Arnold Schwarzenegger, Governor
State of California
Business, Transportation and Housing Agency

Department of Managed Health Care
980 9th Street, Suite 500
Sacramento, CA 95814-2725
916-322-6727 - Phone
916-322-3968 - Fax
sream@dhmc.ca.gov

September 11, 2008

VIA MAIL AND EMAIL lark.park@sen.ca.gov

Lark Park
Consultant, Senate Health Committee
California State Senate
State Capitol, Room 2191
Sacramento, CA 95814

RE: CALIFORNIA PUBLIC RECORDS ACT REQUEST
PRA No. 2008-1793

Dear Ms. Park:

The Department of Managed Health Care ("DMHC") is in receipt of your records request submitted to the DMHC on September 3, 2008 pursuant to the California Public Records Act ("CPRA"). In that request you asked for "the white paper on Applied Behavioral Analysis Therapy that the Maximus Center for Health Dispute Resolution (CHDR) has supplied to DMHC."

Enclosed with this letter please find a copy of the white paper CHDR provided to the DMHC.

Should you have any questions or concerns regarding your CPRA request, please do not hesitate to contact me at (916) 324-2522.

Sincerely,

SARAH REAM
Staff Counsel III

SR:sr

Enclosure

cc: Bobbie Reagan, Assistant Deputy Director, Department of Managed Health Care

DRAFT

MAXIMUS
Federal Services

Considerations in the Treatment of Autism Spectrum Disorders

Prepared for the State of California
Department of Managed Health Care
May 20, 2008

DRAFT

MAXIMUS
Federal Services

Considerations in the Treatment of Autism Spectrum Disorders

Autism is a neurodevelopmental syndrome of uncertain etiology that has been the center of psychological and medical research, educational and political debate, and media attention for over three decades. Despite increased public awareness, earlier attempts at diagnosis, increased funding from the National Institutes of Health and other sources, and hundreds of studies; there remains no clear etiology and no definitive cure for autism. Treatment approaches have varied widely although in the 5 years a measure of consensus has been achieved. This paper will briefly summarize the characteristics and diagnostic considerations of autism, as well as recent data regarding epidemiology and etiology. Initial treatment approaches and their repercussions will also be outlined. Finally, a summary of current evidence for the major treatment modalities will be discussed, beginning with applied behavioral analysis (ABA) treatment.

Historical Perspective

Autism as a disorder was first described by Kanner (1943). He described a series of 11 children with severe language delay, unusual repetitive and stereotypical behavior, and an inability to relate to other people. He used the term "infantile autism" in reference to the solitary preference of those affected. Almost simultaneously, Asperger (1944) described a group of boys that he followed who exhibited fluent but odd, pedantic speech, a restricted set of interests with almost obsessional behavior, and social delays and idiosyncrasies Asperger's work describing the less restrictive variant was not widely known in the United States until the 1980s.

Initially, autism was felt to be in the group of childhood psychoses, possibly even the earliest manifestation of schizophrenia. The typical picture was of a withdrawn, often mute child who exhibited self-stimulatory and often self-injurious behaviors. Alternatively, it was postulated that the cause of autism was a result of withdrawal or failure of maternal attachment to the infant ("refrigerator mothers"). With the publication of the Diagnostic and Statistical Manual 3rd edition (DSM-III), autism was organized under a new classification of disorders (pervasive developmental disorders) that placed emphasis on the developmental nature of the syndrome. The definition of autism and the related disorders were further defined in the DSM-IV.

Today, the two syndromes originally described in the 1940s are felt to exist on the autism continuum or "spectrum", with the syndrome as described by Kanner being identified as the more severe form of autism, while the syndrome described by Asperger (Asperger's syndrome) is considered the less restrictive end of the spectrum. While all affected individuals have deficits in language, communication, and social interactions (as well as degrees of restricted or odd behavior), the degree of disability along each of these areas varies widely amongst individuals. Therefore, the term "autism spectrum disorder" or ASD has come to describe the broad phenotypic variation that characterizes these disorders. Included are autistic disorder, pervasive developmental disorder not otherwise specified (PDD-NOS), and Asperger's syndrome. Also under the general heading of the Developmental Disorders are two distinct neurodegenerative disorders with autistic features: Rett's syndrome and Childhood Disintegrative Disorder (CDD), although for the purposes of this paper the diagnostic and therapeutic issues with these two syndromes will not be considered.

Autism Spectrum Disorders: Diagnosis

The diagnosis of autism is made based on the presence of the clinical features of the disorder. The evaluation process is detailed and its components have been well described by Volkmar, et al. (1999). Evaluation by a team of professionals is standard of care, including medical (developmental pediatrician or psychiatrist), psychology, and speech language pathology expertise. A detailed history including the pregnancy, birth and neonatal course is taken. A full developmental history is performed, including all milestones of development and the timeline of any abnormal behaviors that have developed. A family history is taken including any history of ASD, other developmental disorders, and psychiatric or neurologic disorders. A comprehensive medical history is obtained with particular emphasis on any neurologic symptoms. A full medical examination is undertaken, with particular emphasis on the neurologic examination. There is no specific laboratory test for autism at present, but specific testing is completed to rule out medical conditions that may cause a developmental or behavioral picture that mimics autism. For example, DNA testing may be performed to rule out Fragile X syndrome, but there is no specific gene testing available to establish the diagnosis of autism.

Adjunctive medical testing such as an electroencephalogram, audiology testing, or brainstem auditory evoked response testing may be performed. Although research studies have found correlations between certain anatomic differences and autism, routine radiographic imaging is generally not employed in the work up of autism currently.

A full psychological assessment is indicated, including developmental and intelligence testing, as well as an assessment of adaptive and social skills. Standardized testing may be supplemented by a variety of parental questionnaires. Behavioral assessments often include an observation of the child at home or in other settings (i.e., preschool). Speech and language assessment is also performed using standardized testing and observational methods. Pragmatic and social communication is an area of particular difficulty for most children with ASD, and detailed supplemental information regarding this area of development may be solicited from family, teachers, and others familiar with the child.

The culmination of this diagnostic process is to establish the diagnosis and to have a resulting functional assessment that will assist with treatment planning and serve as a baseline for future comparison as treatment progresses. Despite the diagnostic differences described in the DSM-IV, the variability in presentation of the ASDs makes precise diagnostic differentiation difficult. There has been much debate in the literature regarding the difference in prognosis that may be associated with the different ASDs, and there is no current consensus in this regard. Since there is no definitive testing that differentiates autism from PDD-NOS, for example, the specific diagnostic label is somewhat subjective. It has been documented that the specific diagnosis given may be influenced by practitioner bias, parental preference, or eligibility for services if one diagnostic label is used over another (Shattuck, 2007). Imprecision in the assignment of diagnostic labels has potential consequences from both an epidemiologic and research perspective. Definitive conclusions about treatment efficacy and prognosis will be further complicated if the diagnostic lines are blurred across studies. The structure of funding for services may continue to influence the magnitude of this issue, as well, since in some states "carve out" benefits and treatment availability are designated for only certain types of ASDs.

Prevalence of Autism Spectrum Disorders

The prevalence of autism has changed dramatically in the last 30 years, although the precise reasons for the change are not fully understood. In the 1980s, the estimated prevalence of autism was approximately 2 children per 10,000. The majority of individuals diagnosed were of the classical (Kanner) type, with more severe restrictive symptoms. With the change in diagnostic categorization of ASDs there has been a substantial increase in prevalence, with most authorities estimating the number of children with an ASD to be 50-60 per 10,000 (1 in 166). This dramatic increase in prevalence has been the subject of intense scrutiny and debate. Reasons cited for the increase include the broader diagnostic criteria for ASDs, increased emphasis on screening, increasing pediatrician and public awareness, early intervention service availability, and a diagnostic "backlog" of children who were either unclassified or misclassified under an alternate diagnosis. Changes in reporting also may have contributed to the increased prevalence. Until 1992, there was no separate enrollment/reporting category for autism reported by public school districts. After 1992, a separate autism category was required to be reported, although the diagnostic criteria to be used were not specified.

In addition to the above factors, a number of experts argue that the true incidence of the disorder is increasing, albeit for unknown reasons. The etiology of ASD is unknown, and the current thought is that it may involve a complex interaction of genetic and environmental factors. Furthermore, public attention to ASD and its treatment has grown considerably. Widespread attention was given to the concern for vaccine-related cases of autism; however studies to date have not demonstrated any causal link between vaccine administration and autism. The enormous number of potential environmental factors and the variability of exposures across affected children greatly complicate the identification of specific environmental factors that may play a role in the disorder. Some experts argue that given the heterogeneity of the affected individuals, it is likely that there is more than one etiology or mechanism in ASDs. If that is the case, it obviously complicates the research regarding treatment efficacy and prediction of outcome. Until the etiology of the ASDs is better understood, diagnostic precision and true incidence of the disorders will remain unknown.

DRAFT

Evidence-Based Analysis of Autism Treatments: Current Summary

Prior to the 1970s, the treatment of autism was largely supportive and involved a highly structured environment. Virtually all patients were educated outside of the mainstream setting, with the vast majority requiring institutionalization or a life long supervised living arrangement. Only 2% were able to hold employment or live independently. Even once classified as a developmental disorder, no treatments were identified that were associated with improved outcome. The problematic behaviors that complicated the disorder made even occupational rehabilitation in a structured setting difficult. In recent years, as the diagnosis of ASDs began to rise, so have the number of treatments which are reported to be effective in the treatment of autism. The treatments include a wide array of modalities including:

- Intensive behavioral therapy
- Sensory integration therapy
- Auditory integration training
- Speech therapy
- DIR-floortime™ therapy
- Play therapy
- Music therapy
- Megavitamin therapy and other nutritional supplements
- Psychotropic medications
- Restriction diets
- Holding therapy
- Psychotherapy
- Facilitated communication
- Medical interventions including intravenous secretin or immunoglobulin treatments, and others.

In the following sections, a consideration of the medical evidence regarding efficacy for the major treatment modalities will be presented.

Behavioral Therapy

In the 1970s, researchers began studying the efficacy of using behavioral strategies to reduce targeted undesirable behaviors in individuals with autism. The rise of the behavioral theory that behavior patterns (regardless of etiology) can be altered by a specific programmed series of interactions was applied more globally to the treatment of autism in the UCLA Young Autism Project (Lovaas, 1987). This study reported the outcome of 19 children in the experimental group who received 40 hours of early intensive behavioral intervention (EIBI) on a one-to-one basis from trained therapists, primarily in the home setting. Most subjects received the training for more than 2 years, and all were less than 4 years of age at the onset of training. The outcomes of the experimental group were compared with that of two control groups. The first group received fewer than 10 hours per week of one-on-one behavioral treatment, and the second control group had children with autism who received traditional (non-behavioral) treatment such as special education preschool or speech therapy. All three groups had comparable developmental levels, pre-treatment IQ, and adaptive skills. The behavioral treatment group received structured behavioral therapy that targeted multiple areas including language, attention, imitation, social behaviors, play skills, and adaptive/self-help skills. All subjects were re-evaluated between the ages of 6 and 7. The experimental group had gained an average of 20 IQ points, and had improved in terms of their adaptive skills and language. In addition, 9/19 subjects were able to successfully complete first grade in a regular classroom without assistance and attained normal IQ scores. An additional 8/19 students were able to successfully complete first grade in a class for language delayed or learning disabled students. Only 2 students were in classes for autistic/mentally retarded students and had IQs in the profoundly mentally retarded range.

In comparison to the experimental group, only 1/40 control children was able to successfully complete first grade in a regular classroom and achieved a normal IQ. Eighteen of the forty control children were placed in a language delayed/learning disabled classroom and had IQs in the mildly retarded range. The remaining 21/40 were placed in classes for autistic/mentally retarded children and did not have any change in their IQ scores.

This study was criticized on two major issues. First, it did not have random assignment of children to the experimental and control groups. Rather, the children were assigned based upon availability of therapists at the time of enrollment in the study. Enrollment took place over a period of approximately two years, and therapist availability was greater at the onset of the study. Therefore, the initial children that were enrolled were entered into the treatment group, until there was no further therapist availability. Although statistical analyses were performed that demonstrated that the two groups were comparable, the lack of randomization was strongly criticized by some researchers (Schopler, 1989). Additionally, the idea that the 9 best responders from the treatment group had "recovered" was highly challenged. It was argued that the outcomes as measured by IQ, adaptive scores, teacher reports and independent physician/psychologist assessments do not provide a comprehensive or long term view of functioning to allow assertions that children had recovered from the syndrome. The Lovaas study also raised some concern from parents and other advocacy groups due to the use of some aversive stimuli reinforcers (mild physical restraint, abrupt verbal redirection), and the role of aversives in the treatment outcomes was unknown.

A follow up study on the original experimental group subjects McEachin, et al. (1993) retested the experimental subjects at a mean age of 11.5 years. It found that the gains in IQ and functioning were almost uniformly preserved. Eight of the nine high responders remained in regular classrooms, and the one remaining child had been moved to a classroom for students with language delay. As in the first study, assessments were completed by clinicians who were blinded to the original diagnosis and treatment, and did not identify any findings that would be consistent with signs of residual autism.

A number of studies have been completed since to attempt to replicate the results or to address additional questions regarding the treatment effects seen. Several studies were performed to see if lower intensity interventions in the home based setting were of equal efficacy. Anderson, et al. (1987) studied a group of 14 children with autism who received between 15 and 25 hours per week of EIBI in their home. The children had extensive standardized testing and developmental assessments beforehand to provide a baseline for comparison, but there was no control group. Children also received treatment for a shorter duration (1-2 years) than in the Lovaas study. While significant gains were seen in language and standardized testing scores, none of the children achieved full inclusion in regular classrooms by the end of the study.

Another study by Birnbrauer, et al. (1993) studied 9 children with autism who received and average of 19 hours per week of home based behavioral programming for 1.5-2 years. A group of 5 autistic children that were age and IQ matched and received no treatment served as the control group. At the end of 2 years, 4/9 children in the treatment group had shown significant gains in language, adaptive behavior and IQ, attaining scores of at least 80. An additional 4/9 children showed moderate gains in IQ, with only one of the experimental group children failing to show any gains. Only 1/5 children from the control group had shown significant gains, with an additional child in the control group showing moderate gains.

Attenuated improvements were also seen in the study by Smith et al. (2000). They looked at a group of children who received 25 hours per week of home based EIBI for 33 months. At the end of the study, 4/15 subjects had attained IQ scores of more than 85 and were placed in a regular classroom. Also of note was that the subjects in this study had a higher rate of complete mutism than the children in the earlier studies. Eldevik, et al. (2006) looked at children who received just 12 hours per week of home based EIBI for 2 years, versus a matched control group that did not receive any treatment. At the end of the study period the EIBI treated group showed improvements, but these were not statistically significant. None of these studies had randomized control groups although the control subjects were matched in terms of age and IQ at the onset. There are also differences in the specific outcome measurements and the length of treatment. However, the findings of these studies were consistent in showing that while gains were made in the experimental group relative to their controls; the magnitude of the gain did not equal that from the original Lovaas study. It was postulated that these differences are likely at least partially due to the reduced number of hours per week of behavioral programming.

Another group of studies looked at whether the favorable results of EIBI could be replicated in a community based setting. Fenske (1985) reported the results of a study of 9 children who received 27.5 hours per week of center-based EIBI before 60 months of age for a period of 2 years. The results were compared with a control group of children with the same diagnoses and objective performance who enrolled in the center for EIBI after 60 months of age. The results showed that 67% of the younger group achieved a "positive outcome" of full inclusion in a regular class setting, as opposed to just 11% of the older group. While the other outcome measurements (IQ, etc) from previous studies were not repeated, this study did replicate the favorable results from the Lovaas study in terms of regular classroom placement. Harris, et al. (1991) also demonstrated significant gains in IQ scores for children with autism after attending a preschool utilizing an

Applied Behavioral Analysis (ABA) curriculum, compared with their age matched normal controls who attended the preschool's integrated program. The autistic group showed an average IQ gain of 18.8 points, while the scores for the normal children in the control group did not change. Another study by Sallows and Grapner (2005) demonstrated results of clinic based EIBI that were comparable to those obtained in the Lovaas home based program. They took 23 children with the diagnosis of autism who were matched on the basis of pretreatment IQ and then randomly assigned them to receive either clinic directed EIBI at 40 hours per week or parent directed therapy with intermittent professional supervision at 32 hours per week. Therapy lasted for between 2 and 4 years, based on the progress of the child. A large number of children in both groups also received other supplemental treatments (speech therapy, occupational therapy, special elimination diets, etc). At the end of the study, there was no significant difference between the groups. However, for each group 48% of the children were able to be placed in regular classrooms. There were significant gains in IQ demonstrated, with the average IQ gain of 25 points. The study findings were consistent with those of the Lovaas study, and additionally demonstrated that clinic based treatment can be as effective as home based therapy. The lack of difference between the parent directed treatment group and the clinic based group was somewhat unexpected, but it was suggested that the 32 hours per week of treatment with the amount of available supervision likely allowed for a sufficiently intensive treatment delivery.

Another study by Cohen et. al (2006) replicated these findings, assigned a group of IQ and age matched children to either a center based EIBI program or a special education program with less than 9 hours of behavioral intervention. Those children in the EIBI group achieved an average increase in IQ of 25 points and an average increase in language comprehension test scores by 20 points. Eleven of the 21 children in the experimental group were placed in regular education programs by the third year with support, and 6 of the 21 were placed in regular education placements without need for support. By contrast, the control group achieved less impressive gains with only 1 of the 21 children in the control group able to be placed in a regular education classroom.

A recent study by Remington, et al. (2007) again replicated significant gains in a group of preschool children receiving intensive (40 hours per week) clinic based behavioral instruction. While the programs implemented by the centers in these studies were not identical, it is clear that an intensive center based program can attain favorable results for a comparable number of children when compared with intensive home based programs.

Concerns related to the above studies were raised regarding the possibility that the children's progress was due to the intensive attention the child was receiving and not the specific type of therapy per se. Several studies have addressed this issue. Eikeseth, et al. (2002) compared the outcomes of a group of 13 children receiving 30 hours of EIBI instruction per week with a group of similar matched controls who had 30 hours of intensive but "eclectic" non-behavioral therapy per week. At the end of 1 year, the EIBI group had experienced similar gains to the Sallows study above. Seven out of 13 children in the EIBI group had IQ scores in the normal range, with average gains of 25 points. Significant gains were also seen in communication and adaptive behaviors. In contrast, the control group had average IQ gains of only 8 points, but had decreases in their adaptive behavior scores. Only 1/12 children scored in the average IQ range at the end of the study. At follow-up in 2007, students in the behavioral group maintained gains made after intensive behavioral treatment. This was replicated in a study by Howard, et. al. (2005) who also compared 20 children receiving center based EIBI with a control group of 16 children receiving intensive "eclectic" non-behavioral treatment and a second control group of 16 children in a usual special education setting. The experimental group exhibited significant gains across all domains, while there was no demonstrated difference between the control groups. These studies demonstrate that intensive, non-behavioral treatment is not associated with gains above that to be expected from traditional special education.

While significant gains are seen across studies for a proportion of "fast" learners, and somewhat lesser gains are achieved by another proportion, it has been noted by many researchers that some children make only very modest gains even with intensive behavioral therapy over several years. Several studies have attempted to identify factors that are predictive of response to treatment. Harris and Handelman (2000) noted that in their study higher IQ (mean of 78) and younger age at onset of treatment (42 months) was associated with a better outcome. Smith ,et al. (2000) and Eldevik, et al. (2006) also noted a correlation between lower IQ at start of treatment and more modest treatment gains. All authors noted, however, that gains were still made in the majority of the other experimental subjects as well, and that this data should not be used to suggest that treatment availability should be limited to only the children with higher initial IQ scores.

The majority of the studies cited above have some methodological flaws. Many do not involve random assignment of experimental versus control subjects. In several, assignment to the treatment group was determined by therapist availability, a critical commodity in delivery of the therapy. Also, some of the studies did not blind the evaluators to what treatment group the child was in. In almost all cases, the parents were aware of whether the child was in the behavioral group or not. As was mentioned earlier, there were likely some differences in content and focus between the various EIBI programs across studies, and most certainly in the therapeutic effectiveness of the various providers. In many studies, the behavioral therapy was overseen by a PhD or Master's prepared psychologist, generally with a specific behavioral background and training. However, many of the therapists providing one to one instruction on a daily basis were college or graduate students, or paraprofessionals, who had received training in the specific protocol that also likely varied across studies. The studies with parental directed programs had similar variability in terms of motivation and compliance, as well as skill level across parent providers.

In addition to the above concerns, some of the early studies utilized aversive reinforcers as well as positive reinforcement, while the more recent studies utilized extinction techniques to combat aberrant behavior in lieu of aversives. The length of treatment obviously varied greatly across studies. Even within a study, the idiosyncrasies of schedule changes, illness, personnel attrition, compliance and other factors certainly varied amongst subjects, and would be difficult to measure and impossible to control.

Finally, the dissemination of the data from the Lovaas study and other favorable early studies, along with a best-selling book recounting "recovery" of two siblings after following an EIBI home based program (Maurice, 1993) led to an unprecedented amount of media attention and growth of autism advocacy networks. In the face of this, researchers found it increasingly difficult to design studies that did not take parent preference into account when assigning treatment groups. Even though there was a shortage of trained therapists to implement programs on the scale required by the early 1990s, the dissemination of parental training materials (Lovaas et. al., 1981; Maurice et. al 1996) led to a number of parents undertaking training on their own, in lieu of participation in a control group or in traditional therapy.

DRAFT

Analysis of the Strength of Evidence and Recommendations

In evaluating the strength of evidence for a given treatment or procedure, a standardized framework for assessment is required. Any assessment framework rates the quality of the evidence available for a given treatment or intervention. There are a number of different rating scales that have been published to evaluate strength of medical evidence on a treatment or procedure, as well as to stratify strength of recommendations from such evaluations. One of the most widely used is that from the US Preventative Services Task Force (2003) which is provided below:

Level of Evidence Stratification

Level I: Evidence obtained from at least one properly designed randomized controlled trial.
Level II-1: Evidence obtained from well-designed controlled trials without randomization.
Level II-2: Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one center or research group.
Level II-3: Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled trials might also be regarded as this type of evidence.
Level III: Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees.

The USPSTF grades the quality of the overall evidence for a service on a 3-point scale (good, fair, poor):

Quality of Evidence

Good: Evidence includes consistent results from well-designed, well-conducted studies in representative populations that directly assess effects on health outcomes.
Fair: Evidence is sufficient to determine effects on health outcomes, but the strength of the evidence is limited by the number, quality, or consistency of the individual studies, generalizability to routine practice, or indirect nature of the evidence on health outcomes.
Poor: Evidence is insufficient to assess the effects on health outcomes because of limited number or power of studies, important flaws in their design or conduct, gaps in the chain of evidence, or lack of information on important health outcomes.

Recommendations based on the analysis of evidence are also ranked by USPSTF as follows:

Categories of Recommendations

Level A: Good scientific evidence suggests that the benefits of the clinical service substantially outweighs the potential risks. Clinicians should discuss the service with eligible patients.

Level B: At least fair scientific evidence suggests that the benefits of the clinical service outweighs the potential risks. Clinicians should discuss the service with eligible patients.
Level C: At least fair scientific evidence suggests that there are benefits provided by the clinical service, but the balance between benefits and risks are too close for making general recommendations. Clinicians need not offer it unless there are individual considerations.
Level D: At least fair scientific evidence suggests that the risks of the clinical service outweighs potential benefits. Clinicians should not routinely offer the service to asymptomatic patients.
Level I: Scientific evidence is lacking, of poor quality, or conflicting, such that the risk versus benefit balance cannot be assessed. Clinicians should help patients understand the uncertainty surrounding the clinical service.

In evaluating the evidence for efficacy of EIBI in the treatment of autism, the evidence for several specific questions will be considered. In general, all of the study designs were minimally at Level II-2, with a few studies having a design at Level II-1. None of the studies were double-blinded, and all of the studies had relatively small numbers. The studies were from a variety of investigators. For each of the specific questions, there was more than one study supporting the recommendation. In terms of the risks of the treatment, no risk of treatment was identified in any of the studies, although identifying risk was not a specific objective of any study. It should also be noted that no other treatment or intervention for autism (i.e., speech therapy, traditional special education techniques) has been shown to have the same effects on either IQ or ability to transition to a regular school setting.

Evidence Assessment and Recommendations for EIBI Treatment of Autism

- Early Intensive Behavioral Intervention provided in a **home based setting** is effective in the treatment of autism in young children (Level II-1, 1 study; Level II-2, 2 studies. Quality of Evidence: Fair to Good. Recommendation: Level B)
- Early Intensive Behavioral Intervention provided in a **center based setting** is effective in the treatment of autism in young children (Level II-1, 1 study; Level II-2, 4 studies. Quality of Evidence: Fair to Good. Recommendation: Level B)
- Early Intensive Behavioral Intervention is effective in the treatment of autism in young children when provided for a **minimum of 2 years** (Level II-2, 3 studies. Quality of Evidence: Fair to Good. Recommendation: Level B)
- Early Intensive Behavioral Intervention is effective in the treatment of autism in young children when provided for a minimum of:
    20 hours per week (Level II-2, 2 studies. Quality of Evidence: Fair)
    30 hours per week (Level II-2, 4 studies. Quality of Evidence: Fair to Good)
    40 hours per week (Level II-1, 2 studies. Quality of Evidence: Fair to Good)

It is important to note that the optimal minimum number of hours per week necessary to produce significant changes has not been clearly delineated from the studies. The recommendations published by at least one panel of experts (Clinical Practice Guidelines NYS Dept. of Health, 1999) recommended evaluating individual child characteristics and needs, program features, and periodic assessments of progress, in order to individualize the therapeutic program. Similarly, while most of the studies demonstrated changes after a minimum of two years, a few studies showed significant changes in a subgroup of subjects prior to that time, and some children did not respond significantly even after several years of treatment. Again, prudent judgment is necessary when planning and evaluating results of individual treatment plans. Future studies in this area will be helpful to further refine recommendations.

Work Cited

1. American Psychiatric Association. (1994) Diagnostic and Statistical Manual of Mental Disorders, 4th ed. (DSM-IV). Washington, D.C: American Psychiatric Association.
2. Anderson, S. R. et al. (1987). Intensive home-based early intervention with autistic children. Education and Treatment of Children, 10, 352-366.
3. Asperger, H. (1944) Die "Autistichen Psychopathen" im Kindesalter. Archiv. Psychiatr. Nervenkr 117:76-136 (Reprinted in Autism and Asperger's Syndrome. Frith, U. ed. Cambridge, England: Cambridge University Press, 1991)
4. Bibby, P. et al. (2001). Progress and outcomes for children with autism receiving parent-managed intensive interventions. Research in Developmental Disabilities, 22, 425-447.
5. Birnbrauer, J. S., and Leach, D. J. (1993). The Murdoch Early Intervention Program after 2 years. Behaviour Change, 10, 63-74
6. Cohen H., et al. (2006) Early Intensive Behavioral Treatment: Replication of the UCLA model in a community setting. Developmental & Behavioral Pediatrics. 27, 145-155.
7. Eikeseth, S. et al. (2002). Intensive behavioral treatment at school for 4-7-year-old children with autism: A 1-year comparison controlled study. Behavior Modification, 2002, 49-68.
8. Eikeseth, S. et al. (2007). Outcome for children with autism who began intensive behavioral treatment between ages 4 and 7. Behavior Modification, 31(3), 264-278.
9. Eldevik, S. et al. (2006) Effects of low intensity behavioural treatment for children with a autism and mental retardation, J. Autism and Developmental Disorders, 36, 211-224.
10. Fenske, B. C. et al. Age at intervention and treatment outcome for autistic children in a comprehensive intervention program. Analysis and Intervention in Developmental Disabilities. 5, 49-58.
11. Harris, SL et al. (1991). Changes in cognitive and language functioning of preschool children with autism. J. Autism and Developmental Disorders, 21, 281-290.
12. Harris, SL, Handleman JS. (2000). Age and IQ at intake as predictors of placement for young children with autism: a four- to six-year follow-up. J Autism Dev Disord, 30(2), pp. 137-142.
13. Howard et al. (2005). A comparison of intensive behaviour analytic and eclectic treatments for young children with autism. Research in Developmental Disabilities, 26, 359-383.
14. Kanner, L. (1943). Autistic disturbances of affective contact. Nerv Child 2: 217-250.
15. Koegel, R.L. and Koegel, L.K. (1995) Teaching Children with Autism: Strategies for Initiating Positive Interactions and Improving Learning Opportunities. Baltimore: Brooks.

16. Lovaas, O. I. et al. (1981). Teaching Developmentally Disabled Children: The Me Book. Austin, TX: Pro-Ed.
17. 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.
18. Maurice, C. et. al. (Eds.). (1996). Behavioral Intervention for Young children with Autism. Austin, TX: Pro-Ed.
19. Maurice, C. (1993) Let Me Hear Your Voice: A Family's Triumph Over Autism. New York: Knopf.
20. McEachin, J. J., et. al. (1993). Long-term outcome for children with autism who received early intensive behavioral treatment. American Journal on Mental Retardation, 97, 359-372.
21. Remington B. et al. (2007). Early intensive behavioral intervention: outcomes for children with autism and their parents after two years. Am J Ment Retard, Nov, 112(6), pp. 418-438.
22. Sallows G. O. Graupner T. D. (2005). Intensive behavioral treatment for children with autism: four-year outcome and predictors. American Journal of Mental Retardation, 110(6), pp. 417-438.
23. Schopler, E. et. al. (1989). Relation of behavioural treatment to "normal functioning": Comment on Lovaas. Journal of Consulting and clinical Psychology. 57, 162-164.
24. Shattuck, P.T. and Grosse, S.D. (2007). Issues related to the diagnosis and treatment of autism spectrum disorders. Mental Retardation and Developmental Disabilities Research Reviews. 13: 129-135.
25. Sheinkopf, S.J., & Siegel B. (1999). Home-Based Behavioural Treatment of Young Children with Autism. Journal of Autism and Developmental Disorders, 28(1), 15 - 23.
26. Smith, T., Groen, A. D., & Wynne, J. W. (2000). Randomized trial of intensive early intervention for children with pervasive developmental disorder. American Journal on Mental Retardation, 105 (4), 269-285.
27. Smith T et al. (1997). Intensive behavioral treatment for preschoolers with severe mental retardation and pervasive developmental disorder. Am J Ment Retard, 102(3), 238-249.
28. National Research Council: Committee on Educational Interventions for Children with Autism (2001). Educating Children with Autism. Ed. Catherine Lord and James P. McGee, Division of Behavioral and Social Sciences and Education, National Research Council. Washington, D.C.: National Academy Press
29. New York Department of Health: Clinical Practice Guideline: The Guideline Technical Report – Autism/Pervasive Developmental Disorders, Assessment and Intervention for Young Children (Age 0-3 Years) (1999). New York: Publications, New York Department of Health

30. U.S. Surgeon General: Department of Health and Human Services (1999). Mental Health: A Report of the U.S. Surgeon General. Rockville, MD: Department of Health and Human Services, Substance Abuse and Mental Health Services Administration,

Center for Mental Health Services, National Institutes of Health, National Institute of Mental Health

31. Volkmar, F. et. al. (1999) Practice parameters for the assessment and treatment of children, adolescents, and adults with autism and other pervasive developmental disorders. J. of Am. Acad. Child. Adolesc. Psychiatry. 38: (12 Suppl), 32S-54S.