

# SEPARATING FACT FROM FICTION IN THE ETIOLOGY AND TREATMENT OF AUTISM

## A Scientific Review of the Evidence

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### Abstract

*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.”<sup>1</sup> 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 science, 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 Orthogenic 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 candida 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 co-occur 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.<sup>2</sup> 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, Mulick, & 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 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 in the 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, this does not provide evidence of any significant, long-lasting benefits in the child's behavior or in any underlying neurological deficits. Furthermore, applying brushes of increasing firmness to the arms of autistic children, a common SIT activity, may help to desensitize them to certain tactile stimuli, but such benefits are most parsimoniously explained by well-known behavioral principles (e.g., habituation) rather than anything specific to SIT (Seigel, 1996). In conclusion, the general null effects for SIT relative to control conditions in treating other developmental disabilities, combined with the results of the Reilly et al. (1984) study with autistic children, suggest little benefit of SIT for autism.

## ***Psychotherapies***

Various forms of psychotherapy have been applied to autism, although there is a dearth of research on their effects. The American Academy of Child and Adolescent Psychiatry (AACAP) recently issued a statement of practice parameters for the assessment and treatment of autism and related developmental disorders. The AACAP work group concluded that "it now appears that the usefulness of psychotherapy in autism is very limited" (AACAP, 1999). Nevertheless, various forms of psychotherapy continue to be used with autism. We briefly discuss three of the currently most popular psychotherapies: psychoanalytic psychotherapy, holding therapy, and options therapy.

### ***Psychoanalysis***

As discussed earlier, psychoanalytic theories have long been applied to the etiology of autism despite considerable evidence that many of the basic tenets of these theories are inaccurate; nonetheless, psychoanalytic conceptualization and treatment of autism continues (Beratis, 1994; Bromfield, 2000). Far from being innocuous, psychoanalytic treatments for autism can be quite harmful. The focus on parental (and especially maternal) rejection in the etiology and treatment of autism can lead to a misplaced blame

and a deep sense of guilt in parents. The highly unstructured nature of many psychoanalytic treatments, including granting autistic individuals wide latitude to pursue preferred activities in treatment and the lack of focus on contingencies between behaviors and their consequences, can lead to a worsening of problems (Smith, 1996).

### *Holding Therapy*

Holding therapy has been promoted for numerous childhood problems, including autism (Welch, 1988). Proponents of holding therapy theorize that autism results from a lack of appropriate attachment of child to mother. This deficit in mother-child bonding presumably causes the child to withdraw inward, thereby resulting in social and communicative deficits. It therefore follows that if the mother provides intense physical contact with the child, the previously deficient bond can be reestablished and the “normal” child can emerge. As is evident from this discussion, holding therapy is largely based on psychoanalytic theories of autism, and no researchers have examined its efficacy.

### *Options Therapy*

Options therapy grew out of the book *Son Rise* (Kaufman, 1976), written by parents of an autistic child. The parents reported that they spent many hours every day mirroring the actions of their autistic child without placing demands on him. They theorized that they could enter the world of their son and in turn gradually draw him out. Following the reported success of this treatment with their son, the couple began charging fees to teach this method in workshops. Questions have been raised as to whether the boy was actually autistic (Siegal, 1996). We could locate no published studies investigating the use of options therapy for autism.

### ***Biological Treatments***

Several factors have resulted in the increased popularity of biologically oriented treatments for autism. These include the increased consensus that autism is fundamentally a neurological condition, the increased popularity of psychotropic medications in psychiatry, and the increased popularity of homeopathic, herbal, vitamin, and other “alternative medicine” interventions. Several such treatments have been widely promoted as producing extraordinary benefits for autistic individuals, despite the absence of supportive data, or in some cases even in the face of disconfirming data.

### *Secretin*

Secretin is a hormone involved in the control of digestion that stimulates the secretion of pancreatic juices. It is used in a single dose to help diagnose such gastrointestinal problems as pancreatic disease or ulcers, and it is not approved by the Food and Drug Administration for other uses. Nevertheless, the use of secretin in the treatment of autism gained significant attention following a report in 1998 of a child who appeared to show significant improvement following a single dose (Horvath et al., 1998). Parents of

thousands of autistic children began requesting and receiving injections of secretin for their children based solely on this single case.

In 1999, a study published in the *New England Journal of Medicine* reported the effects of a single dose of secretin on 56 children with autistic-spectrum disorders. The researchers found that a single dose of secretin had no effect on standard behavioral measures when compared with placebo (Sandler et al., 1999). Several other studies have since found similar results. For example, a study recently completed by researchers at the University of California, San Francisco found no effects of secretin on standard measures of expressive or receptive language skills in 20 autistic children (see [www.ucsf.edu/pressrel/2000/05/051401.html](http://www.ucsf.edu/pressrel/2000/05/051401.html) for a description of the study). Similarly, Chez et al. (2000) recently published a two-part study that found no clinically significant differences between secretin and placebo. Some parents reported improvements in their children's functioning following the initial open-label trial phase of the study after receiving an injection of secretin. However, in the second part of the study that was a double-blind trial, children given secretin did not show clinically meaningful improvements compared with those given placebo injections. Chez and Buchanan (2000) concluded that they "cannot rationalize the use of secretin at this point as a 'treatment' modality" (p. 97). Two additional studies likewise found no differences between secretin and placebo in autism (Dunn-Geier et al., 2000; Owley et al., 1999).

Despite these results, interest in secretin in the treatment of autism continues. In fact, in the face of disconfirming research, an influential psychologist and autism advocate, writing on the Internet site of the Autism Research Institute, described secretin as "the most promising treatment yet discovered for the treatment of autism" (Rimland, 1999). Furthermore, likely due to the large consumer demand for secretin for autism, the biopharmaceutical company Repligen secured exclusive rights to a series of patent applications that cover the use of secretin for autism (New update, 1999).

### *Gluten- and Casein-Free diets*

Gluten is a mixture of proteins found in grain products such as wheat bread. Casein is a protein found in milk. Anecdotal reports have abounded that some persons with autism demonstrate increased negative behaviors following the consumption of milk, wheat bread, or similar products. There is some evidence that eliminating these proteins from the diet of some autistic individuals can lead to improvements in behavior (Kvinsberg, Reichelt, Nodland, & Hoiem, 1996; Whitely, Rodgers, Savery, & Shattock, 1999). Due to methodological weaknesses, however, these studies cannot rule out alternative explanations for any observed improvements following gluten- and casein-free diets. The vast majority of the evidence for the benefits of these diets derives from anecdotal reports or case studies (e.g., Adams & Conn, 1997). More rigorous research is needed before the inclusion of these diets as part of a comprehensive treatment plan can be recommended.

### *Vitamin B6 and Magnesium*

Smith (1996) reported that there have been at least 15 studies demonstrating that vitamin B6 with magnesium can be somewhat helpful for children with autism. However, the reports are mixed, with some studies showing no positive effects of high doses of pyroxidine and magnesium (HDPM) (Tolbert, Haigler, Waits, & Dennis, 1993) or no difference between HDPM and placebo (Findling et al., 1997). Critics have argued that a major methodological weakness in most of the studies is that they rely on parent and staff reports instead of assessments from independent observers (Smith, 1996). Also, there are some questions regarding the safety of megadoses of these substances. One potential risk is that high doses of B6 can cause nerve damage and high doses of magnesium can cause reduced heart rate and weakened reflexes (Deutsch & Morrill, 1993). More research is needed to evaluate the safety and effectiveness of long-term use of B6 and magnesium before it can be considered as an efficacious treatment for autism.

### *Dimethylglycine*

Dimethylglycine (DMG) is an antioxidant that can be purchased over the counter as a dietary supplement. In addition to its purported usefulness in increasing energy and enhancing the immune system, DMG is often marketed as a treatment for autism. Some professionals claim that DMG increases eye contact and speech and decreases frustration levels among individuals with autism (Rimland, 1996). In response to the proliferation of anecdotal reports for the effectiveness of DMG, Bolman and Richmond (1999) conducted a double-blind, placebo-controlled, crossover pilot study of DMG in 8 males with autism. Similar to the results of the secretin studies, this study found no significant differences between DMG and placebo. DMG's proponents are undeterred, however, claiming that controlled studies are not needed to demonstrate DMG's effectiveness for autism (Rimland, 1996).

### *Summary of Questionable Treatments*

A wide variety of treatments for autism abound, and families are often persuaded to try methods that are highly unorthodox and scientifically suspect. The observation that individuals with autism sometimes exhibit sensory and motor abnormalities has resulted in the promotion of treatments that claim either to unlock the hidden communicator trapped by the disorder (e.g., FC) or to correct the underlying neurological deficits that are thought responsible for the impairments (e.g., sensory and auditory integration therapies). Others, relying on scientifically untenable theories of the etiology of autism such as the causal role of dysfunctional infant attachment, seek to repair these relationships through intensive psychotherapies (e.g., holding therapy and psychoanalysis). Among the currently most popular treatments are biologically based interventions including various diets, vitamins, or supplements (e.g., secretin). Even though these intervention approaches are extremely heterogeneous in theory and approach, they all share the characteristic of possessing little or no scientific evidence of effectiveness. What is even more distressing is that some of these treatments continue to be promoted even after controlled studies have clearly demonstrated that they are ineffective.

## **PROMISING TREATMENTS FOR AUTISM: REVIEWING THE EVIDENCE AND REINING IN CLAIMS**

The interventions reviewed thus far give little reason for hope in the treatment of autism. Fortunately, the situation is not so bleak. Several promising programs have been developed. Although some research has been conducted on these programs, none has been sufficiently evaluated using experimental research designs. In effect, no treatment currently meets the criteria established by the American Psychological Association's Committee on Science and Practice as an empirically supported treatment for autism (Gresham, Beebe-Frankenberger, & MacMillan, 1999; Rogers, 1998). Nevertheless, the intervention programs reviewed in the following section are based on sound theories, are supported by at least some controlled research, and clearly warrant further investigation.

### ***Applied Behavior Analysis***

Among the currently most popular interventions for autism are programs based on applied behavior analysis (ABA), an approach to behavior modification rooted in the experimental analysis of behavior, in which operant conditioning and other learning principles are used to change problematic behavior (Cooper, Heron & Heward, 1989). Several intervention programs for autism based on ABA methods have been developed. Rogers (1998) noted that many studies of behavioral interventions for autism have focused on a single discrete symptom, and that such interventions have often been shown to be quite effective for such limited targets. In contrast to the single-symptom approach, some programs have been designed to target the core deficits of autism and thereby improve the overall functioning of autistic individuals. By far the most popular of these programs are modeled after the Young Autism Project (YAP) developed at the University of California at Los Angeles by O. Ivar Lovaas and colleagues. Initiated in 1970, the YAP aims to improve the functioning of young children with autism through the use of an intensive, highly structured behavioral program delivered one-on-one by specially trained personnel. The program is designed to be implemented full-time during most of the child's waking hours, and family involvement is deemed to be critical. Treatment is initially delivered in the client's home, with eventual progression to community and school settings. The program is often referred to as "discrete trial training," reflecting the fact that each specific intervention utilizes a discrete stimulus-response-consequence sequence. For example, a child might be presented with three blocks of different colors, and given the verbal stimulus "touch red." If the child touches the red block, a reward is provided (e.g., a small snack, verbal praise). Lovaas (1981) described the program in a treatment manual designed for parents and professionals.

The YAP was evaluated in a widely cited study by Lovaas (1987), with long-term follow-up data reported by McEachlin, Smith, and Lovaas (1993). Lovaas (1987) treated 19 young children with the ABA program described above for 40 or more hours per week for at least 2 years. Two control conditions were employed, one in which 19 children received 10 hours or less per week of the ABA program (minimal treatment condition), and another in which 21 children received unspecified community interventions but no ABA. Outcome measures were IQ and educational placement.

Lovaas (1987) reported dramatic results: After at least 2 years of intervention, almost half (47%) of the experimental group was found to have IQ scores in the normal range, and were reported to be functioning in typical first grade classrooms without special support services. Lovaas described these children as having “recovered” from autism. Only one child from either of the two control groups demonstrated similar gains. In addition, there were large differences in IQ scores between the experimental group and the two control groups. McEachlin et al. (1993) followed up participants from the experimental and minimal ABA treatment conditions several years later. The difference in IQ scores between the two groups was maintained. Of the 9 children with the best outcomes from the original report, 8 continued to function in regular education classrooms.

Not surprisingly, a great deal of enthusiasm was generated by these reports, and demand for ABA programs modeled after the YAP has grown rapidly since their publication. Unlike other treatment or educational programs, the YAP offered not only the possibility of significant improvement in functioning, but also suggested that a substantial number of autistic youngsters could achieve completely normal functioning. Several commentators, however, raised serious concerns about the conclusions reached by Lovaas (1987) and McEachlin et al. (1993). Schopler, Short, and Mesibov (1989) noted that the outcome measures employed, IQ and school placement, might not reflect true overall functional changes. Increases in IQ scores, for example, could reflect increased compliance with testing rather than true changes in intellectual abilities, and school mainstreaming may be more a function of parental and therapist advocacy and changing school policies than increased educational functioning per se. In addition, Schopler et al. argued that the participants in the YAP study appeared to be relatively high-functioning individuals with good prognosis, and were unrepresentative of the larger population of autistic children. Most importantly, they pointed out that the study design was not a true experiment, as subjects were not randomly assigned to the experimental and control groups. They suggested that the procedures for assigning subjects to groups likely resulted in important differences between the experimental and control conditions that may have contributed to the observed outcome differences. Schopler et al. (1989) concluded that that “it is not possible to determine the effects of this intervention” from this study (p. 164).

Others subsequently raised similar criticisms. Gresham and MacMillan (1997, 1998) expanded on the threats to both internal and external validity raised by Schopler et al. (1989) and called for “healthy skepticism” in evaluating the claims of the YAP studies. Mesibov (1993) expressed concerns about pretreatment differences between the experimental and control groups, and about the many domains of functioning in which deficits commonly associated with autism (e.g., social interactions and conceptual abilities) that were not assessed. Mundy (1993) raised similar concerns, noting that many high-functioning autistic individuals achieve IQ levels in the normal range, thereby raising questions about the use of IQ scores to measure “recovery” from autism.

Although they uniformly take exception with the claims of “recovery” from autism proffered by Lovaas and colleagues, even these critics concede that the YAP study yielded promising results that merit further investigation. Although several studies of similar ABA interventions have now been published, two points about these studies are

noteworthy. First, each is methodologically even weaker than the original YAP study. Second, the results of these studies, although generally promising, fall significantly short of those obtained by Lovaas (1987) and McEachlin et al. (1993). Birnbrauer and Leach (1993) reported on 9 children who received 19 hours per week of a one-on-one ABA program for 2 years, and 5 control children who received no ABA. Four of the 9 children in the experimental group made significant gains in IQ, relative to 1 of the 5 control children, although none of the participants achieved completely normal functioning. Sheinkopf and Siegel (1998) conducted a retrospective study of 11 children who received between 12 and 43 hours per week of home-based ABA programs for between 7 and 24 months, relative to a matched control group of children who received unspecified school-based treatment. Data were obtained through record reviews of an existing database. Relative to the control group, children in the experimental group achieved higher gains in IQ, although few differences emerged between the groups in autistic symptoms. Finally, in an uncontrolled, pre-post design study, Anderson, Avery, DiPietro, Edwards, and Christian (1987) reported on 14 children who received between 15 and 25 hours per week of home-based ABA for 1 year. Modest gains were reported in mental age scores and communication skills for most children, although those with the lowest baseline functioning made essentially no progress. In addition, no children were able to be integrated into regular educational settings.

All of these studies involved ABA programs modeled on Lovaas's YAP, in which services were delivered one-on-one in the child's home, although each study differed from the original YAP study in several respects (e.g., the number of hours per week of intervention, the duration of the program, the nature and training of the therapists). Two additional studies evaluated similar ABA interventions, in which services were delivered in school- or center-based programs. Fenske, Zalenski, Krantz, and McClannahan (1985) compared 9 children who began receiving an ABA program through the Princeton Child Development Institute prior to the age of 60 months, relative to 9 who enrolled after the age of 60 months. After at least 2 years of treatment, 4 of the 9 children in the younger group were enrolled in regular school classes, relative to 1 of the 9 children from the older group. No data were provided on autistic symptoms or functioning level. Harris and colleagues reported pre-post data on children treated with an ABA program through the Douglas Developmental Center of Rutgers University. Harris, Handleman, Gordon, Kristoff, and Fuentes (1991) reported average IQ gains of approximately 19 points after 10 to 11 months of intervention. It should be noted that this sample of children was relatively high functioning, with an average pretreatment IQ of 67.5 and with symptoms rated as "mild to moderate." Nevertheless, despite the observed gains in IQ, all children were described as having significant impairments after treatment.

Taken together, the literature on ABA programs for autism clearly suggest that such interventions are promising. Methodological weaknesses of the existing studies, however, severely limit the conclusions that can be drawn about their efficacy. Of particular note is the fact that no study to date has utilized a true experimental design, in which subjects were randomly assigned to treatment conditions. This fact limits the inferences that can be drawn about the effects of the programs studied. Moreover, these concerns are compounded by pretreatment differences between experimental and control conditions in

each of the studies reviewed. Other methodological concerns include questions about the representativeness of the samples of autistic children, unknown fidelity to treatment procedures, limited outcome data for most studies, and problems inherent in relying on IQ scores and school placement as primary measures of autistic symptoms and functioning.

So what are we to make of the claims that ABA programs, and those modeled after the YAP in particular, can result in “recovery” from autism? After more than 30 years since its initiation and 14 years since the first published outcome report, no study has replicated the results of the original YAP study and several critics have challenged its conclusions. Subsequent research has yielded more modest gains in functioning, casting further doubt on the claims that autistic youngsters can be “cured” through ABA programs. Nevertheless, these caveats have not tempered the enthusiasm of some proponents of ABA programs. Consider, for example, the following quotes from leading advocates of ABA intervention programs for autism:

Several studies have now shown that one treatment approach—early, intensive instruction using the methods of Applied Behavior Analysis—can result in dramatic improvements for children with autism: successful integration in regular schools for many, completely normal functioning for some. . . . No other treatment for autism offers comparable evidence of effectiveness. (Green, 1996b, p. 29; emphasis in original)

There is little doubt that early intervention based on the principles and practices of Applied Behavior Analysis can produce large, comprehensive, lasting, and meaningful improvements in many important domains for a large proportion of children with autism. For some, those improvements can amount to achievement of completely normal intellectual, social, academic, communicative, and adaptive functioning. (Green, 1996b, p. 38)

Furthermore, we also now know that applying effective interventions when children are very young (e.g., under the age of 3–4 years) has the potential for achieving substantial and widespread gains and even normal functioning in a certain number of these youngsters. (Schreibman, 2000, p. 374)

During the past 15 years research has begun to demonstrate that significant proportions of children with autism or PDD who participate in early intensive intervention based on the principles of applied behavior analysis (ABA) achieve normal or near-normal functioning. . . . (Jacobson, Mulick, & Green, 1998, p. 204)

It is difficult to justify such assertions in light of the extant scientific literature on ABA programs for autism. Ironically, many of these same authors have been highly critical of the exaggerated claims made for nonbehavioral interventions. Clearly, ABA programs do not possess most of the features of pseudoscience that typify many of the highly dubious treatments for autism. ABA programs are based on well-established theories of learning and emphasize the value of scientific methods in evaluating treatment effects.

Nevertheless, given the current state of the science, claims of “cure” and “recovery” from autism produced by ABA are misleading and irresponsible.

### *Other Comprehensive Behavioral Programs*

Although ABA programs—the YAP in particular—are the best-known behavioral interventions for autism, other programs have been developed that draw to varying degrees on behavioral learning principles. One of the most significant ways in which these programs differ from the ABA programs described earlier is that they make no claims of “curing” autism. Rather, they strive to ameliorate the functioning of autistic individuals by utilizing a variety of educational and therapeutic strategies. Few studies have been conducted on these programs, and those that have utilize only pre-post research designs, thereby limiting the conclusions that can be drawn.

#### *LEAP*

Hoyson, Jamieson, and Strain (1984) described the effects of a program known as Learning Experiences: An Alternative Program for Preschoolers and Parents (LEAP). The LEAP program is composed of an integrated preschool and a behavior-management skills training program for parents. The preschool program, which was one of the first to integrate normally developing children with those with autism, blends normal preschool curricula with activities designed specifically for children with autism. Peer modeling is encouraged in an effort to develop play and social skills. The parental skills-training component aims to teach parents effective behavior-management and educational skills in natural contexts (i.e., home and community). In a pre-post study, Hoyson et al. (1984) reported accelerated developmental rates in 6 “autistic-like” children over the course of their participation in the LEAP program. Strain, Kohler, and Goldstein (1996) reported that 24 out of 51 children were attending regular education classes, although no information was provided regarding functioning level or special school supports. Although certain aspects of the LEAP program appear promising, the paucity of the available research, and especially the absence of controlled research, preclude judgments about its usefulness.

#### *Denver Health Sciences Program*

Developed by Sally Rogers and colleagues at the University of Colorado School of Medicine, the Denver Health Sciences Program is a developmentally oriented preschool program designed not only for children with autism-spectrum disorders, but varied other behavioral problems. Several pre-post studies have reported that autistic children participating in the program demonstrated accelerated developmental rates in several domains, including language, play skills, and social interactions with parents (Rogers & DiLalla, 1991; Rogers, Herbison, Lewis, Pantone, & Reis, 1986; Rogers & Lewis, 1989; Rogers, Lewis, & Reis, 1987). Once again, the lack of controlled research makes it impossible to draw firm conclusions about the effectiveness of this program.

#### *Project TEACCH*

The program for the Treatment and Education of Autistic and Related Communication Handicapped Children (TEACCH) is a university-based project founded by Eric Schopler at the University of North Carolina at Chapel Hill (Schopler & Reichler, 1971). TEACCH programs have become among the more widely used intervention programs for autism. Project TEACCH incorporates behavioral principles in treating children with autism, but differs from ABA in several fundamental ways. Most significantly, TEACCH focuses on maximizing the skills of children with autism while drawing on their relative strengths, rather than attempting “recovery” from the disorder. The program is designed around providing structured settings in which children with autism can develop their skills. Teachers establish individual workstations where each child can practice various tasks, for example, such visual-motor activities as sorting objects by color. Visual cues are often provided in an effort to compensate for the deficits in auditory processing often characteristic of autism. Like the YAP, LEAP, and Denver programs, TEACCH emphasizes a collaborative effort between treatment staff and parents. For example, parents are encouraged to establish routines and cues in the home similar to those provided in the classroom environment (Gresham, Beebe-Frankenberger, & MacMillan, 1999).

Only two treatment outcome studies to date have investigated the effectiveness of project TEACCH. Schopler, Mesibov, and Baker (1982) collected questionnaire data from 348 families whose children were currently or previously enrolled in the program. Individuals with autism who participated ranged in age from 2 to 26, and ranged cognitively from severe mental retardation to normal intellectual functioning. The majority of respondents indicated that the program was helpful. Also, the institutionalization rate of participants was 7%, as compared with the rates of 39% to 75% reported for individuals with autism in the general population based on data from the 1960s. Nevertheless, this study is marked by many serious methodological weaknesses. These include a highly heterogeneous sample (not all participants had autism), the absence of a meaningful control condition, and the lack of standardized and independent assessment measures. In addition, Schopler and colleagues’ comparison of the institutionalization rate in their study with 1960s data is probably misleading. Changes in government policy during the 1960s and 1970s led to decreased institutionalization rates in general (Smith, 1996).

More recently, Ozonoff and Cathcart (1998) tested the effectiveness of TEACCH home-based instruction for children with autism. Parents were taught interventions for preschool children with autism focusing on the areas of cognitive, academic, and prevocational skills related to school success. The treatment group was composed of 11 preschool children with autism who received 4 months of home programming. The treatment group was assessed before and after treatment with the Psychoeducational Profile–Revised (Schopler, Reichler, Bashford, Lansing, & Marcus, 1990), and results were compared with those from a matched comparison group of children not in the TEACCH program who were similarly assessed. Results showed that the preschool children receiving TEACCH-based parent instruction improved significantly more in the areas of imitation, fine-motor, gross-motor, and nonverbal conceptual skills. Furthermore, the treatment group showed an average developmental gain of 9.6 months after the 4-month intervention. Although this study provides some support for the TEACCH

program, the conclusions are tempered by methodological limitations, including the lack of a randomized control condition and the absence of treatment fidelity ratings.

### ***Summary of Behavioral Intervention Programs***

Several programs utilizing various behavioral and developmental intervention strategies have been shown to yield promising results in the treatment of children with autism. Among the most promising are programs based on the intensive, one-on-one application of applied behavior analysis (ABA). Some proponents of ABA have made sweeping claims about the ability of such programs to “cure” autism that are not supported by the available literature. Other behaviorally based programs (e.g., LEAP, Denver Health Sciences Program, TEACCH) have been less prone to exaggerated claims. However, the available research on these programs is more akin to program evaluations than to traditional studies of treatment efficacy or effectiveness. For example, no studies have employed experimental designs, and none has used objective measures of the full range of symptoms and functional impairments associated with autism. Component analysis studies have not evaluated the specific mechanisms responsible for the programs’ effects, and no research has compared the relative effectiveness of various behavioral programs.

Dawson and Osterling (1997) identified six features that are common to most comprehensive early-intervention programs for autism. They suggested that these “tried-and-true” features, rather than the specific methods emphasized by each program, may be responsible for the observed effects of early-intervention programs. These common features include (a) curriculum content emphasizing selective attention, imitation, language, toy play, and social skills; (b) highly supportive teaching environments with explicit attention to generalization of gains; (c) an emphasis on predictability and routine; (d) a functional approach to problem behaviors; (e) a focus on transition from the preschool classroom to kindergarten, first grade, or other appropriate placements; and (f) parental involvement in treatment. Several of these features were incorporated into the treatment recommendations for autism made by the American Academy of Child and Adolescent Psychiatry (AACAP, 1999). Further research is clearly indicated to assess the effects of each of these components, and to evaluate potential additive effects of the specific elements of various early intervention programs.

### ***Pharmacotherapy***

A detailed review of the psychopharmacologic treatment of autism is beyond the scope of this paper, and several excellent recent reviews are available (AACAP, 1999; Aman & Langworthy, 2000; Campbell, Schopler, Cueva, & Hallin, 1996; Gillberg, 1996; King, 2000). Although not curative, in open-label case reports several medications appeared to improve various symptoms associated with autism, thereby increasing individuals’ ability to benefit from educational and behavioral interventions. With a few noteworthy exceptions, few studies have utilized double-blind, placebo-controlled designs, especially with autistic children.

The most extensively studied agents are the dopamine antagonists, especially haloperidol (Haldol). Several well-controlled studies have shown haloperidol to be superior to placebo for a number of symptoms, including withdrawal, stereotypies, and hyperactivity (Anderson et al., 1984; Campbell et al., 1996; Locascio et al., 1991), although drug-related dyskinesias appear to be relatively common following long-term administration (Campbell et al., 1997). There is growing interest in the atypical neuroleptics, risperidone (Risperdal) in particular. In a double-blind, placebo-controlled trial with autistic adults, McDougle et al. (1998) found risperidone to be superior to placebo on several measures, and to be well tolerated.

Several studies suggest the usefulness of various selective serotonin reuptake inhibitors (SSRIs), including fluvoxamine (Luvox; McDougle et al., 1996), fluoxetine (Prozac; Cook et al., 1992; DeLong, Teague, & Kamran, 1998; Fatemi, Realmuto, Khan, & Thuras, 1998), and clomipramine (Anafranil; Gordon et al., 1992; 1993). However, SSRIs are often associated with intolerable adverse events. For example, recent open-label studies reveal significant rates of adverse side effects of clomipramine, including seizures, weight gain, constipation, and sedation (e.g., Brodtkin et al., 1997). Moreover, there is a growing consensus that children appear to respond less well to SSRIs than do adolescents and adults (Brasic et al., 1994; McDougle, Kresch, & Posey, 2000; Sanchez et al., 1996). Tricyclic antidepressants are less frequently used relative to SSRIs, given the possibility of cardiovascular side effects and lowering of seizure threshold.

Although little research has examined anxiolytic agents in autism, what little research has been conducted suggests that they are of little benefit. In fact, Marrosu et al. (1987) found increases in hyperactivity and aggression following treatment with the benzodiazepine diazepam (Valium). More promising results have been obtained in open-label studies of buspirone (Buspar; McCormick, 1997; Realmuto, August, & Garfinkel, 1989; Ratey, Mikkelsen, & Chmielinski, 1989).

## **THE HARM IN PROMOTING UNPROVEN TREATMENTS**

As the previous review illustrates, even the most promising treatments for autism are typically far from ideally effective, leaving the autistic individual with substantial impairments. It is therefore natural for parents, educators, and even mental health professionals to ask what the harm is in trying an unproven treatment. This is a difficult question for which there is no easy answer. On the one hand, we are not suggesting that parents and professionals not be allowed to explore a range of treatment options. What we are suggesting is that they do so with as much information as possible, and armed with an attitude of healthy skepticism. For several reasons, such skepticism is particularly important in considering treatments for autism.

First, proponents of many treatments, both novel and established, often make impressive claims that are simply not supported by controlled research. Moreover, many mental health and educational professionals who work with autistic individuals have been reluctant to speak out against pseudoscientific theories and practices. This silence places the burden directly on consumers to become educated about the empirical status of

various treatment options. Unless they make efforts to become informed about the research literature themselves, consumers can be easily misled and given false hope.

Second, no treatment is without cost. Aside from the obvious financial burden, there are always other costs to consider when contemplating a new treatment. In particular, time and resources spent on an unproven therapy are time and resources that could have been spent on an intervention with a greater likelihood of success (what economists term “opportunity cost”). This point is especially critical with respect to early-intervention programs, as a growing literature suggests the importance of early intervention with specialized behavioral and educational programs (Fenske, Zalski, Krantz, & McClannahan, 1985). The issue of cost is complicated by the tendency, in the absence of appropriate control conditions, to misattribute any positive changes that may be observed to an intervention and then expend even more resources on that intervention when the improvement may not be due to the treatment. Alternatively, repeated experience with treatments that are promoted with much fanfare but turn out to be ineffective might cause family members of autistic individuals to become unnecessarily cynical about even legitimate interventions.

Finally and perhaps most importantly, one must always be aware of the potential for harm. There are numerous examples in the history of pharmacotherapy of substances that were initially believed to be therapeutically useful and devoid of harmful side effects that turned out to be quite harmful (e.g., combined fenfluramine and dexfenfluramine, thalidomide). The effects of long-term use of substances like secretin and DMG have not been investigated and are therefore unknown. The risk of harm is not limited to pharmacologic interventions, however. Consider, for example, the case of FC. The cases of family members being convicted of abuse and sent to prison based on alleged communications provides a sobering example of the harm that can arise from unvalidated interventions. Despite the wealth of scientific data demonstrating that the “facilitator” is the source of such messages, some courts still permit communications derived via FC to be used as evidence (Gorman, 1999).

## **CAVEAT EMPTOR**

Autistic-spectrum disorders are associated with serious psychiatric symptoms, often profound developmental delays, and impairments in many areas of functioning. Although the etiology of autism remains largely unknown and there is currently no cure for the disorder, some promising interventions appear to be useful in helping persons with autism lead more productive lives. The nature of autism renders family members and other stakeholders vulnerable to highly dubious etiological theories and intervention strategies, many of which can be characterized as pseudoscientific. We believe that parents and professionals alike would do well to adopt the position of caveat emptor, or “let the buyer beware,” when considering novel treatments for autism. If something sounds too good to be true, it often is.

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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.

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).

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## REFERENCES

Adams, L., & Conn, S. (1997). Nutrition and its relationship to autism. *Focus on Autism & Other Developmental Disabilities, 12*, 53–58.

Allen, J., DeMeyer, M. K., Norton, J. A., Pontus, W., & Yang, E. (1971). Intellectuality in parents of psychotic, subnormal, and normal children. *Journal of Autism & Childhood Schizophrenia, 3*, 311–326.

Aman, M. G., & Langworthy, K. S. (2000). Pharmacotherapy for hyperactivity in children with autism and other pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 30*, 451–459.

American Academy of Child and Adolescent Psychiatry (1999). Practice parameters for the assessment and treatment of children, adolescents, and adults with autism and other pervasive developmental disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*, 32–54.

American Academy of Pediatrics (1998). Auditory integration training and facilitated communication for autism. American Academy of Pediatrics. Committee on children with disabilities. *Pediatrics, 102*, 431–433.

American Psychiatric Association (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.

Anderson, L. T., Campbell, M., Grega, D. M., Perry, R., Small, A. M., & Green, W. H. (1984). Haloperidol in the treatment of infantile autism: Effects on learning and behavioral symptoms. *American Journal of Psychiatry, 141*, 1195–1202.

- Anderson, S. R., Avery, D. L., DiPietro, E. K., Edwards, G. L., & Christian, W. P. (1987). Intensive home-based early intervention with autistic children. *Education and Treatment of Children, 10*, 352–366.
- Ayres, A. J. (1979). *Sensory integration and the child*. Los Angeles, CA: Western Psychological Services.
- Berand, G. (1993). *Hearing equals behavior*. New Canaan, CT: Keats.
- Beratis, S. (1994). A psychodynamic model for understanding pervasive developmental disorders. *European Journal of Psychiatry, 8*, 209–214.
- Bettelheim, B. (1967). *The empty fortress*. New York: Free Press.
- Bettison, S. (1996). The long-term effects of auditory training on children with autism. *Journal of Autism & Developmental Disorders, 26*, 361–374.
- Birnbrauer, J., & Leach, D. (1993). The Murdoch early intervention program after 2 years. *Behavior Change, 10*, 63–74.
- Bolman, W. M., & Richmond, J. A. (1999). A double-blind, placebo-controlled, crossover pilot trial of low dose dimethylglycine in patients with autistic disorder. *Journal of Autism and Developmental Disorders, 29*, 191–194.
- Brasic, J. R., Barnett, J. Y., Kaplan, D., Sheitman, B. B., Aisemberg, P., Lafargue, R. T., Kowalik, S., Clark, A., Tsaltas, M. O., & Young, J. G. (1994). Clomipramine ameliorates adventitious movements and compulsions in prepubertal boys with autistic disorder and severe mental retardation. *Neurology, 44*, 1309–1312.
- Brodkin, E. S., McDougle, C. J., Naylor, S. T., Cohen, D. J., & Price, L. H. (1997). Clomipramine in adults with pervasive developmental disorders: A prospective open-label investigation. *Journal of Child and Adolescent Psychopharmacology, 7*, 109–121.
- Bromfield, R. (2000). It's the tortoise's race: Long-term psychodynamic psychotherapy with a high-functioning autistic adolescent. *Psychoanalytic Inquiry, 20*, 732–745.
- Bryson, S. (1997). Epidemiology of autism: Overview and issues outstanding. In D. J. Cohen & F. R. Volkmar (Eds.), *Handbook of autism and pervasive developmental disorders* (2nd ed., pp. 41–46). New York: Wiley.
- Bryson, S. E., Clark, B. S., & Smith, I. M. (1988). First report of a Canadian epidemiological study of autistic syndromes. *Journal of Child Psychology and Psychiatry, 29*, 433–445.
- Bunge, M. (1984). What is pseudoscience? *Skeptical Inquirer, 9*, 36–46.

Burgess, C. A., Kirsch, I., Shane, H., Niederauer, K. L., Graham, S. M., & Bacon, A. (1998). Facilitated communication as an ideomotor response. *Psychological Science, 9*, 71–74.

Campbell, M., Armenteros, J. L., Malone, R. P., Adams, P. B., Eisenberg, Z. W., & Overall, J. E. (1997). Neuroleptic-related dyskinesias in autistic children: A prospective, longitudinal study. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 835–843.

Campbell, M., Schopler, E., Cueva, J. E., & Hallin, A. (1996). Treatment of autistic disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 134–143.

Case-Smith, J., & Bryan, T. (1999). The effects of occupational therapy with sensory integration emphasis on preschool-age children with autism. *American Journal of Occupational Therapy, 53*, 489–497.

Chez, M. G., & Buchanan, C. P. (2000). Reply to B. Rimland's "Comments on 'Secretin and autism: A two-part clinical investigation.'" *Journal of Autism & Developmental Disorders, 30*, 97–98.

Chez, M. G., Buchanan, C. P., Bagan, B. T., Hammer, M. S., McCarthy, K. S., Ovrutskaya, I., Nowinski, C. V., & Cohen, Z. S. (2000). Secretin and autism: A two-part clinical investigation. *Journal of Autism & Developmental Disorders, 30*, 87–94.

Cook, E. H., Rowlett, R., Jaselskis, C., & Leventhal, B. L. (1992). Fluoxetine treatment of children and adults with autistic disorder and mental retardation. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*, 739–745.

Cooper, J. O., Heron, T., & Heward, W. (1989). *Applied behavior analysis*. Columbus, OH: Merrill.

Dales, L., Hammer, S. J., & Smith, N. J. (2001). Time trends in autism and in MMR immunization coverage in California. *Journal of the American Medical Association, 285*, 1183–1185.

Darnton, N. (1990, September 10). Beno Brutaheim? *Newsweek, 111*(11), 59–60.

Dawson, G., & Osterling, J. (1997). Early intervention in autism: Effectiveness and common elements of current approaches. In M. Guralnick (Ed.), *The effectiveness of early intervention* (pp. 307–326). Baltimore: Brookes.

Dawson, G., & Watling, R. (2000). Interventions to facilitate auditory, visual, and motor integration in autism: A review of the evidence. *Journal of Autism & Developmental Disorders, 30*, 415–421.

DeLong, G. R., Teague, L. A., & Kamran, M. M. (1998). Effects of fluoxetine treatment in young children with idiopathic autism. *Developmental Medicine and Child Neurology*, *40*, 551–562.

Denson, J. F., Nuthall, G. A., Bushnell, J., & Horn, J. (1989). Effectiveness of a sensory integrative therapy program for children with perceptual-motor deficits. *Journal of Learning Disabilities*, *22*, 221–229.

Deutsch, R. M., & Morrill, J. S. (1993). *Realities of nutrition*. Palo Alto, CA: Bull Publishing.

Dunn-Geier, J., Ho H. H., Auersperg, E., Doyle, D., Eaves, L., Matsuba, C., Orrbine, E., Pham, B., & Whiting, S. (2000). Effect of secretin on children with autism: A randomized controlled trial. *Developmental Medicine & Child Neurology*, *42*, 796–802.

Edelson, S. M. (2001). Disappointed [Letter to the editor]. *Priorities for Health*, *13*(1), 4–6.

Edelson, S. M., Arin, D., Bauman, M., Lukas, S. E., Rudy, J. H., Sholar, M., Rimland, B. (1999). Auditory integration training: A double-blind study of behavioral and electrophysiological effects in people with autism. *Focus on Autism and Other Developmental Disabilities*, *14*, 73–81.

Fatemi, S. H., Realmuto, G. M., Khan, L., & Thuras, P. (1998). Fluoxetine in treatment of adolescent patients with autism: A longitudinal open trial. *Journal of Autism & Developmental Disorders*, *28*, 303–307.

Fenske, E. C., Zalenski, S., Krantz, P. J., & McClannahan, L. E. (1985). Age at intervention and treatment outcome for autistic children in a comprehensive intervention program. *Analysis and Intervention in Developmental Disabilities*, *5*, 49–58.

Findling, R. L., Maxwell, K., Scotese-Wojtala, L., & Huang, J. (1997). High-dose pyridoxine and magnesium administration in children with autistic disorder: An absence of salutary effects in a double-blind, placebo-controlled study. *Journal of Autism & Developmental Disorders*, *27*, 467–478.

Folstein, S. E. (1999). Autism. *International Review of Psychiatry*, *11*, 269–278.

Fombonne, E. (1998). Epidemiology of autism and related conditions. In F. R. Volkmar (Ed.), *Autism and pervasive developmental disorders* (pp. 32–63). New York: Cambridge University Press.

Gardner, M. (2000). The brutality of Dr. Bettelheim. *Skeptical Inquirer*, *24*(6), 12–14.

Gardner, M. (2001). Facilitated communication: A cruel farce. *Skeptical Inquirer*, *25*, 17–19.

Gillberg, C. (1996). The psychopharmacology of autism and related disorders. *Journal of Psychopharmacology*, *10*, 54–63.

Gillberg, C., Johansson, M., Steffenburg, S., & Berlin, O. (1997). Auditory integration training in children with autism: Brief report of an open pilot study. *Autism*, *1*, 97–100.

Gordon, C. T., Rapoport, J. L., Hamburger, S. D., State, R. C., & Mannheim, G. B. (1992). Differential response of seven subjects with autistic disorder to clomipramine and desipramine. *American Journal of Psychiatry*, *149*, 363–366.

Gordon, C. T., State, R. C., Nelson, J. E., Hamburger, S. D., & Rapoport, J. L. (1993). A double-blind comparison of clomipramine, desipramine, and placebo in the treatment of autistic disorder. *Archives of General Psychiatry*, *50*, 441–447.

Gorman, B. J. (1999). Facilitated communication: Rejected in science, accepted in court—a case study and analysis of the use of FC evidence under Frye and Daubert. *Behavioral Sciences and the Law*, *17*, 517–541.

Green, D. (2001). Autism and “voodoo science” treatments. *Priorities for Health*, *13*(1), 27–32, 69.

Green, G. (1994). Facilitated communication: Mental miracle or sleight of hand? *Skeptic*, *2*, 68–76.

Green, G. (1996a). Evaluating claims about treatments for autism. In C. Maurice, G. Green, & S. C. Luce (Eds.), *Behavioral intervention for young children with autism: A manual for parents and professionals* (pp. 15–28). Austin, TX: PRO-ED.

Green, G. (1996b). Early behavioral intervention for autism: What does research tell us? In C. Maurice, G. Green, & S. C. Luce (Eds.), *Behavioral intervention for young children with autism: A manual for parents and professionals* (pp. 29–44). Austin, TX: PRO-ED.

Gresham, F. M., Beebe-Frankenberger, M. E., & MacMillan, D. L. (1999). A selective review of treatments for children with autism: Description and methodological considerations. *School Psychology Review*, *28*, 559–576.

Gresham, F. M., & MacMillan, D. L. (1998). Early intervention project: Can its claims be substantiated and its effects replicated? *Journal of Autism & Developmental Disorders*, *28*, 5–13.

Harris, S. L., Handleman, J. S., Gordon, R., Kristoff, B., & Fuentes, F. (1991). Changes in cognitive and language functioning of preschool children with autism. *Journal of Autism and Developmental Disorders*, *21*, 281–290.

Herbert, J. D., Liliensfeld, S. O., Lohr, J. M., Montgomery, R. W., O’Donohue, W. T., Rosen, R. M., & Tolin, D. F. (2000). Science and pseudoscience in the development of

eye movement desensitization and reprocessing: Implications for clinical psychology. *Clinical Psychology Review*, 20, 945–971.

Herbert, J. D., & Sharp, I. R. (2001). Pseudoscientific treatments for autism. *Priorities for Health*, 13(1), 23–26, 59.

Hoehn, T. P., & Baumesiter, A. A. (1994). A critique of the application of sensory integration therapy to children with learning disabilities. *Journal of Learning Disabilities*, 27, 338–351.

Horvath, K., Stefanatos, G., Sokolski, K. N., Wachtel, R., Nabors, L., & Tildon, J. T. (1998). Improved social and language skills after secretin administration in patients with autistic spectrum disorders. *Journal of the Association for Academic Minority Physicians*, 9, 9–15.

Hoyson, M., Jamieson, B., & Strain, P. S. (1984). Individualized group instruction of normally developing and autistic-like children: The LEAP curriculum model. *Journal of the Division of Early Childhood*, 8, 157–172.

Iwasaki, K., & Holm, M. B. (1989). Sensory treatment for the reduction of stereotypic behaviors in persons with severe multiple disabilities. *Occupational Therapy Journal of Research*, 9, 170–183.

Jacobson, J. W., Mulick, J. A., & Green, G. (1998). Cost-benefit estimates for early intensive behavioral intervention for young children with autism—General model and single state case. *Behavioral Interventions*, 13, 201–226.

Jacobson, J. W., Mulick, J. A., & Schwartz, A. A. (1995). A history of facilitated communication: Science, pseudoscience, and antiscience: Science working group on facilitated communication. *American Psychologist*, 50, 750–765.

Jenkins, J. R., Fewell, R. R., & Harris, S. R. (1984). Comparison of sensory integrative therapy and motor programming. *American Journal of Mental Deficiency*, 88, 221–224.

Kanner, L. (1946). Autistic disturbances of affective contact. *American Journal of Psychiatry*, 103, 242–246.

Kanner, L. (1973). *Childhood psychosis: Initial studies and new insights*. Washington, DC: V. H. Winston & Sons.

Kaufman, B. N. (1976). *Son rise*. New York: Harper & Row.

Kaye, J. A., Melero-Montes, M., & Jick, H. (2001). Mumps, measles, and rubella vaccine and the incidence of autism recorded by general practitioners: A time trend analysis. *British Medical Journal*, 322, 460–463.

King, B. H. (2000). Pharmacological treatment of mood disturbances, aggression, and self-injury in persons with pervasive developmental disorders. *Journal of Autism & Developmental Disorders*, *30*, 439–445.

Kirsch, I., & Lynn, S. J. (1999). Automaticity in clinical psychology. *American Psychologist*, *54*, 504–515.

Kvinsberg, A. M., Reichelt, K. L., Nodland, M., Høien, T. (1996). Autistic syndromes and diet: A follow-up study. *Scandinavian Journal of Educational Research*, *39*, 223–236.

Lilienfeld, S. O. (1998). Pseudoscience in contemporary clinical psychology: What it is and what we can do about it. *The Clinical Psychologist*, *51*, 3–9.

Linderman, T. M., & Stewart, K. B. (1999). Sensory integrative-based occupational therapy and functional outcomes in young children with pervasive developmental disorders: A single-subject study. *American Journal of Occupational Therapy*, *53*, 207–213.

Locascio, J. J., Malone, R. P., Small, A. M., Kafantaris, V., Ernst, M., Lynch, N. S., Overall, J. E., & Campbell, M. (1991). Factors related to haloperidol response and dyskinesias in autistic children. *Psychopharmacology Bulletin*, *27*, 119–126.

Lovaas, O. I. (1981). *Teaching developmentally disabled children: The me book*. Austin, TX: PRO-ED.

Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, *55*, 3–9.

Mahler, M. (1968). *On human symbiosis and the vicissitudes of individuation*. New York: International Universities Press.

Manning, A. (1999, August 16). Vaccine-autism link feared. *USA Today*.

Marrosu, F., Marrosu, G., Rachel, M. G., & Biggio, G. (1987). Paradoxical reactions elicited by diazepam in children with classic autism. *Functional Neurology*, *3*, 355–361.

Mason, S. M., & Iwata, B. A. (1991). Artifactual effects of sensory-integrative therapy on self-injurious behavior. *Journal of Applied Behavior Analysis*, *23*, 361–370.

McCormick, L. H. (1997). Treatment with buspirone in a patient with autism. *Archives of Family Medicine*, *6*, 368–370.

McDougle, C. J., Holmes, J. P., Carlson, D. C., Pelton, G., Cohen, D. J., & Price, L. H. (1998). A double-blind, placebo-controlled study of risperidone in adults with autistic

disorder and other pervasive developmental disorders. *Archives of General Psychiatry*, 55, 633–641.

McDougle, C. J., Kresch, L. E., & Posey, D. J. (2000). Repetitive thoughts and behavior in pervasive developmental disorders: Treatment with serotonin reuptake inhibitors. *Journal of Autism & Developmental Disorders*, 30, 427–435.

McDougle, C. J., Naylor, S. T., Cohen, D. J., Volkmar, F. R., Heninger, G. R., & Price, L. H. (1996). A double-blind, placebo-controlled study of fluvoxamine in adults with autistic disorder. *Archives of General Psychiatry*, 53, 1001–1008.

McEachlin, J., Smith, T., & Lovaas, O. I. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal on Mental Retardation*, 97, 359–372.

Mesibov, G. B. (1993). Treatment outcome is encouraging. *American Journal on Mental Retardation*, 97, 379–380.

Mudford, O. C. (1995). Review of the gentle teaching data. *American Journal on Mental Retardation*, 99, 345–355.

Mudford, O. C., Cross, B. A., Breen, S., Cullen, C., Reeves, D., Gould, J., Douglas, J. (2000). Auditory integration training for children with autism: No behavioral benefits detected. *American Journal on Mental Retardation*, 105, 118–129.

Mundy, P. (1993). Normal versus high-functioning status in children with autism. *American Journal on Mental Retardation*, 97, 381–384.

New update. (1999, August). *Psychopharmacology Update*, 10, 2.

Owley, T., Steele, E., Corsello, C., Risi, S., McKaig, K., Lord, C., Leventhal, B. L., & Cook, E. H. (1999). A double-blind placebo-controlled trial of secretin for the treatment of autistic disorder. *Medscape General Medicine*, 1. Retrieved December 6, 2001, from <http://www.medscape.com/medscape/GeneralMedicine/journal/1999/v01.n10/mgm1006.owle/mgm1006.owle-01.html>.

Ozonoff, S., & Cathcart, K. (1998). Effectiveness of a home program intervention for young children with autism. *Journal of Autism & Developmental Disorders*, 28, 25–32.

Pollak, R. (1997). *Creation of Dr. Bettelheim: A biography of Bruno Bettelheim*. New York: Simon & Schuster.

Ratey, J. J., Mikkelsen, E., Chmielinski, H. E. (1989). Buspirone therapy for maladaptive behaviors and anxiety in developmentally disabled persons. *Journal of Clinical Psychiatry*, 50, 382–384.

Realmuto, G. M., August, G. J., & Garfinkel, B. D. (1989). Clinical effect of buspirone in autistic children. *Journal of Clinical Psychopharmacology*, *9*, 122–125.

Reilly, C., Nelson, D. L., & Bundy, A. C. (1984). Sensorimotor versus fine motor activities in eliciting vocalizations in autistic children. *Occupational Therapy Journal of Research*, *3*, 199–212.

Rimland, B. (1988). Candida-caused autism? *Autism Research Review International Newsletter*. Retrieved December 6, 2001, from <http://www.autism.com/ari/editorials/candida.html>.

Rimland, B. (1996). Dimethylglycine (DMG), a nontoxic metabolite, and autism. *Autism Research Review International*. Retrieved December 6, 2001, from <http://www.autism.com/ari/editorials/dmg1.html>.

Rimland, B. (1999). The use of secretin in autism: Some preliminary answers. *Autism Research Review International Newsletter*. Retrieved December 6, 2001, from <http://www.autism.com/ari/editorials/findings.html>.

Rimland, B. (2000, April 26). Do children's shots invite autism? *Los Angeles Times*. Retrieved from <http://www.latimes.com/archives>.

Rimland, B., & Edelson, S. M. (1994). The effects of auditory integration training on autism. *American Journal of Speech-Language Pathology*, *5*, 16–24.

Rimland, B., & Edelson, S. M. (1995). Auditory integration training in autism: A pilot study. *Journal of Autism & Developmental Disorders*, *25*, 61–70.

Rodier, P. M. (2000). The early origins of autism. *Scientific American*, *282*, 56–63.

Rogers, S. J. (1998). Empirically supported comprehensive treatments for young children with autism. *Journal of Clinical Child Psychology*, *27*, 168–179.

Rogers, S. J., & DiLalla, D. (1991). A comparative study of a developmentally based preschool curriculum on young children with autism and young children with other disorders of behavior and development. *Topics in Early Childhood Special Education*, *11*, 29–48.

Rogers, S. J., Herbison, J., Lewis, H., Pantone, J., & Reis, K. (1986). An approach for enhancing the symbolic, communicative, and interpersonal functioning of young children with autism and severe emotional handicaps. *Journal of the Division of Early Childhood*, *10*, 135–148.

Rogers, S. J., & Lewis, H. (1989). An effective day treatment model for young children with pervasive developmental disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *28*, 207–214.

Rogers, S. J., Lewis, H. C., & Reis, K. (1987). An effective procedure for training early special education teams to implement a model program. *Journal of the Division of Early Childhood, 11*, 180–188.

Roser, K. (1996). A review of psychoanalytic theory and treatment of childhood autism. *Psychoanalytic Review, 83*, 325–341.

Sanchez, L. E., Campbell, M., Small, A. M., Cueva, J. E., Armenteros, J. L., & Adams, P. B. (1996). A pilot study of clomipramine in young autistic children. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 537–544.

Sandler, A. D., Sutton, K. A., DeWeese, J., Girardi, M. A., Sheppard, V., & Bodfish, J. W. (1999). Lack of benefit of a single dose of synthetic human secretin in the treatment of autism and pervasive developmental disorder. *New England Journal of Medicine, 341*, 1801–1806.

Schopler, E., Mesibov, G. B., & Baker, A. (1982). Evaluation of treatment for autistic children and their parents. *Journal of the American Academy of Child Psychiatry, 21*, 262–267.

Schopler, E., & Reichler, R. J. (1971). Parents as cotherapists in the treatment of psychotic children. *Journal of Autism and Childhood Schizophrenia, 1*, 87–102.

Schopler, E., Reichler, R. J., Bashford, A., Lansing, M. D., Marcus, L. M. (1990). *Individualized assessment and treatment for autistic and developmentally disabled children (Vol. 1): Psychoeducational profile revised*. Austin, TX: PRO-ED.

Schopler, E., Short, A., & Mesibov, G. (1989). Relation of behavioral treatment to “normal functioning”: Comments on Lovaas. *Journal of Consulting and Clinical Psychology, 57*, 162–164.

Sheinkopf, S., & Siegel, B. (1998). Home-based behavioral treatment of young children with autism. *Journal of Autism & Developmental Disorders, 28*, 15–23.

Siegel, B. (1996). *The world of the autistic child: Understanding and treating autistic spectrum disorders*. New York: Oxford University Press.

Schreibman, L. (2000). Intensive behavioral/psychoeducational treatments for autism: Research needs and future directions. *Journal of Autism & Developmental Disorders, 30*, 373–378.

Shermer, M. (1997). *Why people believe weird things: Pseudoscience, superstition, and other confusions of our time*. New York: W. H. Freeman.

Smith, T. (1996). Are other treatments effective? In C. Maurice, G. Green, & S. C. Luce (Eds.), *Behavioral intervention for young children with autism: A manual for parents and professionals* (pp. 45–59). Austin, TX: PRO-ED.

Steffenburg, S., & Gillberg, C. (1986). Autism and autistic-like conditions in Swedish rural and urban areas: A population study. *British Journal of Psychiatry*, *149*, 81–87.

Stehli, A. (1991). *The sound of a miracle: A child's triumph over autism*. New York: Doubleday.

Stratton, K., Gable, A., Shetty, P., & McCormick, M. (Eds.) (2001). *Immunization safety review: Measles-mumps-rubella vaccine and autism*. Washington, DC: National Academy Press.

Stromland, K., Nordin, V., Miller, M., Akerstrom, B., & Gillberg, C. (1994). Autism in thalidomide embryopathy: A population study. *Developmental Medicine and Child Neurology*, *36*, 351–356.

Sugiyama, T., & Abe, T. (1989). The prevalence of autism in Nagoya, Japan: A total population study. *Journal of Autism & Developmental Disorders*, *19*, 87–96.

Tolbert, L. C., Haigler, T., Waits, M. M., & Dennis, T. (1993). Brief report: Lack of response in an autistic population to a low dose clinical trial of pyridoxine plus magnesium. *Journal of Autism & Developmental Disorders*, *23*, 193–199.

Trottier, G., Srivastava, L., & Walker, C. D. (1999). Etiology of infantile autism: A review of recent advancements in genetic and neurobiological research. *Journal of Psychiatry & Neuroscience*, *24*, 103–115.

Tustin, F. (1981). *Autistic states in children*. Boston: Routledge.

Volkmar, F. R., Szatmari, P., & Sparrow, S. S. (1993). Sex differences in pervasive developmental disorders. *Journal of Autism & Developmental Disorders*, *23*, 579–591.

Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., Malik, M., Berelowitz, M., Dhillon, A. P., Thomson, M. A., Harvey, P., Valentine, A., Davies, S. E., & Walker-Smith, J. A. (1998). Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *Lancet*, *351*, 637–641.

Welch, M. G. (1988). *Holding time: How to eliminate conflict, temper tantrums, and sibling rivalry and raise happy, loving, successful children*. New York: Simon & Schuster.

Wheeler, D. L., Jacobson, J. W., Paglieri, R. A., & Schwartz, A. A. (1993). An experimental assessment of facilitated communication. *Mental Retardation*, *31*, 49–59.

Whiteley, P., Rodgers, J., Savery, D., & Shattock, P. (1999). A gluten-free diet as an intervention for autism and associated spectrum disorders: Preliminary findings. *Autism*, 3, 45–65.

Zollweg, W., Palm, D., & Vance, V. (1997). The efficacy of auditory integration training: A double blind study. *American Journal of Audiology*, 6, 39–47