misinterpret events that cooccur temporally as being causally related. The fact that the MMR vaccine is routinely given at around the same age that autism is first diagnosed reinforces the appearance of a link between the two. Second, the MMR-autism link reveals nicely the self-correcting nature of science.

Like many hypotheses in science, the MMR-autism hypothesis, although reasonable when initially proposed, turned out to be incorrect or at best incomplete. Third, the issue illustrates the persistence of incorrect ideas concerning the etiology and treatment of autism even in the face of convincing evidence to the contrary. For example, Rimland (2000) purported to warn the public of the dangers of child vaccinations because of their link to autism and begins his article with the decree: "First, do no harm." However, recent research indicates that the MMR vaccine cannot be responsible for the sharp increases in diagnosed autism, and the real harm is the public health concern raised by encouraging parents to avoid vaccinating their children from serious diseases that can easily be prevented.

Current Scientific Findings

Research has implicated genetic factors, in utero insults, brain abnormalities, neurochemical imbalances, and immunological dysfunctions as contributing to autism. Siblings of individuals with autism have about a 3% chance of having the disorder, which is 50 times greater than the risk in the general population. In monozygotic twins, if one twin has autism, the second has a 36% chance of being diagnosed with the disorder and an 82% chance of developing some autistic symptoms (Trottier, Srivastava, & Walker, 1999). Although not definitive, the higher concordance rates in monozygotic twins relative to fraternal siblings suggests a genetic contribution to the etiology of autism. Nevertheless, the lack of 100% concordance for monozygotic twins suggests that the disorder probably develops as the result of combined effects of genetic and environmental factors.

Genetic disorders that have been identified as producing an increased risk of developing autism or pervasive developmental disorders include tuberous sclerosis, phenylketonuria, neurofibromatosis, fragile X syndrome, and Rett syndrome (Folstein, 1999; Trottier et al., 1999). Recent findings have also implicated a variation of the gene labeled HOXA1 on chromosome 7 as doubling the risk of autism, although this is only one of the many possible genes linked to the disorder (Rodier, 2000). Nevertheless, although some gene variants may increase the risk of developing autism, other variants may act to decrease the risk, explaining the large variability in the expression of autism.

Rubella infection of the mother during pregnancy and birth defects resulting from ethanol, valproic acid, and thalidomide exposure are also known in utero risk factors (Rodier, 2000). However, these factors can only explain the development of autism in a small subset of individuals. Regarding time for increased vulnerability, evidence from individuals exposed to thalidomide now points to the conclusion that the in utero insults that increase the risk of the autism probably occur quite early, within the first trimester of gestation (Stromland, Nordin, Miller, Akerstrom, & Gillberg, 1994). Other research that has compared individuals with autism with those without the disorder found differences in brain wave activity, brain (e.g., cerebellar) structures, and neurotransmitter levels (Trottier et al., 1999).

Scientific evidence supports the conclusion that autism is a behavioral manifestation of various brain abnormalities that likely develop as the result of a combination of genetic predispositions and early environmental (probably in utero) insults. Although recent scientific discoveries provide important clues to the development of the disorder, the etiology of autism is complex and the specific causes are still largely unknown.

Summary of Etiologic Theories and Research

There is currently no empirical support for theories that implicate unloving mothers, yeast infections, or childhood vaccinations as the cause of autism. The evidence invoked in support of these claims involves uncontrolled case studies and anecdotal reports. The confusion about the causes of autism appears to stem largely from illusory temporal correlations between the diagnosis of the disorder and normal events occurring in early childhood. No research has demonstrated a differential risk for autism due to maternal personality characteristics, the presence of candidiasis, or the use of the MMR vaccine. Scientific evidence points to genetic predispositions and various early environmental insults to the developing fetus as responsible for the development of the disorder.

QUESTIONABLE TREATMENTS FOR AUTISM: BOLD CLAIMS, DUBIOUS THEORIES, AND LITTLE DATA

A number of interventions have been promoted as providing breakthroughs in the treatment of autism. These treatments share many of the features of pseudoscience described earlier. Despite the absence of supportive data and even in the face of contradictory data, these treatments continue to be passionately promoted by their supporters.

Sensory-Motor Therapies

Smith (1996) reported that over 1,800 variations of sensory-motor therapy have been developed to treat individuals with autism. The popularity of these approaches derives from the observation that many individuals with autism exhibit sensory-processing abnormalities, although these types of dysfunctions are neither universal nor specific to the condition (Dawson & Watling, 2000). Furthermore, many individuals with autism exhibit a relatively high prevalence of fine and gross motor impairments. Nevertheless, little controlled research has examined the effectiveness of sensory-motor treatments for autism. We next briefly review the most commonly promoted treatments for autism that emphasize the importance of ameliorating the sensory-motor deficits often associated with the disorder.

Facilitated Communication

Facilitated communication (FC) is a method designed to assist individuals with autism and related disabilities to communicate through the use of a typewriter, keyboard, or similar device.[2] The technique involves a trained "facilitator" holding the disabled person's hand, arm, or shoulder while the latter apparently types messages on the keyboard device. The basic rationale behind FC is that persons with autism suffer from a neurological impairment called *apraxia*, which interferes with purposeful motoric behavior. This neurological abnormality in motor functioning is often hypothesized to be unrelated to intellectual functioning. Thus, many if not all people with autism are believed to possess a "hidden literacy" that can be expressed by overcoming these motoric deficits (Green, 1994).

FC was originally conceived in the early 1970s in Australia by Rosemary Crossley, a teacher at St. Nicholas Hospital in Melbourne. Crossley later cofounded and directed the Dignity Through Education and Language Center, which promoted the use of FC in Australia. Syracuse University education professor Douglas Biklen witnessed Crossley's use of FC in Australia and brought the technique to the United States. In 1992, Biklen formed the Facilitated Communication Institute at Syracuse University and began to promote its use for persons with autism. Biklen continues to maintain the Facilitated Communication Institute at Syracuse University and to be a vocal proponent of FC for autism (Gardner, 2001; Jacobson, Mulick, & Schwartz, 1995).

FC initially inspired great hope in many family members (especially parents) of people with autism. Their heretofore largely uncommunicative son or daughter appeared to begin communicating via typed messages such as "I love you," presenting them with poems, or carrying on highly intellectual conversations. It is not surprising that FC went largely unquestioned by understandably desperate family members and even many professionals, despite several obvious causes for skepticism. For example, autistic individuals often did not even look at the keyboard while apparently typing with a single digit, yet expert typists were unable to type coherent sentences with one finger without looking at the keyboard (Gardner, 2001). Such observations did not dampen the enthusiasm for FC by its proponents.

Despite this enthusiasm, the dramatic claims for FC have not survived scientific scrutiny. A number of scientifically rigorous studies have investigated FC, and the results of these studies clearly point to facilitators as the source of the typed information (Jacobson, Mullick, & Schwartz, 1995). For example, Wheeler, Jacobson, Paglieri, and Schwartz (1993) conducted a study in which autistic participants were asked to type the names of everyday objects that were shown to them on picture cards. The typing was done under three conditions: (a) the facilitators were not shown the picture; (b) the facilitators did not assist the typing, and (c) both the participants and the facilitators were shown pictures that were varied so that the participants and facilitators sometimes saw the same picture and sometimes saw different pictures. Not surprisingly, participants were unable to type the correct response in any of the conditions except when they were shown the same picture as the facilitators. Furthermore, in the condition in which the participants and the facilitators were shown different cards, the typed responses were of the pictures that were shown only to the facilitators. This study provided clear evidence that the facilitators were the source of the typed information.

Much of the controversy surrounding FC has stemmed from many facilitators' vehement denials of responsibility for the typed information. In one study, for example, Burgess et al. (1998) demonstrated that FC involves a form of "automatic writing" (i.e., writing without awareness that one is doing so), technically called an *ideomotor response*, on the part of the facilitator. Forty college students were trained to facilitate communication with a confederate in the role of a person

2. It is important to distinguish facilitated communication from methods of augmentative and alternative communication (AAC), in which disabled persons independently utilize various keyboard devices to communicate. In legitimate AAC, the individual uses the keyboard independently, and there are therefore no questions about the origins of the resulting communications (Jacobson et al., 1995).

with a developmental disability. Each participant was given different information about the confederate, who was then asked questions related to this information. Eighty-nine percent of the responses corresponded to the information provided to the facilitators, yet all but two reported that the information came from the confederate. In discussing the results of the Burgess et al. (1998) study, Kirsch and Lynn (1999) concluded that:

> The attribution of the response to the confederate was clearly an error. Just as clearly, participants were not aware of generating responses. Instead, their responses were automatic behaviors prepared by the intention to facilitate and their knowledge of the answers to the questions. (p. 510)

These are merely two of dozens of studies that have demonstrated conclusively that the source of messages in FC is the facilitator rather than the disabled individual, despite the absence of conscious intent or awareness on the part of facilitators. It is therefore not surprising that so many facilitators became ardent believers in FC.

The dangers of FC extend well beyond the disappointment of family members and the disillusionment of former facilitators who have acknowledged the actual origins of passages produced through the technique. Beginning the in late 1990s, facilitated messages describing vivid instances of sexual abuse at the hands of parents began to emerge. Such reports resulted in several cases of autistic individuals being removed from their homes, and parents being arrested and jailed on charges of sexual abuse. Although such charges were eventually dismissed, some accused parents were forced to spend their family savings on legal defense fees (Gardner, 2001; Jacobson et al., 1995).

Auditory Integration Training

Auditory Integration Training (AIT) involves listening to filtered, modulated music that presents sounds of varying volumes and pitches. AIT was initially developed by French physician Guy Berard as a treatment for auditory disorders. In the late 1970s, Berard began promoting the use of AIT for autism. The technique gained larger recognition with the publication of the book *The Sound of a Miracle* (Stehli, 1991), written by the mother of a child who was allegedly "cured" of autism through the use of AIT.

AIT is typically administered in two daily half-hour sessions for approximately 10 days. Proponents theorize that a major factor in the problem behaviors of people with autism is hypersensitive hearing. 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. 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; Stehli, 1991).

Once again, scientific research casts serious doubt on the claims made for this innovative treatment for autism. One pilot study (Rimland & Edelson, 1995), one uncontrolled study (Rimland & Edelson, 1994), and one small controlled study (Edelson et al., 1999) suggested possible limited benefits of AIT. In the recent controlled study, Edelson et al. (1999) claimed to demonstrate that AIT produced significant improvements in aberrant behavior in a group of autistic children and adults relative to a placebo condition in which participants listened to unmodulated music. In addition to behavioral improvements, the authors further purported to demonstrate that AIT resulted in improved information processing as reflected in brain wave changes. In describing the results of this study, Edelson (2001) recently went so far as to claim that AIT produced "normalization of brain wave activity" in treated subjects.

Nevertheless, this study is plagued by methodological problems, and the actual results are in fact inconsistent with the authors' conclusions and interpretations. For example, Edelson et al. (1999) found a difference between the experimental and placebo groups on only 1 of 3 primary outcome measures and only at 1 of the 4 assessment periods. Given the number of analyses conducted and the absence of a statistical correction for multiple tests, this single finding may well be the result of chance rather than representing a legitimate effect of AIT. At other assessment periods the AIT-treated participants' scores on this measure actually returned to baseline, which the authors acknowledge reflects that one third of the subjects in the experimental group actually became worse. The "normalization of brain wave activity" consisted of a putative increase in P300 event-related potential (ERP) amplitude in a tonal discrimination task. However, only 5 subjects (3 from the experimental group and 2 from the placebo group) completed this task. No information is provided on how representative these 5 subjects were of the larger subject pool, much less the general population of autistic individuals. This small sample precluded statistical analyses of the data. Furthermore, inspection of the raw ERP data reported by the authors reveals apparently large baseline differences between the two groups, casting further doubt on their conclusions.

Four other well-controlled studies (Bettison, 1996; Gillberg et al., 1997; Mudford et al., 2000; Zollweg et al., 1997) failed to find any specific benefit for AIT. In the most recent study, Mudford et al. (2000) compared AIT with a control condition in which children listened to ambient room music through nonfunctional headphones. No benefit of AIT over the control condition was found on measures of IQ, comprehension, or social adaptive behavior. Teacher-rated measures showed no differences between the groups and parent-rated measures of hyperactivity and direct observational measures of ear-occlusion actually nonsignificantly favored the control group. The authors concluded that "no individual child was identified as benefiting clinically or educationally from the treatment" (p. 118).

The American Academy of Pediatrics' Committee on Children with Disabilities published a statement in 1998 in the journal *Pediatrics* on the use of both AIT and FC for autism. The statement suggested that "currently available information does not support the claims of proponents that these treatments are efficacious," and further that "their use does not appear warranted at this time, except within research protocols" (American Academy of Pediatrics [AAP], 1998).

Sensory Integration Therapy

A. Jean Ayres (1979), an occupational therapist, developed Sensory Integration Therapy (SIT) in the 1950s. The treatment is a form of sensory-motor therapy recommended for children with autism, learning disabilities, mental retardation, cerebral palsy, and similar developmental disabilities. Ayres posited that the child with autism possesses deficits in registering and modulating sensory input, and a deficit in the part of the brain that initiates purposeful behavior, which she calls the "I want to do it" system. SIT, typically delivered in individual sessions, purportedly ameliorates these underlying deficits through sensory integration. In an attempt to facilitate this integration, the treatment involves engaging the child in full body movements that are designed to provide vestibular, proprioceptive, and tactile stimulation. Sensory integration activities include swinging in a hammock, spinning in circles on a chair, applying brushes to various parts of the body, and engaging in balance activities (Smith, 1996). These activities are hypothesized to correct the underlying neurological deficits producing the perceptual-motor problems witnessed in many individuals with autism. In other words, SIT is not designed to teach the child new physical/motor activities, but to correct fundamental sensory-motor dysfunctions underlying the disorder in order to increase the individual's capacity for learning new activities (Hoehn & Baumesiter, 1994).

Controlled studies have found little support for the efficacy of SIT for treating children with various developmental disabilities. Mason and Iwata (1990) found SIT ineffective for treating self-injurious behaviors in three patients with mental retardation, although the problematic behaviors were later reduced through behavioral interventions. Furthermore, self-injurious behaviors paradoxically increased in one 3-year-old patient when treated with SIT. Iwasaki and Holm (1989) found no difference between the SIT and control condition (described as informal talk and touch) in decreasing stereotypic behaviors in young children and adults with mental retardation. Jenkins, Fewell, and Harris (1983) found no differences between young children with mild-to-moderate motor delays who received either SIT or small group therapy for 17 weeks. Finally, Densem, Nuthall, Bushnell, and Horn (1989) found no differences between SIT and no-treatment control conditions for children with learning disabilities. In fact, in their review of the literature Hoehn and Baumeister (1994) concluded that controlled studies of SIT demonstrate no unique benefits for the treatment on any outcome areas in children with learning disabilities.

Dawson and Watling (2000) recently reviewed studies that used objective behavioral measures in investigating the efficacy of SIT for autism. Only one of the four studies had more than 5 participants and no study included a comparison group. In the study with the largest sample size, Reilly, Nelson, and Bundy (1984) used a randomized, ABAB counterbalanced design to compare SIT with tabletop activities (e.g., puzzles and coloring). Eighteen children with autism received an hour of SIT and tabletop activities each. The authors reported that verbal behavior was superior in the tabletop as compared with the SIT condition because children spoke more during the fine motor activities. Nevertheless, the brevity of treatment, lack of specific training in SIT for the therapists, and failure of the researchers to assess verbal behavior outside the experimental condition limit the conclusions that can be drawn.

Other single-case studies comparing SIT with no-treatment baseline among autistic children have reported beneficial results (Case-Smith & Bryan, 1999; Linderman & Stewart, 1999). However, these designs cannot demonstrate that the benefits were produced specifically by SIT. As Reilly et al. (1984) demonstrated, simple tabletop activities actually appeared to result in benefits superior to SIT in their study. Green (1996a) pointed out that although children may find SIT activities enjoyable,