# Autism Spectrum Disorders Monograph Benjamin j. Springer, M.S. University of Utah

## Introduction

The universal symbol for autism awareness is a single puzzle piece: alone, fragmented, and out-of-place. Advocates for autism awareness are all too familiar with the significance of the symbol. Faced with limited resources and a sea of opinion, the puzzle of autism can seem overwhelming. Fortunately, some of the pieces are coming together. Increased public awareness, expanded diagnostic criteria, and advanced genetic detection methods have led to a definitive understanding of the disorder (Fombonne, 2005; Gernsbacher, Dawson, & Goldsmith, 2005; Kielinen, Rantala, Timonen, Linna, & Moilanen, 2004; Wing & Potter, 2002).

Autism, once considered to be a rare disorder, has now reached topical status on the national stage. Current prevalence rates for autism spectrum disorders are increasing (Fombonne, 2005) and estimates suggest that every one child in one hundred and fifty children are affected with an ASD (CDC, 2007). Some researchers posit that the rise can be explained by expanding diagnostic nomenclature, improved detection methods, and better public awareness (Fombonne, 2005) Whatever constitutes the increasing rates, as more children are diagnosed with an autism spectrum disorder, it becomes crucial to understand what treatments are most effective in helping to ameliorate the core symptoms children display.

#### Basic Definition and Classification

Autism may be described as a complex neuro-developmental disorder that affects all aspects of human neurological development. While impairments in social interaction and social reciprocity are the hallmark features of this disorder, attention, memory, communication, speech, cognitive patterns, and severe behavioral impairments are presented at high rates in the disorder as well. The social impairments are among the earliest heralds of autism. Delayed orienting to name, aversion to touch, poor visual orientation, and mouthing of objects differentiate children with autistic disorder from those with Down syndrome and from typical infants by 8-12 months (Baranek, 1999; Werner, Dawson, Osterling, & Dinno, 2000). Running parallel to these impairments are the absence or delay in the development of speech, together with preoccupation with one or few restricted interests that are atypical in either intensity or focus. Additionally, rigid adherence to specific, nonfunctional rituals or routines and repetitive and stereotyped motor mannerisms, such as hand-flapping, object manipulation, body rocking, or bizarre-looking whole-body movements are also present in the disorder (Newsom & Hovanitz, 2006).

According the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR), autism falls under the category of Pervasive Developmental Disorder (APA, 2005). Diagnostic criteria are met for this disorder when a total of six (or more) items from: 1) Qualitative impairment in social interaction, 2) Qualitative impairment in communication, and 3) Restricted repetitive and stereotyped patterns of behavior. The disturbance cannot be better accounted for by Rett's Disorder or Childhood Disintegrative Disorder. The DSM-IV-TR also includes the diagnostic criteria for Asperger's Disorder as 1) Qualitative impairment in social interaction, 2) Restricted repetitive and stereotyped patterns of behavior, and 3) Clinically significant impairment in social, occupational, or other important areas of functioning. The most notable differences between the diagnostic criteria of autism and Asperger's Disorder is that in the latter, there is no significant delay in language or cognitive development.

Empirical evidence supporting the difference between autism and Asperger's Disorder is currently lacking. Miller & Ozonoff (1997) found that the four cases Hans Asperger originally described would be diagnosed, according to DSM criteria, as having autism, not Asperger's Disorder. Research suggests that the use of early language delay as a differential criterion between autism and Asperger's has largely disappeared by early adolescence, (Eisenmajer, Prior, Leekam, Wing, Ong, Gould, and Welham, 1998; Ozonoff, South, and Miller, 2000). Despite the lack of support in the separate diagnostic criteria, there is evidence suggesting an autism spectrum. The spectrum is descriptive of the broad symptom patterns, range of abilities, and characteristics expressed in many different combinations and in any degree of severity (Lord, Cook, Leventhal, and Amaral, 2000).

The DSM-IV-TR criterion for autism is not the only important measure of diagnostic criteria. Autism is typically identified in children, and where there are children there are schools. Public schools are subject to federal laws that protect students with disabilities. For a child with autism to qualify as having a disability, the child must be deemed eligible for federal services by an educational classification of autism. The Individuals with Disabilities Education Improvement Act of 2004 (IDEA-04) Regulatory Definitions of Disability Classifications defines autism as "a developmental disability significantly affecting verbal and nonverbal communication and social interaction, generally evident before age 3 that adversely affects a child's educational performance." (2004). The classification continues, "A child who manifests the characteristics of "autism" after age 3 could be diagnosed as having "autism" if the criteria stated above are satisfied." An IDEA evaluation must use a variety of technically sound tools and strategies that assess the relative contribution of cognitive, behavioral, physical, and developmental factors. No single measure is sufficient, the data generated must include relative functional, academic, and developmental information, including information provided by the parent (2004). There is not a full population count of all individuals with an autism spectrum disorder in the United States, however, the Center for Disease Control and Prevention (CDC) and the Department of Health and Human Services provide data indicating approximately 1 in 150 children with an autism spectrum disorder in the U.S. (2007).

#### Assessment

The assessment of autism has proven to be intrinsically difficult as the spectrum of autism presents itself across a wide array of observable behaviors. Thus, it is helpful to know that autism may be defined as syndromal or idiopathic in nature. As autism co-occurs with several syndromes at a high rate, the syndromes are considered "causally" related to autism or "syndromal". Disorders and/or syndromes causally related to autism include any variety of chromosomal disorders, (e.g., Fragile X, 15q duplication, Rett disorder, del22q11, Ring 20). In addition to these chromosomal disorders, Tuberous sclerosis, cytomegalovirus, in-utero thalidomide or valproic acid exposure, and inherited metabolic disorders are also considered causally related to autism. When no such syndrome is present, the autism is considered idiopathic.

Once autism is understood as either syndromal or idiopathic, there are a variety of evidence based assessment tools to assist the in the appropriate identification and subsequent treatment of autism. Two "Gold Standard" measures include the Autism Diagnostic Interview-Revised (ADI-R) and the Autism Diagnostic Observation Schedule (ADOS), both published by Western Psychological Services. The ADI-R provides a structured and extensive standardized developmental history with excellent diagnostic validity. Standardized review of a detailed developmental history is paramount when considering the diagnosis of autism. As a "pervasive developmental disorder", autism can best be identified when structured data is gathered from caregivers regarding the developmental patterns of their child.

The ADOS is an instrument used for direct child observation that assesses the same areas of the ADI-R and even corresponds to the DSM-IV. The unique administration of the ADOS allows the examiner to "press" for behaviors critical to diagnosis and has excellent diagnostic validity as well. In addition to the ADI-R and the ADOS, there are a number of empirically validated behavior checklists and rating scales that assist in rating the severity and pervasiveness of autistic characteristics observed by caregivers and teachers, (e.g., Autism Behavior Checklist ABC b Krug et al., 1980).

Several standardized tests and checklists have been developed to assess the behavior of children with possible autism. These tests are also intended to further evaluate children in whom autism is considered possible (due to parent concerns, clinical cues, and/or positive screening test results).

These assessment instruments can be used in various ways in assessing children with possible autism. Sometimes these instruments can be used to determine if autism is likely, so that a decision can be made to seek a specific diagnosis. At other times, some of these assessment instruments may be used as part of the formal diagnostic process. Finally, in certain instances some of these instruments may be used to rate the severity of symptoms, which may be useful in assessing interventions, periodic monitoring of the child's progress, and assessing outcomes.

# Possible Causes and Contributory Factors

Charting the causes and contributory factors of autism continues to be a profoundly difficult task. Virtually no modern investigator proposes that there is "a" cause of "autism"; instead, most expect to conquer it bit by bit as the causes of characteristic traits or particular subtypes within the ASDs are found (Eigisti & Shapiro, 2003).

Current research in the etiology of autism focuses primarily on possible genetic aberrations and early environmental events, including the now controversial role of routine childhood vaccinations. Because ASDs are present from birth or present in the first 2-3 years of life, most investigators now believe that ASDs result from multiple genes that interact in a variety of combinations resulting in different symptom profiles and degrees of severity that are seen clinically (Folstein & Rosen-Sheildley, 2001.)

Ample evidence for the heritability of autism comes from twin and family studies. In twin studies, concordance for autistic disorder is found at least 60% and for all ASDs in 90% of monozygotic (identical) twins, while low rates (0-10%) are found in dizygotic (fraternal) twins (Bailey et al., 1995; Stodgell, Ingram, & Hyman, 2000). Studies involving families have indicated that 3-7% of the siblings of children with autistic disorder also have autism (a rate 50-100 times higher than the risk in un-affected families), and that 8% of the extended families include another member with autism (Smally & Collins, 1996).

Due to the high ratio of males to females with ASDs, extensive investigations of the sex chromosomes have been made. Two X-linked genes involved in synaptogenesis have been found to be associated with autistic disorder (Jamain et al., 2003). In addition, research into the transmission of the X chromosome from fathers to daughters has provided some insight regarding a possible protective factor that raises the threshold for expression of the autism phenotype in girls. As a result, fewer girls meet the threshold and develop autistic disorder, even if they have other genes rendering them susceptible (Skuse, 2000).

The possibility that ASDs may be the result of the interaction between genetics and early environmental insult remains uncertain. Some researchers hypothesize that ASDs may be the result of an inherited susceptibility combined with a "second hit" from another factor during gestation or infancy (Folstein & Rosen-Shieldy, 2001). Considering factors such as prenatal infection, aberrant immune response, and exposure to tetragens at critical periods of brain development (Rodier & Hyman, 1998), and vaccines or their components (Geier & Geier, 2003a; Wakefield et al., 1998) have all been proposed as possibilities.

While heated controversies exist around the potential "causes" of autism, it is clear that there is a strong genetic component to the disorder. What is also clear yet seemingly less explored when discussing the etiology of autism is the brain. Neuropsychological, neuroimaging, and autopsy studies reveal a variety of brain abnormalities in ASDs. Evidence of deficits in executive functioning, joint attention, emotion perception, and theory of mind point to frontal lobe and midbrain impairment (Dawson, Osterling, Rinaldi, Carver, & McPartland, 2001; McEvoy, Rogers & Pennington, 1993; Russel, 1998). Impairments in these areas significantly impact the most basic functions of daily life (i.e., organizing letters in a name, planning your breakfast, etc.). While identifying specific genetic contributions to autism is of paramount importance, the war on autism is fought on many fronts and an understanding of how the structures of the brain are impacted is of equal importance.

### Interventions

A very basic "Google" search with the words "interventions for autism" yields close to 5 million possible links on the internet. Of course some of these links are variations of one another, but the Google search is representative of a significant dilemma facing families affected by autism: In a sea of opinion, what works?

Fortunately, we are not limited to a Google search alone. A substantial body of research exists in the area of effective interventions for autism and autistic behaviors. Over twenty years have passed since Ivar Lovaas and his colleagues first published data indicating the "recovery" of almost 50% of a young group of children with autism, (Lovaas, 1987; McEachin, Smith, & Lovaas, 1993). While the extent of the "recovery" Lovaas claimed has been found difficult to replicate, there is no question that the "hope of recovery" has lingered since his first studies utilizing applied behavioral analysis were conducted. In a recent issue of the Journal of Clinical Child & Adolescent Psychology, authors Sally Rogers and Laurie Vismara of the M.I.N.D. Institute have established a comprehensive review of the empirical evidence supporting efficacy of early intervention for young children with autism (2008). The authors reviewed the literature of published papers on early intervention in autism published between 1998 and 2006. The papers reviewed had to involve comprehensive treatment approaches for children with autism, predominantly ages 5 or younger, using either controlled group designs or single-subject multiple baseline designs using three or more subjects with published data.

The Rogers and Vismara paper is extensive and will not be reviewed in its entirety here. However, Rogers and Vismara did make seven recommendations when locating appropriate interventions:

Treatment of unwanted or challenging behaviors should follow the principles and practices of
positive behavior supports (Carr et al., 2002; Horner, Carr, Strain, Todd, & Reed, 2002, for
review). These principles and practices include functional analysis, functional behavioral
assessments, instruction of replacement behaviors, and applied behavioral analysis (ABA).

Interventions focusing on communication should emphasize building spontaneous functional communication skills.

- Children with autism need to be engaged in meaningful (to the child as well as others), ageappropriate learning activities that are functional in multiple settings. "Naturalistic Teaching" approaches that begin with child choice and use intrinsic reinforcers.
- Effective early intervention includes a well-defined and coherent set of teaching plans for developing functional skills, fitted to the child's current developmental level. The early intervention must be delivered at a high frequency throughout the day.
- Peer interactions are a crucial part of the intervention programs for children with autism.
- Programs should assure efforts to generalization of new skills and behaviors. Generalization is
  fostered when the skills that are taught are functional and ecologically valid in natural settings
  and daily routines.
- Parents and family members need to be included in the intervention in a variety of ways, (e.g., setting goals, locating supports for themselves, and receiving training in effective ways, etc.)

The strength of these recommendations is found in the application of early intervention. Early detection and intervention of autism have been proven to have the largest effect upon the reduction of problem behaviors associated with autism, (i.e., attending skills, aggression, and communication). In the absence of early intensive behavioral intervention, only 1-2% of individuals with autistic disorder become "normal" in the sense there is little or no difference between them and children who have never been diagnosed with an ASD (Rutter, 1985). Ballaban-Gil et al. re-evaluated 45 clients with ASDs who had been seen as children in adulthood. Over 90% had persisting social deficits of varying severity. Language improved with age, although only about a third showed relative typical speech and comprehension. Only 11% held competitive jobs, and 16% worked in sheltered workshops (1996). As Ramey and Ramey (1998) noted, "...a widespread hope for early intervention (is) . . . that children could be placed on a normative developmental trajectory and thus continue to show optimal development after early intervention ends."

### Popular Non-validated Techniques

As noted earlier, there is no lack of "opinion" when it comes to interventions for autism. Everyone affected by autism is entitled to an opinion as long as no harm comes from it. Unfortunately, harm has occurred because some opinions have led to ineffective treatments perpetuated by fads or publicity instead of evidence-based research. In his review for the journal Pediatrics, Scott O. Lilienfeld highlighted both effective and ineffective treatments for autism (2005).

The effective treatments reviewed in the article mirrored those already discussed in the previous section. The ineffective treatments included fads such as Secretin hormone therapy, elimination diets (Gluten-Casein Free Diets, etc.), Vitamin B6 therapy, Facilitated Communication, and Sensory-Motor Integration. These treatments were deemed "ineffective" due to the lack of rigorous scientific investigation as defined by the American Psychological Association (i.e., absence of randomized, controlled trials or systematic within-subject designs, etc.). Lilienfeld admonishes: "A key challenge for the future will be to place the treatment of childhood psychopathology on firmer scientific footing by combating the spread of [scientifically questionable treatments] and facilitating the transport of scientifically supported interventions to the clinical community. To accomplish the latter goal, considerably more communication and collaboration between researchers and clinicians will be necessary" (2005).

Standard Case Study

Carlo is a 10-year-old male diagnosed with high-functioning autism/Asperger's Disorder by a school psychologist at the age of 8. Assessment methods included an extensive developmental and medical history, parent interview, Autism Diagnostic Observation Schedule (ADOS): Module 3, Wechsler Intelligence Scale for Children, Fourth Edition, (WISC-IV), Behavior Assessment System for Children, Second Edition (BASC-2), Gilliam Autism Rating Scale, Second Edition (GARS-2), Gilliam Asperger's Disorder Scale (GADS), and the Adaptive Behavior Assessment System, Second Edition (ABAS-II). Carlo is currently receiving special education services in a suburban public elementary school under the educational classification of autism. While Carlo performs well within the average range academically, he participates in weekly social skills groups and a structured recess program.

Carlo has been observed to have difficulty relating socially to peers and teachers and will occasionally engage in what seem to be unprovoked aggressive outbursts towards peers, family, and teachers. A functional behavior assessment (FBA) was conducted to determine the function of the aggressive outbursts. The aggressive outbursts were defined specifically as yelling at a high volume while grabbing the clothing near the collar/neck of another person. An assessment of possible antecedents or "triggering events" was conducted. At the time of the FBA, Carlo was not receiving any form of medication treatment, thus any possible interaction with medications was ruled out.

Possible antecedents at home included poor time management and lack of positive behavior supports. Carlo will refuse to stop playing video games and will often stay up as late as 11 p.m. on school nights. The lack of sleep may contribute to his agitated state at school. Possible antecedents at school included his classroom seating arrangement, pace of instruction, access to scratch paper and pencils, and the classroom routine preceding transitions to recess and lunch.

Carlo is seated near the coat rack and exit of the classroom. This position is also at an awkward angle from the teacher's desk. While the desks are close in proximity, it is difficult for the teacher to have Carlo in a line of sight. Carlo would benefit from consistent reinforcement of replacement behaviors such as "nice hands" and "nice voice" and may be at a disadvantage if the teacher has a difficult time keeping an eye on him.

The pace of instruction is also problematic for Carlo. The teacher lectures at a very slow pace with frequent interruptions by class members. As concepts are stretched along these time periods, Carlo quickly loses interest and begins doodling on scratch paper from within his tote tray. Once engaged in the drawing, Carlo is observably fixated on his drawings and ignores his peers and teacher.

Finally, prior to every major transition, (recess, lunch, going home, etc.) the teacher assigned students to pick up trash from the floor. Carlo was observed to struggle shifting from his drawings to participate in the clean up. Then, as the teacher's commands intensified, Carlo felt rushed to find his materials (i.e., coat, lunchbox, backpack, etc.). Carlo was observed to be visibly agitated (i.e., grimacing) when large groups of students brushed passed his desk to retrieve their materials. It is posited that Carlo preferred to have his materials before the rest of the children so he could move out of their way.

The current FBA suggests that Carlo acts aggressively to escape the unwanted demands placed on him (e.g., stop drawing, picking up trash, etc.). Thus, some simple adjustments to Carlo's environment are necessary to help reduce the number of possible antecedents of his aggressive behavior. First and foremost would be assisting his family create a positive behavior support through a token economy and highly preferred reinforcers (i.e., video game time, etc.) By setting positive goals to go to bed at a decent hour and rewards for ending video games within a time frame, Carlo would receive reinforcement for compliance and, possibly, a good night's rest.

At school, the primary adjustment would be another seating arrangement for Carlo--somewhere more visible enabling the teacher to reinforce positive replacement behaviors such as nice hands and a nice voice. Once Carlo is removed from the coat rack area, his new seating may relieve some of his anxiety about retrieving his materials. Additionally, Carlo's teacher could increase the pace of instruction by first stating clear objectives of the lecture, brief lecture times followed by roaming desk-to-desk instruction. Finally, Carlo should have limited or no accesses to scratch paper until his assignments are completed. As far as the "picking-up trash routine", perhaps the teacher could enlist a mere "5-second rule" for cleaning up as opposed to the typical five to seven minutes that were observed. Unless the classroom was particularly messy, the teacher may also consider abandoning the routine all together.

Determining possible antecedents as well as identifying appropriate replacement behaviors is paramount when trying to establish consistent consequences. Consequences for Carlo's positive behavior could consist of access to drawing materials and even extra time on video games! Conversely, consequences for Carlo's negative behavior could be a loss of access to those highly preferred activities. Data of the frequency of Carlo's aggressive outbursts after the FBA recommendations should be compared to those recorded prior to the adjustments to help identify the effectiveness of these interventions.

Addressing these behaviors should still only be one dimension of the services Carlo is receiving at home and school. Specific attention should be paid on assisting Carlo communicate his wants and needs as well as the generalization of specific social skills such as greetings and conversation skills.

#### References

American Psychiatric Association. (2000). Pervasive developmental disorders. Diagnostic and statistical manual of mental disorders (4th ed. Text revision, pp. 377-379). Washington, D.C.: Author.

Bailey, A., LeCouter, A., Gottesman, L., Bolton, P., Simonoff, E., Yuzda, E., et al. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. Psychological Medicine, 25, 63-77.

Ballaban-Gil, K., Rapin, I., Tuchman, R., & Shinnar, S. (1996). Longitudinal examination of the behavioral, language, and social changes in a population of adolescents and young adults with autistic disorder. Pediatric Neurology, 15, 217-223.

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.

Carr, E. G., Dunlap, G., Horner, R. H., Koegel, R. L., Turnbull, A. P., Sailor, W., et al. (2002). Positive behavior support: Evolution of an applied science. Journal of Positive Behavior Interventions, 4, 4-16.

Dawson, G., Osterling, J., Rinaldi, J., Carver, L., & McPartland, J. (2001). Recognition memory and stimulus-reward associations: Indirect support for the role of ventromedial prefrontal dysfunction in autism. Journal of Autism and Developmental Disorders, 31, 337-341.

Department of Health and Human Services Center for Disease Control and Prevention. (2002). Prevalence of the autism spectrum disorders in multiple areas of the United States, Surveillance Years 2000 and 2002: A Report from the Autism Developmental Disabilities Monitoring (ADDM) Network.

Eigisti, I. M., & Shaprio, T. (2003). A systems neuroscience approach to autism: Biological, cognitive, and clinical perspectives. Mental Retardation and Developmental Disabilities Research Reviews, 9, 205-215.

Eisenmajer, R., Prior, M., Leekam, S., Wing L., Ong, B., Gould, J., and Welham M. (1998). Delayed language onset as a predictor of clinical symptoms in Pervasive Developmental Disorders. Journal of Autism and developmental Disorders, 28, 527-533.

Folstein, S. E., & Rosen-Sheildley, B. (2001). Genetics of autism: Complex aetiology for a heterogeneous disorder. Nature Reviews, 2, 943-954.

Fombonne, E. (2005). Epidemiology of autistic disorder and other pervasive developmental disorders. Journal of Clinical Psychiatry, 66 Suppl 10, 3-8.

Geier, M. R., & Geier, D. A. (2003a). Neurodevelopmental disorders after thimersal-containing vaccines: A brief communication. Experimental Biology and Medicine, 228, 660-664.

Gernsbacher, M. A., Dawson, M., & Goldsmith, H. H. (2005). Three reasons not to believe in an autism epidemic. Current Directions in Psychological Science, 14(2), 55-58.

Horner, R. H., Carr, E. G., Strain, P. S., Todd, A. W., & Reed, H. K. (2002). Problem behavior interventions for young children with autism. A research synthesis. Journal of Autism and Developmental Disorders, 32, 423-446.

Jamiain, S., Quach, H., Betancur, C., Rastam, M., Colineaux, C., Gillberg, I. C., et al. (2003). Mutations of the X-linked genes encoding neuroligins NLGN3 and NLGN4 are associated with autism. Nature Genetics, 34, 27-29.

Kielinen, M., Rantala, H., Timonen, E., Linna, S., & Moilanen, I. (2004). Associated medical disorders and disabilities in children with autistic disorder: A population-based study. Autism, 8(1), 49-60.

Lilienfeld, S. O. (2005). Scientifically Unsupported and Supported Interventions for Childhood Psychopathology: A Summary. Pediatrics, 115, 461-464.

Lord C, Cook EH, Leventhal BL, Amaral DG (2000) Autism spectrum disorders. Neuron 28:355-363

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.

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

McEvoy, R., Rogers, S., & Pennington, R. (1993). Executive function and social communication deficits in young autistic children. Journal of Child Psychology and Psychiatry, 34, 563-578.

Miller, J.N. and Ozonoff, S. (1997) Did Asperger's cases have Asperger Disorder? A research note. Journal of Child Psychology and Psychiatry. 38. 247-251.

Newsom, C., & Hovanitz, C. (2006). The Nature and value of empirically validated interventions. In Jacobson, Fox, & Mulick Ed. Controversial Therapies for Developmental Disabilities. New Jersey: Lawrence Erlbaum Associates.

Ramey, C. T., & Ramey, S. L. (1998). Early intervention and early experience. American Psychologist, February, 109-120.

Rodier, P. M., & Hyman, S. L. (1998). Early environmental factors in autism. Mental Retardation and Developmental Disabilities Research Reviews, 4, 121-128.

Rogers, S., J. & Vismara, L. A., (2008). Evidence-based comprehensive treatments for early autism. Journal of Clinical Child & Adolescent Psychology, 37(1), 8-38.

Russel, J. (Ed.). (1998). Autism as an executive disorder. Oxford: Oxford University Press.

Rutter, M. (1985). Infantile autism and other pervasive developmental disorders. In M. Rutter \* L.

Hersov (Eds.), Child and adolescent psychiatry (2nd ed., pp. 545-566). Oxford: Blackwell.

Skuse, D. H. (2000). Imprinting, the X-chromosome, and the male brain: Explaining sex differences in the liability to autism. Pediatric Research, 47, 9-16.

Smalley, S. L., & Collins, F. (1996). Genetic, prenatal, and immunologic factors. Journal of Autism and Developmental Disorders, 26, 195-198.

Stodgell, C. J., Ingram, J. L., & Hyman, S. L. (2000). The role of candidate genes in unraveling the genetics of autism. International Review on Research in Mental Retardation, 20, 57-81. The Individuals with Disabilities Education Improvement Act of 2004, (IDEA-04) Regulatory Definitions of Disability Classifications: 34 C. F. R. § 300.7(c)(2004).

Transcript of the third presidential debate between Senators John McCain and Barack Obama in Hempstead, N.Y., as recorded by CQ Transcriptions. Oct. 15, 2008, New York Times.

Wakefield, A. J., Murch, S. H., Anthony, A., Linnel, J., Casson, D. M., Malik, M., et al. (1998). Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. Lancet, 351, 637-641.

Werner, E., Dawson, G., Osterling, J., & Dinno, H. (2000). Recognition of autism spectrum disorder before one year of age: A retrospective study based on home videotapes. Journal of Autism and Developmental Disorders, 30, 157-162.

Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: is the prevalence rising? Mental Retardation and Developmental Disabilities Research Review, 8(3), 151-161.