

PREDICTING AUTISM IN YOUNG CHILDREN
BASED ON SOCIAL INTERACTION AND SELECTED DEMOGRAPHIC
VARIABLES

By

Dana Kristina Princiotta

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DEDICATION

I dedicate this dissertation to my mother, Alison Princiotta, for supporting me through challenging graduate studies and for encouraging me to see the true value of education and learning above everything else. I also dedicate this dissertation to my loving partner, Paul.

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ABSTRACT

The purpose of the present study was to examine whether an autism diagnosis could be predicted by social interaction as measured by the Ghuman-Folstein Screen for Social Interaction in conjunction with selected demographic variables (i.e., sex, age, ethnicity, mother's educational level, and socio-economic status). Univariate and bivariate analyses were conducted to explore each predictor variable and to explore possible relationships between predictor variables and autism. Binary logistic regression was utilized to examine various models' ability to predict autism. The final model was able to correctly identify 74% of the cases. The GF-SSI was the greatest predictor of autism. The selected demographic variables were not significant predictors of autism. These results were discussed in relation to the literature on sex, age, ethnicity, maternal education and socio-economic status. Future directions for research were also discussed.

CHAPTER I

INTRODUCTION

This chapter summarizes the information regarding early detection of autism for young children using social interaction. The purpose of the study is also discussed.

Overview of Autism

Autism is characterized by the American Psychiatric Association (APA; 2000) as a combination of impairments in social interaction (e.g., poor eye contact, lack of gestures, lack of friendships, lack of empathy), communication (e.g., speech, echolalia, pronoun reversal, lack of pretend play), and in restricted and repetitive patterns of behavior (e.g., narrow interests, rigidity, and hand flapping) must be present. While these criteria form the basis for diagnosing children as having autism, phenotypic heterogeneity exists regarding the level of impairment in these areas (DiCicco-Bloom et al., 2006).

Autism must have an age of onset of not more than 36 months and meet the diagnostic criteria put forth by the APA *Diagnostic and Statistic Manual of Mental Disorders (DSM-IV-TR)*. Autistic Disorder, or “classic autism,” is included under the umbrella of pervasive developmental disorders (Ghaziuddin, 2005).

Autism may be accompanied by secondary characteristics, such as intellectual disability (ID), seizure disorders, sensory impairments, aggression, hyperactivity, and self-injurious behaviors (Ghaziuddin, 2005). The first detailed case studies describing children with autism were published separately by Leo Kanner and Hans Asperger in the early 1940s (e.g., Kanner, 1943; Asperger, 1944). Both described these children as having difficulties with socialization, communication, and performing routine rituals.

While differences existed between the Kanner's and Asperger's two groups of children and heterogeneity existed between all children, the latter common traits were noted in both Kanner's and Asperger's children.

Formal diagnostic criteria for autism appeared in the literature many years after the initial discussions by Kanner and others (Ghaziuddin, 2005). Autism was also often compared initially with childhood schizophrenia (American Psychiatric Association, 1980; Goldstein & Ozonoff, 2009), although this comparison began to decrease as more research on autism was published in the 1950s, 1960s, and 1970s.

Incidence and Prevalence of Autism

Kanner and other early researchers in the field estimated that autism took place in 2 - 4 per 10,000 children (Goldstein & Ozonoff, 2009). However, by the 1990s, prevalence rates increased to as many as 60 per 10,000 children (Goldstein & Ozonoff, 2009) and, most recently, as 110 per 10,000 for children who were between 3 and 17 years-of-age (Kogan et al., 2009). This suggests that approximately 1% of U.S. children have some form of autism spectrum disorder. Rates in other countries are also consistent with this latter prevalence level (e.g., Scott, Baron-Cohen, Bolton, & Brayne, 2002). In terms of sex, the occurrence of autism is four to seven times higher for males compared to females (e.g., Charlop-Christy, Malmberg, Rocha, & Schreibman, 2008; Centers for Disease Control, 2009). In addition, children in families with a sibling with autism spectrum disorder (ASD) have 50 to 100 times greater chance of having a second child with ASD (DiCicco-Bloom et al., 2006). Among the reason for the increasing prevalence rates in autism are such factors as changing diagnostic criteria, use of different

assessment methods, increased awareness of autism on the part of the public, greater access by parents to medical and psychological services agencies that provide diagnostic services, and/or the presence of a true increase across the U.S. in the occurrence of the disorder (Goldstein & Ozonoff, 2009).

Etiology of Autism

Originally perceived to be a psychogenic disorder—being related to a child's attachment to his or her parents---the etiological perspectives on autism have changed over the decades (Matson, 2007). The research connected to the various autism theories has revealed that there is no single cause of autism (Gupta, 2003). Currently, autism is considered to be either a prenatal or postnatal brain developmental disorder. Genetic, neurological and environmental factors are connected to the major theories in this area. In this regard, research using concordance rates between identical twins and siblings have tended to support genetic theories (e.g., DiCicco-Bloom et al., 2006). The research demonstrating a relationship between intellectual disability (ID) and autism has also supported neurological theories (Matson, 2007). Similarly, evidence supports certain environmental theories, with viral infections found to be one of the possible contributing factors (Ghaziuddin, 2005).

Assessment of Autism

Most children with autism receive a diagnosis between 4.5 and 5.5 years of age (Stone, Coonrod, & Ousley, 2000). This average age of diagnosis does not reflect the current research trends that indicate that a diagnosis can be made prior to two years of age and even as early as 18 months of age (CDC, 2009; Ghaziuddin, 2005). Children that

have been identified at these latter earlier ages have demonstrated appreciable deviance in social or language development when compared to typically maturing same-aged peers (Baranek, 1999). In this regard, after the first year of life, measures of social interaction have been found to be valid in identifying autism in young children (Ghuman, Peeble, & Ghuman, 1998).

Many children with autism are detected using comprehensive assessment techniques, including multiple data sources that include, but are not limited to, communicative, emotional, neuropsychological, and intellectual functioning domains (Goldstein & Ozonoff, 2009). The behavior of the child is also observed in multiple contexts as this provides the most information regarding the child's current level of social and emotional functioning. The combination of the *Autism Diagnostic Observation Schedule-General (ADOS-G)* and the *Autism Diagnostic Interview-Revised (ADI-R)* has been labeled as the "gold standard" in autism evaluation (Goldstein & Ozonoff, 2009).

Early Screening Measures for Autism

Screening measures have increased in utility as a result of the time needed and cost of a thorough autism evaluation (Goldstein & Ozonoff, 2009). In this regard, many screening measures have failed to obtain the level of sensitivity and specificity needed to distinguish children with ASD from those having other types of developmental or psychiatric disorders (e.g., Osterling et al., 2009). In addition, most autism screening instruments that have been developed have not been validated in community healthcare settings or shown adequate power in separating children with ASD from those without this disorder (Gray, Tonge, & Sweeney, 2008; Osterling et al., 2009). Without adequate

screening measures, autism evaluations tend to occur later in a child's life (4-5 years of age) or, in some cases early screening measures have been used but have often resulted in misdiagnosing the children (Matson, 2007).

Researchers have recently been highlighting a need for the screening of children prior to the comprehensive evaluations that often take place at the pre-school or grade-school age (Goldstein & Ozonoff, 2009). A screening process to detect children earlier than grade school can include same-aged peer comparisons to decrease the monetary and resource strains invested in a comprehensive assessment of autism. This can be accomplished with a universal screen for children of a certain age or be given only to those children that appear to be at-risk for atypical social development (Osterling et al., 2009). However, many current screening measures have failed to reach adequate levels of sensitivity and specificity in identifying children with autism (Osterling et al., 2009). In order to effectively implement programs of intensive instruction with children with autism, these children need to be identified as in need of such services (Osterling et al., 2009).

Screening measures like the *Checklist for Autism in Toddlers* (CHAT) (Baron-Cohen, Cox, Baird, Sweetenham, Nightingdale, Morgan, Drew, & Charman, 1996), *Modified CHAT* (M-CHAT) (Robins, Fein, & Barton, 1999), *Social Communication Questionnaire* (SCQ) (Rutter, Bailey, & Lord), *Screening Tool for Autism in Two-Year-Olds* (STAT) (Stone, Coonrod, & Ousley, 2000), *Pervasive Developmental Disorders Screening Test* (PDDST-II) (Siegel, 2004), *Early Screening for Autistic Traits* (ESAT) (Dietz et al., 2006), *Autism Spectrum Screening Questionnaire* (ASSQ) (Ehlers, Gillberg,

& Wing, 1999) have all focused on the early identification of children with autism, with some attempting to identify children prior to 18 months of age.

The *Ghuman-Folstein Screen for Social Interaction* (GF-SSI) was developed to measure social interactions in young children utilizing a parent-caregiver questionnaire. Unlike other screening measures, the GF-SSI focuses on typical development and has been found to distinguish children with autism who are between 2 and 6 years of age from those in the same age grouping who have a developmental disorder (Ghuman et al., 1998).

Demographic Variables Associated with Autism

Few studies have been reported in the literature regarding the contribution of broad socio-demographic factors to a diagnosis of autism (Hertz-Pannier et al., 2006). Knowing these factors could create better developmental outcomes for children with autism (Yirmaya & Ozonoff, 2007). For example, research has demonstrated that males are at least four times more likely to have autism than females, a finding that is one of the most stable demographic factors in the autism literature (Gupta, 2003). In regard to ethnicity, there is research that suggests that there is a decreased risk of autism in black or African-American families, although this finding is less consistent across various studies (Mandell et al., 2009). Age, on the other hand, appears to play a role in the manifestation of particular characteristics associated with autism, allowing researchers to distinguish autism at particular ages from other forms of pervasive developmental disorder (Yirmaya & Ozonoff, 2007).

While the GF-SSI was validated in a group of children from 2-6 years of age, it is not yet known if the GF-SSI can successfully predict children with autism from those without autism. In addition, the literature is not yet clear regarding how much the sex or ethnicity of a child contribute to a child being diagnosed as having autism. In addition, the literature is unclear regarding the degree to which socio-demographic factors, in conjunction with a social interaction screening measure will predict a diagnosis of autism versus other developmental disorders. The literature is also vague regarding whether a prediction model could be developed that identifies very young children who are at-risk for autism using an early-childhood screening measure and demographic risk markers.

Purpose of the Present Study

The purpose of the present study was to examine whether an autism diagnosis could be predicted as measured by the GF-SSI screening instrument in conjunction with information on selected demographic variables for each child (i.e., sex, age, ethnicity, mother's educational level, and socio-economic status). Given the inconsistent empirical support in the research literature on the validity of early screening instruments for children who have autism, the following research questions and, where relevant, predictive hypotheses were made:

Question #1: Are scores on the GF-SSI significant predictors of a diagnosis of autism?

Hypothesis #1: Scores on the GF-SSI significantly ($p < .05$) predict a diagnosis of autism.

Question #2: Which independent variable is the best predictor of an autism diagnosis?

Hypothesis #2: The GF-SSI will be the best predictor of an autism diagnosis in the final model.

Question #3: Which set of observed variables gives rise to the best prediction of autism?

Hypothesis #3: The GF-SSI and sex will be the significant predictor variables in this study.

CHAPTER II

REVIEW OF RELATED RESEARCH

This chapter provides an overview of the history of autism, current definitions, prevalence and incidence estimates, etiological theories, assessment techniques, and early detection screening measures.

Leo Kanner, a child psychiatrist at Johns Hopkins University, introduced the concept of autism in 1943. He described 11 children from the Johns Hopkins Hospital and the Child Study Center of Maryland. The children were having difficulty with forming relationships, communicating, and often performing routines and rituals (Kanner, 1943). These children had problems with appetite, did not “conform” to their parent’s body when held as infants, had “unusual” memory, and demonstrated a lack of interest in their parents (Kanner, 1943). Other defining features of these children included: excellent rote memory, delayed echolalia, literalness, and repetition of personal pronouns just heard. In regard to phenotypic characteristics, Kanner found the children to be rather typical; however, he did notice that the heads of the 11 subjects were relatively large (Kanner, 1943).

Kanner compared autism in these children to childhood schizophrenia, stating the following: (1)“the combination of extreme autism, obsessiveness, stereotypy, and echolalia brings the total picture into relationship with some of the basic schizophrenic phenomena” (Kanner, 1943, p. 248); (2) “in spite of the remarkable similarities, the condition differs in many respects from all other known instances of childhood schizophrenia” (Kanner, 1943, p. 248); and, (3) “while the schizophrenic tries to solve his

problem by stepping out of a world of which he has been a part and with which he has been in touch, our children gradually compromise by extending cautious feelers into a world in which they have been total strangers from the beginning" (Kanner, 1943, p. 249).

In addition to the characteristics of the children that he observed, Kanner studied the parents of these children. He reported that the majority of the parents of the 11 children were well educated and from affluent families. He, therefore, began to speculate whether the parents' intelligence played a role in their child's development. He also noticed that these parents had a limited interest in other people (Kanner, 1968). Moreover, in a later article (Kanner, 1949), he suggested that autism was correlated with lack of maternal warmth and that maternal frigidity could cause autism or schizophrenia, using the term, "refrigerator mother," to describe these mothers (Kanner, 1949). This characterization of the refrigerator mother lasted for approximately three decades (Goldstein & Ozonoff, 2009). Austrian pediatrician, Hans Asperger, described patients with whom he worked in a manner that mirrored many of the characteristics of the children that Kanner described (Frith, 1991). The major difference, however, between Kanner's and Asperger's children was that Asperger's children were high functioning. His first paper describing Asperger Syndrome was in 1944 (Frith, 1991). Uta Frith (1991) provides the only translation of Asperger's 1944 paper, "Autistic psychopathy in childhood." He describes the "autist" as "only himself and is not an active member of a greater organism which he is influenced by and which he influences constantly" (Frith, 1991, p. 38). The characteristics that Asperger described in one child (Fritz V.) include

the following: severe impairment in social integration, delayed milestones, having “no real love for anybody,” and speaking early—the latter characteristic being different from those described by Kanner (Frith, 1991). Although Asperger reported delays in the children he worked with, the level of the delays was not as profound as those described by Kanner. He also recognized the heterogeneity of the disorder, as did Kanner, indicating that: “not every case has every feature” (Frith, 1991, p. 67).

The similarities between these two disorders has led to several discussions in the literature regarding whether autism and Asperger’s are separate disorders or if they fall under a single spectrum (Matson, 2007). Although descriptions of autism were presented in the 1940s, formal diagnostic criteria for the disorder did not appear in the diagnostic literature until many years later (Ghaziuddin, 2005); the sole exception was “disintegrative psychosis” which was described in 1908 and was a disorder with a similar description to autism (Gupta, 2003).

History of the Diagnosis of Autism

As discussed in Kanner’s original 1943 article, prior to the 1970s, autism was often compared with childhood schizophrenia; with the distinction between the two not appearing formally in the literature until 1980 with the publication of the third edition of the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM-III) (American Psychiatric Association, 1980; Goldstein & Ozonoff, 2009). One of the distinctions in the DSM-III included the comment that children having autism, unlike those with childhood schizophrenia, are typically characterized by the presence of lower intelligence (Gupta, 2003). The distinction between these two diagnostic groupings were

further differentiated in 1987 with the publication of the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised* (DSM-III-R) (American Psychiatric Association, 1987), when autism was placed in its own category of pervasive developmental disorders (PDD) (Wing & Potter, 2009). In the DSM-III-R, the categories of PDD included the following autism categories: infantile, childhood-onset, atypical, and residual autism. The DSM-III-R replaced infantile autism with “autistic disorder,” removed childhood-onset autism as a diagnosis, and extended the age of onset from 30 to 36 months (Gupta, 2003; Wing & Potter, 2009).

In the *Diagnostic and Statistical Manual for Mental Disorder, Fourth Edition-Text Revision (DSM-IV-TR)* (2000), autistic disorder (sometimes referred to as “classic” autism), Asperger syndrome, Rett’s syndrome, childhood disintegrative disorder and pervasive developmental disorder not otherwise specified (PDD-NOS) were included under pervasive developmental disorders (Ghaziuddin, 2005). In addition to the term PDD which includes the latter terms, the construct “autism spectrum disorder” (ASD), is often utilized as an umbrella term to include the whole range of autistic symptoms from mild to severe, often depicting a child with autism, asperger syndrome or PDD (Ghaziuddin, 2005).

Characteristics of Autism

Autism is best described as a “biologically determined set of behaviors that occurs with varying presentation and severity, probably as the result of varying causes” (Goldstein & Ozonoff, 2009, p.5). The disorder is characterized by a combination of impairments in social interaction, communication, and in restricted and repetitive patterns

of behavior (APA, 2000). Within these three broad domains, an accumulation of six markers of social impairment (e.g., poor eye contact, lack of gestures, lack of friendships, lack of empathy), communication impairment (e.g., speech, echolalia, pronoun reversal, lack of pretend play) as well as restricted patterns of behavior (e.g., narrow interests, rigidity, and hand flapping) must be present (APA, 2000). The combination of markers from these areas should also be manifested in the form of at least two from social interaction, one from communication, and one from restricted patterns of behavior, respectively (APA, 2000). While these criteria form the basis for diagnosing children as having autism, there is also a phenotypic heterogeneity in the level of impairment in the domains of language, social interaction and restrictive or repetitive movements (DiCicco-Bloom et al., 2006).

Secondary Characteristics of Autism

The secondary characteristics that can co-occur with autism include one or more of the following: intellectual disability (ID), seizure disorders, sensory impairments, proprioceptive issues such as walking on tip-toes, short attention span, hyperactivity, impulsivity, aggressiveness, self-injurious behaviors, sleep disturbances, and temper tantrums. In addition, comorbid mental disorders that may be present are anxiety disorder, mood disorder, psychosis, and/or attention deficit hyperactivity disorder (ADHD) (Ghaziuddin, 2005).

The age of onset and the timing of assessment and diagnosis are important components for an appropriate diagnosis since the age of onset of autism is before 36 months of age. The DSM-IV-TR also requires that the diagnosis of autism prevail over

that of Asperger's syndrome, even if the language skills in the child are higher than for those reported for a diagnosis of autism (Ozonoff, Goodlin-Jones, & Solomon, 2007). In this regard, the DSM-IV-TR criteria for autistic disorder are presented below.

[As presented in the DSM-IV-TR (2000), a diagnosis of autistic disorder requires the following:] “A. A total of six (or more) items from (1), (2), and (3), with at least two from (1), and one each from (2) and (3).

(1) Qualitative impairment in social interaction, as manifested by at least two of the following:(a) Marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction (b) failure to develop peer relationships appropriate to developmental level (c) a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest) (d) lack of social or emotional reciprocity.

(2) Qualitative impairments in communication as manifested by at least one of the following: (a) Delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime); (b) in individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others; (c) Stereotyped and repetitive use of language or idiosyncratic language; (d) lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level.

(3) Restricted repetitive and stereotyped patterns of behavior, interests and activities, as manifested by at least two of the following: (a) Encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus; (b) apparently inflexible adherence to specific, nonfunctional routines or rituals; (c) stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole-body movements); (d) persistent preoccupation with parts of objects

B. Delays or abnormal functioning in at least one of the following areas, with onset prior to age 3 years: (1) social interaction, (2) language as used in social communication, or (3) symbolic or imaginative play.

C. The disturbance is not better accounted for by Rett's disorder or Childhood Disintegrative Disorder" (p. 75).

Since autism is included in the DSM-IV-TR under the umbrella of PDD, the descriptions of each PDD will be briefly outlined. There are many overlapping characteristics for these disorders as well as defining features, creating challenges in differential diagnoses for "classic" autism versus PDD.

Rett's Disorder

Rett's disorder is characterized as the onset of deceleration of head growth between 4 and 48 months with loss of previously acquired hand skills, the development of stereotyped hand movements, loss of social engagement, poorly coordinated gait, with impaired expressive/receptive language (APA, 2000). The greatest difference that distinguishes autism from Rett's is that the latter is only diagnosed in females whereas

autism is diagnosed mostly in males.

Childhood Disintegrative Disorder (CDD)

Childhood Disintegrative Disorder (CDD) is marked by the apparent typical development for two or more years, followed by clinically significant loss of skills before age 10 in social, adaptive behavior, bowel or bladder control, play, motor, or expressive/receptive language skills. Deficits must occur in at least two of these categories (APA, 2000). In addition, abnormalities in two of three areas must occur for social interaction, communication and restricted/repetitive behaviors.

Asperger's Syndrome

Asperger's syndrome or disorder is characterized as impairments in social interaction, specifically in the use of nonverbal behaviors, in peer relationships, in lack of sharing enjoyment with others, and in lack of social or emotional reciprocity. In addition to social interactions, impairments must occur in restricted/repetitive patterns of behavior, such as preoccupation with a particular interest, inflexible adherence to a routine, preoccupation with parts of objects, or repetitive motor mannerisms (at least one) (APA, 2000). Asperger's disorder distinguishes itself from autistic disorder with the absence of clinically significant general delays in language (e.g., single words used by age two years, communicative phrases used by age three years) (APA, 2000).

Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS)

This category is used when there is a severe and pervasive impairment in reciprocal social interaction or verbal and nonverbal behavior or interests, however, the criteria are not met for a specific pervasive developmental disorder, or for such mental

disorders as schizophrenia, schizotypal personality disorder, or avoidant personality disorder (APA, 2000).

Diagnosing Autism Versus Asperger's Syndrome

There is significant overlap of diagnostic criteria for autism spectrum disorders (Freeman, Cronin & Candela, 2002). Autism and Asperger's (AS) are both characterized as social disorders typically marked with behavioral problems, restricted range of interests, and an unusual response to sensory stimuli (Myles & Simpson, 2002). Deficits are common to AS and autism in the areas of imitation, imagination, awareness of social rules, nonverbal behavior, spontaneity, and stereotypic movements (Freeman et al., 2002). Diagnostically, a spectrum exists in each disorder and both are pervasive. Whether AS and autism are truly distinct disorders has not yet been established (Freeman et al., 2002). While common traits between autism and AS exist, marked differences also exist. Notably, the greatest disparity between the two is expressed as an individual with AS must possess typical intellectual development as well as typical language development (Myles & Simpson, 2002). In this regard, Matson (2007) suggests that instead of trying to differentiate children with autism from those with AS, it may prove more fruitful to place people along the spectrum in terms of severity.

Incidence and Prevalence of Autism

In 1943, at the time of Kanner's and Asperger's work, the prevalence rates for autism were estimated to be about 2-4 per 10,000 children (Goldstein & Ozonoff, 2009). By the 1990s, prevalence rates increased to as many as 60 per 10,000 children (Goldstein & Ozonoff, 2009). Most recently, however, the October 2009 issue of *Pediatrics* listed

the prevalence of parent-reported diagnosis of ASD for children in the U.S. as 110 per 10,000 for 3-17 year-olds (Kogan et al., 2009). The ratio for males to females in this *Pediatrics* study was 4:1, similar to other literature in the field. The Autism and Developmental Disabilities Monitoring Network of the Center for Disease Control (CDC) found that in a multi-site study in the US among eight year-old children there were rates as high as 10 per 10,000 in New Jersey. The CDC (2009) estimated the overall rates as between 1 per 100 and 1 per 300 children, suggesting approximately as many as 1% of US children have an ASD. Children in the 5-11 age range in the United Kingdom showed that ASD numbers might be significantly higher than previously thought and that the prevalence rates are higher nationally and internationally (Scott, Baron-Cohen, Bolton, & Brayne, 2002). Around the world, studies in Asia, Europe and North America report a rate of 0.6% to 1% of the child population (CDC, 2009).

Autism occurs more often in boys than in girls with a ratio between 4:1 and 7:1 (Charlop-Christy, Malmberg, Rocha, & Schreibman, 2008; CDC, 2009). Children in families with a child with ASD have between 50 and 100 times greater chance of having another child with ASD, with an 8% recurrence rate (DiCicco-Bloom et al., 2006). Researchers have identified significant racial and ethnic disparities for ASD prevalence. For example, Black and Hispanic children have been found to be less likely to have a diagnosis of ASD than their Caucasian counterparts (Mandell et al., 2009). Despite such findings, ASD does appear to cross ethnic, racial, social-economic, and international boundaries.

Some writers have suggested that the number of children reported to be labeled as having an intellectual disability has shifted downward as the frequency of reported autism has shifted upward, hypothesizing that autism has included some of the children that were previously being diagnosed as having an intellectual disability (e.g., Goldstein & Ozonoff, 2009). In this regard, most researchers agree that there is still a complicated issue as to whether there is a greater number of children with autism or whether the numbers are a product of other variables (Wing & Potter, 2009). More specifically, while the incidence of autism appears to be on the rise, there are difficulties in distinguishing the age of diagnosis with the age of onset (Goldstein & Ozonoff, 2009). An added difficulty arises as incidence rates for autism are often confused with incidence rates for ASD, which includes Asperger's syndrome (Wing & Potter, 2009).

There are five major reasons that could contribute to a perceived increase in the incidence of autism. First, there have been changes over the years in the diagnostic criteria that have been used; currently, the observation of behaviors and the detailed developmental history encompass the bulk of the assessment of autism. Second, different diagnostic procedures are used across studies; for example, some studies used the DSM-IV-TR to diagnose the disorder while others used the International Classification of Disorders (ICD) yielding different proportions of diagnosed children. In addition, some studies used different target population sizes and methods of case findings (Wing & Potter, 2009). Third, there has been increased awareness among parents and educators over the years regarding the criteria for autism. For example, in 1991 autism was included in the *Individuals with Disabilities Education Act* (IDEA; 1991), and this point

in time coincides with an increase in reported cases of autism (Wing & Potter, 2009). In addition to the growing interest and awareness on the part of educators, parent support groups were created in the 1960s to increase awareness of autism and promote and support research in the field (Goldstein & Ozonoff, 2009). Fourth, as autism specialty services in the medical, psychological and educational fields increased beginning in the 1980s, prevalence also increased. A fifth reason may be related to the fact that there is an actual rise in the incidence in autism independent of the above possible explanations (Goldstein & Ozonoff, 2009).

Comorbidity and Autism

In children with PDD-NOS, ADHD is usually diagnosed as a comorbid disorder for those receiving psychiatric care (Ghaziuddin, 2005). Often, children who have been diagnosed with autism have been misdiagnosed as having ADHD (Ghaziuddin, 2005). The distinction between the two disorders can be ambiguous—especially in the inattentive subtype of ADHD (Ozonoff, Goodlin-Jones, & Solomon, 2007). Despite overlaps in diagnostic criteria, the DSM-IV-TR does not allow the diagnosis of ADHD in individuals with autism (Ozonoff, Goodlin-Jones, & Solomon, 2007). More specifically, the DSM-IV-TR states that ADHD “does not occur exclusively during the course of a Pervasive Developmental Disorder” (APA, 2000, p. 85). This is problematic in that the diagnosis can then never be given since the course of PDD is life course persistent (Ghaziuddin, 2005). Clinically this can be a problem because the hyperactivity and inattention of a child may worsen, but they could not receive the ADHD diagnosis and subsequent services (Ghaziuddin, 2005). It has been recommended that children with

ADHD who do not respond to the typical treatment interventions be screened for ASD. (Ghaziuddin, 2005).

The most likely disorder to co-occur with autism is ID. Approximately 75% of children with autism also have an ID (Charlop-Christy, et al., 2008). Of these individuals, half are within the range of mild to moderate ID and the other half functions in the severe to profound range (Ozonoff, Goodlin-Jones, & Solomon, 2007). The spectrum of intelligence and heterogeneity under a diagnosis of autism can create difficulties in differential diagnosis with ID.

Etiology of Autism

Perspectives on the etiology of autism have changed over the decades. Kanner (1943) first believed that autism was a psychogenic disorder that was related to a child's attachment to his/her parents and to personality characteristics describing the parents. For example, the term "refrigerator mother" was used to denote a neurotic mother who acted coldly towards her child, instilling a lack of intimacy that he or she would mimic to others. It was not until the 1960s that this theoretical view was deleted from Kanner's theory of autism and the theoretical understanding was placed more on the child (Kanner, 1973). In this regard, etiological research since the 1960s has not yet revealed a single cause of autism—making it a multifactorial disorder. What researchers have concluded to date is that it is now considered a disorder of prenatal or postnatal brain development (e.g., DiCicco-Bloom et al., 2006). In addition, no single animal model has been found that captures all of the phenotypic diversity of this disorder while including the organismic, cellular, and molecular variables (e.g., DiCicco-Bloom et al., 2006). Rather,

autism appears to be a pervasive development disorder that still has an unknown etiology. A discussion follows of some of the most popular theories.

Genetic Theories of Autism

Genetic causes are those in which mutations in genes lead to damage in certain brain centers (Moran, 2004). Geneticists propose that genetic mutations result in the atypicality seen in ASDs. Abnormalities have been suggested for the C4B “null allele,” extended HLA Haplotype B44-S30-DR4, HVR-3 sequence 1, Hox A1 gene, as well as Chromosome 15 (Bristol, 2001). Autism may also co-exist with Fragile X (FraX) since the gene may be loosely linked to that of the Fragile X marker, making autism likely to be a polygenic disorder (Gupta & State, 2007; Autism Genetic Resource Exchange, 2009). Furthermore, there is no chromosomal anomaly associated with autism. The genetic basis for autism is supported by twin studies in which the heritability has been estimated as greater than 90% (Gupta & State, 2007). Research from the 1970s and 1980s yielded studies in which the concordance of autism among monozygotic (MZ) twins was higher than that for dizygotic (DZ) twins (Matson & Minshawi, 2006). In 1995, Bailey and colleagues confirmed previous findings of autism among twins, with a 60% concordance rate among MZ twins and a 5% rate among DZ twins. Currently, the concordance rate among siblings for autism is about 3-6%. Depending on the population prevalence in comparison, this can translate to a sibling prevalence rate at 100 times greater (Matson & Minshawi, 2006). Some studies have targeted siblings of children with ASD in an attempt to lower the age of diagnosis (Yirmiya & Ozonoff, 2007).

Neurological Abnormalities

As indicated previously, approximately 75% of children with ASD also have an ID, suggesting a neurological etiology (Matson & Minshawi, 2006). The most common neurological disorder connected with ASD is epilepsy, with an occurrence rate of 18-42%, suggesting a common genetic basis between the two (Matson & Minshawi, 2006). Postmortem studies have demonstrated evidence of brain abnormalities in children with autism (DiCicco-Bloom et al, 2006). Fragmentation within the typical development of the brain is purported to occur in children with autism by DiCicco-Bloom and colleagues. There are significant abnormalities present in children with autism versus typical controls as seen through electroencephalography (EEG), computed tomography (CT), and magnetic resonance imaging (MRI) techniques. Differences can readily be seen in the gray and white matter with approximately 10% more mass in the brains of children with autism (DiCicco-Bloom et al., 2006). This excess brain tissue can be seen by 3-4 years of age in MRIs (DiCicco-Bloom et al, 2006). Termed macroencephaly, children with autism typically have head circumferences in the 97th percentile (i.e., the brain growth phenotype) (DiCicco-Bloom et al., 2006). MRI has been an instrumental tool in studying more than brain mass; hypoactivation in the fusiform face area (FFA) has been noted, which is integral to perceiving faces (DiCicco-Bloom et al., 2006). A third use with MRI has been the examination of social interaction and perception with children with autism. The findings indicate that there are abnormalities in the neural systems of these children (DiCicco-Bloom et al., 2006). More specifically, the mirror neuron system (MNS) can provide clues into the early development of autism, including deficits in social communication, “theory of mind” (e.g., the ability to represent mental states; failing tests

of false belief understanding) and imitation (Dapretto et al., 2006; Happe, 1995). Mirror neurons are motor neurons that fire when the person observes the actions of other people (DiCicco-Bloom et al., 2006). Dapretto and colleagues investigated whether the MNS would have an effect on the Autism Diagnostic Observation Schedule-Generic (ADOS-G) and the Autism Diagnostic Interview (ADI-R) measures. They found that the lack of MNS activity in imitation and observation during emotional expressions in children with autism demonstrates the dysfunction in the MNS (Dapretto et al., 2006). A second look at theory of mind tasks shows that there are deficits in the medial prefrontal and amygdaloid during tasks in which the child with autism must take someone else's perspective (DiCicco-Bloom et al., 2006).

Considering neurochemical factors, neurotransmitters like serotonin have been highlighted in the literature. Anderson and Hoshimo (1987) found serotonin blood levels to be 17-128% higher for people with autism compared to controls (Matson & Minshawi, 2006). Serotonin is responsible for eating, mood regulation, sleeping, and sex. In persons with autism, the dentatohalamicocortical pathway is affected by abnormal serotonin levels, which is responsible for sensory integration and speech, both problem areas for children with autism. In addition to serotonin, it has been proposed that heightened levels of dopamine in the brain may be responsible for increases in restricted, repetitive, stereotyped behaviors in children (Nebel-Schwalm & Matson, 2009).

Environmental Factors

Although autism is considered a genetic disorder, there are environmental factors that have been associated with the disorder. Purported prenatal factors include the

maternal use of anti-convulsants or thalidomide (a sedative-hypnotic medication), maternal viral infections, folic acid, and teratogens (Bristol, 2001). Postnatal factors have included vaccines (e.g., measles, mumps and rubella vaccination and the mercury-based vaccine preservative), autoimmune disease, and viral infections (Ghaziuddin, 2005; Bristol, 2001) though vaccinations have now been deleted as a possible factor (Nebel-Schwalm & Matson, 2009). In regard to vaccines, a report from the Institute of Medicine of the National Academies stated, "The overwhelming evidence from several well-designed studies indicates that childhood vaccines are not associated with autism" (as cited in Moran, 2004, p. 44).

Assessment of Autism

The median age of diagnosis for autism is currently 4.5 to 5.5 years of age (CDC, 2009; Stone, Coonrod, & Ousley, 2000). Recent research, however, indicates that symptomology of autism may be present prior to 18 months of age, and that a reliable diagnosis can be made by two years of age (CDC, 2009; Ghaziuddin, 2005). While assessing a child prior to three years of age may be more challenging, the typical markers of autism may be present—namely, social and language impairments--that differentiate autism from other developmental disabilities (Matson, 2007). In certain cases, a retrospective video analysis can show signs of autism earlier than a formal assessment (CDC, 2009). A diagnosis of autism requires appreciable impairments relative to typical children in social interactions (i.e., joint attention, offering comfort or sharing), which occur between 18 and 36 months, leading researchers to reconceptualize the earliest age at which autism can be detected. In a typical child, the expectations for social behavior

increase in accordance with the child's developmental level, suggesting that children below two would be more difficult to assess for a lack of social skills (Baranek, 1999).

The assessment of autism requires the collection and utilization of multiple data sources. A thorough assessment for autism includes neuropsychological, communicative, behavioral, emotional, and intellectual functioning (Goldstein & Ozonoff, 2009). Observations, standardized tools and interviews are typically utilized in the assessment of autism (Shriver, Allen, & Matthews, 1999).

Much of the assessment of a child suspected of having autism involves behavioral observations in natural and contrived settings (Shriver et al., 1999). Structured observations as well as observations during free play provide valuable information to the observer for comparisons with a typically developing peer. Another form of observation involves the use of retrospective video analysis. In 1999, Baranek utilized home videos that were edited to include ten minutes of experiences with 9 to 12 months-old children. Baranek's assistants who coded the footage without knowing the purpose of the study, were able to identify subtle symptoms of autism in these infants, suggesting that the observation of sensory-motor functions can demonstrate differences in children with autism versus those without the disorder (Baranek, 1999). In this study, the greatest predictor for a later diagnosis of autism in the video analyses was looking at other people, namely, that children having autism had less eye contact with others. This video analysis provided an avenue for researchers to obtain a novel piece of information about the child in question, in addition to parental interviews of the child's previous behaviors as an infant (Baranek, 1999).

In 2000, the *Autism Diagnostic Observation Schedule-Generic (ADOS-G)* was developed to systematically observe behavior and to addresses social interaction, communication, and play (Lord et al., 2000). The examiner selects one of four modules based on the level of language the child possesses (ranging from non-verbal to fluent). The ADOS-G takes approximately 30-45 minutes to administer, with structured play activities and observations incorporated in the assessment. A summary diagnostic algorithm is derived from activities that look at social interactions, communication, and restricted behaviors, and has the capacity to distinguish children with autism from those without. Excellent interrater and test-retest reliability (0.84 and 0.79, respectively), and good internal consistency (0.78 to 0.89) have been reported. According to Lord et al. (2000) algorithmic specificities and sensitivities for autism and PDD-NOS were excellent (Lord et al., 2000).

The combined application of the ADOS-G and *Autism Diagnostic Interview-Revised (ADI-R)* incorporates information from various sources and settings (Le Couteur, Haden, Hammal, & McConachie, 2008). In this regard, Le Couteur et al. (2008) found generally good agreement between the ADOS-G and the ADI-R for assessing autism (Goldstein & Ozonoff, 2009).

Interviews with parents/caregivers or school personnel provide the developmental and medical histories of the child with indicators of the child's typical versus atypical development (Shriver et al., 1999). The ADI-R is part of the "gold standard" in the assessment of autism when used in conjunction with the ADOS-G, according to Goldstein and Ozonoff (2009). The ADI-R examines the areas of communication, play,

interests, behaviors and social development in a child via a semi-structured interview for caregivers, based on an algorithm (Moore, Titcomb, Goodson & Rolles, 1998). The ADI-R is a standardized, reliable and valid instrument in assessing autism in preschool children. The capacity to distinguish children with an ASD from those without was 1.0 for sensitivity and 0.97 for specificity (Lecavalier et al., 2006). For example, differential diagnosis of autism from CDD and Rett's disorder at an early age can be accomplished with the ADI-R (Matson, 2007).

The *Childhood Autism Rating Scale (CARS)* is a 15-item behavioral rating scale for use with a child older than two years of age. The *CARS* was developed by the Treatment and Education of Autistic and Related Communication Handicapped Children (TEACCH) program at the University of North Carolina. Each item rates a particular behavior, ability, or characteristic on a seven-point scale, measuring the degree to which the child's behavior deviates from a typical child. The outcome represents a total score in which the severity of the disorder is denoted as "severe," "mild/moderate" or "no diagnosis" (Schopler, Reichler, & Renner, 1986). The *CARS* is an empirically valid instrument that obtained three types of reliability: internal consistency (0.94), interrater (0.71), and test-retest (0.88). Criterion-related validity was 0.80 in relation to expert clinical judgments. The *CARS* has an extensive history as a screening measure that includes "norm" groups and has proven superior to the *Autism Behavior Checklist (ABC)* and *Diagnostic Checklist* in distinguishing between children with autism and children with ID. However, the *CARS* does not use the DSM-IV-TR criteria and does not

distinguish autism from other ASDs, making it more useful as a screening tool rather than part of a comprehensive evaluation (Matson, 2007).

Parents, teachers and professionals can identify children with autism aged 3-22 years with the *Gilliam Autism Rating Scale (GARS)*. The GARS follows the criteria as outlined in the DSM-IV-TR. Validity and reliability of this measure are high, with reliability coefficients ranging from 0.80 to 0.90 for internal consistency, test-retest, and inter-score reliability (Matson, 2007).

The findings vary regarding the stability of the diagnosis of autism. Diagnostic stability from ages 2 to 9 was high for autism but not for PDD-NOS, according to Lord et al., 2006. Most studies have reported that a diagnosis of autism is stable from 2 to 3 years of age, but that children that were diagnosed at two years of age showed limited continuity in symptom severity by seven years of age. A diagnosis of autism at five years of age, however, is stable up to late adolescence (Lord et al., 2006). For 112 children assessed at five years of age, stability was 88% from ages 5 to 9 years (Lord et al., 2006). More than half of the children that received a diagnosis of PDD-NOS at the age of two years of age had obtained a label of autism by nine years of age (Lord et al., 2006).

Many factors and confounding variables contribute to the difficulty in assessing autism. The spectrum of intelligence that is under the diagnosis of autism creates difficulty in differential diagnosis with overlaps with severe ID, Rett's disorder and CDD on the lower end and Asperger's disorder, learning disabilities and schizophrenia at the higher end of the spectrum (Matson, 2007). Issues such as age, ID, problematic behaviors, lack of eye contact, lack of reciprocity, etc, may all contribute to difficulties in

initially assessing children with autism. Another challenge lies in the fact that children younger than three years are not very likely to want to interact with a stranger and play activities that are directed by an adult (McConachie, Le Couter, & Honey, 2005). Pragmatic reasons point to why 5 - 6 years of age is an important time to screen for disorders as children are entering school and are required to sit for extended periods, follow many rules, and interact with many children and adults with whom they were previously unfamiliar (Matson, 2007).

Utility of Screening Measures

Osterling, Swinkels, Jan van der Gaag, Visser, Dietz, and Buitelaar (2009) compared different screening instruments in more than 200 children to look at the power in discriminating ASD from non-ASD (2009). Many screening measures fail to obtain the level of sensitivity and specificity needed to distinguish children with ASD from children with abnormal development (Osterling et al., 2009). Many of these instruments have not yet been validated within community healthcare settings as they were intended (Gray, Tonge, & Sweeney, 2008). According to Osterling et al., 2009, no individual item or instrument has shown adequate power in discriminating children with ASD from non-ASD. The general lack of screening for ASD leads to diagnoses late into adolescence or misdiagnosis into adulthood (Matson, 2007). Currently, most of the early detection tools are not utilized prior to two years of age and often are not administered until 6 years of age (Matson, 2007).

Current Assessment Screening Measures for Autism

Prior to the comprehensive assessment for autism, a screening process may be initiated to identify children who are at risk for having autism in comparison to other children. This screening process is initiated in order to decrease the monetary costs and time involved in conducting a comprehensive assessment (Osterling et al., 2009). There are two major screening methods through which autism can be detected early: (1) a systematic population screen for all children between specific ages and conducted by a primary care provider and (2) a specific screening instrument for autism applied only to those children who deviate from a typical developmental pathway (Osterling et al., 2009).

Checklist for Autism in Toddlers (CHAT) (Baron-Cohen, Cox, Baird, Sweetenham, Nightingdale, Morgan, Drew, & Charman, 1996). This instrument was designed to be a screening tool for autism in children as young as 18 months. The CHAT was designed with the intent of early intervention for these children. This screen has been successful in detecting lack of pretend play, protodeclarative pointing, social interest, social play, and joint attention (Baron-Cohen, Allen, & Gillberg, 1992). Baron-Cohen and colleagues also demonstrated that consistent failure for three specific items on the CHAT at 18 months indicated an 83% chance of an autism diagnosis later (Baron-Cohen, et al., 1996). A six-year follow-up study indicated high specificity (0.98) but low sensitivity (0.38) (Gray, Tonge, & Sweeney, 2008).

Modified CHAT (M-CHAT) (Robins, Fein, & Barton, 1999). This screening instrument was created to eliminate the portion of the CHAT to be completed by a healthcare professional making it more accessible for non-healthcare professionals, while still targeting children over 18 months. A child is considered to have failed this screen

when he or she fails two out of six critical items or any three items. The reported specificity and sensitivity (0.97 and 0.95, respectively) are high but the screen was not used in a community setting as purported by the instrument developers (Gray, Tonge, & Sweeney, 2008). In addition, there is a negative relationship between the M-CHAT and measures of cognitive and adaptive functioning, suggesting that this screening would not be a good measure for children that are high functioning (Gray, Tonge, & Sweeney, 2008).

Social Communication Questionnaire (SCQ) (Rutter, Bailey, & Lord). The SCQ uses questions that are derived from the items included in the ADI-R. It evaluates social functioning and communication skills and is composed of 40 yes/no questions to be answered by parents for children over four years of age (Gray, Tonge, & Sweeney, 2008). The sensitivity and specificity were low for most ages (0.71 and 0.72, respectively), as reported by Eaves et al. (2006).

Screening Tool for Autism in Two-Year-Olds (STAT) (Stone, Coonrod, & Ousley, 2000). This instrument was designed with early detection in mind as a second-level screen. Twelve items are administered within a play-like interaction for a time period less than 20 minutes. The STAT uses four social-communicative domains: directing attention, play, requesting, and motor imitation (Stone et al., 2000). The STAT was found to have high inter-rater and test-retest reliability, as well as high agreement for diagnosed children with the ADOS-G, according to Matson, 2007, and a sensitivity of 0.92 and a specificity of 0.85 according to Stone et al. (Matson, 2007; Stone et al., 2000).

Pervasive Developmental Disorders Screening Test (PDDST-II) (Siegel, 2004).

The PDDST-II was normed on pre-school children with ASDs. The screen takes approximately 10 minutes to determine whether a child should be referred for additional assessment. The caregiver completes the screen and the clinician follows-up with probe questions that take approximately five minutes. There are three versions of the PDDST-II: the Primary Care Screener (PCS), the Developmental Clinic Screener (DCS) and the Autism Clinic Severity Screener (ACSS). The sensitivity and specificity for the PCS was 0.92 and 0.91, for the DCS (0.73 and 0.49), and 0.58 and 0.60 for the ACSS.

Early Screening for Autistic Traits (ESAT) (Dietz et al., 2006). This instrument was developed to retrospectively examine children at 14 to 15 months of age and focuses on play activities. This 14-item tool is purported to differentiate children with ASD from non-ASD in children, but not below 24 months (Swinkel et al., 2006).

Autism Spectrum Screening Questionnaire (ASSQ) (Ehlers, Gillberg, & Wing, 1999). The ASSQ is one of two instruments specifically developed to screen for ASDs with primary grade children with an emphasis on screening for Asperger syndrome and other high functioning autism. However, Posserud et al. (2009) have also demonstrated that the ASSQ successfully screens for ASD in lower functioning children (Posserud, Lundervold, & Gillberg, 2009).

Ghuman-Folstein Screen for Social Interaction (GF-SSI) (Ghuman, Freund, Reiss, Serwint, & Folstein, 1998). This instrument is a parent-caregiver questionnaire designed to measure social interactions in young children. Fifty-four items based on the ADI-R and Vineland Adaptive Behavior Scales measure reciprocal social interaction

taking into account the child's cognitive level (Ghuman et al., 1998). In contrast to other screening tools, the GF-SSI focuses on typical social development to detect children with autism. This measure has been found to distinguish children with autism from children with other developmental disabilities (Ghuman et al., 1998). The psychometrics of the GF-SSI were the following: .99 for test-retest reliability, .80 for interrater reliability, and .67 for construct validity (Ghuman et al., 1998).

Social interaction is one of the primary or secondary aspects of developmental disabilities as well as some psychiatric disorders (Ghuman, Peebles, & Ghuman, 1998). Very young children with autism are distinguished from other children with atypical development by social behaviors and communication skills (Eaves & Helena, 2004). In further support, social deficits and delays in language were the two greatest indices from the DSM-IV evidenced by young children with autism (Stone et al., 1999). Social impairments are central to autism as evidenced in the high endorsement by clinicians and alterations to diagnoses in alliance with number of symptoms in the social domain (Stone et al., 1999).

Whereas stereotyped or ritualistic behaviors may not be concerns stated by parents during early development, delays in social interaction and communication will be present earlier (Baranek, 1999). After the first year of life, measures of social interaction have been found to be valid in detecting autism in young children (Ghuman et al., 1998). A parent's knowledge and awareness of deficits becomes more apparent as the child ages and same-aged peers provide comparisons of typical development (Baranek, 1999). Social interactions have three major functions in young children: behavior regulation

(e.g., requesting), joint attention (e.g., indicating), and affiliation (Ghuman, Peebles, & Ghuman, 1998). Further, for both the early detection and diagnosis of autism, lack of social or emotional reciprocity is a salient feature in need of further evaluation.

Demographic Variables Associated With Autism

Contributing factors and causes of autism are not fully understood at this point in time (Hertz-Pannier et al., 2006). Few studies have incorporated broad socio-demographic factors that may affect the development of autism (Hertz-Pannier et al., 2006) and create better developmental outcomes for children with autism (Yirmaya & Ozonoff, 2007). In addition to the tools utilized to screen for autism versus other developmental disorders, certain demographic variables of children and their families have been attributed to a diagnosis of autism versus other developmental disorders. These demographic variables will be discussed in detail as they relate to a diagnosis of autism.

Age and Autism

As indicated in the DSM-IV-TR, the characteristics of autism must be present by three years of age. This criterion is important for distinguishing autism from Asperger's, since the criteria for Asperger's does not include a maximum age of onset. Johnson, Siddons, Frith, and Morton, (1992) found no significant differences between children at 6 and 12 months of age in regard to autism symptomology. Although Johnson et al. (1992) and Baranek (1999) note that autism can be detected on the basis of social delays by 12-18 months, according to some studies, pediatricians have not always referred children suspected of autism until 15.5 months following the parents' stated concerns (Mandell,

2002). In addition, other characteristics associated with autism like restricted and stereotyped behaviors may not appear until later, even up to five years of age. While autism is not always present or clinically evident at birth, some children are at risk and should be identified as such. Further, “signs of autism are certainly present far earlier than diagnosis is currently made, yet they emerge over time and are not always evident even to the expert clinical eye before 12 months” (Yirmiya & Ozonoff, 2007).

The age of formal diagnosis of autism is an integral component to early intervention. In fact, the age of intervention is a strong predictor for the prognosis of a child with autism (Matson & Minshawi, 2006). A diagnosis of autism allows school systems to make the appropriate accommodations for a child, in accordance with FAPE (Free and Appropriate Public Education). Age of intervention has specifically been shown to be a predictive factor for entering a mainstream classroom for children with autism, with children under 42 months having a better prognosis (Harris & Handleman, 2000). Children who are 54 months and older were more likely to be enrolled in a special education classroom following treatment (Harris & Handleman, 2000). There was a significant difference in mean age at diagnosis as found by Rosenberg et al., 2009. Autism is often diagnosed later than 36 months or misdiagnosed as other PDDs, ID, or ADHD (Matson, 2007). The average age of diagnosis for autism is six years of age, when children have already entered the school system (Mandell, 2002). Children with more severe delays in language and cognition may be diagnosed earlier than those with less severe disabilities (Howlin & Asgharian, 1999).

Sex and Autism

Several studies report that there is a 4:1 ratio for males to females found in many studies (Banach et al., 2009; Center for Disease Control, 2009; Charlop-Christy, et al., 2008; Croen, Grether, & Selvin, 2002). In fact, one of the most well established findings in the autism literature is the greater proportion of males with autism than females (Carter et al., 2007; Bhasin & Schendel, 2007). While increased risk has been reported in males (Croen, Grether, & Selvin, 2002), females may have some protective factors against developing autism. Moreover, with this male-female disparity, females with autism have purported to have more severe forms of autism (Charlop-Christy et al., 2008). This severity may include lower IQ and lower adaptive functioning (as measured by the Vineland Adaptive Behavior Scales) (Banach et al., 2009; Carter et al., 2007). The disproportionality between males and females decreased in a study by Croen, Grether, and Selvin (2002) as the level of severity of ID increased. Nevertheless, this study showed that sex was independently associated with autism risk. Researchers have begun to ask whether the sex ratio is of etiological significance or whether there is a sex difference in phenotypic expression (Carter et al., 2007). In conjunction with IQ, external factors like bias in diagnostic criteria that recognize autism-like behavior in higher functioning males may leave out high functioning females (Bhasin & Schendel, 2007).

Ethnicity and Autism

Although a large body of research points to ethnic disparities in the diagnosis of mental health disorders in children (Mandell et al., 2009), the evidence surrounding

autism, has been less conclusive. Nevertheless, various studies have found a relationship between autism and ethnicity. For example, Rosenberg et al. (2009) concluded that ethnicity and race were significantly related to the diagnosis and treatment of autism. In addition, Croen, Grether, and Selvin (2002) found an elevated risk for black children in their sample. Bhasin and Schendel (2007) also found that even though autism was associated with the black race, more white children with autism had cognitive impairments. According to Beeger et al. (2009), ethnicity and sex do not consistently influence the prevalence values of autism. Mandell et al. (2009) found that black and Hispanic children were less likely to have a diagnosis of ASD than white children, with some black children receiving a diagnosis of conduct disorder, only to be diagnosed with autism at a later time.

African American children in a sample of Medicaid-eligible children were 2.6 times less likely to be diagnosed with autism in comparison to Caucasian counterparts (Mandell, Ittenbacg, Leroy, & Pinto-Martin, 2007). However, they were three times more likely to first receive another diagnosis (e.g., adjustment or conduct disorder), meaning that there is different symptomology, clinical interpretations, or different descriptors in diagnosing children of different ethnicities with respect to autism (Mendell et al., 2007). While there is no known difference by ethnicity or race in the epidemiology of autism, diagnoses may be assigned differently by ethnicity (Mandell et al., 2007).

On average, white children received a diagnosis of autism at 6.3 years of age, while black children did not receive a diagnosis until 7.9 years of age (Mandell et al.,

2002). There is also a disparity in the age at which children entered the mental health system, which was six years old for white children and 7.1 years for black children. Disparities might exist for early detection, diagnosis and treatment of autism because there is a difference in help seeking, advocacy and support for people of different ethnic backgrounds (Mandell et al., 2002).

Intellectual Disability and Autism

In Kanner's 1943 paper, Kanner did not suggest that there was a direct association between autism and ID (Kanner, 1943). Currently, with almost 75% of children with autism also having an ID (Charlop-Christy et al., 2008), challenges can occur for the clinician in the differential diagnosis of autism versus ID. However, children with Asperger syndrome and PDD-NOS are excluded from this rate (Matson & Minshawi, 2006). Intellectual functioning was found to be in the normal range for only about 20% of people with autism (Matson & Minshawi, 2006). Intellectual disability is characterized by sub-average intellectual functioning as indicated by an IQ equal to or below 70 and significant deficits in at least two areas of adaptive functioning such as interpersonal skills, work, leisure, and self-care (Matson & Minshawi, 2006). Two of the key elements in distinguishing autism from other developmental disabilities like ID are language and social skills (Matson, 2005). Children with autism and ID have also been shown to have more comorbid problems like mood disorders (Matson & Shoemaker, 2009). However, children with autism with normal intelligence can also be prone to psychiatric disorders, like depression, due to greater insight into their deficits, though this area has rarely been studied (Matson & Minshawi, 2006). In contrast, children with

PDD-NOS and Asperger's have lower rates of ID, at approximately 6-49% (Klinger, O'Kelley, & Mussey, 2009).

In regard to sex, females with autism have also been found to have lower IQ scores than males with autism, and more severe forms of ID, as stated previously (Klinger et al., 2009). More severe forms of autism might also include higher rates of stereotypic behaviors (Matson & Minshawi, 2006). Males were also purported to be 8.8 times more likely to have average intelligence than females (Klinger et al., 2009).

Maternal Education and Autism

Level of maternal education has been associated with child problems behaviors, with lower maternal education being linked with other risk factors for child problem behaviors (Hughes & Ensor, 2009). For example, Luyster, Qui, Lopez and Lord (2007) found that in their sample of children with ASD, one third of the mothers had a high school education or less education. In this study the researchers used a social interaction screen at two years, three years and nine years to see if diagnoses could be predicted using ethnicity, gender and maternal education and found that these variables did not significantly predict scores at nine years of age on the social interaction measure (Luyster et al., 2007). However, Croen, Grether, and Selvin (2002) showed that as maternal education increased, the risk of autism increased in their study. More specifically, women with postgraduate education were more than four times likely to have a child with autism than women with less than a high school education. The findings regarding autism and level of maternal education are inconclusive.

A covariate of maternal education level, maternal and paternal age of parents has been linked to ASD in some studies. Durkin and colleagues (2008) investigated independent effects of maternal and paternal age on risk of ASD. The researchers found that after adjustment for the other parent's age, maternal education, birth order, and other covariates, paternal age and maternal age was independently associated with ASD (Durkin et al., 2008). More specifically, a maternal age of 35 or greater vs. women 25-29 was associated with ASD and a paternal age of 40 or greater vs. men 25-29 was associated with ASD (Durkin et al., 2008).

SES and Autism

SES does not consistently predict autism (Beeger et al., 2009; Croen, Grether, & Selvin, 2002) and did not effect a social interaction screening's predictive ability in the Luyster et al. 2007 study described previously. However, in the Bhasin and Schendel (2007) study, higher family income was significantly associated with autism. This investigation also showed that higher maternal education and higher family income were associated with autism without ID, but not with autism with ID (Bhasin & Schendel, 2007). The combination of SES and level of maternal education in relation to autism was also examined by Larsson et al. (2005) and they found that parental wealth was associated with autism but not maternal education.

Overview of the Literature

One of the most stable findings regarding autism is that males are at an increased risk for this disorder in comparison to females. However, a decreasing risk is found for males when IQ is factored in. Studies regarding ethnicity have found some decreased

risks for children in black families, though the findings are less established than those for sex. Age is an integral component in distinguishing autism from other pervasive developmental disorders since behaviors may be present at particular developmental stages while not at others. This leads to autism often being diagnosed late or misdiagnosed, despite having screens for autism at two years of age. Adding the socio-demographic variables purported to contribute to a prediction of autism with a social interaction screening tool can allow for earlier diagnosis and intervention in children. In this regard, the literature has shown that there is a better prognosis for younger children in terms of the development of speech and language. The question, therefore, is whether these latter variables in conjunction with a social screening instrument predict a diagnosis of autism versus other pervasive developmental disorders. Distinguishing between autism and other PDD can result in early intervention for children with autism.

CHAPTER III

METHODOLOGY

This chapter describes the methodology that was employed in the present study. The methodological components include: participants, independent measures, dependent measures, procedure and statistical analyses.

Participants

Participants in the present study were chosen from a dataset established on children referred to a psychiatric clinic at the Kennedy Krieger Institute of Maryland and the Center for Autism and Related Disorders, Ohio State University. The original dataset consisted of 761 children between 0 months and 20 years of age who were referred to these clinics. The GF-SSI was given to different caregivers of the children (e.g., mother, father, grandmother, guardian). A truncated dataset was developed for the purpose of this study to include only data on children between the ages of 24 and 60 months who were diagnosed as having an autism diagnosis versus a non-autism diagnosis (e.g., PDD-NOS, Depressive Disorder, Attention Deficit/Hyperactivity Disorder, Generalized Anxiety Disorder, Attention Deficit Disorder, Communication Disorder, Fragile X Syndrome, Oppositional Defiant Disorder, Mood Disorder, Separation Anxiety Disorder, or no diagnosis). All diagnoses were based on the *Autism Diagnostic Interview-Revised* (ADI-R) (Lord, Rutter, & Le Couteur, 2000; Lord, Storoschuk, Rutter, & Pickles, 1993), *Childhood Autism Rating Scale (CARS)* (Schopler, Reichler, & Renner, 1986), or DSM-IV diagnoses (APA, 1994). Children with missing diagnoses were not included in the truncated sample since they did not receive a clinical evaluation; therefore all children in

the sample had a clinical diagnosis. The final sample consisted of 171 children with 59 children in the “Autism” group and 112 children in the “Non Autism” group. More specifically, the Non Autism Group consisted of 44 children with PDD-NOS, and 68 children with assorted clinical diagnoses as described above. All children in the sample were clinically referred. The database included scores on the GF-SSI screening instrument, as well as demographic information including the participants’ sex, age, ethnicity, mother’s level of education, and socio-economic status.

In regard to demographic characteristics of the sample, 77.2% were males and 22.8% females. Moreover, 71.3% of the sample was Caucasian, 22.2% African American and less than 6% consisted of Hispanic, Native American, Asian American, and “Other” ethnic categories. In terms of SES, 12.9% of the sample was from the upper class, 26.3% upper-middle class, 19.9% middle class, 17% lower-middle class, and 11.7% from the lower class, as determined by Hollingshead Four Factor Scale. For maternal education, 49.1% of the children had a mother with a bachelor’s degree or higher level of college degree (e.g., J.D., M.D., Ph.D.) and 44.4% were mothers with less than a bachelor’s degree (e.g., high school diploma, associate’s degree). Finally, with respect to an autism diagnosis, 65.5% of the children did not have an autism diagnosis while 34.5% had an autism diagnosis. Demographic information for the sample is included in Table 1.

Table 1

Demographic Characteristics of the Sample

Demographic Variable	N	% of the Sample
Gender:		
Male	132	77.2
Female	39	22.8
Total	171	100
Ethnicity:		
Caucasian	122	71.3
African American	38	22.2
Hispanic	3	1.8
Native American	2	1.2
Asian American	2	1.2
Other	2	1.2
Missing	2	1.2
Total	169	
Maternal Education:		
Bachelor's degree and above	76	44.4
Below Bachelor's degree	84	49.1
Missing Information	11	6.4
Total	171	100
SES Code:		
Upper	22	12.9
Upper-Middle	45	26.3
Middle	34	19.9

Lower-Middle	29	17
Missing	21	11.1
Total	150	

Autism:

Autism	59	34.5
Not autism	112	65.5
PDD-NOS	44	
Depressive Disorder	1	
ADHD	14	
Anxiety Disorder	3	
ADD	1	
Communication Dis.	1	
Fragile X	2	
Language Dis.	8	
ODD	7	
ID	2	
Separation Anxiety	1	
Unknown Clinical	28	
Total	171	100

Permission to use this dataset was granted from the Psychiatry Department at the University of Arizona Medical Center (See Appendix B). The Human Subjects Protection Program at the University of Arizona approved this study (See Appendix C).

Instruments and Measures

The GF-SSI score is the primary independent variable. The GF-SSI (See Appendix A) is a parent-caregiver questionnaire measuring social interactions in children under 60 months of age and was derived from the Socialization domain of the *Vineland Adaptive Behavior Scales (VABS; Sparrow, Balla, & Cicchetti)* and the *Autism Diagnostic Interview-Revised (ADI-R; Lord, Router, & Le Couter, 2000)* (Ghuman, Freund, Reiss, Serwint, & Folstein, 1998). The instrument consists of 54 items that

examine reciprocal social interactions in typical children. The GF-SSI takes into account the cognitive and developmental levels of a child as well as the child's language (Ghuman, Freund, Reiss, Serwint, & Folstein, 1998).

The items in the GF-SSI are scored using a Likert scale, including "Almost Never," "Some of the Time," Most of the Time," "Almost all the Time," and "Not Applicable." Children of different ages can be compared by examining the GF-SSI percentile score, which is the ratio of the child's GF-SSI score to the maximum GF-SSI score for the child's age range. Questions that are worded negatively are reversed scored so that a child scoring higher indicates more typical social development. Examples of questions from the GF-SSI are provided below in Appendix A.

In the initial psychometric evaluation of the instrument, the GF-SSI was administered to 60 healthy control children and 51 clinically referred children in order to assess the reliability and validity. Test-retest reliability and inter-rater reliability was established for the GF-SSI in 24-61 month-old children. The GF-SSI scores successfully differentiated the clinically referred participants from the healthy control participants and between children with autism from other PDD (Ghuman, Freund, Reiss, Serwint, & Folstein, 1998). While most tools like the *Childhood Autism Rating Scales* (CARS) or *Child Behavior Checklist* (CBCL) use a deficit approach to measure deviancy, the GF-SSI uses typical development acquisition to measure for disorders that may be present.

In regard to psychometric properties, test-retest reliability was established by giving the parents/caregivers the GF-SSI at two different intervals (noted as SSI-1 and SSI-2) ($r=.88$). For interrater reliability, a second caretaker was asked to complete the

SSI, making this SSI-3, with $r=.80$. Internal consistency of the scale was satisfactory, with a Cronbach's alpha at .756. Construct validity was established by comparing the total score on the GF-SSI with SVABS (Socialization domain of the Adaptive Behavior Scales). The SVABS was administered to the 111 children. In addition, GF-SSI scores were compared with *ADI-R* algorithm items for children in the clinical group ($n=51$). Clinical validity was established by comparing total GF-SSI scores between the control and clinical groups. GF-SSI scores were also compared within the clinical group (e.g., among various diagnoses). Construct validity was established with SSI-1 scores in concert with SVABS scores. The correlation coefficient for the SSI-1 percent score and the SVABS was $r=.67$. The SSI-1 total percentile had a significant negative correlation with the ADI-R total scores, RSI score and the ADI-COM score as expected, $r=-.71$, $r=-.71$, and $-.62$.

In addition to the GF-SSI, the independent predictor variables were sex, age, ethnicity, mother's educational level, and socio-economic status. The GF-SSI total score for the SSI-1, SSI-2, and SSI-3 was obtained through a ratio of the total score for a child to the maximum score allowed for that age range (Ghuman et al., 1998).

The other predictive variables were coded as follows:

1. Age was coded by months and children between 24.0 months and 60.0 months were included in the study.
2. Sex was coded as: Male (0) and Female (1). One indicator variables was created entitled "Female."

3. Ethnicity was coded as: Caucasian (1), African American (2), Hispanic (3), Native American (4), Asian American (5), and Other (6). Caucasians as well as African Americans were compared against all other groups in the final analyses because Hispanic, Native American, Asian American, and Other children had only two children in each of the latter groups. In the logistic regression analyses, the interpretation of the indicator variable, “Caucasian,” in the model allowed the researcher to compare Caucasians to all other ethnicities. Similarly, the indicator variable, “African American” in the model allowed the researcher to compare African Americans to all other ethnicities.
4. Maternal education was coded as: Bachelor’s degree and above (e.g., law degree, M.D., B.S., M.S., etc.) (1) and below a bachelor’s degree (0) (e.g., AA, H.S. diploma, etc.), with one indicator variable entitled, “College.”
5. SES was coded using the Hollingshead Four Factor Index of Social Status (Hollingshead, 1975), yielding an SES code that consisted of a continuous variable which was used in this study.

Dependent Measure

The dependent measure for this investigation was the diagnosis of the child. The diagnosis included two categories: autism (1) or not autism (0). The autism diagnosis had been confirmed by licensed or certified mental health clinicians, including psychiatrists using one of the following standard assessment tools: *Autism Diagnostic Interview-Revised* (ADI-R) (Lord, Rutter, & Le Couteur, 2000; Lord, Storoschuk, Rutter,

& Pickles, 1993), *Childhood Autism Rating Scale* (CARS) (Schopler, Reichler, & Renner, 1986), or DSM-IV diagnoses (APA, 1994).

1. The outcome variable of autism is coded into two groups so that it is a binary variable: In the Autism Group (1) children are diagnosed as an autism diagnosis. Those in the Non-Autism Group (0) include those children with PDD-NOS, non-spectrum diagnoses (e.g., Depressive Disorder, Attention Deficit/Hyperactivity Disorder, Generalized Anxiety Disorder, Attention Deficit Disorder, Communication Disorder, Fragile X Syndrome, Oppositional Defiant Disorder, Mood Disorder, Separation Anxiety Disorder no diagnosis).

Procedure

A pre-existing dataset was obtained from the Psychiatry Department at the University of Arizona Medical Center. The database was stripped of any identifying information prior to the researcher receiving the dataset. The cleansed database was delivered electronically in Microsoft Excel formatting and SPSS formatting.

Statistical Methodology

Data analyses were completed using the SPSS for Windows statistical package 18.0 (PASW, 2010). Descriptive analyses were conducted first, to compare the dependent variable with each independent variable. Bivariate analyses were computed using phi correlations between the dependent variable and dichotomous independent variables. Spearman's rank correlations were computed between the dependent variable and continuous independent variables. Binary logistic regression was used to predict the probability of an autism diagnosis by fitting the logistic model to the data (McCulloch,

2000). Logistic regression is a commonly used technique for modeling a binary response variable as a function of predictor variables (McCulloch, 2000).

CHAPTER IV

RESULTS

This chapter presents the results of the statistical analyses based on the hypotheses tested.

Table 2 shows the age range, mean age, and standard deviation of the Autism and Non-Autism groups. In addition, the GF-SSI range of scores and mean scores are presented.

Table 2

Means and Standard Deviations of Age and GF-SSI Total Scores for the Autism and Non-Autism groups

	<i>N</i>	<i>Minimum</i>	<i>Maximum</i>	<i>M</i>	<i>SD</i>
Autism					
Age (Months)	59	24.772	59.959	43.552	8.837
GF-SSI Total Scores	59	24	146	70.54	26.179
Non-Autism					
Age (Months)	112	25.298	59.992	45.433	9.475
GF-SSI Total Scores	112	29	150	97.31	27.065

Table 3 presents the results of the bivariate correlational analyses between the predictor variables and the outcome variable. The phi correlation coefficients (ϕ) did not show any significant correlations between autism and the categorical predictor variables. The Spearman's rho correlation coefficients between autism and the continuous predictor variables revealed that there was a significant negative relationship

between autism and the GF-SSI, $r(171)=-.433, p<.001$. Therefore, as scores increased on the GF-SSI, a child was less likely to be diagnosed with autism.

Table 3 Phi Correlations and Spearman's Rho for Autism Variable with Predictor Variables

Predictor*Autism/Non Autism	ϕ	r_s	p
Female	-.072		.346
Caucasian	.067		.386
African American	-.097		.207
Hispanic	-.098		.201
Native American	.035		.652
Asian American	.035		.652
Other	.149		.052
College	.099		.431
SES		-.101	.217
Age		-.113	.141
GF-SSI		-.433	.000

Logistic Regression Analyses

The binary logistic regression was constructed based upon the theoretical literature and historical predictor variables discussed in the autism literature. The demographic variables sex, age, ethnicity, maternal education and socio-economic status were included in the model to be able to attribute the odds ratios for each predictor in the final model.

The classification table (Table 4) of the null model demonstrated that the null model with no predictors (just the intercept) was able to correctly predict the dependent variable of autism 63.3% of the time. The prediction that an individual does not have

autism was 95% of the time, and the prediction was 0 that a person with autism did not have autism.

Table 4

*The Observed and Predicted Frequencies for Autism in the Null Model (no predictors)
With the Cutoff of 0.50*

Observed	Predicted		% Correct
	Yes	No	
No	95	0	100
Yes	55	0	0
<u>Overall Percentage</u>			63.3

Table 5

Logistic Regression with Constant Only (Null Model)

	β	SE β	Wald's χ^2	df	p	e^β (OR)
Constant	-.547	.169	10.405	1	.001	.579

The null model (Table 5) indicated that the null hypothesis in which the constant would be zero could be rejected. The null model explained that the odds ratio (OR) of having autism was .579 in this sample.

Table 6 demonstrates the likelihood ratio test and the model summary for the null model. The log likelihood (-2LL) is the odds that the observed values of the dependent

variable may be predicted from the observed values of the independent variables. The log likelihood reflects the significance of the unexplained variance in the dependent variable. As the model is built, it is hoped that the -2LL will decrease. The results show that the null model was not significant ($p=.565$).

Table 6

Likelihood Ratio Test and Model Summary for the Null Model

	χ^2	<i>df</i>	<i>p</i>	-2LL
Constant	.332	1	.565	196.816

In building the model, the first predictor added was the sex variable based upon consistent finding that more males than females have a diagnosis of autism (Table 7). Age was the second variable, as the age of diagnosis and the age at which social behaviors are observable are an important variable in the autism diagnosis. The third variable added was ethnicity to determine if ethnic disparities exist in the diagnosis of autism. The final model only includes Caucasian and African American because the other ethnic categories did not have enough members to be included in the final model. Maternal education (“College”) was added to the model in the fourth position, and socio-economic status in the fifth position. Theoretical considerations and previous research findings determined which predictor variables entered the model at which point in the positioning of the variables. Finally, the GF-SSI was added to the model last as the least

amount of literature surrounds this assessment instrument. When the GF-SSI was added, it produced significant results for the final model, $p<.001$.

Table 7

Logistic Regression Analysis of Children for Autism with Predictor Variables

Predictor	β	SE β	Wald's χ^2	df	p	e^β (OR)
Female	.076	.497	.024	1	.878	1.079
Age	.022	.022	1.006	1	.316	1.022
Caucasian	-.115	.891	.030	1	.862	.857
African A.	.206	.988	.043	1	.835	1.229
College	.003	.012	.049	1	.826	1.003
SES	-.015	.010	2.032	1	.154	.986
GF-SSI	-.041	.008	24.832	1	.000	.959
Constant	2.549	1.418	3.228	1	.072	12.789

Test	χ^2	df	p
Overall model evaluation			
Likelihood Ratio Test	35.525	7	.000
Goodness-of-fit test			
Hosmer & Lemeshow	7.856	8	.448

Tests were conducted to assess the overall model, including the likelihood ratio test (Garson, 2008) and the Hosmer & Lemeshow (Hosmer & Lemeshow, 1989) goodness-of-fit test. The likelihood ratio test produced significant results for the overall model evaluation, $\chi^2=35.525(7)$, $p<.01$. This test indicates the probability of obtaining the chi-square statistic if there is no effect of the independent variables taken together, on the dependent variable. The likelihood ratio test permits one to compare models and test

how well the model fits. For significant variables, the larger the chi-square, the greater the loss of model fit if that variable is dropped; therefore, the model was found to be statistically significant ($p < .01$). Since the model chi-square is less than .05, the null hypothesis is rejected; therefore, suggesting that the independent variable make a difference in predicting the dependent variable. As the Hosmer and Lemeshow test produced a non-significant result ($p > .05$), the null hypothesis is rejected, suggesting that there is a difference between the observed and model predicted values.

The results of the full model suggest that being male increases the logit for autism by .076; for every unit increase in age, the logit for autism increases by .022; being Caucasian (versus all other ethnic groups) decreases the logit for autism by .155; being African American (versus all other ethnic groups) increases the logit for autism by .206; having less than a bachelor's degree for maternal education increases the logit for autism by .003; and for every unit increase in SES, the logit for autism is increased by .015. Finally, for every unit increase in the GF-SSI, the logit for autism decreases by .041.

Explained in odds ratios (OR), the OR for males was 1.079, .857 for Caucasians, 1.229 for African Americans, and 1.003 for children of mothers with an educational attainment of less than a bachelor's degree. For increasing age the OR was 1.022; for increasing SES the OR was .986. The OR for the GF-SSI was .959. OR can be interpreted based on how close to 1.000 they are. Since all of the odds ratios for the predictors fall within a 95% confidence interval from 1.000, this means that the probability of having autism is close to 50/50. Specifically, higher scores on the GF-SSI

decreased the probability of autism (OR=0.959) and the effect size was small, even though a person's score was a significant predictor variable for autism.

Table 7 shows that the Wald Statistic for the GF-SSI scores is significant, but not for the other predictors. To bolster this explanation, the results for the Likelihood Ratio Test are provided in Table 7 for assessing the fit of the model, otherwise known as the Omnibus Tests of Model Coefficients. For the GF-SSI, a loss of model fit would occur if this variable were dropped, since the model was statistically significant when it was added in the final step. The GF-SSI, therefore, makes a difference in predicting autism.

The model was built and evaluated for fit using the Bayesian information criterion (BIC), also known as Schwarz's Bayesian criterion (SBC; Garson, 2008), for one or several fitted models in which the log-likelihood value can be obtained. The BIC operates under a more stringent criterion than the Alkaline Information Criterion (AIC) as the number of parameters is accounted and penalized. These values are located in Table 8 for each model. The model with the lowest BIC becomes the final model, which was model number 6, with BIC=177.254, which decreased from 199.049 in the first model.

Table 8

Model building and model evaluation using Bayesian Information Criterion

<i>Model</i>	<i>Variables Included</i>	<i>Log Likelihood</i>	<i>df</i>	<i>BIC</i>
1	Female	196.816	1	199.049
2	Female Age	196.698	2	201.164

3	Female Age Caucasian African American	196.547	4	205.479
4	Female Age Caucasian African American College	196.529	5	207.694
5	Female Age Caucasian African American College SES	195.082	6	208.480
Final	Female Age Caucasian African American College SES GF-SSI	161.623	7	177.254

Table 9

*The Observed and Predicted Frequencies for Autism by Logistic Regression by Model
With the Cutoff of 0.50*

Block	Observed	Predicted Not Autism	Predicted Autism	% Correct	Overall %

1	Not Autism	95	0	100	
	Autism	55	0	0	63.3
2	Not Autism	95	0	100	
	Autism	55	0	0	63.3
3	Not Autism	95	0	100	
	Autism	55	0	0	63.3
4	Not Autism	95	0	100	
	Autism	55	0	0	63.3
5	Not Autism	95	0	100	
	Autism	55	0	0	63.3
6	Not Autism	77	18	81.1	
	Autism	24	31	56.4	72.0

In comparison to the null model predicting correctly 63.3% (Table 4), Table 9 shows that the full model was able to predict 72% of the cases correctly when the GF-SSI was added to the model.

CHAPTER V

DISCUSSION & CONCLUSION

This chapter provides a summary of the research findings and compares these results to current literature on predictive factors and screens of social interaction in the early detection of autism. Implications of the results and limitations of the study are addressed as well as future directions for research in this area.

The purpose of the present study was to examine whether an autism diagnosis could be predicted on the basis of a child's social interactions, as measured by the Ghuman-Folstein Screen for Social Interaction, and selected demographic variables (i.e., sex, age, ethnicity, mother's educational level, and socio-economic status) that were chosen based on literature in the area of autism that has focused on factors that may predict (or be associated with) autism (Mandell et al., 2009; Matson & Minshawi, 2006; Bhasin & Schendel, 2007; Croen, Grether, and Selvin, 2002). Binary logistic regression equations were utilized to determine the extent to which various models could predict a diagnosis of autism.

The results showed that an autism diagnosis was significantly predicted, as hypothesized, on the basis of children's social interactions as measured by the GF-SSI. Specifically, the GF-SSI score aided in the prediction of a diagnosis of autism when it was added in the final step of the model. This resulted in the final model being able to correctly identify 72% of the diagnosed cases, an increase from 63.3% in the null model. The GF-SSI scores of children were also found to be negatively correlated with a diagnosis of autism as discovered in the bivariate analyses. The model was not found to

be significant until GF-SSI scores were added during the final step of the model building process.

Factors that were not found to be significant predictors of the diagnosis of autism were: sex, age, ethnicity, mother's educational level, and socio-economic status. Since many studies have reported a 4:1 ratio for males versus females in the general pediatric population with regard to the diagnosis of autism (e.g., Banach, 2009; Center for Disease Control, 2009; Charlop-Christy, et al., 2008; Croen, Grether, & Selvin, 2002), it was hypothesized that in the present clinical sample being male would, in fact, be a predictive factor; however, this was not the case. There is also a lack of consistent findings amongst the literature as well as the samples used in studies sometimes include all clinical groups and sometimes include normal controls. Miles and Hillman (2000) studied homogeneous autism subgroups for genetic study. Of 88 children in the sample, the group had a male to female ratio of 4.2:1. However, the morphologically normal subgroup had a sex ratio of 7.5:1 and the phenotypically abnormal subgroup had a sex ratio of 1.7:1. In addition, much of the research on the age ratio focuses on children that span the age of 4-10 years of age, rather than the 2-5 age range in the present study. There is also some conflicting information as the 4:1 ratio can include all autism spectrum disorders in some studies, not just autism.

With respect to age, the final model did demonstrate that as children in the sample increased by one unit in age, their OR was 1.022. While this increase is small in effect since it is close to one, it is consistent with the literature that most children are not diagnosed until 4 ½ to 5 ½ years of age (e.g., school age children). As posited by Matson

(2007), autism is often diagnosed later than 36 months or misdiagnosed as other PDDs, ID, or ADHD. The average age of diagnosis for autism is six years of age, when children have already entered the school system (Mandell et al., 2002).

In terms of ethnicity, while a large body of research points to ethnic disparities in the diagnosis of mental health disorders in children (Mandell et al., 2009), the evidence is less conclusive for autism, as was also demonstrated the current study. Consistent with the findings of Beeger et al., (2009), ethnicity and sex did not appear to contribute to the prevalence values of autism. In addition, in the present study, the majority of the children in the sample were Caucasian, followed by African American, and, contrary to Mandell et al. (2009), there was not a significant difference by ethnicity in the diagnosis of autism. Within the realm of the mental health field disparities might exist for early detection, diagnosis and treatment of autism because there is a difference in help-seeking, advocacy and support for people of different ethnic backgrounds (Mandell et al., 2002), which could not be measured in this investigation.

Through various methods of coding, maternal education was created into the “indicator variable, “college,” and was not a significant predictor of autism. For example, Luyster, Qui, Lopez and Lord (2007) found that in their sample of children with ASD that one third of the mothers had a high school or less education. However, Croen, Grether, and Selvin (2002) showed that as maternal education increased, the risk of autism increased in the children in their study. More specifically, women with postgraduate education were more than four times more likely to have a child with autism than women with less than a high school education (Croen, Grether, & Selvin, 2002).

Although the findings in the present study do not support the latter findings, there may be several reasons for these apparent conflicting results. First, the present study used a different assessment instrument. In addition the children in the present study were younger than in the other studies.

Bhasin and Schendel (2007) found in previous research that higher family income was significantly associated with autism. However, consistent with the present findings, SES has not, in general, been found to predict autism (e.g., Beeger et al., 2009; Croen, Grether, & Selvin, 2002 Luyster et al. 2009).

Last, the GF-SSI was found to be a significant predictor of an autism diagnosis. In this regard, a strong bivariate relationship was found between GF-SSI and autism prior to the binary logistic regression analyses. Following the binary logistic regression analyses, the GF-SSI was the strongest predictor for autism of all the predictor variables, and also a significant predictor of autism as well. This points to the predictive capacity of the GF-SSI and the ability of the tool to be used in further clinical studies and the possibility of using this instrument in children 2-6 years of age to screen for autism. The full regression model was able to predict correctly 72% of the autism cases, which was an improvement from the null model. The GF-SSI can be implemented with children from ages 2-6 years since it has shown predictive ability for children with the diagnosis of autism versus those who do not have diagnoses of autism. Osterling, Swinkels, Jan van der Gaag, Visser, Dietz, and Buitelaar (2009) compared different screening instruments in more than 200 children to look at the power in discriminating ASD from non-ASD (2009) and found that many screening measures fail to obtain the level of sensitivity and

specificity needed to distinguish children with ASD from children with abnormal development (Osterling et al., 2009).

Limitations of the Present Study

While the present study offers some empirical evidence for the utility of the GF-SSI, the other theoretical predictors were limited in predicting autism. In this regard, the present study has several limitations. First, the sample contained only participants that had been referred to clinics involved in the study; therefore, the findings may not be generalizable to children entering other clinics.

Second, the dataset was developed for the purpose of this study to include only data on children between the ages of 24 and 60 months who were diagnosed as having an autism diagnosis versus a non-autism diagnosis. Much of the research on the age ratio focuses on children that span the age of 4-10 years of age, rather than the 2-5 age range in the present study. This truncation of the dataset lead to loss in sample size and disallowed claims of causality to be made.

In addition the sample size was less than one-quarter of the total sample, leading to smaller power and effect sizes. While 171 participants in the present study is a robust sample size, in general, and meets the criteria of having 10-20 participants per predictor variable, this only captures a fraction of the sample for which data were available. This fraction resulted from extracting participants that were between the ages of 24.0 and 60.0 months and by only including children in this age range that had a diagnosis of autism, or a diagnosis that was not autism. Although the sample size was robust, the sample was created through a process of truncating the sample, meaning that it is not normally

distributed and, therefore, claims of causality cannot be made. The effect sizes in the present study are small and the ORs are close to one, suggesting that they may be more important clinically.

Last, more randomized data for sex would be beneficial since no notable differences were found in the present study, even when the dataset was split by sex.

In regard to SES, SES was used as a continuous variable in the current study, based on Hollingshead Four Factor Scale. This scale utilizes gender, marital status, occupation and education. This scale has been criticized for a lack of validity and practicality due to its conception in New Haven Connecticut in 1975, and the scale lacking occupations outside of New Haven. This might ignore fluctuations in the component's of a family's SES (Duncan & Magnuson, 2001). Opponents to this scale also argue that the components in SES are not interchangeable and that they all have distinct impacts on children. In a comparison of methods for measuring SES by occupation or postal area, it was found that the Hollingshead Four factor scale had moderate agreement with other scales (Blishe, Pineo-Porter, and British Registrar General). These scales estimated SES better than the postal area codes (Deonandan et al., 2000). Overall, "SES is too multi-faceted to be captured by a single index or even a multi factor index." (Duncan & Magnuson, p. 15), meaning that SES is extremely difficult to be used in any study.

Future Directions for Research

The present study highlights the need to continue studying the demographic factors associated with a diagnosis of autism. Although significant results were found for

the GF-SSI, the study should be enlarged to include more children with diagnoses in the sample. Networks like the IAN (International Autism Network) and other very large databases might be able to alleviate some of these problems as various researchers (with permission) will have access to datasets that might be very rich in information and might have more of this information readily available across a variety of settings, children, demographics, etc.

With particular attention to the GF-SSI, it may be beneficial to compare different groups within the sample to each other, using binary logistic regression analyses. While the current study compared children with an autism diagnosis to those with a non-autism diagnosis, breaking down the non-autism group into further subgroups could be beneficial in explaining the children in this group more effectively. Additional studies in which sub-group analyses could be conducted with the autism versus PDD-NOS group would be helpful in showing how the GF-SSI might predict autism against only PDD-NOS children. Similarly, a sub-group analysis of the autism group versus the clinical non-ASD cases would demonstrate the predictive capability of the GF-SSI in that sample, removing other spectrum disorders (PDD-NOS) from the sub-group analyses. Last, a missing cases analysis may be conducted to better understand the children that were not included in the final truncated sample used in this study. Perhaps finding commonalities between the children that were excluded will enhance the predictive capability of the GF-SSI for use with a wider range of children in the future.

APPENDIX A: GF-SSI

SCORING TEMPLATE
GHUMAN-FOLSTEIN SCREEN FOR SOCIAL INTERACTION*
(FOR CHILDREN 6 MONTHS TO 5 YEARS OF AGE)

	0	1	2	3
YOUR OBSERVATION	ALMOST NEVER	SOME OF THE TIME	MOST OF THE TIME	ALMOST ALL THE TIME
WHEN YOU TALK TO YOUR CHILD, DOES HE/SHE:				
1. look at you?	0	1	2	3
2. smile at you?	0	1	2	3
DOES YOUR CHILD TRY TO GET YOUR ATTENTION:				
3. to get things for him/her?	0	1	2	3
4. when he/she can't do something by himself/herself?	0	1	2	3
5. to show you things?	0	1	2	3
DOES YOUR CHILD INTERACT WITH YOU BACK AND FORTH OVER A				
6. speech or sounds?	0	1	2	3
7. gestures?	0	1	2	3
DOES YOUR CHILD GREET YOU WHEN YOU RETURN HOME BY:				
8. looking at you?	0	1	2	3
9. making sounds or talking to you?	0	1	2	3
10. wanting to be picked up or coming to you?	0	1	2	3
11. hugging you?	0	1	2	3
DOES YOUR CHILD SHOW AFFECTION BY:				
12. cuddling up to you?	0	1	2	3
13. When you show affection does your child return your affection?	0	1	2	3
14. Does your child want you to hug or pick him/her up when he/she gets hurt?	0	1	2	3
DOES YOUR CHILD RESPOND PLAYFULLY BY:				
15. laughing when you make silly sounds?	0	1	2	3
16. playing games like peek-a-boo, patty cake, rolling ball, blowing bubbles?	0	1	2	3
17. playing "dressing up" games?	0	1	2	3
18. playing pretend games with dolls, cars, action figures, dollhouse?	0	1	2	3
DURING MEALS DOES YOUR CHILD:				
19. look at you?	0	1	2	3
20. interact with you by making sounds or talking?	0	1	2	3
SHOW WHAT FOOD HE/SHE LIKES BY:				
21. look on his/her face?	0	1	2	3
22. pointing or other gestures?	0	1	2	3
23. speech or making sounds?	0	1	2	3

24. Does your child copy you by washing dishes, pretending to cook or mow the lawn, etc.?	0	1	2	3
25. Does your child show you things that he/she has done and wants you to praise?	0	1	2	3
26. Does your child smile when you praise him/her?	0	1	2	3
YOUR OBSERVATION	ALMOST NEVER	SOME OF THE TIME	MOST OF THE TIME	ALMOST ALL THE TIME
27. Does your child have a favorite toy, blanket, stuffed animal or another object that he/she takes with	0	1	2	3
28. Does your child get upset when you leave him/her with an unfamiliar babysitter?	0	1	2	3
29. Does your child follow you around in the house?	0	1	2	3
30. Does your child come looking for you if you are not in the same room with him/her?	0	1	2	3
31. Is your child shy around people he/she does not know well?	0	1	2	3

DOES YOUR CHILD:

* 32. have a blank face?	*3	*2	*1	*0
* 33. look distant or removed?	*3	*2	*1	*0

CAN YOUR CHILD TELL FROM THE LOOK ON YOUR FACE OR THE TONE OF YOUR VOICE THAT YOU ARE:

34. happy?	0	1	2	3
35. angry?	0	1	2	3

DOES YOUR CHILD SHOW INTEREST IN OTHER CHILDREN BY:

36. watching them?	0	1	2	3
37. moving towards them?	0	1	2	3
38. staying close to them?	0	1	2	3
39. trying to play with them?	0	1	2	3
40. joining in the play if they invite him/her?	0	1	2	3
41. showing concern if another child is upset?	0	1	2	3

IS YOUR CHILD ABLE TO:

42. start social exchange with other children?	0	1	2	3
43. join in when other children start the social exchange?	0	1	2	3
44. keep a social exchange going back and forth with other children over few turns?	0	1	2	3

DO OTHER CHILDREN (OF HIS/HER AGE):

45. like him/her?	0	1	2	3
46. want to play with him/her or want to be around him/her?	0	1	2	3

DOES YOUR CHILD "TURN OFF" SIBLINGS OR OTHER CHILDREN

* 47. is aggressive with them?	*3	*2	*1	*0
* 48. avoids other children?	*3	*2	*1	*0
* 49. takes toys away from them?	*3	*2	*1	*0

* 50. yells and screams?	*3	*2	*1	*0
51. Is your child able to take turns in play?	0	1	2	3
52. Does your child have playmates who he/she prefers to play with?	0	1	2	3
DOES YOUR CHILD SHARE TOYS AND FAVORITE OBJECTS WITH:				
53. you or other adults?	0	1	2	3
54. siblings or other children his/her age?	0	1	2	3

APPENDIX B: SAMPLE SITE AUTHORIZATION LETTER

December 09, 2009

Dana Princiotta
825 E. 5th St., Apt. #414D
Tucson, AZ 85719

Dear Dana Princiotta:

I have reviewed your request regarding your study and am pleased to support your research project entitled "Predicting autism in young children based on social interaction and selected demographic variables." Your request to use the Ghuman-Folstein Screen for Social Interaction dataset for your dissertation is granted. The research will include secondary analysis of the existing dataset and presenting the results to the dissertation committee. This authorization covers the time period of December, 2009 to January, 2011.

Sincerely,

Jaswinder Ghuman, M.D.
Associate Professor of Psychiatry and Pediatrics
Child and Adolescent Psychiatry
University of Arizona

APPENDIX C: IRB APPROVAL



Human Subjects
Protection Program

1618 E. Helen St.
P.O. Box 245137
Tucson, AZ 85724-5137
Tel: (520) 626-6721
<http://www.irb.arizona.edu>

HSPP Correspondence Form

Date: 02/05/10

Investigator: Jaswinder K. Ghuman, M.D.

Department: Psychiatry

Project No./Title: 05-0538-00 Retrospective Chart Review for Social Interaction Problems in Children with Developmental and Psychiatric Disorders

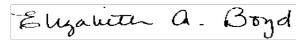
Current Period of Approval: 08/29/05 – No Expiration

IRB Committee Information	
<input checked="" type="checkbox"/> Administrative Action	<input checked="" type="checkbox"/> Administrative/Exempt Review – 02/05/10
FWA Number: FWA00004218	
Nature of Submission	
<input checked="" type="checkbox"/> Amendment	
Documents Reviewed Concurrently	Appr: Approved Ack: Acknowledged Rev: Reviewed
<input checked="" type="checkbox"/> Request for Amendment Form – PI Initiated Changes (dated 01/25/10)	
<input checked="" type="checkbox"/> VOTF (signed 01/26/10)	
<input checked="" type="checkbox"/> Other (define): Student PI's Abstract	

Description of Modifications

Personnel change [changing the research role of Princiotta to Student-PI from Research Assistant]; new study document [Student PI's Abstract].

Committee/Chair Determination	
<input checked="" type="checkbox"/> Approved as submitted effective 02/05/10	
Additional Determination(s)	
<input checked="" type="checkbox"/> Not Applicable	



02/05/10

Elizabeth A. Boyd, Ph.D.
Assistant Vice-President, Research Compliance & Policy
Office for the Responsible Conduct of Research

EAB:deg

Reminders: Continuing Review materials should be submitted 30–45 days prior to the expiration date to obtain project re-approval

- Projects may be concluded or withdrawn at any time using the forms available at www.irb.arizona.edu.
- No changes to a project may be made prior to IRB approval except to eliminate apparent immediate hazard to subjects.
- Original signed consent forms must be stored in the designated departmental location determined by the Department Head.

/
Arizona's First University – Since 1885

Form version: 09/23/09



APPENDIX D: REQUEST FOR AMENDMENT FORM

Request for Amendment Form PI Initiated Changes	
Name of Principal Investigator: Jaswinder K. Ghuman, M.D.	Telephone Number: 520-626-3603
Email Address: jkghuman@email.arizona.edu	
Name of Alternate Contact: Natalie Mai-Dixon/Dana Princiotta	Telephone Number: 626-8733/845-548-5445
Email Address: nmaidixon@uph.org / danap@email.arizona.edu	
Department: Psychiatry	PO Box/Mailstop: 245002
Project Number: exempt—approved 8/29/05 (revised VOTF apr 12/23/09)	Date of Request: January 25, 2010
<u>Title of Project:</u> Retrospective Chart Review For Social Interaction Problems In Children With Developmental And Psychiatric Disorders	
<u>Request reflects changes in radiation exposure to subjects and requires review by Radiation Control?</u> <input type="checkbox"/> Yes <input checked="" type="checkbox"/> No <u>(If yes, please submit applicable materials to the Radiation Control Office.)</u>	
<u>What changes do you want to make to the study?</u>	
<p>Revise Dana Princiotta's role on the study to student PI. She will be analyzing a subset of the study's data set for her doctoral dissertation.</p> <p>More specifically, the sample will be data on children between the ages of 24 and 60 months who were diagnosed as having autism, PDD-NOS, non-spectrum diagnoses, and no diagnoses. The final dataset will consist of 450 children with diagnoses of autism (N=108), PDD-NOS (N=61), non-spectrum (N=103), and no diagnosis (N=178). Scores on the GF-SSI screening instrument as well as demographic information on the participants' sex, age, ethnicity, mother's level of education, socio-economic status, and presence vs. absence of intellectual disability will also be included in the database.</p> <p>Descriptive analyses will be conducted first, using the appropriate methods to compare the dependent variable with each independent variable. Once significant variables are identified, in conjunction with the aforementioned historical predictors of autism, analysis with logistic regression will be utilized to predict diagnostic class and to examine the independent effects. (see attached abstract for additional information.)</p>	
<u>Provide the rationale for these changes.</u>	
<p>The original study purpose is as follows:</p> <p>"The purpose of this retrospective chart review is 1) to collect preliminary information regarding the nature and correlation of social interaction deficits with diagnosis, cognitive and language development, and other factors in young children with development and psychiatric disorders; and 2) to explore if social interaction deficits can be used to identify at-risk children who should be carefully monitored and/or referred for further assessment. It is hoped that screening for and identification of social interaction deficits can lead to the development of specifically targeted assessments, interventions and improve treatment outcomes."</p> <p><u>Because Ms. Princiotta's study aims are within the original study purpose, but her study aims and defined data set are distinct, adding her to the currently approved study as a student PI seemed appropriate based on the definition of that role and the limited project oversight it provides.</u></p>	
<u>Does this change the risk/benefit ratio (increases OR decreases risk to subjects?)</u> Yes <input checked="" type="checkbox"/> No <u>(If No, explain why/If Yes, explain how)</u>	
<p>A pre-existing dataset obtained from the Psychiatry Department of a University of Arizona Medical Center will be utilized in the proposed study. The database will be stripped of any identifying information prior to the investigator receiving the dataset.</p>	
<u>Will subjects be notified of this/these change(s)?</u> <input type="checkbox"/> Yes <input checked="" type="checkbox"/> No	

(If No, explain why/If Yes, explain how)

N/A (The original dataset was collected through chart review.)

Due to the requested changes, are revisions to the consenting instruments necessary?

Yes No

(If No, explain why/If Yes, explain how)

N/A (The original dataset was collected through chart review; no consenting instruments were used.)

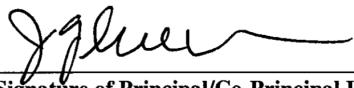
If applicable, revisions should be incorporated into the currently approved IRB documents (e.g., Research Proposal/Protocol, Informed Consent document, Recruitment Materials, Verification of Training Form, etc.) The revised documents must be submitted with the Request For Amendment Form.

List attachments and/or revised documents and include the version and/or version date:

Abstract

Revised VOTF

Statement of Principal Investigator: I have personally reviewed this form and propose the above changes.



Signature of Principal/Co-Principal Investigator

1/26/10

Date

Abstract
(Princiotta study – data set from J Ghuman study)

Parent Study Title: Retrospective Chart Review For Social Interaction Problems In Children With Developmental And Psychiatric Disorders

Substudy Title: Predicting Autism in Young Children Based on Social Interaction and Selected Demographic Variables

The purpose of the present study is to examine whether an autism diagnosis can be predicted by social interaction as measured by the GF-SSI in conjunction with selected demographic variables (sex, age, ethnicity, intellectual disability, mother's educational level, and socio-economic status).

Given the related literature review, the predictive hypotheses are the following:

Question #1: Are scores on the GF-SSI significant predictors of a diagnosis of autism?

Hypothesis #1: Scores on the GF-SSI significantly explain variance in predicting a diagnosis of autism.

Question #2: How much variance will the GF-SSI explain in the final model?

Question #3: Which independent variable is the best predictor of an autism diagnosis?

Hypothesis #3: The GF-SSI will account for the greatest level of variance in the final model.

Question #4: Which set of observed variables gives rise to the best prediction of autism?

Hypothesis #4: The GF-SSI and sex will account for the greatest level of variance in the final model.

The sample of the present study will be data on children between the ages of 24 and 60 months who were diagnosed as having autism, PDD-NOS, non-spectrum diagnoses, or no diagnoses. Their data will be extracted from a larger database of children (N=802) between 0 months and 20 years of age who were referred to the latter clinics. The final dataset will consist of 450 children with diagnoses of autism (N=108), PDD-NOS (N=61), non-spectrum (N=103), and no diagnosis (N=178). Scores on the GF-SSI screening instrument as well as demographic information on the participants' sex, age, ethnicity, mother's level of education, socio-economic status, and presence vs. absence of intellectual disability will also be included in the database.

The dependent measure for this investigation will be the diagnosis of the child. The diagnosis will include two categories: autism or not autism. A logistic regression will be used in the present study to predict the probability of a diagnosis by fitting the logistic model to the data (McCulloch, 2000). Logistic regression is a commonly used technique for modeling a binary response variable as a function of predictor variables (McCulloch, 2000). Logistic regression can quantify the impact of various simultaneous influences on a single dependent variable.

Descriptive analyses will be conducted first, using the appropriate methods to compare the dependent variable with each independent variable. Once significant variables are identified, in conjunction with the aforementioned historical predictors of autism, analysis with logistic regression will be utilized to predict diagnostic class and to examine the independent effects.

UNIVERSITY OF ARIZONA HUMAN SUBJECTS PROTECTION PROGRAM
Verification of Human Subjects Training (VOTF)

Title of Project: **Retrospective Chart Review For Social Interaction Problems In Children With Developmental And Psychiatric Disorders [exempt— approved 8/29/05 (revised VOTF apr 12/23/09)]**

All individuals conducting research involving human subjects (with or without financial support of any sponsoring organization or agency) must complete Human Subjects training. Those individuals include principal investigators, co-investigators and all other individuals involved in the conduct of research. Students and their advisors must meet the same standard as faculty and staff.

I hereby certify that individuals involved in this proposal have completed the required Human Subject training.


 Principal Investigator's Signature
1/26/09
 Date

Please list all individuals involved in the above-cited research study.

Name	Research Role (PI, Co-PI, Collaborator, Sub-I, Data Mgr, Research Ass., etc.)	Affiliation UA/NA/ Other	Will this person be involved in the consenting process?*	Training Title Indicate type of training: CITI-Biomed, CITI-SBS **	Completion Date(s) For each Human Subjects (mm/dd/yy)
Jaswinder Ghuman, M.D.	PI	UA	NO	CITI-Biomed & SBS	9/26/08
Dana Princiotta	Student PI	UA	NO	CITI-SBS	5/21/09
			YES NO		
			YES NO		
			YES NO		
			YES NO		

*Consent forms are to be signed and dated by the subject (or their legal representative) and by the Principal Investigator or Co-Principal Investigator (no other study personnel may sign as Investigator without prior approval of the IRB). Other study personnel involved in the consenting process may sign as Presenter, but not as Investigator.

**CITI-Biomed, CITI-SBS: Collaborative Institutional Training Initiative – www.citiprogram.org
 Author: University of Miami

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