Autism and Child Psychopathology Series
Series Editor: Johnny L. Matson

Jonathan Tarbox
Dennis R. Dixon
Peter Sturmey
Johnny L. Matson *Editors*

Handbook of

Early Intervention for Autism Spectrum Disorders

Research, Policy, and Practice

Springer

Recovery and Prevention

21

Doreen Granpeesheh, Jonathan Tarbox
and Angela Persicke

Keywords

Recovery · Prevention · Rapid learning

Recovery and prevention are among the two most sought after achievements in the treatment of any disorder. Recovery from autism continues to be a controversial topic, receiving everything from total acceptance in some circles to complete denial in others. Recovery is talked about widely in the community of families affected by autism spectrum disorders (ASD), as well as amongst practitioners of complementary and alternative medical treatments (CAMs), but little has been written on the topic in peer-reviewed scientific publications. The unfortunate result is that families of individuals with ASD are left primarily with unsubstantiated claims that are propagated on the World Wide Web. In the first half of this chapter, we will review definitions of recovery from autism, elaborate our working definition, review existing scientific evidence on the topic, and discuss directions for future research on recovery.

If the concept of recovery from autism is controversial, the concept of prevention of autism is virtually unheard of. In the second half of the chapter, we will discuss a behavioral approach to the concept of preventing autism and describe some initial clinical impressions. Since research on preventing autism has not even begun, our discussion of prevention will necessarily be primarily conceptual. The chapter will then conclude with overall directions for future research.

Recovery from Autism

Our Definition of Recovery

We have described our definition of recovery in two previous publications (Granpeesheh 2008; Granpeesheh et al. 2009), but we will elaborate here. Essentially, our concept of the behavioral manifestation of autism is that a set of skills has not developed or has developed more slowly and inconsistently than those skills in typically developing children of the same age. Recovery is simply the name for the acceleration of the development of those skills, such that clinically significant impairment no longer exists for the child. Some analogies of other skill deficits may be helpful to illustrate the basic point. An adult who never learned to read is called illiterate. Without effective reading intervention, he will likely remain illiterate for the rest of his life. In other words, illiteracy is a "lifelong disorder." With effective reading intervention, he can learn to read. He is now no longer illiterate, and it would be

A. Persicke (✉) · D. Granpeesheh · J. Tarbox
Center for Autism and Related Disorders, Tarzana, CA, USA
e-mail: a.persicke@centerforautism.com

J. Tarbox et al. (eds.), *Handbook of Early Intervention for Autism Spectrum Disorders,*
Autism and Child Psychopathology Series, DOI 10.1007/978-1-4939-0401-3_21,

plainly absurd to say that he still "has illiteracy" even though he can read. Traumatic brain injury (TBI) is another useful example. Major skill loss is a common effect of TBI, and the remediation of skill loss is the goal of treatment for it. If a person who incurred a TBI receives rehabilitative treatment and subsequently regains all their lost skills, it is entirely common to say the person "recovered from their brain injury." The same should be true for autism. If a child who was once diagnosed with autism receives treatment that remediates all language and social deficits and eliminates any clinically significant problems with repetitive interests, it is no longer logical to say the child "has autism." He has recovered from autism. Of course, autism is far more complicated than illiteracy and has different etiology from TBI, but there is not a single shred of scientific evidence to suggest that the skill deficits that comprise autism cannot be fully remediated, at least in some children.

Measuring Recovery

The seminal paper by Lovaas (1987) was the first study that attempted to document recovery of children with autism. It is also important to note that it was the first controlled outcome study to document large-scale and relatively long-term treatment effects for individuals with autism. Prior to this study, it was still widely believed that autism was not treatable. In this study, children with autism received 40 h per week of one-to-one behavioral intervention, starting before the age of 3.5 and continuing for more than 2 years. At followup, 47% of the children who received intensive intervention no longer suffered from clinically significant impairment related to autism. Unfortunately, the only evidence of this outcome that the study contained was IQ scores in the normal range and success in regular education placement without support. McEachin et al. (1993) followed up with the group of children who had recovered and found that eight of nine retained their gains and continued to function successfully with little to no clinically significant impairment.

Stating that a child can recover from autism was quite a large claim to make in 1987. Not surprisingly, many in the autism community had strong negative reactions to the Lovaas paper. It is probably fair to say that most reactions were based as much on bias as on science, but legitimate criticisms were raised as well. For example, Mundy (1993) raised concerns over residual symptoms that may be similar to those displayed by individuals with high-functioning autism. He stated that much more rigorous evaluation of outcome should be conducted before it is prudent to state that children have recovered. Mundy's paper stressed that measuring the phenomenon of recovery from autism demands precise and comprehensive measurement. Unfortunately, most research to this day has fallen short of comprehensively measuring all areas of functioning that are critical, and no consensus yet exists on how to measure recovery from autism.

In 2008, our group proposed the following system for measuring recovery from autism (Granpeesheh 2008). An individual can be considered to have recovered from autism if he once had a confirmed diagnosis of autism, receives a treatment of some sort, and then achieves all of the following:

1. Standard scores of 85 (one standard deviation below the mean), or higher, on valid tests of language, intelligence, and socialization
2. Is included in regular education with no special supports or modifications of any kind
3. Is evaluated by an expert diagnostician and no longer qualifies for any ASD diagnosis, according to DSM-IV criteria, on the basis of his current level of functioning

In the same year, Deborah Fein's research group proposed a similar system of measuring recovery from autism (Helt et al. 2008). They proposed that recovery consists of:

1. History:
   a. Diagnosed with ASD in early childhood
   b. Language delay: No words by 18 months or no word combinations by 24 months
   c. Blind review of charts confirming diagnosis
2. Current functioning
   a. Does not meet DSM-IV criteria for any ASD by best clinical judgment

b. Does not meet ASD cutoff on communication or social subdomains of the ADOS
c. No special education services are being rendered specific to autism symptoms (support for attention, organization, or academic difficulties is acceptable)
d. Individual is functioning without an assistant in a regular education classroom
e. Verbal, performance, and full-scale IQ are all at 78 or above
f. Vineland communication and socialization subscales are at 78 or above

Fein's definition of recovery is similar to ours in most respects, except that it is slightly less stringent in terms of the standard scores that meet the cutoff (one-and-a-half standard deviations below the mean vs. one), slightly more precise in terms of how the history must be measured (i.e., number of words spoken at particular ages), and somewhat more stringent in terms how diagnostics are measured after recovery (i.e., including ADOS in addition to clinical judgment by an expert diagnostician). However, the similarities stand out more than the differences because both models of measuring recovery from autism essentially amount to one basic proposition: If valid measures of all areas of functioning relevant to autism produce results within the average range and the individual is functioning successfully in day-to-day life, it is reasonable to say he or she has recovered from autism.

Research Documenting Recovery from Autism

Sallows and Graupner (2005) evaluated the effectiveness of EIBI, consisting of 38 h per week of behavioral intervention, starting before the age of 3.5 and lasting for 4 years. In addition to the primary purpose of the study, which was to evaluate overall outcome, the study evaluated the characteristics and outcome of a subset of children they labeled "rapid learners." Rapid learners were children who achieved non-impaired functioning on measures of intelligence, language and socialization and were succeeding in regular education. Eight of the eleven rapid learners were receiving no specialized supports in public school, while three had aids because of inattentiveness. Eight of eleven also scored in the non-ASD range on the ADI-R. The Sallows and Graupner study was important because it was among the first to apply a well-accepted diagnostic measure, the ADI-R, to evaluating outcome of children who recovered from autism.

Zachor et al. (2007) evaluated the effects of 1 year of EIBI for children with autism in Israel. They included the ADOS in their battery of assessments. By the end of treatment, 20% of children who had received EIBI no longer met criteria for any ASD according to the ADOS, a gold-standard diagnostic measure. In contrast, none of the children in the control group achieved this outcome.

Most existing research on recovery included relatively small numbers of individuals. In 2009, our group published a retrospective review of the charts of children who we observed to have recovered from autism at our clinic (Granpeesheh et al. 2009). We interviewed our most senior clinicians and asked them to identify all past clients who had recovered prior to discharge from treatment, from 1995 to 2007. This process produced a list of 204 names. The charts of all 204 clients were then reviewed for usable data and 38 charts were identified that had IQ scores taken within 6 months of the beginning of treatment and within 6 months of discharge from treatment. Twenty-four of the charts also had data from the Vineland Adaptive Behavior Scales (VABS). All charts showed IQ in the average to above average range at discharge, and increases were generally seen on the VABS although they were less robust. This was the first study to document recovery in a relatively large group of children with ASD, although the fact that the study consisted of a retrospective chart review severely limited the data that were available for analysis. Further research using thorough measures of recovery, as well as a valid experimental design, was still needed.

In a recent study, Fein's research group published a study that contained comprehensive evaluations of functioning in children with autism who had recovered (Fein et al. 2013). Kelley and colleagues evaluated three groups

of children: (1) typically developing children, (2) children who were reported to have recovered from autism, and (3) children who retained their ASD diagnoses but were reportedly high functioning. One of the primary purposes of the study was to determine whether children who recovered from autism had no detectable clinically significant impairment or whether they retained symptoms similar to those observed in high-functioning individuals with ASD. The rationale behind this comparison was to address the notion proposed by some that children do not recover from autism; they merely become higher-functioning individuals with autism. However, overall, the study found no differences between the recovered and typically developing groups, whereas differences were found in several comparisons between recovered children and high-functioning children who retained their ASD diagnoses. The only exceptions were three of the 34 recovered children who showed below average scores on facial recognition. Another common criticism of the concept of recovery from autism is the claim that children who recovered likely never really had autism to begin with. In order to address this concern, the Fein study used blind reviewers to review the original diagnostic charts of both the recovered group and the high-functioning ASD group. The reviewers, blind to group assignment, found that the participants in the recovered group did indeed qualify for ASD diagnoses at the time they were diagnosed. Similar to the Granpeesheh study, the Fein study was retrospective but included significantly more thorough evaluations across a wider range of skills. Taken together, these studies strongly support the notion that some percentage of children recover from autism.

Objections to Recovery

One objection to the concept of recovery is that it denies the individuality of people with autism and seeks to make them "normal." This objection is based on a fundamental misunderstanding of what most, if not all, EIBI practitioners mean by the term recovery. The goal of EIBI is never to make people normal; it is to give them the skills to be whoever they want to be. Social proclivities are a good example. Many individuals with autism prefer to spend time alone rather than making friends with peers. Effective EIBI treatment that aims toward recovery does not attempt to change this. Rather, EIBI treatment aim to teach the individual the skills necessary to make friends if one wants to make friends. If one has never learned the language necessary to make friends with peers, then one can hardly be said "choose" not to make friends. When children recovers from autism following EIBI treatment, they still have their own interests, intricacies, and unique personalities, but they also have the skills they need to live independently and to access the full range of human experiences in life, if they so choose.

The position we are advocating here is that autism is not a personality, a unique perspective, or a different way of looking at the world. All humans, regardless of whether they have autism, already have unique personalities, perspectives, and preferences. Autism is a name for the failure to develop critical skills that allow one to get the most out of life. For example, being particularly aware of visual stimuli does not make one autistic. It may well be true that many individuals with autism display this trait, but so do many typically developing individuals, and it is not something that needs to be addressed by treatment. Similarly, many individuals with autism excel at memorizing facts and enjoy spending free time doing so. Memorization can be a critically important skill for everyone, and effective EIBI treatment does not attempt to remove it; it merely establishes additional leisure and social skills that may be more effective in helping the individual make friends and have fun with peers, if he so chooses.

Another objection to the notion of recovery from autism is exemplified by the statement that one can "still tell the person used to have autism." This is an empirical question that could be settled through blind evaluation. Prospective studies of recovery from autism could include blind evaluation of children who have recovered from autism, as well as typically developing peers of the same age, and the ability to distinguish between

these two groups could be directly studied. But more importantly, the very nature of objection to recovery lacks credibility. Being able to tell that someone used to have a disorder does not mean they currently have the disorder. Some describe recovered children as "quirky" or "different," but quirky and different are not disorders. Certainly, we would not want to live in a world without quirkiness and individuality.

Still another objection to discussing recovery from autism is that it implies that recovery is the only meaningful outcome of treatment and may therefore increase societal stigma attached to individuals who have not recovered. An ASD diagnosis can be quite stigmatizing and a source of significant stress for the individuals and their families, so it is critical that we, as a community, do nothing to contribute to this. Therefore, honest discussion of whether the concept of recovery increases stigma for individuals who have not recovered is probably healthy for the autism community. It is conceivable that an *overemphasis* on recovery as the outcome of treatment could unintentionally imply that it is the only meaningful goal. For example, if the percentage of participants in a treatment study who recovered was the only outcome that was discussed to a significant degree, and no attention was paid to the very real and important gains that the rest of the participants made, the mistaken impression could be given that recovery is the only valued outcome. However, the goal of EIBI has always been clearly stated as maximizing the skill development of each individual person with ASD, such that each can reach his maximum potential, whatever that maximum may be. For some, the maximum potential is recovery. For most, it is not. In the worst case scenario, a child learns basic functional communication (perhaps through pictures or sign language), his challenging behavior decreases, he learns to use the toilet, and he learns to function more independently. These gains, though modest in comparison to recovery, are not modest to the individual who made them and his/her family. Any gains that maximize independence and self-determination and minimize upheaval and frustration in an individual's life are valuable, and it is important for the EIBI community to be clear on this issue.

Yet another objection to discussing recovery is that most individuals with ASD are too old for recovery to be a realistic possibility. The hopes, dreams, and aspirations of families of adolescents and adults with ASD are critically important and represent the vast majority of families living with ASD today. Some complain that too large an emphasis is made on early intervention and that, when their children reach adolescence, the available supports and opportunities decrease dramatically. Perhaps the very topic of this book (early intervention) reflects this problem. Particularly since the majority of children with ASD will still not recover, even when given the best possible treatment, it is critical that the needs of older individuals with ASD not be ignored. The large emphasis that is currently placed on early intervention is likely due to a number of factors, including the larger amount of funding currently available for research and practice, the relatively larger treatment gains that can be made, and the fact that it is simply easier for practitioners to manage young children, particularly when severe challenging behavior is present. Regardless of the reason for the overwhelming emphasis on early intervention that is present today, a greater degree of attention and resources is needed for older children and adults with ASD.

Finally, some object to the use of the term recovery because they believe it implies that a medical or biological cure has been produced. In other words, since ASD is a biological disorder and no biological or physiological intervention has been done, then there must be some underlying biological disorder and therefore the child cannot have been recovered from the biological disorder. It is our position that autism is not merely a biological disorder. In fact, it is worth noting that, so far, it is still a disorder that is diagnosed *purely* on the basis of behavior, that is, on the basis of how the individual interacts with his or her environment. It is our position that autism is the name used to describe a complex interaction between behavior, environment, and disordered biology. In the case of recovery from autism, the behavior and environment components are removed and what is left is an individual with disordered biology (although this is usually undetectable) who interacts

with his or her environment in a non-disordered manner. This is no longer usefully referred to as autism. This is a non-autistic person with an unidentified—and apparently unproblematic—disordered biology.

Sociopolitical Implications of Recovery from Autism

The concept of recovery from autism is very rarely acknowledged in the mainstream scientific community. Today, virtually all mainstream informational websites on autism (e.g., National Institutes of Health, etc.) assert clearly that autism is either a "lifelong condition" or that there "is no cure for autism." Perhaps, by design, such mainstream communities are conservative and not early adopters of information. In science, it is common to reject new ideas until there is overwhelming evidence in support of them, and this tendency is usually a good thing because it staves off premature adoption of erroneous and therefore unfruitful ideas. However, we argue that, in the case of recovery from autism, the clinical evidence is far too large to ignore any longer. Indeed, even in 1987, a small but sufficient number of individuals had recovered from autism, such that the concept should have been wholeheartedly engaged and studied, rather than criticized and marginalized. No particular researchers will be pointed out here (pointing fingers is probably not fruitful), but a large chorus of dissent on the topic of recovery was audible in the scholarly community in response to the Lovaas (1987) paper. It is interesting to note that few or none of the dissenters were experts in autism treatment, and few or none actually engaged the possibility of recovery by familiarizing themselves with the treatment that was said to produce it and contributing to the rigorous scientific study of it. Instead, the vast majority of these researchers merely said "nay" and continued to do research on a myriad of variables that had little or no direct relevance to treatment for individuals with ASD. Recovery was happening more than 25 years ago, and it was clear to anyone who was directly involved in the provision of top-quality EIBI services. Yet, only now the mainstream scientific community is beginning to acknowledge it. Legions of experts in autism research denied the possibility of recovery and discouraged its study and therefore, quite possibly, slowed the progress of scientific research on autism treatment. Conservatism is generally good in science, but we hope that the mainstream scientific community is beginning to recognize that many have been on the wrong side of the recovery debate for more than 2 decades.

On the opposite side of the continuum from the mainstream scientific community, several parent groups and hundreds of practitioners of CAM treatments embrace the concept of recovery far less judiciously than is justified. Virtually every week, a claim is made on the Internet that a new treatment has recovered or cured a child with autism. An exhaustive review of these treatments is far beyond the scope of this chapter, but they run the gamut from ones that have at least minimal legitimate rationale to ones that are wholesale fabrication (e.g., exorcism). CAM practitioners often complain that there is a very real lack of progress in research on medical treatment of autism, and this complaint is indeed justified. Today, there is not a single FDA approved medical treatment for autism. The atypical antipsychotic risperidone is approved for the treatment of challenging behaviors in individuals with autism, but this hardly amounts to a treatment for autism, per se, and hundreds of studies already support the effectiveness of behavioral interventions, which are far less intrusive, for challenging behavior in individuals with autism and other developmental disabilities.

In response to the lack of scientific progress in medical treatment for autism, many CAM practitioners prematurely adopt treatments that may indeed have tertiary data supporting the rationale behind them but have no real treatment data supporting their use or safety. In the overwhelming majority of these cases, the practitioners solicit verbal reports from their patients' parents on the effects of the treatment. It is no surprise that, when parents go to a doctor who thinks a treatment is going to work, and the parents are desperate for something that will work, the parents are likely to believe they see at least some effects.

This is the very reason for placebo-controlled treatment research. However, in the absence of real scientific research, both CAM practitioners and parents alike hold onto any evidence they can, anecdotal reports proliferate on the Internet, and soon tens of thousands of families are implementing treatments for their children that have no research support. It is no surprise, then, that this movement lacks credibility in the mainstream scientific community. An unfortunate side effect is that the world likely notices that the loudest proponents of recovery from autism are also the least judicious in which treatments they advocate. The inevitable result is that recovery is seen as just another outlandish anecdotal story on the Internet.

Practitioners of top-quality EIBI have been producing recovery in some proportion of their clients for decades. It is interesting, then, to note the almost total lack of use of the term recovery by EIBI researchers and practitioners. To our knowledge, less than a handful of EIBI practitioners in the USA publicly acknowledge recovery from autism at the time this chapter was written. It is not clear why this would be the case. However, a likely reason is the harsh criticism that Lovaas received in response to his 1987 paper. Indeed, he even publicly stated that he regretted using the term recovery in print. However, those close to him knew that he fully believed in recovery and continued to see it on a regular basis until he passed away.

EIBI practitioners and researchers generally avoid the topic of recovery, but when it is addressed, euphemisms such as "rapid learning," "optimal outcome," "removal of diagnosis," or "loss of diagnosis" are used. Occasionally, EIBI practitioners will say that a client has "graduated" or has "finished the program." Often, when pressed for more details, they indicate that the child no longer has clinically significant impairment in any domain and is succeeding in his everyday social, educational, and family life, with no specialized supports. Such practitioners often admit, verbally, that they are afraid to use the term "recovery" because of the negative backlash they anticipate from others. To quote a well-known fable, we believe "the emperor is wearing no clothes" here. The time has come for the EIBI community to stand up and acknowledge the outcome they have been producing for decades. Indeed, it could be argued that, when we produce recovery, we as EIBI practitioners, have an ethical obligation to acknowledge that recovery from autism exists.

Ethics of Recovery from Autism

Some question the ethics of discussing the possibility of recovery from autism, a priori. This view must ultimately reduce to prejudice because no one would question the ethics of discussing the possibility of recovery from cancer, diabetes, depression, alcoholism, or phobias. An anecdotal observation serves as an example. At a conference presentation on the topic of recovery from autism (Granpeesheh et al. 2008), an audience member stated that she believed it was unethical to discuss the possibility of recovery because children with autism in her region were not able to receive funding for intensive services and were therefore highly unlikely to recover. Therefore, she stated, telling their parents that recovery from autism following EIBI is possible for some children would harm the parents by making them distressed. We would suggest quite the opposite. We believe that clinicians have the ethical responsibility to tell their patients the possible outcome of the various treatment options that exist. If economic or logistical variables prohibit patients from receiving the treatment they need and to which they have a right, then the patient should feel distressed, just as any patient should when denied access to a proven treatment for any seriously debilitating disorder. Replace "autism" with "cancer" and the point is clear. Consider vaccine-preventable diseases in third world countries. Would it be more ethical to hide knowledge of disease prevention from citizens merely because they cannot afford access to the vaccine?

Future Research on Recovery

It is the opinion of these authors that recovery from autism exists and that early intensive behavioral intervention produces it in some portion

of children who receive it. However, much more scientific work has yet to be done. The future of research on recovery from autism is wide open. Unfortunately, much of the most important research will necessarily be large and costly. The first question that many ask is *Who will recover*? A significant amount of research has been done on predicting response to EIBI treatment, and space does not permit a thorough review of this research. However, several variables have been found to be related to positive response to EIBI, including higher IQ and younger age at intake (Harris and Handleman 2000), as well as less impaired language at intake and higher rates of learning early in treatment (Sallows and Graupner 2005). However, results have often differed across studies, and additional research is needed with larger samples.

Predicting who is likely to recover from autism, in itself, is not likely to improve the lives of individuals with ASD. Indeed, this information, alone, may enable funding sources to deny access to treatment to those who are less likely to recover. What is really needed is research that uses this information as a springboard from which to push the effectiveness of EIBI to new heights. For example, questions such as the following must be directly addressed by research: What is the optimal model of EIBI to produce recovery? How can EIBI be made to produce higher recovery rates? How can EIBI be altered to recover children who otherwise would not recover? It seems likely that a subset of children with ASD learn more slowly in EIBI programs because of particular deficits at intake. For example, children for whom social attention is not already a conditioned reinforcer are likely to be less motivated by the highly social nature of EIBI than children for whom attention is already a large source of motivation at intake. Perhaps focusing more heavily on establishing attention as a conditioned reinforcer at intake would help those children respond better to treatment overall. There are presumably scores of such possibilities, and research into them has scarcely begun.

Future research on recovery should focus on identifying particular ways in which EIBI can be modified or enhanced to contribute to recovery, and then randomized controlled trials should commence, wherein children with ASD are randomly assigned either to a standard EIBI group (in which some percentage of children are going to recover) or to the enhanced EIBI group. The goal of the study would then be to evaluate whether a larger percentage of children in the enhanced EIBI group recovered relative to the standard EIBI group. Research of this sort will need to be very large scale and will therefore be very costly. Even if an important variable is identified, it is not likely to increase recovery rates by more than 5%, and so a small but important effect of this sort would require a very large sample size in order to achieve sufficient power, particularly given the high degree of intersubject variability that is inherent in ASD research. But logistic and monetary challenges are not a reason to abandon important research endeavors; they are simply barriers that must be systematically addressed and overcome.

Preventing Autism

The concept of preventing autism is all but unprecedented and has not yet been documented in sound scientific research. However, we believe it may be possible to prevent autism via very early behavioral intervention, at least for some children. Furthermore, preventing autism may be less expensive and more efficient than treating it. More importantly, preventing autism should prevent at least some degree of the suffering that the disorder causes for families affected by it. Therefore, we devote the remainder of this chapter to a discussion of the topic. Since little or no research has yet been done on preventing autism, the discussion will necessarily be primarily conceptual.

Defining Prevention

Our concept of preventing autism through very early behavioral intervention is based on our position on recovery from autism. As described above, we believe that it is reasonable to say that,

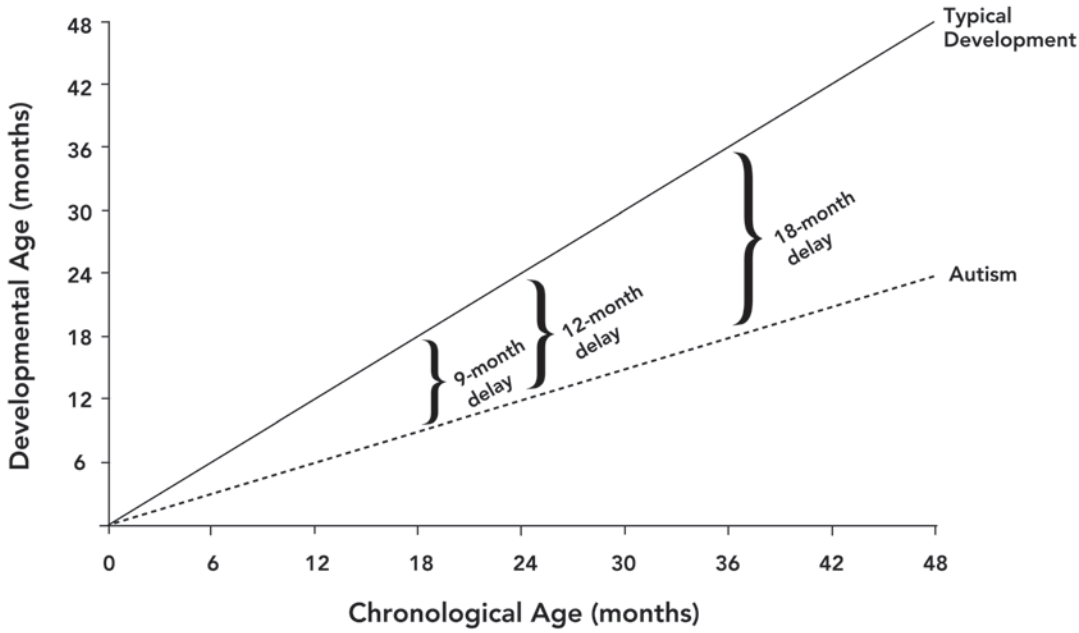

Fig. 21.1 Hypothetical data describing the course of child development in typical children and in children with non-regressive autism. The lower rate of development in autism produces a gradually increasing overall amount of developmental delay as time passes.

if all of a child's clinically significant skill deficits have been remediated via intervention such that he no longer exhibits symptoms characteristic of a diagnosis of autism, he has recovered from autism. We propose that the same basic logic should apply to prevention: If a child exhibits skill deficits that would—without intervention—qualify him for an ASD diagnosis, but EIBI prevents the deficits from reaching a degree of severity to merit an ASD diagnosis, then it is reasonable to say that one has prevented autism.

Figure 21.1 depicts hypothetical child development over time. The horizontal axis depicts chronological age, and the vertical axis depicts developmental age. The solid line at a 45° slope depicts an average rate of development; for example, at chronological age three, the child's developmental age is three. The dashed line depicts hypothetical data for a child with non-regressive autism. The slope of the line is less steep than average—the child with autism is learning at a lower rate—and the vertical distance between the two lines depicts the degree of developmental delay that the child suffers from, at any given chronological age. Since the rate of development is slower for the child with autism, his overall amount of developmental delay gets larger as time passes. For children who present with more severe forms of autism, this gap may be particularly large. Whenever behavioral intervention begins, the goal of intervention is to increase the rate of child development such that the child's developmental age increases to meet his chronological age. If intervention begins when the child is diagnosed at age three, then a delay of 18 months of development must be remediated. As treatment progresses, however, the child's chronological age also increases. Therefore, it is not enough merely to increase the child's learning rate to an average rate because this would merely maintain the total months of delay that were already present. In order to remediate delay entirely, the child's learning rate must *exceed* that of typical development—no small feat for a child with a pervasive developmental disorder.

Since the amount of developmental delay increases over time, it follows that one would want to intervene at the earliest possible time in order to have the least amount of deficit to remediate. For example, if one begins intervention at age two

instead of three (on this hypothetical chart), then one has 12 months of delay to remediate, as opposed to 18 months if intervention begins at age three. Our concept of prevention is based on the possibility of beginning intervention before the degree of delay is sufficient to warrant an ASD diagnosis and, therefore when the size of the delay is far smaller than it is when intervention typically begins. Beginning intervention at a chronological age of 18 months is not unreasonable and has become increasingly common in EIBI, as will be discussed below. Beginning at this time would require remediating only 9 months of delay—that is, a hypothetical reduction of 50% in the amount of remediation, compared to beginning intervention at 3 years of age. But if intervention can begin at 18 months of age, there is no reason to think it could not begin at 15 or 12 months of age—perhaps even 8 or 10 months of age.

It is also worth noting that intervening with particularly young children often entails working on more basic skills, which in some cases should be easier to teach. For example, the play skills that a 2-year-old must learn to "catch up" to her typically developing peers are relatively simple (e.g., parallel functional pretend play), versus those that are displayed by a typically developing 5-year-old (e.g., imaginary and sociodramatic play). In short, if one begins intervening when a child is a very young age, one has fewer and more basic skills to teach.

Another possible benefit of very early intervention is the potential for remediating skill deficits before a significant amount of challenging behavior has been learned and reinforced. It is commonly believed that a large portion of challenging behavior that is displayed by children with autism occurs because of a lack of other, more appropriate means of communication (Carr and Durand 1985). If the child is taught successful communication and social interaction skills very early on, then it may be possible to prevent the development of challenging behavior. This, in turn, would likely make treatment more efficient because there would be little or no need to spend the first several months decreasing challenging behavior, as is often done when intervention starts at 3 or 4 years of age.

The hypothetical data in Fig. 21.1 imply that preventing autism should be more efficient than treating it. Research on predictors of successful outcomes for children with autism has indicated that a younger age at intake is strongly correlated with better outcomes (Perry et al. 2011; Harris and Handleman 2000), and it seems reasonable that this same basic logic should stand for prevention, too. In this light, prevention is less of a categorical concept and is probably better conceptualized as a continuum, where the earlier one starts intervention, the less intervention is needed. If one starts it before the diagnosis is made and removes clinically significant impairment before the child is old enough to receive the diagnosis, then one has "prevented" autism. From a purely behavioral perspective, this is no different than behavioral intervention at any other age, except that there is less work to do and it is therefore likely to be more efficient. Additionally, providing early intervention prior to a diagnosis will result in reduction of the "red flag" symptoms commonly used to identify children who may qualify for a diagnosis. These "red flag" symptoms (e.g., poor eye contact, stereotypical patterns of play behavior, etc.) are behaviors that are subject to intervention and can be treated early.

It is important to note that the data depicted in the figure are hypothetical, and it is not known whether the true function describing developmental delay in autism is linear, nor is it known what degree of skill development is possible for any given child. And of course, every child with ASD is different, with some presumably being more severe than the hypothetical data depicted in the chart and some less severe. In addition, beginning EIBI at such a young age has not yet been evaluated in rigorous scientific research, so a large number of variables warrant discussion, several of which we address in detail below.

Research on Autism Prevention

No published studies of which we are aware have specifically set out to prevent autism via very early behavioral intervention. However, one case study described results of very early behavioral

intervention for a toddler at high risk for autism. Green et al. (2002) reported the case of a little girl, Catherine, who received an "at risk" diagnosis of autism based on multiple screenings indicating communication and language delays, as well as stereotypic patterns of behavior. The parents of this child sought professional evaluations at the first signs of these delays because of their prior experience with their first child, who had received a diagnosis of autism and was receiving behavioral intervention. At the early age of 14 months, Catherine began an intensive in-home behavioral program consisting of 1:1 direct instruction for 25–36 h per week. At the age of 4 years and 5 months, Catherine completed her 1:1 in-home instruction but continued to have monthly follow-up observations in her preschool classroom. At the age of five, Catherine entered a general education kindergarten classroom without a diagnosis of autism, an individualized education plan (IEP), or a classroom aide, and she did not meet diagnostic criteria for an autism spectrum diagnosis. This case study represents a critical first step in research on using very early behavioral intervention for the prevention of autism, but much more research using valid experimental designs is still needed.

A highly controversial 2004 paper proposing a purely behavioral etiology of autism also contains a discussion of the prevention of autism (Drash and Tudor 2004). Space does not permit a discussion of the main thrust of the Drash and Tudor paper—that autism is caused solely by parent-child interactions—nor is one needed. It will suffice to say that such an idea is bordering on irresponsible in that it is essentially a return to the notion that autism is caused by bad parenting. We find this notion plainly absurd and not even worth discussing. The authors do specifically state that "Our analysis in no way attempts to blame parents" (p. 60), but it seems plainly obvious that denying the contribution of any factor other than parent behavior will carry that extremely negative implication for many parents.

Aside from the controversial aspects of the argument, the Drash and Tudor paper is one of the very few existing papers that discusses the possibility of preventing autism through very early behavioral intervention, and it makes an important practical point: Regardless of the genetic contributions to the etiology of autism, the only level at which we can intervene now or any time soon is the level of behavior—environment relations. The authors give specific recommendations for how this might be done. They propose that within the first 18 months of life or less, at-risk children should be identified, and parent–child interactions should be modified in order to encourage the development of adaptive forms of child communication and decrease avoidant behavior on the part of the child. Furthermore, they describe several case studies in which this was done and report that development for all the children was corrected in a much shorter period of time than is typically required of EIBI for children already diagnosed with autism. Like the Green case study, these were uncontrolled case reports, and further replication with sound experimental designs is still needed.

Identifying Who Should Receive Preventive Intervention

The first obvious roadblock to preventing autism via EIBI is simply detecting at a very young age which children will later be diagnosed with an ASD. Although warning signs can be observed at very young ages, the vast majority of diagnosticians are highly hesitant to provide an ASD diagnosis before 2–3 years because diagnostic evaluations were not validated with younger children (Crane and Winsler 2008). Research in this area has advanced significantly, but results still vary dramatically across studies. Retrospective studies have been published that reviewed home videos of typically developing children and children who later received a diagnosis of autism, indicating that some deficits can be observed as early as 4–6 months for motor anticipation (Brisson et al. 2012), and on average around 7 months for social attention, affective responsiveness, and prelinguistic vocalizations (Crane and Winsler 2008). Another retrospective video analysis has reported that some sensory-motor and social symptoms may categorize children

later diagnosed with autism from those later diagnosed with developmental delays and children of typical development at 9–12 months (Baranek 1999). These symptoms include poor visual orientation/attention to nonsocial stimuli, prompted or delayed response to name, excessive mouthing of objects, and aversion to social touch. Similar results were found by Osterling et al. (2002) in a retrospective video analysis of 1-year-old infants later diagnosed with autism versus intellectual disabilities showing that the children who were later diagnosed with autism rarely looked at others or showed an orienting response when their names were called as compared to children later diagnosed with intellectual disabilities.

Although several autism screening tools exist, very few were developed and have been validated for children younger than 2 or 3 years old. One promising screening tool is the Checklist for Autism in Toddlers (CHAT; Baron-Cohen et al. 1992) which includes screening items for children as young as 18 months. The CHAT includes nine items asked to the caregivers and an additional five items that require direct observation in the home. To validate the accuracy of the CHAT, Baron-Cohen et al. (1992) administered the checklist to 41 children considered at high risk for autism based on genetic predisposition and determined that four children consistently failed items assessing gaze monitoring (e.g., looking in the direction of a caregiver's gaze), protodeclarative pointing (e.g., pointing at objects to direct another's gaze toward the object), and pretend play. These behaviors develop in typically developing children around 14 months of age and appear to be distinctively deficit among children with autism. All four children in the sample received a later diagnosis of autism, whereas the remaining 37 children did not fail more than one of these three items and none received a diagnosis of autism. Extending on this initial analysis, Baron-Cohen et al. (1996) administered the CHAT to 16,000 children in Great Britain, and 12 were identified as being at high risk for autism based on failing the three critical items from the initial analysis. Of the 12 children identified as at risk, 10 later received a diagnosis of autism and the remaining two were diagnosed with developmental delays, demonstrating that the CHAT was a fairly accurate indicator of autism at 18 months. A follow-up study investigating the total number of diagnoses on the autism spectrum from all 16,000 participants identified that 94 children had a diagnosis of either autism or PDD at 7 years. Using less stringent criteria to assess which items on the CHAT were most likely to indicate which children may have been identified at 18 months, only 38 % of the 50 cases would have been flagged at 18 months. Although the sensitivity is quite low, the specificity, or likelihood that the instrument will not falsely predict that a child has autism, is quite high (97.5 %) because, out of the identified cases, very few did not actually receive a diagnosis. The concern with using this instrument is that a child may not be flagged on the screening at 18 months, thus missing the opportunity to receive intervention until later, when greater deficits are apparent.

The requirement for direct observation necessary to administer the CHAT makes this screening tool less likely to be used despite its potential benefits of detecting early signs of autism for some children exhibiting symptoms at 18 months. Additionally, some behavioral deficits may not present during a single observation. Because of these potential limitations, a modified version of the CHAT was developed that can be conducted during normal visits to a family pediatrician by relying on parent report of current behaviors. The Modified Checklist for Autism in Toddlers (M-CHAT: Robins et al. 2001) includes 23 items that require a caregiver to provide a yes or no response. The authors validated the M-CHAT by administering it to 1,293 children between 18 and 30 months of age resulting in 58 cases identified as at risk for autism. A full evaluation was then conducted with the children identified as at risk and resulted in diagnoses of autism or pervasive developmental disorder (PDD) for 39 of these children. The remaining 19 children were found to have other language or global delays but did not meet full diagnostic criteria for autism or PDD NOS. Based on these findings, the M-CHAT may be a valuable screening tool that can be easily administered during a child's 18-month pediatric visit, but follow-up research is necessary to determine the number of children who were not identified by the M-CHAT

and later received a diagnosis. Although both the CHAT and M-CHAT are promising early screening tools, they are both designed for children at least 18 months of age. With research suggesting that red flag symptoms may be observed within the first year of life, there is a need for much earlier screening tools to identify children who may benefit from even earlier intervention and the possibility of preventive intervention.

There are a few other early screening measures with initial studies demonstrating promising results, but further evaluation of these tools is necessary. These include: the First Year Inventory (FYI: Reznick et al. 2007), a parent report measure administered during 12-month pediatric checkups; the Autism Observation Scale for Infants (AOSI: Bryson et al. 2008), a direct observation assessment that can be administered to children as young as 6 months; and the Infant-Toddler Checklist (ITC: Wetherby et al. 2008), a parent questionnaire that can be administered repeatedly with children 6–24 months old. Efforts are underway to improve the predictive power of these and other assessments for children at risk for developing a later diagnosis of autism, with the goal to identify children that will benefit from early treatment and potentially prevent a diagnosis altogether.

Appropriateness of EIBI Procedures for Infants and Toddlers

One potential concern with very early behavioral intervention, for preventing autism is that the treatment procedures used in EIBI may not be appropriate for infants and toddlers (Dawson et al. 2010). Adapting procedures from one population to another always presents unique challenges, but the concern over using behavioral procedures for very young children is primarily due to a confusion of principles with procedures. The basic principles that are the foundation of EIBI (e.g., reinforcement, extinction, stimulus control, generalization, etc.) were originally derived from research with animals, and their generality has been replicated across scores of species and populations, including human infants (Pelaez et al. 2012). EIBI *procedures*, however, are specific operations derived from the basic principles, such as discrete trial training, natural environment training, chaining, and so on, and these clearly need to be customized to each different population to which they are applied, indeed, to each individual client within each individual population. Just as a smaller scalpel may be needed for pediatric surgery than for surgery on adults, the specific operations by which positive reinforcement are delivered, prompting is delivered, and so on, will need to be adjusted to be appropriate for infants and toddlers. But such adjustment is a natural part of EIBI treatment already and represents minor changes in how a procedure is done, not a question of *whether* it is done. For example, prompting consists of providing extra assistance to ensure a correct response—the particular form of extra assistance does not matter as long as it is nonintrusive, is successful in aiding a correct response, and can be faded out when no longer needed. Any time a child is attempting to learn something new, prompting will be helpful and will therefore still be included in whatever form is developmentally appropriate for the learner.

Similarly, discrete trial training (DTT) is probably the most empirically supported teaching procedure for children with ASD (see chapter on DTT in this volume). It seems unlikely that DTT at the level of intensity and duration that is often done with 3-year-olds would be appropriate for a 1-year-old. But the basic principles behind DTT—many learning opportunities, clear expectations, clear consequences, and providing assistance when needed—would still be needed if maximum learning rate is desired. Therefore, it seems likely that a somewhat "softened" version of DTT would be implemented, perhaps more in the context of play, or that very short blocks of DTT would be implemented—perhaps only two or three trials at a time.

Long-term Outcome

Children who receive preventive behavioral intervention and subsequently do not receive an ASD diagnosis would still need to be followed up in order to ensure that clinically significant ASD symptoms do not emerge at a later time.

Ideally, a lack of ASD diagnosis for life would constitute the purest definition of prevention. In reality, initial research that shows the absence of symptoms qualifying for a diagnosis at age 3–5 would be enormously valuable. After this effect is documented, more longitudinal research documenting continued success in middle childhood and adolescence would be needed. Since it is still not known what causes autism, it seems possible that the genetic and environmental contributing factors may still be present when preventive intervention is discontinued and that they may exert further influence at future times of stress in the child's life, such as when he/she enters school full-time, transitions to middle school, or enters puberty.

Objections

One potential objection to our position on prevention is that, even if it works, one would not be preventing autism; one would merely be preventing the child from receiving the diagnosis of autism. This is not a very meaningful objection because autism, as a disorder, is identified solely on the basis of a behavioral diagnosis, so prevention of that diagnosis amounts to prevention of the disorder. Clearly, this assumes that the child is evaluated by an expert diagnostician and no information is withheld, and they still do not qualify for the diagnosis. Very early behavioral intervention would, of course, no prevent the child from inheriting the genetic susceptibility of developing autism, but the genetic susceptibility is just that—*susceptibility*; it is not a disorder unless it manifests in clinically significant impairment in functioning. A similar objection is sometimes made against the concept of recovery by saying that treatment may change behavior, but it does not change "who the person really is" or that the "person is still autistic, even if they are able to fully function in life without impairment." This objection to recovery is particularly absurd and seems to be based on the assumption that autism is "who the person is," rather than a name for clinically significant impairment across three areas of functioning. We believe that a person is himself, not a disorder. A unique and interesting personality paired with a genetic susceptibility to autism is not autism. It is a unique and interesting person.

Discussing the prevention of autism long before scientific evidence has documented it may be a further source of ethical concern for some. It is important to point out that our definition of preventing autism cannot be applied in a practical way to any particular child and should not be used in such a way by clinicians or researchers. By definition, if our definition of prevention is achieved, one would never know with any degree of certainty whether the child would have been diagnosed with autism if he had not received very early behavioral intervention. Therefore, we do not feel it is ethical at this point to tell parents that one *is going to prevent* their child from developing autism or, after implementing very early behavioral intervention, that one *has prevented* the child from developing autism. All that can be honestly said is that skill deficits should be addressed and that this should be a particular priority for a child who is showing warning signs of autism and has an older sibling with the disorder. It is therefore somewhat alarming that, at the time this volume is being printed, multiple websites exist that provide non-research-based medical information on how one can prevent autism. Thus, it appears as though the concept of prevention is already beginning to receive the same treatment that the concept of recovery has received for the last 25 years: Virtually no research by the mainstream scientific community and irresponsibly premature adoption by the CAM community.

Discussing the concept of preventing autism must be done with care, but a strong argument can be made that *avoiding* the discussion is ethically questionable. A lively interdisciplinary engagement of the topic is going to be needed if sufficient research is going to be allocated to it. Furthermore, we believe it is important to use the word *prevention*, not some other euphemism that is less controversial. Just as in the case of the term recovery, we believe avoiding the use of the word prevention merely serves to marginalize the concept and therefore make it less likely that it will be addressed by serious scientists. To avoid

giving the term the full respect of calling it what it is does a disservice to the population of children who may develop autism in the future if effective preventive treatments are not researched.

Funding Preventive Treatment

At the time this volume is going to print, very little funding is available for preventive treatment. This is no surprise, given the lack of research supporting it. However, as discussed already, it makes sense to begin treatment as soon as deficits are observed, therefore preventing the child from falling further behind. Unfortunately, intensive behavioral intervention can exceed US$ 40,000–US$ 60,000 on average per year, and funding is often contingent on a diagnosis (Chasson et al. 2007); therefore, funding for services is usually not available until global deficits are demonstrated and a diagnosis is provided. Nevertheless, funding for early intervention is occasionally available, particularly when a child has an older sibling with a confirmed ASD diagnosis, and so it has become increasingly common for EIBI treatment providers to begin treatment of younger siblings at earlier and earlier ages.

Future Research on Preventing Autism

Our hypothesis, that preventing autism is possible via very early behavioral intervention, must be tested with sound, controlled research. However, such research is going to be enormously challenging. An ideal outcome study on prevention might include the following steps: (1) identify a treatment that is likely to prevent ASD, (2) identify very young children who are highly likely to be diagnosed later with an ASD, (3) randomly assign the participants either to receive the treatment or control, and (4) follow-up at age three or four and evaluate whether more children in the control group have a current diagnosis of ASD than in the treatment group.

One major potential methodological problem is that a very small number of children are likely to later receive any ASD diagnosis, regardless of treatment. Even in the highest risk groups, many children will not develop autism even without intervention. Therefore, many individuals in the control group would not develop autism as well. Detecting an effect this small, especially relative to the overall makeup of the group, would be statistically very challenging. It could require group sizes in the hundreds of participants, making such research very costly and logistically challenging. It is likely that large-scale grant funding and multi-site collaborations would be required for a study of this scale.

A major potential ethical concern with such a study is the need for a control group that does not receive very early behavioral intervention. In other words, in order to produce a controlled demonstration of prevention, one needs a group of children whose autism was *not* prevented. At first blush, this would appear clearly ethically unacceptable. However, the study might be ethically acceptable because treatment would not be *withheld* from children in the control group; they would merely receive what everyone else in their community receives if they had not participated in the study. This ethical concern could be further mitigated by strongly recommending to families in the control group that they seek behavioral intervention elsewhere. If they succeed in doing so, they would need to be dropped from the study, but at least they would be receiving treatment. In addition, some families still choose not to receive behavior analytic treatment, even when they are fully informed of its research base and even when the treatment is fully funded by a third party.

Conclusion

In conclusion, it is our position that recovery from autism exists, and preventing autism may be possible. Both concepts are based on the same basic rationale: If clinically significant impairment is not present, the diagnosis of autism is not appropriate. If an individual once had an ASD, received treatment, and no longer suffers from clinically significant impairment, it is reasonable to say this individual has recovered from autism. Much more controlled research is still needed on

recovery. Research is needed to identify which children are likely to recovery, but much more important, research is needed to identify which children will not recover and how EIBI can be enhanced to help these individuals achieve more than was ever possible before.

If children who were going to develop an ASD receive behavioral intervention early enough and they no longer suffer from clinically significant impairment that justifies an ASD diagnosis, then it is reasonable to say that autism has been prevented. Of course, it is not, and perhaps will never be, possible to confirm this at the level of the individual child, but through comparisons across groups, it is entirely possible to test this hypothesis. Not only is it ethical to discuss the possibility of preventing autism, we propose that it is our ethical *responsibility* to do the research that is necessary to determine whether the prevention of autism is possible.

References

Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders, 29,* 213–224.

Baron-Cohen, S., Allen, J., & Gillberg, C. (1992). Can autism be detected at 18 months? The needle, the haystack, and the CHAT. *The British Journal of Psychiatry, 161,* 839–843.

Baron-Cohen, S., Cox, A., Baird, G., Swettenham, J., Nightingale, N., Morgan, K., & Charman, T. (1996). Psychological markers in the detection of autism in infancy in a large population. *The British Journal of Psychiatry, 168,* 158–163.

Brisson, J., Warreyn, P., Serres, J., Foussier, S., & Adrien-Louis, J. (2012). Motor anticipation failure in infants with autism: A retrospective analysis of feeding situations. *Autism: The International Journal of Research and Practice, 16,* 420–429.

Bryson, S. E., Zwaigenbaum, L., McDermott, C., Rombough, V., & Brian, J. (2008). The autism observation scale for infants: Scale development and reliability data. *Journal of Autism and Developmental Disorders, 38,* 731–738.

Carr, E. G., & Durand, V. M. (1985). Remediating behavior problems through functional communication training. *Journal of Applied Behavior Analysis, 18,* 111–126.

Chasson, G. S., Harris, G. E., & Neely, W. J. (2007). Cost comparison of early intensive behavioral intervention and special education for children with autism. *Journal of Child and Family Studies, 16,* 401–413.

Crane, J. L., & Winsler, A. (2008). Early autism detection: Implications for pediatric practice and public policy. *Journal of Disability Policy Studies, 18,* 245–253.

Dawson, G. et al. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The EARLY START DENVER MODEL. *Pediatrics, 125,* 17–23.

Drash, P. & Tudor, R. (2004) An analysis of autism as a contingency-shaped disorder of verbal behavior. Analysis of Verbal Behavior, 20, 5–23.

Fein, D. et al. (2013) Optimal outcome in individuals with a history of autism. Journal of Child Psychology and Psychiatry, Feb;54(2):195–205.

Granpeesheh, D. (2008). Recovery from autism: Learning why and how to make it happen more. *Autism Advocate, 50,* 54–58.

Granpeesheh, D., Tarbox, J., Guerrero, F., Dixon, D., Najdowski, A., & Kenzer, A. (2008, May). *Defining recovery from autism*. Paper presented at the 34th annual convention of the Association for Behavior Analysis, International.

Granpeesheh, D., Tarbox, J., & Dixon, D. (2009). Retrospective analysis of clinical records in 38 cases of recovery from autism. *Annals of Clinical Psychiatry, 21,* 195–204.

Green, G., Brennan, L. C., & Fein, D. (2002). Intensive behavioral treatment for a toddler at high risk for autism. Behavior Modification, 26, 69–102.

Harris, S. L., & Handleman, J. S. (2000). Age and IQ at intake as predictors of placement for young children with autism: A four- to six-year follow-up. *Journal of Autism and Developmental Disorders, 30,* 137–142.

Helt, M., Kelly, E., Kinsbourne, M., Pandey, J., Boorstein, H., Herbert, M., & Fein, D. (2008). Can children with autism recover? If so, how? *Neuropsychological Review, 18,* 339–366.

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

McEachin, J. J., Smith, T., & Lovaas, I. O. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. *American Journal on Mental Retardation, 55,* 359–372.

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

Osterling, J. A., Dawson, G., & Munson, J. A. (2002). Early recognition of 1-year-old infants with autism spectrum disorder versus mental retardation. *Development and Psychopathology, 14,* 239–251.

Pelaez, M., Virues-Ortega, J., & Gewirtz, J. L. (2012). Acquisition of social referencing via discrimination training in infants. *Journal of Applied Behavior Analysis, 45,* 23–36.

Perry, A., Cummings, A., Geier, J. D., Freeman, N. L., Hughes, S., Managhan, T., Reitzelg, J., & Williamsh, J. (2011). Predictors of outcome for children receiving intensive behavioral intervention in a large, community-based program. Research in Autism Spectrum Disorders, 5, 592–603.

Reznick, J. S., Baranek, G. T., Reavis, S., Watson, L. R., & Crais, E. R. (2007). A parent-report instrument for identifying one-year-olds at risk for an eventual diagnosis of autism: The first year inventory. *Journal of Autism and Developmental Disorders, 37,* 1691–1710.

Robins, D. L., Fein, D., Barton, M. L., & Green, J. A. (2001). The Modified checklist for autism in toddlers: An initial study investigating the early detection of autism and pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 31,* 131–144.

Sallows, G. O., & Graupner, T. D. (2005). Intensive behavioral treatment for children with autism: Four-year outcome and predictors. *American Journal on Mental Retardation, 110,* 417–438.

Wetherby, A. M., Brosnan-Maddox, S., Peace, V., & Newton, L. (2008). Validation of the Infant—Toddler Checklist as a broadband screener for autism spectrum disorders from 9 to 24 months of age. *Autism: The International Journal of Research and Practice, 12*(5), 487–511.

Zachor, D. A., Ben-Itzchak, E. L., Rabinovich, A. L., & Lahat, E. (2007). Change in autism core symptoms with intervention. *Research in Autism Spectrum Disorders, 1,* 304–317.