Early Characteristics of Young Siblings of Children with Autism

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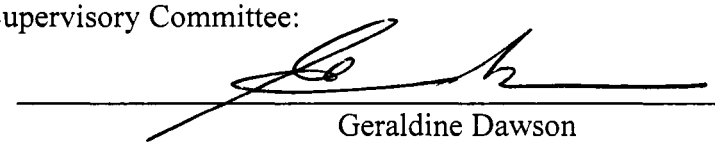

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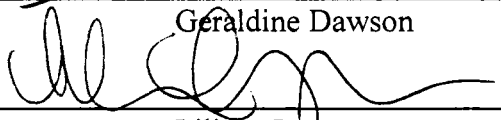

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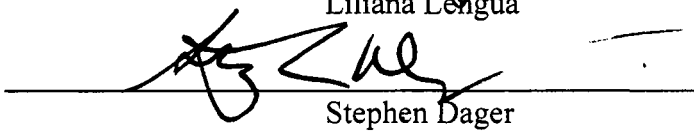

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Abstract

Early Characteristics of Young Siblings of Children with Autism

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Background: Studies are needed to better understand the broad autism phenotype in young siblings of children with autism, and to identify areas of impairment, so that interventions can begin at as young an age as possible. **Method**: Multiple measures were used to examine cognitive, adaptive, social communication, social-emotional functioning, imitation, play, and language abilities in 42 non-autistic siblings and 20 toddlers with no family history of autism, ages 18-27 months. **Results**: Siblings had lower mean receptive language and adaptive behavior skills, lower overall rates of social communication and social-emotional functioning, and used fewer words, distal gestures, responsive social smiles, and pointing during social interactions. Additionally, parents reported social impairments in siblings by 13 months of age. In other domains, such as imitation and play, siblings performed similarly to controls. **Conclusions**: These results suggest that certain aspects of language and social communication are affected at an early age in young non-autistic siblings. As such, the development of such children should be closely monitored by parents and professionals, with appropriate interventions implemented as needed.

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ACKNOWLEDGEMENTS

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DEDICATION

In loving memory of my parents, Alfred and Wilma Toth.

CHAPTER I

Introduction

Autism is a severe, neurodevelopmental disorder characterized by impairments in social and communication behaviors, and a restricted range of activities and interests. Once considered a rare disorder, autism is now believed to affect as many as 3 in 500 individuals (Baird et al., 2000; Bertrand et al., 2001; Chakrabarti & Fombonne, 2001; Fombonne, 2003). Historically, individuals with autism have had very poor prognoses; however, more recent research suggests that early intensive intervention can lead to improved outcomes for individuals with autism spectrum disorders (Birnbrauer & Leach, 1993; Dawson & Osterling, 1997; McEachin, Smith, & Lovaas, 1993; Rogers, 1998; Sheinkopf & Siegel, 1998). Thus, there is a critical need for research that aims to improve existing methods of early detection of infants and toddlers at risk for autism. Furthermore, it is now recognized that a familial-genetic basis for autism exists, evidenced by an increased concordance for autism in monozygotic twins (Bailey, LeCouteur, Gottesman, & Bolton, 1995; Folstein & Rutter, 1977), and an increased risk of recurrence in siblings of children with autism (Jorde et al., 1990; Jorde et al., 1991). There is also now evidence for a behavioral phenotype in relatives (parents, siblings) that is much milder but qualitatively similar to the defining features of autism, referred to as the broad autism phenotype (Baron-Cohen & Hammer, 1997; Bolton et al., 1994; Bolton & Rutter, 1990; Starr et al., 2001). So far, however, most broader phenotype studies that have included siblings have focused on children 5 years of age and older.

The current study sought to further our understanding of the early characteristics of very young children at risk for autism and broad autism phenotype impairments by

examining cognitive, adaptive, language, imitation, play, and social abilities in 18-27 month old non-autistic siblings of children with autism, as compared with toddlers with developmental delay without autism and typical development. The final sample included two groups of 18-27 month old children: 42 non-autistic siblings of children with autism and 20 typically developing toddlers with no family history of autism. Only 6 toddlers with idiopathic developmental delay meeting eligibility criteria were recruited, which was too few to include in analyses. Older siblings were given cognitive and diagnostic evaluations to confirm the presence of an autism spectrum disorder. All of the 18-27 month old children were administered a comprehensive battery of measures assessing IQ, adaptive functioning, play, language, social communication, social-emotional functioning, face recognition, imitation, and sensory/motor behaviors. Detailed data on children's early development (birth through two years of age) were also obtained by parent interview. The information gained in this study will be important in helping to alert parents to the kinds of difficulties that are likely to manifest early in siblings of children with autism. By recognizing these broad autism phenotype characteristics as early as possible, parents will be able to seek appropriate early intervention for siblings. Furthermore, such characteristics can offer clues to what facets of the autism syndrome tend to aggregate in families, which is useful for genetic studies.

Background and Rationale

Broad Autism Phenotype

Autism is a disorder that is known to have a strong genetic component (Bailey, Palferman, Heavey, & Le Couteur, 1998; Szatmari, Jones, Zwaigenbaum, & MacLean, 1998) as well as a "unique environmental-familial experience" (Bauminger & Yirmiya,

2001, p.61; see also Rutter et al., 1997). In the general population, autism occurs at a rate of about 3 in 500, or 0.6% (Baird et al., 2000; Bertrand et al., 2001; Chakrabarti & Fombonne, 2001; Fombonne, 2003). However, in families with one child with autism, the likelihood of having a second child with autism is approximately 4.5% (Jorde et al., 1990; Jorde et al., 1991), or 45-90 times greater than the general population risk (Cook, 1998); in families with two children with autism, the recurrence risk rate has been estimated to range from 16-35% (Szatmari, Jones, Zwaigenbaum & MacLean, 1998). Additionally, children in the same family share not only 50% of their genes, but the same environment (parental characteristics such as depression, level of external social support, socio-economic status, etc.). Siblings of children with autism also experience nonshared aspects of the environment, including living with a sibling with a disability (Anderson, Hetherington, Reiss, & Howe, 1994). These genetic-environmental-familial factors all combine to set siblings of children with autism at a higher risk of developing a range of impairments. These impairments in family members of individuals with autism have been referred to as the broad autism phenotype, a lesser variant of autism. This broader phenotype has been defined as having impairments in any *one* of the following areas: reciprocal social interaction, communication, and stereotyped interests/behaviors – the same areas that are affected in autism, but of insufficient severity to meet criteria for a specific diagnosis on the autism spectrum.

Most broader phenotype studies to date have interpreted their findings as support for the genetic basis of autism. These studies are typically twin and family studies; the rare occurrence of autism precludes adoption studies which would provide the strongest support for a genetic component. Some researchers have proposed that the broader autism

phenotype is simply a lower dose of the genetic predisposition to autism, while others argue a "two-hit" mechanism. In this view, one set of factors predisposes the individual to the broader phenotype, while a separate set of factors is involved in the development of autism (Bailey, Phillips, & Rutter, 1996). A number of other studies have examined environmental factors associated with sibling functioning. For instance, Boutin and colleagues (1997) reported that cognitive disabilities in siblings were found more often in low-SES families, while Gold (1993) revealed that depression in siblings was correlated with the ability to express feelings about having a sibling with autism. The following is a review of broad autism phenotype studies of siblings.

Sibling Studies

Piven and colleagues (1990) used a family history interview method to examine the frequency of social, cognitive, and affective disorders in 67 adult siblings of 37 autistic probands. They found that 3% of siblings met criteria for autism, while 4.4% had severe social dysfunction and isolation, 15% had cognitive disorders, and 15% had received treatment for an affective disorder. Bolton and colleagues (1994) also used a family history interview method to study 195 parents and 137 siblings of autistic probands and found that 12-20% of siblings demonstrated broader phenotype impairments, including language and communication deficits, social impairments, and/or learning disabilities, as compared to only 2-3% of siblings of individuals with Down syndrome. A more recent study reported that 12% of siblings and 10% of parents showed broader phenotype characteristics (Starr et al., 2001). Relatives of autistic probands from multiplex families (i.e., families with two or more children with autism) are at greater risk of having broader phenotype impairments than relatives of probands from families

with only one child with autism (Szatmari et al., 2000). Like autism, broader phenotype characteristics are more common in males than females, although this is less true of the mildest cases (Bailey et al., 1996; Szatmari et al., 2000). However, unlike autism, individuals with broader phenotype characteristics are generally of normal intelligence (Bailey et al., 1996).

What characteristics define the broader autism phenotype? Many broader phenotype studies have relied on a family history interview developed by researchers from Rutter and Folstein's groups (Family History Interview; Bolton et al., 1994) to assess behavior in the three domains affected in autism: social interaction, communication, and restricted and repetitive interests and behaviors. Items in the social domain include lack of affection, impaired friendships, impaired social play, impaired conversation, and odd or socially inappropriate behavior. Communication items include language delay, reading, articulation, and spelling difficulties. The restricted and repetitive behavior domain includes items intended to capture circumscribed interests, rigidity, obsessions/compulsions, and repetitive behaviors. Additionally, the Family History Interview assesses behaviors both in childhood and adulthood and yields a composite score for each of the three domains. Using this method, social impairments were observed in a majority of nonautistic monozygotic twins (Bailey, Le Couteur, Gottesman, & Bolton, 1995; Le Couteur et al., 1996). Follow-up into adulthood of nonautistic monozygotic twins from Folstein and Rutter's study (1977) indicated persistent impairments in confiding relationships, limited independence, and difficulties in employment (Le Couteur et al., 1996). Piven and colleagues (Piven, Palmer, Jacobi, Childress & Arndt, 1997) used the family history method to examine rates of social

deficits, communication impairments, and stereotyped behaviors in relatives (parents, aunts, uncles, grandparents, and siblings) in families with two or more children with autism and in families with children with Down syndrome. Increased rates of social and communication impairments as well as stereotyped behaviors were found in parents, aunts, uncles, and grandparents in the multiplex autism families. Although there were only 12 siblings in this sample, a higher rate of social deficits was found (33% versus 0% among siblings in Down syndrome families).

In the communication domain, Bailey and colleagues (1995) found language delay, articulation disorders, and specific reading and spelling difficulties in half of nonautistic monozygotic twins and in 10% of dizygotic twins. In another study, siblings with a history of language delay also exhibited significantly lower verbal and full-scale IQ scores (approximately 9 points lower, although still within the normal range) compared to nonaffected relatives (Fombonne, Bolton, Prior, Jordan, & Rutter, 1997). In that same study, the group of siblings with *both* language delay and reading or spelling difficulties had verbal IQ scores 20 points lower on average compared to nonaffected siblings. Plumet and colleagues (1995) demonstrated that lower verbal scores were specific to brothers of females with autism compared to siblings of Down syndrome controls. Other studies have identified elevated rates of specific language impairments in family members, including impairments on tests of executive function, especially set-shifting and planning, among siblings (Hughes, Plumet, & Leboyer, 1999), reading ability, including passage comprehension and rapid automotized naming (Piven & Palmer, 1997), verbal fluency in siblings (Hughes et al., 1999), and pragmatic language (Landa, et al., 1992). Folstein and colleagues (1999) have argued that manifestations of

the language-related component of the broader phenotype are separate from the social-related component. Incorporating language phenotypes has strengthened evidence for linkage at loci on chromosomes 2 and 7 (Bradford et al., 2001; Buxbaum et al., 2001). In a recent study of siblingships with at least one autistic proband and another sibling with either autism or autism-related impairments, a number of familial features emerged including delays in and presence of useful phrase speech, repetitive behavior, and nonverbal communication as assessed by the Autism Diagnostic Interview – Revised (Silverman et al., 2002).

Broader phenotype traits in the domain of repetitive and restricted interests and behaviors have not been studied as extensively as social and communication impairments. The Family History Interview includes circumscribed interests, rigidity, obsessions/compulsions, and repetitive behaviors as a composite score. Using this method, Bolton and colleagues (1994) found that although these behaviors occurred more frequently among first degree relatives of autistic probands than controls, they were less common than social or communication impairments and never occurred in isolation. Piven, Palmer, Jacobi et al. (1997) reported an increased rate of these behaviors, as well as social and communication impairments, among relatives of multiplex probands (as reported above). Studies assessing personality traits have found higher rates of anxiety (Murphy et al., 2000) and rigidity (Piven, Palmer, Landa, et al., 1997) in parents of autistic probands than in controls. In sum, impairments in this domain occur in a smaller minority of relatives and generally in association with social and/or communication difficulties (Bailey, Palferman, Heavey, & Le Couteur, 1998).

Studies of environmental factors, the broad autism phenotype, and social adjustment and emotional functioning of siblings in general are reported next. These include a study by Boutin and colleagues (1997) that revealed that cognitive disabilities in siblings were found more often in low-SES families. In another study by Gold (1993), there was an association between elevated depression scores in female non-autistic siblings and characteristics of the sibling with autism, including age and length of time since diagnosis. In male siblings, elevated depression scores were related to a feeling that there was noting good about having a brother with autism. Further, adolescent siblings were more depressed than siblings below age 12 years. Mates (1990) found that parent participation in the TEACCH (Treatment and education of autistic and communication-handicapped children) program was a possible protective factor for sibling adjustment. Henderson and Vandenberg (1992) reported that improved family adjustment was associated with a supportive social network, parental characteristics such as an internal locus of control, and the severity of the disability of the child with autism in the family. Higher marital satisfaction among parents of children with autism was correlated with higher levels of self-esteem in the non-autistic siblings in these families (Rodrigue, Geffken, & Morgan, 1993). However, Fisman and colleagues (1996) examined marital satisfaction and family cohesiveness in families of Down syndrome children, typically developing children, and children with pervasive developmental disorders and found that these served as protective factors only for siblings of Down syndrome and typical children. Systematic studies of other possible influences on sibling adjustment, such as parental attention, are only in preliminary stages, and studies that examine the transaction of both genetic and environmental factors as it relates to the development of siblings have

not yet been conducted to our knowledge. In the meantime, utilizing different comparison groups can help to clarify both genetic and environmental contributions to the impairments evidenced by siblings (Bauminger & Yirmiya, 2001).

Early Development of Siblings

While there has been an increase in broader phenotype studies in recent years, and thus in studies of siblings, the early development of non-autistic siblings of children with autism under age 2 has not been fully investigated. The following is a review of studies of very young siblings of children with autism to date.

In a recently published study of 21 siblings of children with autism and 21 siblings of typically developing children with no family history of autism, Yirmiya and colleagues (2006) found that, as early as 4 months, dyads of mothers and siblings of children with autism were less synchronous during mother-child interactions led by the infant, and the siblings were less upset by a still-face paradigm, as compared to dyads and sibs of typical children. Surprisingly, however, siblings of children with autism responded to their name being called more often than sibs of typical children at 4 months. At 14 months, sibs of children with autism were found to use requesting gestures less often, and had lower language scores, than sibs of typical children. In another recent study, Goldberg and colleagues (2005) found that 14-19 month old non-autistic siblings of children with autism obtained lower scores on the Early Social and Communication Scales (ESCS; Mundy, Delgado, Hogan, & Doehring, 2003; Seibert & Hogan, 1982) as compared to toddlers with typical development. Non-autistic siblings actually performed similarly to children with autism on measures of responding to social interaction, initiating joint attention, and requesting behavior. However, this study was limited by a

very small sample size (8 children with autism, 8 siblings, and 9 typically developing children). Landa and Garrett-Mayer (2006) used the Mullen Scales of Early Learning (Mullen, 1997) to assess 60 siblings of children with autism and 27 age-matched low-risk (i.e., no family history of autism) toddlers at 6, 14, and 24 months of age. The children were classified at 24 months of age into three groups: children with an ASD (22 siblings and 2 low risk), children with language delay (LD; 9 siblings and 2 low risk), and unaffected children (29 siblings and 23 low risk). Results showed that, at 14 months, the ASD group had lower scores in all Mullen domains than the unaffected group, and the LD group (largely composed of non-autistic siblings) had lower fine motor and receptive language skills than the unaffected group. By 24 months, the LD group performed similarly to the unaffected group in motor skills, but continued to show lower scores in visual problem solving and receptive and expressive language skills. At 14 months, the LD group also showed a pattern of higher visual vs. receptive language skills, similar to the ASD group, although this difference was no longer apparent at 24 months of age for the LD group.

In an ongoing prospective study of siblings being conducted by Zwaigenbaum and colleagues (2005), an 18-item observation-based scale, the Autism Observation Scale for Infants (AOSI; Bryson, Rombough, McDermott, Brian, & Zwaigenbaum, 2000), was used to assess for the presence/absence of behavioral risk markers at 6, 12, 18, 24, and 36 months of age. Thus far, 65 of 150 siblings, and 23 low-risk infant controls, have been followed to 24 months of age (Zwaigenbaum et al., 2005). Results indicated that siblings of children with autism showed more behavioral risk markers at 12 months than controls, and that risk markers at 12 months predicted social and communication impairments at

24 months of age. The most prominent risk markers at 12 months of age included impairments in eye contact, visual tracking, disengagement of visual attention, orienting to name, imitation, social smiling, reactivity, social interest and affect, and sensory-oriented behaviors. Of siblings who exhibited these behavioral risk markers at 12 months of age, 7 met criteria for Autistic Disorder, and 12 met criteria for Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), at 24 months of age. In addition, these children showed language delays and a characteristic pattern of temperament, with passivity and decreased activity level at 6 months of age, followed by extreme distress reactions and decreased positive affect by 12 months. Group differences were also found between non-autistic siblings and control infants, with non-autistic siblings showing more early risk markers than controls. Two siblings in this group showed as many as 7 or more behavioral risk markers at 12 months of age, but to a milder degree than those children meeting full criteria for an autism spectrum disorder. Stereotypical sensory and motor behaviors were also assessed in this study, but did not distinguish non-autistic siblings from controls.

Studies focusing on language abilities in very young siblings have yielded mixed results. Ozonoff, Rogers, and Sigman (2005) recently reported on a large sample of toddlers (86 sibs of children with autism and 37 sibs of children with typical development). They found that at 18 months of age, siblings of children with autism obtained lower receptive and expressive language scores on the Mullen Scales of Early Learning (Mullen, 1997) compared to siblings of typically developing children. However, in this study, 36 month data had not yet been obtained and so it is not clear if the group means were lowered primarily by siblings who later were diagnosed with autism. In

another study that followed infant siblings from 4 to 54 months, Yirmiya and colleagues (2005) reported on the cognitive and verbal abilities of 30 non-autistic siblings of children with autism as compared to 30 siblings of typically developing children. At 14 months, non-autistic siblings of children with autism had lower language scores on the Bayley Scales of Infant Development (Bayley, 1993) than siblings of typically developing children. However, by 24 months of age, there were no differences on the Bayley or the Reynell Developmental Language Scales (Reynell & Gruber, 1990), although a significant number of siblings of children with autism scored 1 and 2 SDs below the mean in receptive language skills as compared to sibs of typically developing children. At 36 months, siblings of children with autism had lower receptive language scores on the Clinical Evaluation of Language Fundamentals-Preschool (CELF-P; Wiig, Secord, & Semel, 1992) compared to sibs of typical children, but by 54 months, there were no overall group differences (although a greater number of siblings of children with autism scored 1 SD below the mean as compared to siblings of typical children).

In sum, there have been only a few studies published to date with adequate sample sizes that have focused on the early behaviors of non-autistic siblings. In addition, these studies have tended to focus primarily on cognitive and language abilities, and have yielded mixed results. The current study represents a unique contribution to the literature by using multiple measures to examine a broad range of early abilities – cognitive and adaptive functioning, language, social communication, social-emotional functioning, face recognition, imitation, and functional and symbolic play skills – in a group of 18-27 month old non-autistic siblings of children with autism as compared to toddlers with no family history of autism. Further, detailed data on children's development from birth to

two were obtained by parent interview. Finally, multiple measures were used to assess parent mental health, marital relationship, and stress to determine whether differences in child behavior could be accounted for by differences in parental well-being.

The literature on early indicators of autism, reviewed below, guided the selection of specific measures used in the current study.

Early Indicators of Autism

Language

The acquisition and development of language in autism is often delayed and/or deviant, with approximately 30% of individuals never acquiring spoken language (Bryson, 1996; Lord & Paul, 1997). In addition to delays in language acquisition, persons with autism often exhibit atypical speech patterns, including immediate or delayed echolalia (i.e., verbatim repetition of words or phrases), unusual prosody (e.g., atypical intonation, rhythm, stress, and volume) and pronoun reversal (e.g., "*you* want a drink" instead of "*I* want a drink"), which can persist into adulthood (Cantwell, Baker, Rutter, & Mawhood, 1989; Kanner, 1943; Lee, Hobson, & Chiat, 1994).

Social Orienting

One of the earliest social attention impairments in autism is a lack of normal "social orienting," namely, the tendency to spontaneously orient to naturally occurring social stimuli in one's environment (Dawson, Meltzoff, Osterling, & Rinaldi, 1998; Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998). In typical development, infants devote particular attention to social stimuli, including faces, voices, and other aspects of human beings (Rochat & Striano, 1999). Indeed, by six months of age, typically developing infants will actively orient (i.e., turn head and/or eyes) to novel stimuli,

particularly social stimuli (e.g., being called by name) (Trevarthen, 1979). Children with autism, however, exhibit early impairments in social orienting as evidenced by home videotape studies of infants later diagnosed with autism (Osterling & Dawson, 1994; Osterling, Dawson, & Munson, 2002; Werner, Dawson, Osterling, & Dinno, 2000). In two experimental studies of preschool aged children with autism and mental-aged matched children with developmental delay, children with autism more frequently failed to orient to both social and nonsocial stimuli, but the impairment was more severe for social stimuli (Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998; Dawson, Toth, et al., 2004). It has been argued that a failure to orient to social stimuli represents one of the earliest and most basic social impairments in autism and may contribute to later social and communicative impairments by depriving the child of critical early social experiences (Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998; Mundy & Neal, 2001).

Joint Attention

Joint attention behaviors include sharing attention in regard to an object or event (e.g., through the use of alternating eye gaze), following the attention of another (e.g., following a gaze or point), and directing attention (e.g., showing and pointing to objects/events). Some infants display some aspects of joint attention (e.g., matching direction of mother's gaze to a visible target) as early as 6 months of age (Morales, Mundy, & Rojas, 1998), and most infants display all of these skills by 12 months of age (Carpenter, Nagell, & Tomasello, 1998; Leekam & Moore, 2001). Research has established joint attention ability as a core social-communication impairment in children with autism, present by one year of age and incorporated into the diagnostic criteria for

autism (Mundy, Sigman, Ungerer, & Sherman, 1986; DSM-IV, American Psychiatric Association, 1994). Impairments in joint attention skills have been found to distinguish preschool age children with autism from those with typical and delayed development (Bacon, Fein, Morris, Waterhouse, & Allen, 1998; Charman et al., 1998; Dawson, Meltzoff, Osterling & Rinaldi, 1998; Dawson, Munson, et al., 2002; Mundy et al., 1986). Additionally, impairments in protodeclarative joint attention behaviors (e.g. pointing to show, sharing) seem to be more severe than impairments in protoimperative joint attention behaviors (e.g., pointing to make a request) in children with autism (Mundy et al., 1986; Mundy, Sigman, & Kasari, 1990; Sigman et al., 1986). Joint attention ability is predictive of both concurrent language ability, and future gains in expressive language skills, for children with autism (Mundy, Sigman, & Kasari, 1990; Mundy, Sigman, Ungerer, & Sherman, 1987; Sigman & Ruskin, 1999; Toth, Dawson, Meltzoff, & Munson, e-pub ahead of print 7-15-06). Taken together, these findings suggest that joint attention ability is a "pivotal" skill in autism as it appears to lay a foundation for the development of more complex abilities, such as pretend play, language, and theory of mind (Charman, 2003, 1997; Mundy & Crowson, 1997; Sigman, 1997).

Dawson and colleagues (Dawson, Toth, et al., 2004) recently examined which of these impairments best discriminated 3-4 year old children with autism spectrum disorder from matched DD and typical children. They found that, based on measures of joint attention ability alone, it was possible to correctly classify 83% of children with ASD and 63% of children without ASD at 3-4 years of age in this sample. When children with only autistic disorder were considered (i.e., excluding those with PDD-NOS), 97% of children with autistic disorder and 67% of children without autism were correctly classified, based

on their joint attention ability. Adding social orienting ability decreased the sensitivity somewhat, but added greater specificity. When both joint attention and social orienting abilities were considered, 75.5% of the children with ASD and 81.5% of children without autism were correctly classified. When only children with autistic disorder were considered, 88.6% of the children with autistic disorder and 89% of children without autism were correctly classified. Thus, the combination of joint attention and social orienting improved classification overall.

These results suggest that joint attention impairment is highly characteristic of autism, but that some children without autism also fail on tasks of joint attention. These findings are not surprising since joint attention abilities appear toward the end of the first year of life. Therefore, if a child's mental age is at or below 12 months, he or she might not have yet achieved joint attention skills. On the other hand, social orienting is a very early emerging ability, apparent by at least 6 months of life (Rochat & Striano, 1999). Thus, unless a child was very young or much delayed in his or her development, it is unlikely that he or she would fail on social orienting tasks simply based on his or her developmental level. In the current study, we assessed both social orienting and joint attention in siblings and controls.

Face Recognition

In typical development, a visual preference for faces (Goren, Sarty, & Wu, 1975) and the capacity for very rapid face recognition (Walton & Bower, 1993) are present at birth. By 4 months, infants recognize upright faces better than upside down faces (Fagan, 1972). Therefore, an impairment in face recognition may be one of the earliest indicators of abnormal brain development in autism. In a study of home videotapes of 1st birthday

parties of infants with autism, a failure to attend to others' faces was the single best discriminator between 1-year-olds with autism vs. typical development (Osterling & Dawson, 1994). Face recognition impairments have been found in many studies of older children and adults with autism (Boucher, Lewis, & Collis, 1998; Cipolotti, Robinson, Blair, & Frith, 1999; Hauk, Fein, Maltby, Waterhouse, & Feinstein, 1999; Jambaque, Mottron, Ponsot, & Chiron, 1998; Klin et al., 1999; Ozonoff, Pennington, & Rogers, 1990; Teunisse & DeGelder, 1994). In fMRI studies of face recognition, the fusiform gyrus is activated, typically more on the right than left (Gauthier, Tarr, Anderson, Skudlarski, & Gore, 1999; Kanwisher, McDermott, & Chun, 1997; McCarthy, Puce, Gore, & Allison, 1997). However, in individuals with autism and Asperger syndrome, a recent fMRI study showed a failure to activate the fusiform face area during face processing (Schultz et al., 2000).

Recently, Dawson and colleagues (Dawson, Carver, Meltzoff, Panagiotides, & McPartland, 2002) studied face recognition in preschool aged children with autism to better determine when such impairments emerge. Both face and object recognition were measured using a passive viewing paradigm (deHaan & Nelson, 1997; 1999) in which a child is shown a picture of mother's face versus an unfamiliar female face, and a favorite toy versus an unfamiliar toy, while event-related potentials (ERPs) are collected. Results showed that both preschool aged children with typical development and those with developmental delay showed differential ERP responses to mother's face versus a stranger's face and to a favorite object versus an unfamiliar object (Dawson, Carver, et al., 2002). In contrast, children with autism failed to show differential ERPs to mother's versus stranger's face, but did show differential ERPs to a favorite versus unfamiliar toy.

In fact, their ERP patterns in response to toys were quite similar to those of the chronological age-matched typical children. These data add to the growing body of evidence indicating an early-emerging impairment in face processing in autism.

In addition to face processing impairments in individuals with autism, a recent study by Dawson and colleagues (Dawson, Webb, et al., 2004) provides evidence for altered face processing in parents of children with autism. Parents (n=143) from multiplex families were administered standardized cognitive tasks as well as a test of face recognition ability. Previous work has shown that typically developing adults with no family history of autism demonstrate a right hemisphere response that is greater and faster to faces than to non-face stimuli. Results revealed that 29% of parents from multiplex families showed a significant deficit in face recognition ability relative to their performance on verbal and visual cognitive tasks. Electrophysiological data were then obtained for a subset of parents in response to viewing upright and inverted faces and chairs. Results showed that parents of children with autism exhibited reduced right hemisphere amplitude when viewing faces, and showed no significant difference in latency of brain response when processing faces compared to chairs, as compared to adults with no familial history of autism (Dawson, Webb, et al., 2004). These authors conclude that impaired face processing ability may be a functional neural trait marker of genetic susceptibility to autism.

Few studies using ERPs have been published with children between 12 and 36 months of age and often good data are only available for 50% of the sample. For these reasons, a visual habituation paradigm (described in detail below), which measures looking behavior, was used in the current study to assess face and object recognition.

Imitation

Meltzoff and Moore (1977, 1983, 1989, 1994) demonstrated that newborns are able to imitate facial expressions, which suggests that this is an innate ability. Children with autism, however, show impairments in both immediate and deferred motor imitation (Charman et al., 1998; Dawson, Meltzoff, Osterling, & Rinaldi, 1998; Sigman & Ungerer, 1984; Stone, Ousley, Yoder, Hogan, & Hepburn, 1997). Importantly, imitation skills in children with autism have been shown to predict later social and language learning (Charman et al., 2000, 2003; Stone & Yoder, 2001; Stone, Ousley, & Littleford, 1997; Toth, Dawson, Meltzoff, & Munson, e-pub ahead of print 7-15-06). In one study, body imitation was found to predict expressive language ability, whereas object imitation predicted play skills (Stone, Ousley, Yoder, et al., 1997). Additionally, it has been theorized that a failure to engage in social imitative play may interfere with the development of joint attention, social reciprocity, and later theory of mind abilities (Dawson, 1991; Meltzoff & Gopnik, 1993; Rogers & Pennington, 1991).

In preliminary, unpublished analyses of a large sample of 3-4 year old children with autism spectrum disorders, developmental delay, and typical development, Dawson, Meltzoff, and colleagues recently discovered that children with the full syndrome of autism are significantly more impaired in terms of their imitation ability than mental age matched children with idiopathic developmental delay or those with fewer symptoms of autism (i.e., pervasive developmental disorder – not otherwise specified). Thus, motor imitation ability may be diagnostic of autistic disorder and/or prognostic of level of severity of the disorder. In the current study, we examined motor imitation abilities in siblings and controls.

Play

Representational, or symbolic, play typically emerges between 14 and 22 months of age and includes using an object to represent another object (e.g., a block to represent a car), using absent objects as if they were present (e.g., food that does not exist), or animating objects (e.g., pretending that stuffed animals can talk) (Leslie, 1987). In children with autism, symbolic play is an early ability that is often absent at 18 months of age (Baron-Cohen et al., 1996) or delayed relative to mental age matched developmentally delayed and typical children (Charman et al., 1998; Dawson, Meltzoff, Osterling, & Rinaldi, 1998; Mundy et al., 1987; Wing & Gould, 1979). For those children with autism who do acquire symbolic play skills, their level of symbolic play often remains below that of their language abilities (Amato, Barrow, & Domingo, 1999; Ungerer, 1989; Wing, 1978) and is often less diverse and elaborate compared to that of developmentally delayed and typical children (Ungerer & Sigman, 1981). Further, symbolic play has been associated with both language and social ability in young children with autism (Sigman & Ruskin, 1999; Toth, Dawson, Meltzoff, & Munson, e-pub ahead of print 7-15-06; Toth, Dawson, Munson, Estes, & Abbott, 2003). There exists some controversy as to the cause of this impairment; some believe that it results from impairments in joint attention and understanding others, while others believe it hinges on deficits in symbolic thinking and executive functioning (Charman, 1997). In any case, symbolic play is an early impairment in autism and so was included in the current study.

Purpose of Study

The current study had the following specific aims and hypotheses.

Research Aim 1

To determine whether there exist differences in social attention, language, play, imitation, and stereotyped behavior in 18-27 month old non-autistic siblings of children with autism as compared to 18-27 month olds with no family history of autism (i.e., to define the characteristics that comprise the broad autism phenotype at 18-27 months of age).

Hypothesis 1. Based on previous findings that 12-month-old siblings of children with autism exhibit deficits predominantly in social attention, language, and imitation, it was hypothesized that non-autistic siblings of children with autism would perform less well on tests of social attention (social orienting and joint attention), language, and imitation, but not on measures of toy play or atypical behavior, as compared to children with no family history of autism. Furthermore, prior work by Dawson and colleagues (Dawson, Webb, et al., 2004) provided evidence of a specific face processing deficit in parents of children with autism, suggesting that altered face processing may be familial and possibly a marker of genetic susceptibility to autism. Thus, it was hypothesized that non-autistic siblings of children with autism would show specific deficits in face recognition, but not in object recognition, compared to children with no family history of autism.

Research Aim 2

To determine whether parents of non-autistic siblings versus parents of toddlers with no family history of autism report differences in social attention, language, and stereotyped behavior as early as 12 months of age.

Hypothesis 2. Based on previous findings from studies of 12-month-old siblings of children with autism, it was hypothesized that, using the Early Development Interview (EDI) – a detailed developmental history method based on retrospective parent report – non-autistic siblings of children with autism would be reported to show significantly more symptoms in the social and communication domains as compared to toddlers with no family history of autism. Non-autistic siblings were not expected to show elevated rates of atypical behavior at 12 months of age compared to comparison children.

CHAPTER II

Method

Participants

As part of an ongoing NIMH-funded early detection study (Dawson, PI, at UW site), 150 siblings of children with autism were recruited and screened for an autism spectrum disorder using the Modified Checklist for Autism in Toddlers (M-CHAT; Robins, Fein, Barton, & Green, 2001). Of this group of 150, 62 siblings (i.e., *all* siblings between the ages of 18 and 27 months, both those who passed the screen and those who failed) participated in the present study. Twelve of these 62 siblings were diagnosed with an autism spectrum disorder using the diagnostic measures described below. Additionally, 8 siblings were excluded from the study because the older sibling did not meet criteria for an autism spectrum disorder using the diagnostic measures described below. The 42 remaining non-autistic siblings comprise the group of interest for this study. These 42 siblings were compared to 20 typically developing toddlers (11 had older siblings, 9 did not) with no family history of autism recruited through the NIMH-funded UW STAART Center early characteristics of autism study. Table 1 summarizes the characteristics of these two groups. A Pearson chi square test confirmed that the gender ratio for the two groups was non-significant (χ^2 (1, N = 62) = 1.231, p = .267). On average, non-autistic siblings were approximately 2 months younger than comparison children, both in terms of chronological age (CA; $F(1, 61) = 10.33$, $p < .01$) and mental age (MA; $F(1, 61) = 6.03$, $p < .05$). However, when CA was covaried, the groups no longer differed in mental age. Therefore, CA was entered as a covariate in analyses, except when standard scores were used as these already correct for CA.

Table 1. *Participant characteristics*

Diagnostic Group	*n*	M:F	*M* CA in mos. (*SD*)	*M* MA in mos. (*SD*)
Non-autistic siblings	42	21:21	20.31 (2.18)	20.29 (4.21)
Comparison children	20	13:7	22.40 (2.80)	22.85 (2.91)

Siblings and their parents were recruited from the Center on Human Development and Disability, the UW Autism Center, local public health agencies, pediatric offices, and Birth-to-Three Centers; typically developing children were recruited from the Psychology Department Infant Subject Pool. Exclusionary criteria included significant sensory or motor impairment, major physical/medical problems, and history of serious head injury and/or neurological disease. Children in the comparison group were excluded if they exhibited accelerated development (a score of > 115) as assessed by the Mullen Scales of Early Learning (Mullen, 1997) or if they had a family history of autism. The justification for this is that there tends to be a selection bias in university-recruited samples and we did not want to create artificial differences by having a comparison group that was above average in IQ. Over the course of the study, 13 children were excluded from the comparison group based on above average composite IQ scores on the Mullen. All groups were matched on SES. Ethnicity was as follows: for the sibling group: 28 European American, 9 Multiracial, 4 Asian/Pacific Islander, and 1 Native American; for the comparison group: 18 European American, 1 Multiracial, and 1 African American.

This study was approved by the University of Washington (Seattle) institutional review board and informed consent for human investigation was obtained from parents of all children participating in this study.

Measures

The following measures were collected over three days of testing (see Tables 2 and 3). For all tests, the child sat in the parent's lap and breaks were given as needed. The clinicians administering these measures were not blind to group membership.

Table 2. *Measures*

Autism screen	Modified Checklist for Autism in Toddlers (M-CHAT)	Parent report
Diagnosis	Autism Diagnostic Interview – Revised, Toddler (ADI-R)	Parent report
	Autism Diagnostic Observation Schedule (ADOS)	Direct observation
	DSM-IV Clinical Diagnosis	Direct observation
Autism symptoms	Early Development Interview (EDI)	Parent report
	Repetitive Behavior Scale-Revised (RBS-R)	Parent report
IQ/adaptive behavior	Mullen Scales of Early Learning	Direct observation
	Vineland Adaptive Behavior Scales	Parent report
Language	Mullen Scales of Early Learning receptive/expressive language	Direct observation
Social	Vineland Social-Emotional Early Childhood Scales (SEEC)	Parent report
	Social orienting	Direct observation
	Communication and Symbolic Behavior Scale (CSBS-DP)	Direct observation
	Joint attention (Butterworth & Jarrett; CSBS-DP; ADOS)	Direct observation
	Face and object recognition	Direct observation
	Immediate and deferred motor imitation (Meltzoff battery)	Direct observation
Play	Functional toy use (PAS)	Direct observation
	Symbolic toy use (PAS)	Direct observation
Parent Factors	Brief Symptom Inventory (BSI)	Parent self-report
	Dyadic Adjustment Scale (DAS)	Parent self-report
	Life Experiences Survey (LES)	Parent self-report

Autism screening measure. The Modified-Checklist for Autism in Toddlers (M-CHAT; Robins, Fein, Barton, & Green, 2001) was used to screen all of the siblings for symptoms of autism. The M-CHAT is a 23-item, yes/no, parent report measure that assesses behaviors in the following domains: play, nonverbal communication, social orienting, social interaction, and repetitive motor behavior. Preliminary studies have shown the M-CHAT to have a specificity of 95% and a sensitivity of 97% in detecting

Table 3. *Dependent variables by domain*

Social	Social orienting
	Joint attention: initiating and responding scores
	CSBS-DP social communication scores
	Face and object recognition
	Early Development Interview (EDI) social domain score
	Vineland Social-Emotional Early Childhood Scales (SEEC)
	ADOS social scores
Communication	Mullen Scales of Early Learning language scores
	Early Development Interview (EDI) communication domain score
Imitation	Immediate and deferred motor imitation
Toy play	Functional toy use
	Symbolic toy use
Stereotyped behavior	Repetitive Behavior Scale-Revised (RBS-R)
	Early Development Interview (EDI) sensory/rep behavior domain score
Parent Factors	Brief Symptom Inventory (BSI) domain and global indices scores
	Dyadic Adjustment Scale (DAS) domain and total scores
	Life Experiences Survey (LES) domain and total scores

autism in very young children (Robins, Fein, Barton, & Green, 2001). All of the children (n = 42) in the group of non-autistic siblings passed the screen, meaning they failed no more than one critical item and no more than two items total.

Diagnostic assessment. All children were administered a standardized diagnostic assessment consisting of the following three measures: (1) the Autism Diagnostic Interview – Revised, Toddler version (ADI-R; Lord, Rutter, & Le Couteur, 1994), which was administered only to the sibling group, (2) the Autism Diagnostic Observation Schedule (ADOS; Lord, Rutter, DiLavore, & Risi, 1999; Lord et al., 2000), administered to all children, and (3) clinical judgment of an experienced clinician based on presence/absence of autism symptoms per DSM-IV (DSM-IV; American Psychiatric

Association, 1994), completed for all children. Diagnosis of autism was defined as meeting criteria for Autistic Disorder on the ADOS and ADI-R and meeting DSM-IV criteria for Autistic Disorder based on clinical judgment. In addition, if a child received a diagnosis of Autistic Disorder on the ADOS and based on DSM-IV clinical diagnosis, and came within 2 points of meeting criteria on the social or communication domains of the ADI-R, the child was also considered to have Autistic Disorder. Diagnosis of PDD-NOS was defined as meeting criteria for PDD-NOS on the ADOS, meeting criteria for Autistic Disorder on the ADI-R or missing criteria on the social or communication domains of the ADI-R by 2 or fewer points, and meeting DSM-IV criteria for PDD-NOS based on clinical judgment. Of 62 siblings in the larger sample, 12 met criteria for Autistic Disorder or Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), and 8 had an older sibling who did not meet criteria on all three diagnostic instruments as described above, and so were excluded from the sample. Children in the comparison group were administered the ADOS to ensure that they did not meet criteria for Autistic Disorder or PDD-NOS on the ADOS or based on clinical judgment, and did not show elevated symptoms on these measures. The ADI-R Toddler was not administered to parents of typical children however.

Cognitive, motor, language, and adaptive behavior. The Mullen Scales of Early Learning (Mullen, 1997) is a standardized measure for use with infants and preschool children from birth through 68 months. The Mullen assesses gross motor, visual reception, fine motor, receptive language, and expressive language abilities, and also yields a composite score. The Vineland Adaptive Behavior Scales (Sparrow, Balla, &

Cicchetti, 1984), a well-standardized measure of adaptive functioning, was administered to all children.

Social-emotional functioning. The Vineland Social-Emotional Early Childhood Scales (SEEC; Sparrow, Balla, & Cicchetti, 1998), which assesses social-emotional functioning in early childhood and yields domain scores, including Interpersonal Relationships and Play and Leisure Time, as well as a composite score, was administered to all children.

Social orienting. In this procedure, developed by Dawson (Dawson, Meltzoff, Oserling, & Rinaldi,1998; Dawson, Meltzoff, Oserling, Rinaldi, & Brown, 1998), the child sat across from a familiar examiner and was allowed to play with a toy chosen to be only mildly interesting. Once the child was engaged in play, a second examiner delivered the stimuli. There were four social stimuli consisting of live sounds created by the human voice and body: (a) humming a neutral tone, (b) calling the child's name, (c) snapping fingers, and (d) patting hands on thighs. Four nonsocial stimuli consisted of mechanical sounds of inanimate objects, although activated by a human being: (a) a timer beeping, (b) a phone ringing, (c) a whistle blowing, and (d) a tape recording of a car horn, delivered while the examiner held a toy car. Each stimulus was delivered three times with a 1-s interstimulus interval, for a total presentation time of approximately 6 s. A decibel meter placed on the table next to the child for a subset of participants confirmed that social and nonsocial stimuli were matched in terms of loudness. To control for acoustics, the same testing room was used for all children for this task. Stimulus order and location (behind vs. in front of the child, 30° to the right or the left) were counterbalanced across

participants. Delivery of the stimuli occurred only when the child was not looking at the second examiner, and the second examiner remained in each location for 15 s following delivery to allow for a delayed orienting response. The examiner assumed a neutral facial expression when delivering social stimuli and looked down at the floor when delivering nonsocial stimuli to ensure that the child's response was not unduly influenced by personal or social characteristics specific to the examiner. Parent ratings of the degree of familiarity on a 5-point scale ranging from 1 (*very unfamiliar*) to 5 (*very familiar*) were obtained for each of the stimuli. There were no significant between-group ($F(1, 58) = .00, p = .98$) or within-group (siblings $t(41) = -.18, p = .86$, typical $t(17) = .09, p = .93$) differences in degree of familiarity for social versus nonsocial stimuli, nor was there a group by stimulus interaction ($F(1, 58) = .03, p = .86$).

Children's behavior was videotaped. The two examiners coded live whether the child oriented to the stimulus. An error was defined as a failure to turn head and/or eyes toward the stimulus within 15 s of delivery of the stimulus. Coding discrepancies, though rare, were resolved immediately following the task by viewing the videotape. To assess inter-observer agreement, a second independent coder reviewed the videotapes while remaining uninformed as to the initial scoring and group status. There were few disagreements and inter-observer agreement was high at .97. The dependent variables are percentages of head and/or eye turns for social and for nonsocial stimuli.

Joint attention: Responding to joint attention. This procedure is based on an experimental method originally developed by Butterworth and Jarrett (1991) to assess response to joint attention in infants and toddlers. The child sat at a table across from a familiar examiner and was allowed to play with a mildly interesting toy. Four red

cardboard stars, approximately 8 inches tall, were mounted on the wall at the child's eye level, 62 inches from the center of the room (where the child is seated) and 30 degrees in front of the child to right and left, and 30 degrees to right and left behind the child. Two types of joint attention probes were each delivered twice: (a) the examiner gazed at a star, and (b) the examiner pointed and gazed at a star. The examiner first gained the child's attention and eye contact, then delivered the probe with an audible intake of breath. Type and location of probe were counterbalanced across participants and interspersed among stimuli in the social orienting procedure (see above). The examiner and an assistant coded live whether the child looked toward the stimulus. An error was defined as a failure to turn head and/or eyes toward the stimulus within 15 s from delivery of the stimulus. The dependent variable is the percentage of head and/or eye turns given four trials.

Joint attention: Initiating and responding to joint attention. Additional measures of joint attention were derived from the Communication and Symbolic Behavior Scale-Developmental Profile (CSBS-DP; Wetherby & Prizant, 1998, 2002) and the ADOS. The CSBS-DP is a 20-minute procedure providing opportunities for sampling and rating a variety of communicative and symbolic behaviors. The child sat at a table next to a familiar examiner and the child's parent. Two colorful posters hung on the walls of the room. One at a time, the child was presented with a variety of toys, including a wind-up toy, a balloon, bubbles, a jar filled with cereal, a bag filled with toys, books, a Kermit doll, a toy kitchen set, and blocks. Twice during the assessment, the examiner gained the child's attention and then pointed to one of the posters saying, "Look." The assessment was rated from videotape by a clinician trained and reliable on this measure (i.e., intraclass correlation coefficients of .90 or greater for each item). Dependent variables

included initiating (range is 0-6) and responding (range is 0-2) to joint attention, as well as a broad range of other social communication variables detailed in the section on social communication below.

The ADOS is a 30-minute, semi-structured play observation that includes measures of both initiating and responding to joint attention. The ADOS response to joint attention item was coded based on the child's response to the following joint attention probes. While the child was playing quietly, the examiner placed herself directly in front of the child and established eye contact by calling the child's name or, if necessary, providing a physical prompt. Upon making eye contact, the examiner said, "Look, *(child's name)*," and looked towards a toy that had been placed in front and 65 degrees to the side of the child. If the child did not respond to this joint attention probe by following the examiner's gaze to the toy, it was repeated, appending the phrase, "Look at that" to the verbal prompt. If the child failed to respond to this bid, the examiner stated, "*(Child's name)*, look at that" and pointed to the toy. Scores ranged from zero to two. A score of zero indicated that the child had successfully used the orientation of the examiner's face and eyes as a cue to attend to the toy. A score of one indicated that the child had required a point to attend to the toy. A score of two indicated that the child had not responded to any of the joint attention probes or that the examiner had been unable to obtain the child's attention in five attempts and so was unable to deliver the probe. The ADOS initiating joint attention item was scored based on the examiner's judgment of the child's attempts at protodeclarative attention bids throughout the course of the entire play interview. Scores ranged from zero to two. A score of zero indicated that on at least one occasion the child directed an adult's attention to a *distal* object by gazing at the object, then

making eye contact with the examiner, and then gazing again at the object. Using a point or a vocalization was acceptable but not necessary to receive a score of zero. A score of one indicated that on at least one occasion a child partially referenced a distal object by either looking at the object and pointing or vocalizing, or by looking or pointing at an adult without redirecting attention back to the object. A score of two indicated that the child did not initiate a bid for joint attention to reference a distal object.

Social communication. The CSBS-DP provided measures of a range of social communication behaviors, including gaze shifts, shared positive affect, overall rate of communicating, behavior regulation (requesting), social interaction, gestures, sounds, words, language comprehension, and functional and symbolic object use. In addition, this measure yielded three composite scores: social, speech, and symbolic. The concurrent and predictive validity of the CSBS-DP has been well established (Wetherby, Allen, Cleary, Kublin, & Goldstein, 2002; Wetherby & Prizant, 1998, 2002).

Face and object recognition. An infant-controlled visual habituation procedure (e.g., Cohen & Strauss, 1979) was used to assess face and object recognition. This experiment addressed the following questions: (1) the length of time needed for a toddler to become habituated to a visual stimulus and if this differs based on stimulus type or group, and (2) whether recovery of interest when presented with a novel but similar looking stimulus differed based on stimulus type, group, or delay. This infant controlled visual habituation procedure allowed the amount of visual fixation necessary to meet the habituation criterion to differ across individuals based on the child's information processing speed and not on a predetermined value. Dependent variables included (a)

length of first fixation; (b) number of fixations till habituation; (c) total time to habituate; (d) visual fixation to novel stimulus at test (novelty preference) and (e) fixation to habituated stimulus at test (dishabituation). During all phases, the toddler was seated in front of a monitor and a video camera recorded looking behavior. A trained observer watched the child's eyes on a video monitor and recorded visual fixation times via a button press by observing the reflection of the stimulus in the child's pupil. The computer signaled stimulus presentation based on the subject's visual fixation (greater than 1 second) of the computer screen. Once the subject fixated on the screen, the stimulus was presented. During the habituation phase, the same stimulus was displayed until the observer signaled that the toddler was looking away from the monitor for greater than 1 second. In order to re-focus the child's attention on the computer monitor, whenever the subject looked away from the computer for longer than 1 second, a series of flashing colored boxes accompanied by sound appeared on the monitor. Once the child again attended to the computer monitor, the flashing boxes were replaced by the habituation stimulus. The computer recorded the length of each visual fixation and the number of fixations, and calculated habituation using an algorithm (see below).

There were 3 phases to this experiment. 1) In the habituation phase, the child was repeatedly exposed to a single stimulus until the mean fixation time for the 2 shortest fixations was less than half of the mean fixation time for the 2 longest fixations (habituation algorithm). 2) In the delay phase, a delay of either 10 seconds or 5 minutes was imposed prior to the start of the test phase. 3) In the test or dishabituation phase, the habituated stimulus and a novel stimulus were presented serially. The stimulus was present for the length of one visual fixation (greater than 1 second). This experiment is a

two by two design: Stimulus (face vs. house) by delay (10 s vs. 5 min). Each subject participated in 4 habituation experiments, 2 per visit (visits were on different days): face 10 s delay; face 5 minute delay; house 10 s delay; house 5 minute delay. Each subject received one face and one house experiment per visit and one 10 s and one 5 minute delay experiment per visit. The following variables were counterbalanced across subjects: order of delay (short then long or long then short); stimulus pairs (faces A/B and faces C/D; houses A/B and houses C/D—see Figure 1 for sample stimuli); habituation and test stimuli (habituation to A and novel B; habituation to B and novel A); and order of stimulus presentation (face/house or house/face). Inter-rater reliability was assessed by the following paired ratings: First, a second trained observer recorded visual fixation times via a button press at the same time the primary observer recorded the child's fixation times (81.5% of sessions were conducted in this manner). Second, independent paired ratings were made from videotapes for 58% of the remaining sessions, which were conducted with only one observer. For both types of sessions – those with one observer and those with two observers – only data with inter-rater correlations above .80 were accepted as valid and included in analyses. Across all of the children in the study, only 34 files (i.e., experiments, such as "house long") were invalid and removed from final analyses: 28 of these were due to low correlations, primarily related to child behaviors (e.g., fussiness), 4 were the result of computer error, and 2 were due to parent behavior (i.e., cueing the child to look). The average correlation for valid files was .98.

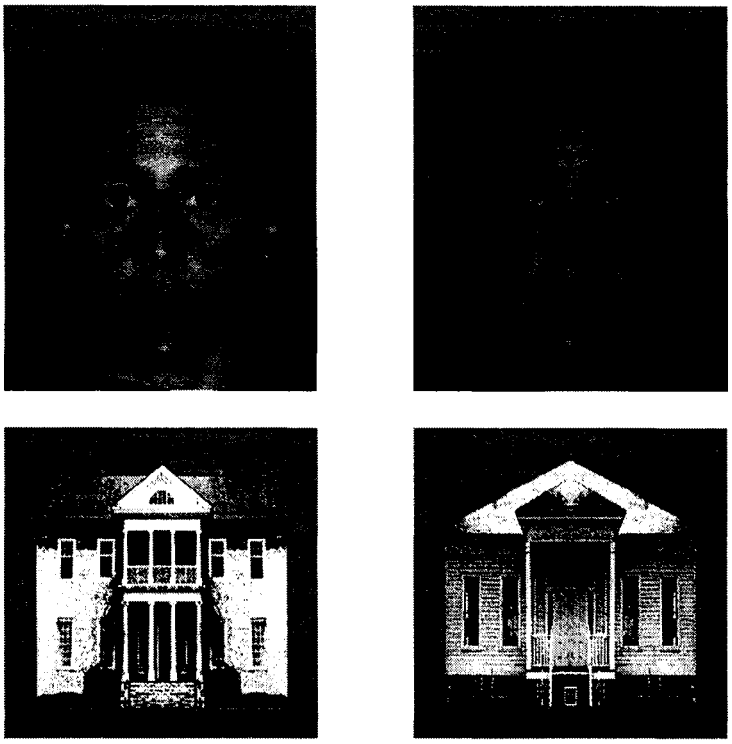

Figure 1. *Sample face and house stimuli for visual habituation task*

Imitation. Immediate and deferred motor imitation abilities were assessed based on a battery developed by Meltzoff (Meltzoff, 1988; Rast & Meltzoff, 1995). The battery consisted of 15 items, 10 immediate imitation tasks and 5 deferred imitation tasks. A range of tasks was used, including body movements (e.g., eye-blinking), novel acts on objects (e.g., touching elbow to a panel), and familiar acts on objects (e.g., banging wooden blocks). The tasks were administered while a familiar examiner was seated across a small table from the child. After gaining the child's attention, the experimenter demonstrated each target act three times in about 20 seconds. There was no verbal description of the tasks, and no physical prompting of the child to try to elicit a response. The tasks were administered in two blocks, one for the immediate imitation and the other for the deferred imitation. For the deferred imitation, the examiner demonstrated all 5 of

the target acts, and then a 10-minute memory interval was interposed. After the delay, the child was presented with the test objects one at a time in their original order. For immediate imitation, the same general procedure was followed, except that the child was given a chance to imitate directly after the demonstrated target acts. Correct or incorrect response to each imitation task was coded from videotape by a primary and reliability coder (20% of the sample). Intra- and inter-scorer agreement exceeded $r = .90$. The dependent measure was the total number of target acts performed, with a possible range of 0-15 (0-5 for each, immediate body, immediate object, and deferred object).

Play. The Play Assessment Scale (PAS; Fewell, 1992) was used to assess functional and symbolic play skills and contains 31 items ordered developmentally for use with children 2-30 months. The child was presented with a variety of play materials and the child's functional and symbolic use of the objects was scored as follows. Raw scores were calculated based on basal (three consecutive 4-point scores) and ceiling (three consecutive scores of 0) performances, with 5 points possible for each item: spontaneous play (4 points), after a general verbal prompt (e.g., "What can you do with these?") (3 points), after a specific verbal instruction (e.g., "Feed the doll") (2 points), after a specific verbal instruction with the examiner modeling the action (1 point), or the child did not engage in the target action (0 points). Symbolic play scores included all acts with a doll and all items using objects to represent other objects (see Table 4).

Stereotyped behavior. The Repetitive Behavior Scale-Revised (RBS-R) (Bodfish, Symons, & Lewis, 1999; Bodfish, Symons, Parker, & Lewis, 2000) was also administered to all children to assess for the presence/absence of the following atypical behaviors: Compulsive, Restricted, Ritualistic, Sameness, Self-injurious, Stereotyped,

Table 4. *Play assessment scale symbolic play items*

Item	Examples
Single act on doll	Talks, babbles to doll, puts spoon or bottle to doll's face or mouth, puts comb to doll's head
Appropriate serial acts involving doll or adult	Stirs in cup with spoon, picks up doll, feeds doll w/spoon
Places doll in appropriate position to 2 objects within one play scheme	Puts cup and teapot next to doll sitting up
Appropriate 3-step serial acts involving adult, doll, or other props in dramatic play with a theme	Pours from one container to another, stirs, serves to adult or doll as if having dinner
Substitutes doll for self in play; play indicates child thinks doll has senses/reacts to sensations	Holds Ernie to mirror to see himself, holds telephone to doll's ear and moves doll as if talking
Substitutes object in single meaningful act	Uses paper as a blanket to cover doll, uses peg as spoon to feed Ernie
Makes doll act on self as though doll is capable of performing actions independent of child	Places comb in doll's hand then moves doll's arm to indicate doll can brush own hair
Substitutes multiple objects in same scenario	Uses paper for a blanket and shoe for doll's bathtub
Verbalizes play plan for assigned pretend roles	"I am mother...you be baby, I cook dinner and you watch TV"
Shows adult how to perform simple motor act using a body part	Child uses finger to represent toothbrush, hand as hair brush, or fingers to eat cereal
Verbalizes play plan and uses pretend props which are identified for benefit of adult	"This is our house" (a box); "This will be my pot" (saucer)
Demonstrates functions with dissimilar object substitutions	Engages in play by pretending with dissimilar objects (e.g., brushes teeth with a wad of paper)

Total Repetitive.

Parent report of early development. The Early Development Interview (EDI; Werner, Dawson, & Munson, 2001; Werner, Dawson, Munson, & Osterling, 2005), a 96-item parent-report interview that incorporates mnemonic techniques to improve accuracy

of parent recall, inquires about symptoms in four domains (regulatory/sensory, social, communication, and repetitive) from birth to two. Items assess eye contact, orienting to social cues, interest in reciprocal social games, communicative babbling, joint attention, and atypical sensory and motor behaviors, among others. The EDI also measures family changes (birth of a sibling, move to a new home, temporary or permanent absence of a family member, etc.), child medical events (high fevers, seizures, head injuries, etc.), and stressors during pregnancy (financial/employment problems, conflict with spouse, diagnosis of older child, etc.). Children with autism versus DD versus typical development have been shown to significantly differ on the EDI beginning by 12-15 months (Werner, Dawson, Munson, & Osterling, 2005). See Table 5 for a description of EDI items by domain.

Parent mental health, marital relationship, and stress. Three measures of parental functioning were administered to both mothers and fathers of all children in the study. The Brief Symptom Inventory (BSI), a 53-item self-report scale, measured nine primary symptom dimensions (somatization, obsessive-compulsive behavior, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism) and yielded global indices (Global Severity Index and Positive Symptom Total (Derogatis, 1975). The Dyadic Adjustment Scale (DAS), a 32-item self-report measure, assessed the quality of the current marriage or relationship with partner and yielded the following domain scores: affectional expression, dyadic cohesion, dyadic consensus, and dyadic satisfaction, as well as a total score (Spanier, 1976). The Life Experiences Survey (LES), a 57-item self-report measure, assessed significant events experienced during the preceding 12 months, and the extent to which each event has had

Table 5. *Early Development Interview (EDI)*

Child age (mos)	Regulatory/Sensory	Social	Communication	Repetitive Behavior
0-3	Difficult to hold/cuddle? Exceptionally fussy? Sleeping problems? Feeding problems? Overly sensitive to noise/ touch?	None	None	None
4-6	Difficult to hold/cuddle? Exceptionally fussy? Sleeping problems? Feeding problems? Overly sensitive to noise or touch?	Lack of smiling at people? Long attention span for objects?	None	None
7-9	Difficult to hold/cuddle? Exceptionally fussy? Sleeping problems? Feeding problems? Overly sensitive to noise/ touch?	Lack of smiling at people? Long attention span for objects? Lack of orienting to name? Difficult to catch his/her eye?	Failure to use vocalizations socially?	None
10-12	Sleeping problems? Overly sensitive to noise /touch? Excessive mouthing?	Lack of smiling at people? Difficult to catch his/her eye? Failure to orient to name? Failure to follow point?	Failure to use vocalizations socially? Lack of babbling?	Repetitive/stereotyped motor movements?
13-15	Sleeping problems? Overly sensitive to noise/touch?	Difficult to catch his/her eye? Failure to orient to name? Failure to follow point? Lack of interest in interactive games? Failure to initiate simple, ritualized social interaction? Lack of pointing to express interest? Failure to show objects?	Failure to use words meaningfully?	Repetitive/stereotyped motor movements?

Table 5 (continued)

Child age (mos)	Regulatory/Sensory	Social	Communication	Repetitive Behavior
16-18	Overly sensitive to noise/ touch?	Difficult to catch his/her eye? Failure to orient to name? Failure to initiate simple, ritualized social interaction? Lack of pointing to express interest? Failure to follow point? Failure to imitate actions on objects?	Failure to use words meaningfully on regular basis? Failure to use formal gestures? Placed adult's hand on desired objects/moved adult's hand?	Repetitive/stereotyped motor movements?
19-21	Overly sensitive to noise/ touch?	Difficult to catch his/her eye? Failure to orient to name? Failure to initiate simple, ritualized social interaction? Lack of pointing to express interest? Failure to follow point/gaze? Failure to imitate actions on objects?	Failure to use words meaningfully on regular basis? Failure to use formal gestures? Placed adult's hand on desired objects/moved adults hand?	Repetitive/stereotyped motor movements?
22-24	Overly sensitive to noise/ touch?	Difficult to catch his/her eye? Failure to orient to name? Failure to initiate simple, ritualized social interaction? Lack of pointing to express interest? Failure to follow point/gaze?	Failure to use words meaningfully on regular basis? Failure to use formal gestures? Placed adult's hand on desired objects/moved adult's hand? Failure to combine words?	Repetitive/stereotyped motor movements? Had routines/rituals? Had unusual attachments to objects?

Table 5 (continued)

	Family Changes	Child Adverse Medical Events	Pregnancy Stressful Events	
	Birth of sibling Death of familiar family member Divorce of parents Marriage of a parent Move to a new home Temporary absence of a family member Permanent absence of a family member Changes in caretakers Changes in schedule/work	Seizure Head injury Other injuries and minor physical traumas High fever Hospitalization Minor illness	Injury/illness to relatives or friends Death of relative/friend Conflict in relationship with spouse/partner Legal matters for self or partner Employment/financial problems for self or partner Criminal matters for self, partner, or family	

a positive or negative impact on one's life, and yielded domain scores for positive change, negative change, and total change, as well as scores for number of positive and negative events (Sarason, Johnson, & Siegel, 1978).

CHAPTER III

Results

All children and parents with complete data were included in analyses. General linear models (e.g., repeated measures analysis of variance, univariate and multivariate analyses of variance) were used to test for mean differences between groups. The groups differed on chronological age (CA); therefore, CA was entered as a covariate, except when standard scores were examined (Vineland, Vineland SEEC, Mullen Scales of Early Learning), as these already correct for chronological age. Tables 6-12 show the means, standard deviations (or standard errors), effect sizes (Cohen's *d*), and statistical results for each of the variables examined.

Diagnostic Measures

Autism Diagnostic Observation Schedule (ADOS). The ADOS yields domain scores in social, communication, social + communication, play, and stereotyped behavior, as well as scores for individual items. Multivariate analysis of covariance (MANCOVA) revealed group differences (Wilks' Lambda = .521, $F(20, 40) = 1.84$, $p = .05$), but not in overall domain scores (see Table 6). See section on Social Abilities and Table 8 below for group differences on individual items.

Autism Diagnostic Interview – Revised: Toddler (ADI-R Toddler). The ADI-R Toddler yields scores in three domains: social, communication, and repetitive behavior. Cutoff scores are provided for each domain, with scores above cutoff indicating autistic symptomatology. Scores at or above 10 points in social, 7/8 points in communication (7

for nonverbal children, 8 for verbal children), and 3 points in repetitive behavior are considered in the autism range. It should be noted that very young children with

Table 6. *Autism Diagnostic Observation Schedule domain scores for siblings and comparison children*

	Siblings		Comparison		MANCOVA[a]			
	M[b]	*SE*	*M*	*SE*	*F*	*df*	*p*	*d*
Social (0-20)	2.60	0.38	2.11	0.56	0.48	1, 59	.492	0.36
Communication (0-16)	1.94	0.21	1.53	0.31	1.13	1, 59	.293	0.63
Social + Communication (0-36)	4.53	0.51	3.64	0.75	0.91	1, 59	.344	0.54
Play (0-4)	1.54	0.15	1.21	0.22	1.47	1, 59	.230	0.57
Stereotyped Behavior (0-8)	0.28	0.09	0.37	0.13	0.35	1, 59	.558	0.02

On the ADOS, lower scores reflect less impairment.
[a] CA was entered as a covariate.
[b] Estimated marginal means and standard errors are reported.

developmental delays without autism sometimes obtain scores above cutoff on the ADI-R, particularly in communication and repetitive behavior; the ADI-R is better at discriminating autism from overall delays at older ages (3 and older). In this study, 11 children in the sibling group (26% of siblings) obtained scores above cutoff in the communication domain on the ADI-R Toddler, 4 children (10%) obtained above cutoff scores in the social domain, and 2 children (5%) obtained above cutoff scores in repetitive behavior.

Cognitive, Motor, Language, and Adaptive Behavior

Cognitive and motor skills. Multivariate analysis of variance was initially used to test for group differences in developmental level (cognitive functioning or IQ) as assessed by the Mullen Scales of Early Learning. However, the MANOVA omnibus test was non-significant (Wilks' Lambda = .845, $F(5, 56) = 2.05$, $p = .09$), possibly due to

small sample size and inadequate power on this measure; therefore, separate univariate ANOVAs were used to examine group differences. Note that the Type I error rate was kept at .05 for each ANOVA, a liberal standard given the number of tests conducted. Results indicated that the two groups of children obtained comparable overall mean scores on composite IQ, visual reception, gross motor, and fine motor scales (see Table 7). Mean scores on each of these scales fell within the average range for both groups of children. However, 12 siblings (29% of the sibling group) had below average (i.e., below 85) composite IQ scores while none of the children in the comparison group scored below 85 in overall IQ. The Fisher exact test indicated that this difference was significant ($p = .005$). Receptive and expressive language scores are discussed below.

Language. Results of ANOVAs on the Mullen receptive and expressive language scores revealed a significant group difference in receptive language, with siblings obtaining, on average, lower scores than children in the comparison group (see Table 7). The groups did not differ in mean expressive language ability. However, while the overall mean scores fell within the average range for both groups on these measures, 14 children (33%) in the sibling group had below average receptive language scores (a significant difference using the Fisher exact test, $p = .002$) and 8 (19%) had below average expressive language scores (Fisher exact test, $p = .035$). It should be noted that there was a significant correlation between receptive language and chronological age only for the sibling group ($r = .37$). CA was not related to any of the other scales of the Mullen for either group.

Adaptive behavior. On the Vineland Adaptive Behavior Scales, results of MANOVA indicated a significant group difference (Wilks' Lambda = .819, $F(5, 56) = 2.48$, $p = .04$), with significant mean differences on all domains except communication. On average, siblings scored below that of comparison children in daily living skills, socialization, motor skills, and on the overall adaptive behavior composite (see Table 7).

Table 7. *Cognitive, motor, language, and adaptive behavior of siblings and comparison children*

	Siblings		Comparison		ANOVA/MANOVA			
	M	*SD*	*M*	*SD*	*F*	*df*	*p*	*d*
Mullen[a]								
Composite IQ	99.05	16.94	100.80	6.34	0.20	1, 60	.657	0.14
Visual Reception	51.50	8.50	48.90	7.49	1.37	1, 60	.247	0.32
Gross Motor	45.46	6.27	45.80	8.50	0.03	1, 57[b]	.863	0.05
Fine Motor	49.26	8.11	49.55	6.65	0.02	1, 60	.891	0.04
Receptive Language	*46.55*	*15.60*	*54.50*	*6.40*	*4.78*	*1, 60*	*.033*	*0.67*
Expressive Language	49.07	12.24	48.45	6.65	0.05	1, 60	.833	0.06
Vineland[c]								
Communication	95.24	14.53	100.90	7.32	2.70	1, 60	.106	0.50
Daily Living Skills	*85.83*	*7.27*	*92.90*	*8.32*	*11.67*	*1, 60*	*.001*	*0.91*
Socialization	*91.48*	*9.50*	*96.15*	*7.37*	*3.76*	*1, 60*	*.057*	*0.55*
Motor	*94.02*	*7.50*	*99.20*	*9.91*	*5.22*	*1, 60*	*.026*	*0.59*
Composite	*88.48*	*9.42*	*95.95*	*8.11*	*9.29*	*1, 60*	*.003*	*0.85*

Italics indicate significant differences.
[a] On the Mullen, composite IQ scores between 85 and 115 are considered to be in the average range; for the subscales, T-scores between 40 and 60 are in the average range.
[b] Fewer children completed the gross motor subtest of the Mullen.
[c] On the Vineland, scores between 85 and 115 are in the average range.

Social Abilities

Social orienting ability. A repeated measures analysis of covariance (ANCOVA) was conducted, with group (non-autistic siblings, typical) entered as a two-level between

subjects factor, stimulus type (social vs. nonsocial) entered as a two-level within subjects factor, and CA entered as a covariate. There was no main effect of group ($F(1, 57) = .43$, $p = .52$), nor was there a group by stimulus interaction, $F(1, 57) = 2.93$, $p = .09$ (see Table 8 for means and standard errors). Both groups of children oriented at similar rates to both social and nonsocial stimuli.

Joint attention ability. MANCOVA was conducted using mean scores from the Butterworth task (response to joint attention), the CSBS-DP measure (joint attention and gaze/point following), and the ADOS (initiating joint attention (IJA) and responding to joint attention (RJA)), with CA as a covariate (see Table 8). No differences in joint attention ability were found for the two groups of children (Wilks' Lambda = .934, $F(5, 52) = .73$, $p = .60$).

Social communication. MANCOVA revealed significant group differences on the CSBS-DP (Wilks' Lambda = .448, $F(22, 38) = 2.13$, $p = .02$), with siblings demonstrating fewer behaviors than comparison toddlers in rate of communicating and use of distal gestures (see Table 8). Siblings also obtained lower cluster scores in overall social use of gestures and words, lower Social Composite scores, Symbolic Composite scores (reflecting frequency and variety of symbolic play as well as language comprehension), and Total Composite scores. The two groups did not differ in frequency of gaze shifts, shared positive affect, gaze/point following, behavior regulation, social interaction, and use of conventional gestures, nor in overall Speech Composite scores.

On the ADOS, MANCOVA indicated a significant group difference (Wilks' Lambda = .521, $F(20, 40) = 1.84$, $p = .05$), with siblings responding to social smiles,

pointing to request and/or show objects to others, and engaging in functional play less often than comparison children. There were no group differences in shared enjoyment, showing, or responding to name (see Table 8).

Social-emotional functioning. On the Vineland SEEC, MANOVA indicated a significant group difference (Wilks' Lambda = .784, $F(3, 58) = 5.31$, $p = .003$), with siblings obtaining significantly lower scores in the Interpersonal Relationships domain, and in the overall Social-Emotional Composite, as compared to comparison toddlers. The two groups did not differ, however, in the Play and Leisure Time domain (see Table 8).

Table 8. *Social orienting, joint attention, social communication, and social-emotional functioning of siblings and comparison children*

	Siblings		Comparison		MANCOVA/MANOVA[a]			
	M[b]	*SE*	*M*	*SE*	*F*	*df*	*p*	*d*
Social Orient Task								
Social orienting (0-4)	3.20	0.13	3.31	0.14	--	--	--	--
Nonsocial orienting (0-4)	3.71	0.21	3.28	0.22	--	--	--	--
Joint Attention								
Butterworth RJA (0-4)	3.08	0.14	3.48	0.22	2.04	1, 56	.158	0.61
CSBS-DP Joint attention	3.54	0.28	3.77	0.44	0.19	1, 56	.666	0.38
CSBS-DP Gaze/point follow	1.91	0.06	1.86	0.09	0.23	1, 56	.630	0.03
ADOS IJA	0.70	0.13	0.68	0.21	0.01	1, 56	.945	0.05
ADOS RJA	0.30	0.12	0.15	0.18	0.43	1, 56	.514	0.43
CSBS-DP Social Communication								
Gaze shifts	4.99	0.18	5.32	0.27	0.96	1, 59	.331	0.33
Shared positive affect	2.52	0.24	2.05	0.35	1.16	1, 59	.287	0.03
Rate of communicating	*15.74*	*0.36*	*17.34*	*0.53*	*5.85*	*1, 59*	*.019*	*0.88*
Behavior regulation	5.34	0.12	5.58	0.18	1.13	1, 59	.292	0.48
Social interaction	1.49	0.24	1.66	0.36	0.15	1, 59	.702	0.25
Conventional gestures	4.36	0.22	4.89	0.33	1.65	1, 59	.203	0.42
Distal gestures	*1.90*	*0.23*	*3.01*	*0.35*	*6.68*	*1, 59*	*.012*	*0.87*

Table 8 (continued)

	Siblings		Comparison		MANCOVA/MANOVA[a]			
	M[b]	*SE*	*M*	*SE*	*F*	*df*	*p*	*d*
Cluster Scores								
Speech: Words	*10.80*	*0.42*	*12.47*	*0.62*	*4.70*	*1, 59*	*.034*	*0.92*
Social: Gestures	*7.45*	*0.36*	*9.00*	*0.53*	*5.47*	*1, 59*	*.023*	*0.70*
Composite Scores								
Social Composite	*7.43*	*0.36*	*8.75*	*0.53*	*3.96*	*1, 59*	*.051*	*0.65*
Speech Composite	10.52	0.41	11.87	0.62	3.10	1, 59	.083	0.83
Symbolic Composite	*8.52*	*0.32*	*9.80*	*0.48*	*4.63*	*1, 59*	*.035*	*0.94*
Total Composite	*92.67*	*1.97*	*101.34*	*2.93*	*5.64*	*1, 59*	*.021*	*1.02*
ADOS[c]								
Responsive social smile	*1.09*	*0.13*	*0.35*	*0.19*	*9.81*	*1, 59*	*.003*	*1.10*
Pointing	*1.18*	*0.13*	*0.63*	*0.19*	*5.48*	*1, 59*	*.023*	*1.03*
Functional play w/objects	*0.51*	*0.08*	*0.23*	*0.11*	*4.08*	*1,59*	*.048*	*0.70*
Shared enjoyment	0.26	0.09	0.46	0.14	1.36	1, 59	.249	0.07
Showing	0.66	0.11	0.46	0.16	1.05	1, 59	.310	0.21
Response to name	0.38	0.10	0.35	0.15	0.02	1,59	.890	0.05
Vineland SEEC								
Interpers Relationships	*85.67*	*9.82*	*93.10*	*11.08*	*7.14*	*1, 60*	*.010*	*0.71*
Play/Leisure Time	93.64	8.10	96.60	8.38	1.77	1, 60	.189	0.36
Soc-Emot Composite	*88.05*	*8.94*	*95.40*	*9.21*	*8.99*	*1, 60*	*.004*	*0.81*

Italics indicate significant differences.

[a] CA was entered as a covariate, except for the Vineland SEEC analyses, which used standard scores.

[b] Estimated marginal means and standard errors are reported for the CSBS-DP and ADOS; raw means and standard deviations are reported for the Vineland SEEC.

[c] On the ADOS, lower scores reflect less impairment; scores range from 0-2.

Face and object recognition. A repeated measures ANCOVA was originally planned, but requires data in all four cells (experiments) for each subject and thus would have resulted in a reduced *n* and power for analyses. Therefore, univariate analyses of variance with CA entered as a covariate were conducted separately for the following dependent variables collected for each stimulus type (face, house) and delay (short, long):

number of fixations (i.e., looks) until habituation, total time to habituate in seconds, and visual fixation to novel stimulus at test (i.e., novelty preference, calculated as follows: percentage of total time at test spent looking at novel, defined per the literature (Fagan, Singer, Montie, & Shepard, 1986) as .55 or greater; no preference = .46-.54; familiarity preference = .00-.45). The Type I error rate was kept at .05 for each ANOVA, a liberal standard given the number of tests conducted. Based on prior work (Dawson, Carver, et al., 2002), siblings were expected to exhibit a specific deficit in face recognition (i.e., longer total time to habituate to faces vs. objects, failure to show a novelty preference for faces). Results, however, did not support this hypothesis (see Table 9).

Table 9. *Face and object recognition, imitation, and play abilities of siblings and comparison children*[a]

	Siblings		Comparison		ANCOVA			
	M[b]	*SE*	*M*	*SE*	*F*	*df*	*p*	*d*
Face and Object Recognition								
# looks to habituate								
Face Long	5.34	0.43	3.93	0.73	2.67	1, 51	.108	0.67
Face Short	5.02	0.29	4.34	0.46	1.51	1, 53	.225	0.50
House Long	5.72	0.39	5.10	0.59	0.73	1, 50	.397	0.37
House Short	4.73	0.31	5.40	0.46	1.44	1, 52	.237	0.34
Total time to habituate (secs)								
Face Long	29.44	2.03	26.64	3.43	0.47	1, 49	.500	0.30
Face Short	32.86	2.70	27.93	4.26	0.92	1, 51	.340	0.44
House Long	31.24	2.64	28.68	3.98	0.27	1, 48	.600	0.23
House Short	30.95	2.57	34.84	3.83	0.68	1, 50	.410	0.27
Novelty preference (% time at test)								
Face Long	0.59	0.03	0.56	0.05	0.26	1, 51	.611	0.23
Face Short	0.59	0.04	0.54	0.05	0.62	1, 50	.436	0.42
House Long	0.55	0.04	0.63	0.05	1.55	1, 48	.220	0.32

Table 9 (continued)

	Siblings		Comparison		ANCOVA			
	M^b	*SE*	*M*	*SE*	*F*	*df*	*p*	*d*
House Short	0.58	0.03	0.49	0.05	2.04	1, 50	.160	0.37
Imitation								
Total[c]	9.25	0.59	8.20	0.89	0.88	1, 58	.352	0.15
Immediate	5.90	0.48	5.01	0.71	0.95	1, 58	.333	0.16
Deferred	3.35	0.18	3.18	0.27	0.24	1, 58	.625	0.07
Play								
Functional Play	87.72	2.17	80.92	3.42	2.65	1, 57	.109	0.09
Symbolic Play	7.38	0.60	6.67	0.95	0.38	1, 57	.542	0.30

[a] CA was entered as a covariate.
[b] Estimated marginal means and standard errors are reported.
[c] A separate ANOVA was used for the Total variable, as this variable is the sum of the immediate and deferred scores

Imitation. MANCOVA was conducted on three imitation variables (total imitation score, immediate imitation, and deferred imitation). No significant group differences in immediate and deferred imitation, as measured by Meltzoff's imitation battery, were found (Wilks' Lambda = .984, $F(2, 57) = .468$, $p = .63$; see Table 9).

Play

MANCOVA was conducted to test for group differences in functional and symbolic play, with CA entered as a covariate. Based on the fact that toy play has not been identified as an area of impairment in siblings of children with autism in prior research, non-autistic siblings were not expected to show impairments on measures of toy play. Consistent with hypotheses, there were no significant group differences in either functional or symbolic play skills as measured by the Play Assessment Scale (Wilks' Lambda = .947, $F(2, 56) = 1.58$, $p = .22$; see Table 9).

Stereotyped Behavior

To test for group differences in atypical behaviors, scores from the Repetitive Behavior Scale – Revised were analyzed using MANCOVA. Based on previous studies of 12-month-old siblings (Zwaigenbaum personal communication, 2004), elevated rates of atypical behaviors in non-autistic siblings as compared to comparison children were not expected. Consistent with hypotheses, no group differences were found in the following behavior domains: Compulsive, Restricted, Ritualistic, Sameness, Self-injurious, Stereotyped, and Total Repetitive (Wilks' Lambda = .906, $F(6, 50) = .86$, $p = .53$; see Table 10).

Table 10. *Stereotyped behaviors in siblings and comparison children*

	Siblings		Comparison		MANCOVA			
	M	*SE*	*M*	*SE*	*F*	*df*	*p*	*d*
Compulsive (0-24)	0.98	0.28	0.88	0.47	0.33	1, 55	.857	0.06
Restricted (0-12)	0.63	0.19	0.67	0.32	0.01	1, 55	.908	0.02
Ritualistic (0-18)	0.92	0.28	0.78	0.47	0.07	1, 55	.800	0.09
Sameness (0-33)	1.09	0.30	0.89	0.49	0.13	1, 55	.725	0.11
Self-injurious (0-24)	0.72	0.22	0.37	0.35	0.68	1, 55	.412	0.28
Stereotyped (0-18)	0.87	0.20	0.46	0.33	1.13	1, 55	.293	0.38
Total repetitive (0-129)	5.20	1.23	4.03	2.03	0.24	1, 55	.629	0.12

Early Development Interview (EDI)

Symptoms from the Early Development Interview from each 3-month time period (0-3 mos., 4-6 mos., 7-9 mos., 10-12 mos., 13-15 mos., 16-18 mos., and 19-24 mos.) were analyzed. Sensory items (i.e., sensitivity to noise and touch) were derived from the regulatory domain. Additionally, information on family changes, child medical events, and

stressors during pregnancy were also analyzed. Separate univariate ANOVAs were used to test for group differences in these areas as well as the following behavior domains: social, communication, regulatory, sensory, and repetitive. Although several comparisons were made in these analyses, the alpha level was kept at .05 for each analysis as we were interested in each symptom domain at each time point (Perneger, 1998). A summary of results is presented in Table 11. Note that a number of items are included in each domain score, and the possible range of scores for each individual item is 0-2, with higher scores indicating greater impairment. Siblings were reported to show greater overall social difficulties, first apparent at 13-15 months of age, than comparison children.

Table 11. *Early symptoms in siblings and comparison children*[a]

	Siblings		Comparison		ANOVA			
	M	*SD*	*M*	*SD*	*F*	*df*	*p*	*d*
0-3 months								
Regulatory (0-10)	*1.50*	*1.27*	*3.11*	*2.37*	*11.03*	*1, 54*	*.002*	*0.85*
Sensory (0-2)	0.03	0.16	0.11	0.47	1.00	1, 54	.322	0.23
4-6 months								
Regulatory (0-10)	1.16	1.13	1.44	1.79	0.53	1, 54	.468	0.19
Sensory (0-2)	0.00	0.00	0.11	0.47	2.16	1, 54	.148	0.33
Social (0-4)	0.08	0.36	0.17	0.51	0.55	1, 54	.462	0.20
7-9 months								
Regulatory (0-10)	1.08	1.08	1.50	1.20	1.74	1, 54	.193	0.37
Sensory (0-2)	0.05	0.23	0.22	0.65	2.11	1, 54	.152	0.35
Social (0-8)	0.24	0.59	0.17	0.51	0.19	1, 54	.667	0.13
Communication (0-2)	0.08	0.27	0.06	0.24	0.10	1, 54	.756	0.08
10-12 months								
Regulatory (0-2)	0.29	0.65	0.50	0.79	1.11	1, 54	.297	0.29
Sensory (0-4)	*0.13*	*0.41*	*0.50*	*0.79*	*5.32*	*1, 54*	*.025*	*0.59*
Social (0-8)	0.37	0.97	0.06	0.24	1.80	1, 54	.185	0.44
Communication (0-4)	0.32	0.96	0.22	0.55	0.15	1, 54	.703	0.13

Table 11 (continued)

	Siblings		Comparison		ANOVA			
	M	*SD*	*M*	*SD*	*F*	*df*	*p*	*d*
10-12 months								
Repetitive (0-2)	0.05	0.23	0.06	0.24	0.00	1, 54	.965	0.04
13-15 months								
Regulatory (0-2)	0.29	0.65	0.44	0.78	0.60	1, 54	.441	0.21
Sensory (0-2)	*0.08*	*0.27*	*0.44*	*0.78*	*6.67*	*1, 54*	*.013*	*0.62*
Social (0-14)	*1.68*	*2.28*	*0.50*	*0.86*	*4.52*	*1, 54*	*.038*	*0.69*
Communication (0-2)	0.45	0.80	0.28	0.67	0.61	1, 54	.438	0.23
Repetitive (0-2)	0.03	0.16	0.11	0.47	1.00	1, 54	.322	0.23
16-18 months								
Sensory (0-2)	*0.08*	*0.27*	*0.44*	*0.78*	*6.67*	*1, 54*	*.013*	*0.62*
Social (0-10)	*1.13*	*1.96*	*0.11*	*0.47*	*4.70*	*1, 54*	*.035*	*0.72*
Communication (0-6)	0.87	1.42	0.50	0.79	1.06	1, 54	.309	0.32
Repetitive (0-2)	0.08	0.27	0.11	0.47	0.10	1, 54	.748	0.08
19-21 months								
Sensory (0-2)	*0.08*	*0.27*	*0.50*	*0.79*	*8.82*	*1, 54*	*.004*	*0.71*
Social (0-10)	0.61	1.48	0.00	0.00	2.98	1, 54	.090	0.58
Communication (0-6)	0.58	1.03	0.50	0.86	0.08	1, 54	.779	0.08
Repetitive (0-2)	0.03	0.16	0.11	0.47	1.00	1, 54	.322	0.23
22-24 months								
Sensory (0-2)	*0.08*	*0.27*	*0.50*	*0.79*	*8.82*	*1, 54*	*.004*	*0.71*
Social (0-10)	0.32	0.74	0.00	0.00	3.25	1, 54	.077	0.61
22-24 months								
Communication (0-8)	0.66	1.34	0.44	0.78	0.39	1, 54	.535	0.20
Repetitive (0-6)	*0.26*	*0.72*	*0.94*	*1.21*	*6.91*	*1, 54*	*.011*	*0.68*
Total Scores								
Regulatory (0-34)	*4.32*	*3.60*	*7.00*	*4.59*	*5.68*	*1, 54*	*.021*	*0.65*
Sensory (0-18)	*0.53*	*1.57*	*2.83*	*4.61*	*7.77*	*1, 54*	*.007*	*0.67*
Social (0-64)	*4.42*	*6.60*	*1.00*	*1.68*	*4.66*	*1, 54*	*.035*	*0.71*
Communication (0-28)	2.95	4.32	2.00	2.57	0.74	1, 54	.395	0.27
Repetitive (0-14)	0.45	1.03	1.33	2.59	3.38	1, 54	.072	0.45

Table 11 (continued)

	Siblings		Comparison		ANOVA			
	M	*SD*	*M*	*SD*	*F*	*df*	*p*	*d*
Family Changes (0-18)	2.61	2.35	3.39	2.95	1.15	1, 54	.289	0.29
Child Medical Events (0-12)	1.87	1.86	2.00	1.82	0.06	1, 54	.804	0.07
Pregnancy Stress Events (0-16)	1.97	1.81	1.17	1.79	2.45	1, 54	.124	0.44

Italics indicate significant differences.

[a] Higher scores on this measure indicate greater impairment.

Siblings were also reported to show fewer regulatory difficulties (analysis of individual items revealed that these differences were due to differences in fussiness and sleep in particular) at 0-3 months of age as compared to comparison children. Finally, children in the comparison group were reported to show a greater number of sensory behaviors (from 10-24 months) and repetitive behaviors (at 22-24 months) than siblings. There were no differences reported in family changes and child medical events during the first two years of life, nor in stressful events occurring during pregnancy, suggesting that these variables were unlikely to account for the differences found in the children's early behaviors.

Parent Mental Health, Marital Relationship, and Stress

Not all parents completed the three measures of parental mental health, marital functioning, and stress. Of fathers in the sibling group, only n=27 completed the BSI and DAS, and n=35 completed the LES; n=9 fathers in the comparison group completed the BSI and DAS, and n=13 completed the LES. Of mothers, only n=31 in the sib group completed the BSI and DAS, and n=38 completed the LES; n=15 in the comparison group completed the BSI and DAS, and n=17 completed the LES. There were no significant group differences for mothers and fathers on any of the symptom dimensions

or global indices of the Brief Symptom Inventory (for mothers: Wilks' Lambda = .939, $F(10, 35) = .23, p = .992$; for fathers: Wilks' Lambda = .702, $F(10, 25) = 1.06, p = .424$). On the Dyadic Adjustment Scale, the omnibus test indicated group differences for mothers (Wilks' Lambda = .727, $F(4, 38) = 3.58, p = .014$), but there were no group differences on any of the individual variables. Results for fathers indicated no group differences (Wilks' Lambda = .928, $F(4, 31) = .60, p = .667$). On the Life Experiences Survey, there were no group differences for mothers (Wilks' Lambda = .963, $F(4, 50) = .48, p = .749$) or for fathers (Wilks' Lambda = .955, $F(4, 43) = .51, p = .727$). Table 12 shows means and standard deviations for each variable. These results suggest that levels of parental mental health, marital functioning, and stress as measured by these three questionnaires were essentially the same for both parents of comparison toddlers and parents of children with autism in this sample.

Table 12. *Results of parent measures*[a]

	Siblings		Comparison	
	Mother	Father	Mother	Father
BSI				
Anxiety	0.59 (0.56)	0.30 (0.28)	0.53 (0.48)	0.50 (0.68)
Depression	0.56 (0.77)	0.48 (0.62)	0.60 (0.69)	0.50 (0.98)
Hostility	0.71 (0.70)	0.50 (0.48)	0.64 (0.56)	0.69 (1.25)
Interpersonal sensitivity	0.66 (0.74)	0.28 (0.31)	0.63 (0.72)	0.39 (0.40)
OCD behavior	1.08 (0.77)	0.68 (0.68)	1.04 (0.71)	0.96 (0.78)
Paranoid ideation	0.47 (0.55)	0.44 (0.54)	0.47 (0.72)	0.60 (0.94)
Phobic anxiety	0.12 (0.30)	0.07 (0.14)	0.13 (0.22)	0.07 (0.14)
Psychoticism	0.32 (0.47)	0.24 (0.35)	0.28 (0.43)	0.51 (0.96)
Somatization	0.47 (0.55)	0.20 (0.32)	0.34 (0.37)	0.27 (0.44)
Positive Symptom Total	29.40 (26.10)	18.50 (16.27)	27.21 (22.20)	26.93 (35.93)

Table 12 (continued)

	Siblings		Comparison	
	Mother	Father	Mother	Father
Global Severity Index	0.57 (0.50)	0.36 (0.31)	0.52 (0.43)	0.52 (0.69)
DAS				
Affectional expression	3.53 (1.50)	4.00 (1.62)	4.08 (1.26)	3.44 (1.42)
Dyadic cohesion	14.47 (3.07)	15.22 (2.93)	15.85 (3.16)	16.11 (2.52)
Dyadic consensus	14.50 (5.73)	15.52 (6.32)	14.08 (5.65)	16.00 (2.83)
Dyadic satisfaction	33.83 (4.14)	33.41 (4.08)	31.92 (6.25)	34.56 (3.50)
Total	66.33 (4.27)	68.15 (5.30)	65.92 (7.08)	70.11 (4.96)
LES				
Positive change	6.26 (6.03)	5.80 (5.67)	7.24 (6.84)	6.15 (7.95)
Negative change	6.87 (7.75)	5.94 (4.96)	5.88 (7.00)	5.15 (5.06)
Total change	-0.61 (7.83)	-0.14 (7.26)	1.35 (6.08)	1.00 (5.13)
# of positive events	3.13 (2.60)	3.11 (2.69)	3.71 (2.82)	2.92 (3.35)
# of negative events	3.55 (3.24)	3.86 (2.94)	3.41 (3.12)	3.31 (2.98)

[a] Means are reported with SDs in parentheses.

CHAPTER IV

Discussion

Summary of Early Impairments in Siblings

A broad range of early cognitive, adaptive, social cognition, social communication, social-emotional functioning, imitation, play, and language abilities was examined in 18-27 month old non-autistic siblings of children with autism as compared to children with no family history of autism to identify the kinds of impairments that may be evident early on in young siblings. In addition, a detailed, retrospective parent interview method was used to determine if siblings demonstrated impairments during the first two years of life as compared to comparison infants. Finally, measures of child temperament and parent mental health, marital relationship, and stress were examined to determine if such characteristics might explain differences found in siblings' early abilities. Findings based on multiple measures, utilizing both direct observation and parent report, indicated that non-autistic siblings demonstrated a variable profile, with decrements in some aspects of social communication, language, and adaptive behavior, and intact skills in other areas. Specifically, we found that, on average, siblings had lower receptive language skills, lower adaptive behavior skills, and lower overall rates of social communication and social-emotional functioning than comparison children. In addition, a significant number of siblings demonstrated below average expressive language and overall cognitive abilities. Finally, siblings demonstrated fewer symbolic behaviors overall (a combination of language comprehension and symbolic object use during play), as well as less frequent use of words, distal gestures including pointing, and responsive

social smiles during social interactions than comparison children. Since multiple comparisons were made without correction for family-wise error, it is possible that some of these findings achieved statistical significance by chance. However, in light of the fact that we actually excluded children who had above average IQ scores in the comparison sample, the finding of group differences in a number of areas can be considered a conservative result.

Further findings based on retrospective parent report indicated that siblings showed greater overall social difficulties, evident by 13 months of age, fewer early regulatory difficulties (i.e., less fussiness and fewer sleep problems at 0-3 months), and fewer sensory and repetitive behaviors during the second year of life than toddlers in the comparison group. This may reflect higher levels of passivity in siblings of children with autism. In autism, repetitive behaviors are often not yet present at this young of an age, and such behaviors have not been shown to reliably distinguish children with autism at such an early age. Perhaps this is true of non-ASD siblings as well. In certain other domains, such as face recognition, imitation, and play, and in other aspects of social communication, such as initiating and responding to joint attention, siblings performed similarly to typical children. In fact, the young siblings in this study were a heterogeneous group, and their performance on each of the measures ranged from below average to, in some cases, above average.

In terms of family environmental factors, parents of children with autism and parents of comparison children in this sample reported similar levels of mental health symptoms, marital adjustment and satisfaction, and stressful life events, suggesting that

these parental factors were unlikely to account for the group differences found between comparison children and siblings.

Explanation of Findings

The strengths of this study included the sample size (as compared to other published studies of young siblings), a broader range of measures than has been used in other studies of young siblings, and the use of multiple measures for many of the skill domains examined. Although several of our findings were consistent with those of previous studies, there were some skill domains, such as joint attention, where we did not obtain similar results (e.g., Goldberg et al., 2005). There are several possible explanations for this. First, it is possible that some of our measures were less sensitive in capturing subtle differences. For instance, the CSBS-DP yields a more restricted range of scores of use of eye gaze, joint attention, behavior regulation, and social interaction than the Early Social Communication Scales (ESCS; Mundy, Delgado, Hogan, & Doehring, 2003; Seibert & Hogan, 1982), a comparable instrument that yields a greater range of frequency scores. In terms of the face recognition measure used in the present study, infant-controlled habituation paradigms have been shown to be most appropriate for children with mental ages of 18 months and younger. At ages above 18 months, looking behavior becomes more volitional and children may be disengaging from the task for a variety of reasons, not just upon habituation to the stimuli. Therefore, it is possible that another measure of face recognition may have detected subtle differences in our group of siblings. Second, it is possible that, over time, many non-autistic siblings develop more frank broader phenotype characteristics that are not as evident at very young ages. For

instance, regarding face recognition impairments, which have been found in parents of children with autism (Dawson et al., 2004), it is possible that these difficulties emerge over time in siblings, as siblings with early, subtle social communication deficits may engage in fewer face-to-face social interactions that eventually result in more frank impairments in face recognition ability. Further, some of the impairments that have been observed in older siblings, including executive function deficits and verbal fluency impairments (Hughes, Plumet, & Leboyer, 1999), are not easily detected until children reach later preschool and school ages. Asperger syndrome is rarely identified in preschool, and symptoms of autism (e.g., poor joint attention, imitation, play) may not be apparent at that age or may not be part of the Asperger phenotype (see McConachie, Le Couteur, & Honey, 2005). Third, it is possible that previous studies have overestimated the number of non-autistic siblings that demonstrate broader phenotype characteristics. Many studies have relied on a family history interview method (e.g., Bolton et al., 1994) and have focused on older children. These studies require parents to report retrospectively on broad constructs, such as "impaired friendships." The fact that prospective observations of younger siblings thus far have yielded mixed results likely reflects the variable presence of impairments in the sibling population at different ages as well as the types of measurements used across studies. For example, results may vary depending on whether data are collected by direct testing by experimenters in a laboratory setting versus parent report of behavior at home. Additionally, characteristics of the broad autism phenotype may vary across development, with some delays being transient while others are more lasting. It is also possible that at least some of the children

in these studies may go on to develop autism; therefore, the differences that are being captured at very early ages may in fact reflect early symptoms of autism, rather than broader phenotype characteristics in non-autistic siblings. Finally, it is possible that more frank broader phenotype characteristics would exist and/or develop at such an early age, but that siblings are benefiting from the intensive treatments that their older sibling with autism is receiving, many of which are conducted at home. Anecdotally, many of the parents participating in the study commented that their younger child enjoyed being included in their older child's treatment, and enjoyed the attention from the service providers working with their older child. In this way, these young siblings may actually be receiving early behavioral treatment that mitigates the expression of the broad autism phenotype.

What might account for the differences found in siblings' early social, language, and adaptive functioning? Although this study did not address the question of causation, both genetic and environmental factors should be considered. Siblings are at heightened genetic risk for impairments due to the fact that they share genes in common with their sibling with autism. Added to this genetic vulnerability are environmental factors, including modeling of atypical behaviors by the sibling with autism, reduced opportunities for engaging in typical sibling social interactions, and possibly fewer interactions with parents who are overly involved with the child with autism in the family. Sibling and parent-child interactions may not only be less frequent but also qualitatively different. It will be informative, in future studies, to examine early parent-child, and child-child, interactions as these play an important role in the development of

early social and language abilities. Although these family factors may not cause the impairments found in siblings, it is possible that these factors may contribute additional risk (or protection) during a particular time in development (Bauminger & Yirmiya, 2001). For some siblings, the genetic involvement may be strong enough that under even the most optimal environment, these children develop impairments, while for other children, the genetic predisposition may be so weak that only under certain environmental conditions will these children exhibit the broader autism phenotype (Bauminger & Yirmiya, 2001, p. 75). This explanation has been referred to as the diathesis-stress model, and integrates both genetic and environmental factors in explaining development (Brown & Harros, 1989; Rende & Plomin, 1992, Walker, Downey, & Bergman, 1989, among others). According to this model, a genetic predisposition may be essential but not sufficient for the expression of a phenotype.

Future Directions and Conclusion

In conclusion, certain aspects of language, adaptive behavior, social communication, and social-emotional functioning appear to be affected at an early age in non-autistic siblings of children with autism and should be closely monitored by parents and professionals. In some cases, interventions, such as speech and language therapy and behavioral therapy approaches focusing on social skills, may be warranted. However, we are just now starting to learn about the early development of siblings of children with autism. Future research would benefit from the use of common measures to enable replication across studies and to ensure that differences that are reported reflect true differences in siblings' early abilities and are not simply the result of measurement issues.

Studies should also include comparison groups of developmentally delayed children, as well as siblings of children with other developmental disorders, to determine whether the early characteristics evidenced by siblings of children with autism are specific to families with a history of autism, or are more generally related to families with children with special needs. In addition, young siblings should be followed over time to determine how these early characteristics change with development, and to assess whether any of these children go on to a later diagnosis of autism. It is possible that, for siblings, there are periods of development that are especially critical, such as early childhood, although impairments may also develop later or worsen over time. Finally, additional environmental factors and family variables, including sibling interactions, parent-child interactions, and parental responsivity, should be considered.

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CURRICULUM VITAE

RESEARCH INTERESTS

Autism spectrum and related disorders, with a particular focus on early recognition and predictors of outcome in autism.

EDUCATION

B.A. — **University of California, San Diego (UCSD), San Diego, CA**
Political Science, 1986

M.S. — **University of Washington, Seattle, WA**
Child Clinical Psychology, 2001
Thesis: Defining the early social attention impairments in autism: Social orienting, joint attention, and response to emotion

Ph.D. — **University of Washington, Seattle, WA**
Child Clinical Psychology, expected June 2007
Dissertation: Early characteristics of young siblings of children with autism

HONORS

2005-2006	Gatzert Child Welfare Fellowship
2004	Wagner Memorial Scholarship
2003	Wagner Memorial Scholarship
2001	Wagner Memorial Scholarship
1986	Phi Beta Kappa
1986	magna cum laude
1983-1986	UCSD Provost's Honors

PROFESSIONAL AFFILIATIONS

American Psychological Association
Society for Research in Child Development
Society of Clinical Child and Adolescent Psychology (Division 53, APA)
International Society for Autism Research (INSAR)
Autism Society of America

EDITORIAL ACTIVITIES

Ad hoc Reviewer:
Autism: International Journal of Research and Practice (2006)
Development & Psychopathology (2006)
Journal of Developmental & Behavioral Pediatrics (2005)
Journal of Autism & Developmental Disorders (2005-2007)
Journal of Child Psychology & Psychiatry & Allied Disciplines (2004)

RESEARCH EXPERIENCE

7/2002 – 5/2006 **Associate Director**
Infant and Toddler Sibling Study, UW Autism Center
Directed from start-up all aspects of a longitudinal, multi-site study of young siblings of children with autism aimed at improving early detection of autism spectrum and related disorders. Responsibilities included overseeing the recruitment of 150 subject families, subject enrollment, institutional review board documentation, and data entry, as well as training and supervision of a study Project Coordinator and undergraduate research assistants. Additionally, served as primary clinician on this project, conducted diagnostic evaluations of eighty 14-30 month old toddlers and their older siblings, and provided clinical feedback to parents. Have co-authored several conference presentations based on data from this project (see below).

1/1998 – 5/2006 **Research Assistant**
UW Autism Center
Assisted with all aspects of two large program project grants: the Collaborative Program of Excellence in Autism (CPEA) and the STAART (Studies to Advance Autism Research and Treatment) Center of Excellence in Autism. These program projects involved a number of individual studies that sought to better understand the etiology, neurobiology, and course of autism, as well as an intervention study to assess an early intensive behavioral treatment for autism and its impact on brain and behavioral outcomes. Responsibilities included a major role in grant writing; design of individual projects, experimental measures, and testing protocols; recruitment and scheduling of subjects; data collection (conducted cognitive and neuropsychological batteries with children and adults, as well as structured diagnostic interviews with parents), data entry, and analysis; development and management of a behavioral coding project; formulation of research questions and preparation of manuscripts, conference presentations, and a book chapter; training of staff psychologists, graduate, and undergraduate research assistants on standardized and experimental measures; and supervision of staff. Presented data at professional conferences, provided training in autism to medical residents at the University of Washington Medical Center, and gave numerous community lectures to parents and professionals in the education and health fields.

2/2002 – present **Consultant**
Infant Learning Project
University of Washington/Children's Hospital and Regional Medical Center
This longitudinal, multi-site study seeks to better understand the early cognitive and neuropsychological development of children with craniosynostosis. Responsibilities have included training Principal Investigators and research staff at four national sites to conduct a battery

of neuropsychological measures of prefrontal and medial temporal lobe functioning. These measures include A not B, A not B with Invisible Displacement, Visual Paired Comparison, Object Discrimination Reversal, Spatial Reversal, and Delayed Non-Matching to Sample. Conducted initial testing on 18-month-old children with craniosynostosis and typical development. Continue to provide quarterly consultation via teleconferencing to all sites, as well as monthly ongoing consultation regarding the reliability and validity of data collected. Currently assisting with interpretation of data and preparation of manuscripts.

3/1997 – 9/1997 **Research Assistant**
Cognitive Electrophysiology Laboratory
The Scripps Research Institute, La Jolla, CA
Department of Neuropharmacology
Assisted with all aspects of several electrophysiological studies, including design of experiments, recruitment and scheduling of subjects, data collection, and analysis of electroencephalographic (EEG), event-related potentials (ERP), and cognitive data. Study participants included cerebral stroke patients, Alzheimer's patients, subjects with a family history of alcoholism, and subjects with heavy nicotine use.

CLINICAL EXPERIENCE

7/2006 – present **Psychology Resident**
Children's Hospital and Regional Medical Center
Rotations include pediatric neuropsychology, outpatient psychiatry, inpatient psychiatry, and pediatric consultation liaison service.

5/2005 – 5/2006 **Practicum – Eating Disorders Clinic**
Children's Hospital and Regional Medical Center
Conducted diagnostic intake interviews with pre-adolescent and adolescent patients and their parents. Diagnoses included Anorexia Nervosa (both Restricting and Purging Subtypes), Eating Disorder NOS, Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, Major Depressive Disorder, and Adjustment Disorder with Disturbances in Mood and Conduct; many patients also presented with acute medical symptoms, including orthostasis and bradycardia. Worked within a multidisciplinary team in a collaborative effort to provide comprehensive patient care. Developed familiarity with medication management issues. Other responsibilities included co-leading two therapy groups with pre-teens and adolescents hospitalized on an inpatient psychiatric unit, including a psychoeducational therapy group focusing on body image and a CBT therapy group focusing on coping strategies and decreasing eating disordered thoughts and behaviors. Developed materials for use in these groups to assist patients in challenging their disordered thoughts and behaviors. Also provided individual and family therapy to adolescents with eating disorders, and case management services for patients receiving outpatient psychotherapy.

1/1998 – 5/2006 **Clinician – UW Autism Center, Research Program**
Center on Human Development and Disability, University of Washington
Conducted cognitive, diagnostic, adaptive, and neuropsychological assessments of children ages 15 months to 18 years with a variety of childhood disorders: autism spectrum disorders (Autistic Disorder, Pervasive Developmental Disorder, Not Otherwise Specified, and Asperger's Disorder), mental retardation,
receptive and expressive language disorders, selective mutism, as well as children with typical development. Also conducted cognitive and neuropsychological assessment of parents from multiplex families (families with 2 or more children with ASD) and adults with ASD, as well as psychological history and family history interviews with parents. Provided clinical and diagnostic feedback and recommendations to families, many of whom were receiving the diagnosis of autism for the first time.

3/2002 – 6/2005 **Pre-Doctoral Intern – UW Autism Center, Clinical Services Center**
Center on Human Development and Disability, University of Washington
Lead therapist for five social skills groups for children and adolescents with high-functioning autism and Asperger's Disorder from diverse cultural backgrounds. Developed the social skills curriculum for the Center, which included complete 10-week lesson plans for three age-levels: preschool and Kindergarten students, elementary age students, and secondary school age/adolescent students. Provided numerous social skills training lectures and workshops to education and health field professionals. Provided individual and family therapy to child and adolescent clients with autism spectrum and related co-morbid disorders, such as Tourette's Disorder, anxiety, depression, ADHD, conduct disturbances, and emotion regulation problems. Also provided ongoing school consultation services for individual therapy clients, including working directly (one-on-one) with school staff to develop and implement behavioral and coping strategies for clients, and attending IEP meetings. Conducted diagnostic evaluations of toddlers and preschool aged children with autism spectrum disorders.

11/2000 – 5/2003 **Therapist – Psychological Services and Training Center**
Department of Psychology, University of Washington
Clinical experience at this outpatient clinic included individual therapy with clients of various ages (child and adult) and from diverse ethnic and religious backgrounds, as well as family and group therapy. Casework included treatment of children with Oppositional Defiant Disorder, Separation Anxiety Disorder, language disorders, and learning disabilities; cognitive-behavioral treatment of adults with depression and

anxiety; and cognitive-behavioral/family systems group therapy for adult women trauma survivors.

9/2001 – 6/2002 **Practicum – Child Development Clinic**
Center on Human Development and Disability, University of Washington
Conducted clinical diagnostic evaluations of children ages 3 to 13 years with a wide variety of childhood disorders, including Obsessive-Compulsive Disorder, ADHD, Oppositional Defiant Disorder, Adjustment Disorder, Bipolar Disorder, anxiety, depression, Down Syndrome, Tuberous Sclerosis, mental retardation, language disorders, and attachment disorders. Assessment included cognitive and neuropsychological testing, as well as school observations. In addition, collaborated with a multidisciplinary team, acted as team leader for several cases, and provided feedback and recommendations to parents.

1/2001 – 4/2001 **Clinician – LEARN Clinic**
Department of Psychology, University of Washington
Conducted diagnostic assessments of children and adolescents with learning disabilities, depression, and social impairments, and provided feedback and recommendations to families.

6/1998 – 8/1998 **Site Coordinator**
Whatcom County Best S.E.L.F. Summer Program
Managed summer educational and recreational program for children and adolescents with developmental and behavioral disorders from diverse ethnic backgrounds and low-income families; utilized behavioral interventions as needed; supervised a staff of 20 teachers and aides.

SPECIALIZED CLINICAL TRAINING

Assessment of Psychopathology

K-SADS Diagnostic Interview
Russ Hanford, PhD, Children's Hospital & Regional Medical Center

Personality Assessment

Clinical Personality Assessment: MMPI-2 and MCMI-III
Ronald E. Smith, Ph.D., University of Washington

Autism Spectrum Disorders

Autism Observation Scale for Infants (AOSI)
Jessica Brian, Ph.D., Autism Research Unit, Hospital for Sick Children, Toronto

Autism Diagnostic Observation Schedule (ADOS)
Training for Researchers:
Christina Corsello, Ph.D., University of Michigan Autism & Communication Disorders Center (UMACC)

Training for Clinicians:

Catherine Rice, Ph.D., Western Psychological Services

Autism Diagnostic Interview – Revised (ADI-R)
Christina Corsello, Ph.D., University of Michigan Autism & Communication Disorders Center (UMACC)

Eating Disorders

Maudsley Treatment for Anorexia Nervosa
Karen Pavlidis, Ph.D.

Assessment and Treatment of Eating Disorders
Rose Calderon, Ph.D., Children's Hospital & Regional Medical Center

Behavior Disorders

Behavioral Family Therapy
Robert McMahon, Ph.D., University of Washington

Mood Disorders

Behavioral Activation Therapy
Christopher Martell, Ph.D.

Borderline Personality Disorder

Dialectical Behavior Therapy (DBT)
Kate Comtois, Ph.D., Harborview Medical Center

DBT Adolescent Skills Group, Jul-Dec 06
Barbara Kleine, MA, Children's Hospital & Regional Medical Center

TEACHING EXPERIENCE

10/2006 **University of Oklahoma Child Study Center**
Provided 2-day workshop to multidisciplinary team on developing procedures to assess children for autism spectrum disorders.

1/2003 – 5/2006 **University of Washington School of Medicine and Children's Hospital**
Lecture on autism spectrum disorders delivered monthly to medical residents and fourth year medical students on rotation at the Center on Human Development and Disability (CHDD) and UW Autism Center. Lecture included diagnostic criteria, associated behaviors/disorders, and early detection of autism.

10/2005 **UW Autism Center**
Provided training to research and clinical staff at the UW Autism Center on a measure designed to screen for early symptoms of autism in infants and toddlers: the Autism Observation Scale for Infants (AOSI).

1/2005 **North Thurston School District, Lacey, WA**
Provided workshop to teachers in the North Thurston School District on *Social Skills and Emotion Regulation Strategies for Pre-K – Secondary Students with HFA and AS.*

10/2004	**Lake Washington School District, Seattle, WA** Leader of half-day seminar for the Lake Washington School District's Elementary Special Education Department Chair Meeting. Lectures included *Current Research Findings in Autism* and *Social Skills Strategies for Elementary Students with Autism.*
3/2004	**Collaborative Program of Excellence in Autism (CPEA) Network Training in the Broader Phenotype Autism Symptom Scale (BPASS)** Provided training on the BPASS, an instrument developed at the UW Autism Center to capture the broad phenotype of autism in family members of individuals with autism, to a group of Principal Investigators and senior psychology staff from a number of large autism research centers in the U.S. Collaborated on the development of this instrument, including refining the constructs measured and establishing reliability.
7/2003 and 7/2004	**Combined Summer Institute, Yakima, WA** Leader of two-day seminar for educators and other service and care providers for children with special needs and their families. Sponsored by the Office of Superintendent of Public Instruction, Northwest ESD, North Central ESD 171, the Washington Sensory Disabilities Services, and the Autism Outreach Project. Lectures included: *Current Research Findings in Autism; An Introduction to High Functioning Autism and Asperger's Syndrome; Social Skills Strategies for Elementary Students with Autism; Social Skills Strategies for Secondary Students with Autism;* and *Practical Educational Strategies for Students with Autism*
9/2003 and 10/2003	**Educational Service District 189** Guest lecturer. *Current Research Findings in Autism.* Lecture delivered to educators via teleconferencing to districts throughout the state of Washington.
10/2003	**Psychology 560C: Graduate Research Seminar in Child Clinical Psychology** Professor: Bob McMahon, Ph.D., University of Washington. Guest lecturer. *The UW Autism Center Research Program.*
10/2003	**Psychology 4900: Senior Seminar in Psychology** Professor: Stephen Smith, Ph.D., North Georgia College & State University. Guest internet lecturer. *Autism Spectrum Disorders.*
3/2002	**Psychology 560I: Graduate Psychology Seminar** Professor: Davida Teller, Ph.D., University of Washington. Guest lecturer. *Defining the early social attention impairments in autism: Social orienting, joint attention, and response to emotion*
2/2001 and 3/2000	**Psychology 499: Undergraduate Research** Professor: Geraldine Dawson, Ph.D., University of Washington. Guest lecturer. *The UW Autism Center Research Program.*

10/2000 **UW Autism Center**
Lecture delivered on the broader autism phenotype to physicians and other health professionals from Alaska.

3/2000 – 12/2000 **Psychology 499: Undergraduate Research**
Professor: Geraldine Dawson, Ph.D., University of Washington. Responsible for an upper division course in undergraduate research. Instructed students in developing behavioral coding skills related to observation and classification of early nonverbal social-communication abilities of children with both typical and atypical development.

INVITED PRESENTATIONS

Autism Spectrum Disorders: Current Research Findings (2006, October). Grand Rounds Lecture on Autism, University of Oklahoma College of Medicine, Oklahoma City, OK.

PROFESSIONAL PRESENTATIONS

Elder, L., Dawson, G., Toth, K., Fein, D., & Munson, J. (2007, April). Head circumference as an early predictor of autism symptoms in young children at risk for autism. Paper symposium at the biennial meeting of the Society for Research in Child Development, Boston, MA.

Dawson, G., Toth, K., & Elder, L. (2006, June). UW Infant and Toddler Sibling Study. Presented at the Autism Speaks workshop, London, UK.

Toth, K., Dawson, G., Meltzoff, A., Greenson, J., & Fein, D. (2006, June). Early social and communication impairments in young siblings of children with autism. Presented at the annual International Meeting for Autism Research, Montreal, Canada.

Pandey, J., Toth, K., Esser, E., Wilson, L., Boorstein, H., Sutera, S., Verbalis, A., Rosenthal, M., Dumont-Mathieu, T., Hodgson, S., Barton, M., Dawson, G., & Fein, D. (2006, June). An update on the modified checklist for autism in toddlers (M-CHAT) sibling study. Presented at the annual International Meeting for Autism Research, Montreal, Canada.

Collett, B., Toth, K., and Dawson, G. (2006, April). Use of novel neuropsychological tasks to assess prefrontal functions in infants with single-suture craniosynostosis. Panel presentation at the annual meeting of the American Cleft-Palate/Craniofacial Association, Vancouver, B.C.

Pandey, J., Toth, K., Ventola, P., Kleinman, J., Esser, E., Wilson, L., Boorstein, H., Sutera, S., Verbalis, A., Rosenthal, M., Hodgson, S., Dumont-Mathieu, T., Barton, M., Dawson, G. & Fein, D. (2006, February). The modified checklist for autism in toddlers (M-CHAT) sibling study: An update. Poster presented at the annual meeting of the International Neuropsychological Society, Boston, MA.

Webb, S., Dawson, G., Toth, K., Greenson, J., Groen, R., Merkle, K. (2005, November). Early learning about faces and objects: Habituation speed differs in toddlers with ASD. Poster presented at the annual meeting of the Collaborative Program of Excellence in Autism/Studies to Advance Autism Research and Treatment Network, Bethesda, MD.

Webb, S., Dawson, G., Toth, K., & Carlberg, M. (2005, May). Face and object memory in toddlers with autism, siblings of children with autism, and controls. Paper symposium at the annual International Meeting for Autism Research, Boston, MA.

Pandey, J., Toth, K., Sutera, S., Kleinman, J., Dixon, P., Wilson, L., Boorstein, H., Esser, E., Barton, M., Hodgson, S., Dumont-Mathieu, T., Green, J., Marshia, G., Dawson, G., & Fein, D. (2005, May). Using the M-CHAT to detect autism spectrum disorders in young siblings of ASD children. Paper symposium at the annual International Meeting for Autism Research, Boston, MA.

Pandey, J., Toth, K., Kleinman, J., Dixon, P., Barton, M., Allen, S., Green, J., Dawson, G., & Fein, D. (2005, April). Detecting autism spectrum disorders in young siblings of ASD children. Paper symposium at the biennial meeting of the Society for Research in Child Development, Atlanta, GA.

Pandey, J., Toth, K., Kleinman, J., Dixon, P., Barton, M., Allen, S., Green, J., Dawson, G., & Fein, D. (2005, February). Detecting autism spectrum disorders in young siblings of ASD children. Poster presented at the annual meeting of the International Neuropsychological Society, St. Louis, MO.

Toth, K., Dawson, G., Meltzoff, A., & Munson, J. (2004, May). Early predictors of language growth in young children with autism: Joint attention, imitation, and toy play. Poster presented at the annual International Meeting for Autism Research, Sacramento, CA.

Toth, K., Dawson, G., Meltzoff, A., & Munson, J. (2004, May). Early predictors of language growth in young children with autism: Joint attention, imitation, and toy play. Poster presented at the CPEA/STAART Annual Meeting, Bethesda, MD.

Toth, K., Dawson, G., & Fein, D. (2003, August). The UW infant and toddler sibling study. Presented at the first annual NAAR-NICHD High-Risk/Baby Sibs Autism Consortium Meeting, Washington, D.C.

Toth, K., Dawson, G., Munson, J., Estes, A., & Abbott, R. (2003, April). Role of joint attention, social interaction, and play in language and social growth in young children with autism. Paper symposium at the biennial meeting of the Society for Research in Child Development, Tampa, FL.

Toth, K., Dawson, G., Munson, J., Abbott, R., Estes, A., & Osterling, J. (2001, April). Defining the early social attention impairments in autism: Social orienting, joint attention, and responses to emotions. Poster presented at the biennial meeting of the Society for Research in Child Development, Minneapolis, MN.

PUBLICATIONS

Pandey, J., Toth, K., Hodgson, S., Green, J., Dawson, G., & Fein, D. (in preparation). Characteristics of younger siblings with ASD: Are they representative of the larger ASD population?

Pandey, J., Toth, K., Hodgson, S., Green, J., Dawson, G., & Fein, D. (in preparation). Similarities within sibling pairs with ASD.

Toth, K., Breiger, D., Jones, K., & Speltz, M. (in preparation). Neuropsychological and cognitive profiles of young children with conduct problems from preschool to school age.

Toth, K., Collett, B., Kapp-Simon, K., Keich Cloonan, Y., Gaither, R., Cradock, M. M., Buono, L., Cunningham, M. L., Dawson, G., Starr, J., & Speltz, M. L. (submitted, under revision). Memory and response inhibition in young children with single-suture craniosynostosis.

Elder, L., Dawson, G., Toth, K., & Fein, D. (submitted). Head circumference trajectory as a risk factor for developing autism symptoms in infant siblings of children with autism.

Toth, K., Dawson, G., Meltzoff, A. N., Greenson, J., & Fein, D. (e-pub ahead of print, 1-11-07). Early social, imitation, play, and language abilities in young non-autistic siblings of children with autism. *Journal of Autism and Developmental Disorders.*

Dawson, G., Munson, J., Webb, S. J., Nalty, T., Abbott, R., & Toth, K. (2007). Rate of head growth decelerates and symptoms worsen in the second year of life in autism. Biological Psychiatry, 61(4), 458-464.

Toth, K., Munson, J., Meltzoff, A. N., & Dawson, G. (2006). Early predictors of communication development in young children with autism spectrum disorder: Joint attention, imitation, and toy play. *Journal of Autism and Developmental Disorders, 36,* 993-1005.

Dawson, G., & Toth, K. (2006). Autism spectrum disorders. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology, second edition, volume three: Risk, disorder, and adaptation* (pp. 317-357). Hoboken, NJ: Wiley.

Toth, K. (2004). Definition of regression in autism. In J. T. Neisworth & P. S. Wolfe (Eds.), *The autism encyclopedia* (p.172). Baltimore, MD: Paul H. Brookes Publishing Co., Inc.

Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Social attention impairments in young children with autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology, 40,* 271-283.

Dawson, G., Munson, J., Estes, A., Osterling, J., McPartland, J., Toth, K., Carver, L., & Abbott, R. (2002). Neurocognitive function and joint attention ability in young children with autism spectrum disorder versus developmental delay. *Child Development, 73,* 345-358.

COMMUNITY PRESENTATIONS AND INVOLVEMENT

2/2006 **Researcher Night, National Alliance for Autism Research (NAAR) and Autism Speaks**
Presentation on current research projects at the UW Autism Center delivered to educators, parents, and supporters of NAAR.

8/2005	**Zero to Three Leadership Fellows, UW Center on Human Development and Disability (CHDD) and Center for Infant Health and Development** Lecture on the state of the science in autism delivered to scholars in psychology, psychiatry, social work, and the law.
6/2005	**Healthy Start Program** Lecture on early recognition of autism spectrum disorders delivered to healthcare and social support providers in east King County, Seattle, Washington as part of the Healthy Start program for young mothers.
2/2005	**First Signs, Inc.** Provided input on First Signs parent brochure to the First Signs Clinical Advisory Board. The brochure is designed to assist parents in early detection of developmental delays including autism and will be distributed in states launching the First Signs program.
10/2004	**Child Health Institute, UW Department of Pediatrics** Lecture on early identification of autism spectrum disorders delivered to researchers in pediatrics and psychologists, Work in Progress Program.
4/2004	**UW Autism Center Open House** Answered questions and described current work on early identification and early intervention to invited guests.
3/2004	**Queen Alexandra Centre for Children's Health, Vancouver Island Health Authority, B.C.** Provided consultation to clinical psychologists on early identification of autism spectrum disorders and behavioral coding of home videotapes.
3/2004	**Infant Toddler Early Intervention Program, CHILD Profile, Washington State Department of Health** Provided input on items to include in the Watch & Help Me Grow series of brochures to capture early symptoms of autism. These brochures are sent to all new parents in Washington State.
6/2002	**UW Autism Center Research Program Open House** Answered questions and described individual research projects to families participating in the autism research project at the University of Washington.
6/2002	**Autism Spectrum & Related Disorders Resource Center website** Designed the home page of a website for individuals and families with ASD.
7/2000	**Center for Autism and Related Disorders (CARD), Orlando, FL** Public lecture on the genetic basis of autism delivered to parents of children with autism spectrum disorders.

8/1998 **Learn to Win Camp**
Children's Hospital and Regional Medical Center
Volunteer camp counselor for a group of eight adolescent males participating in a one-week summer camp program for children with psychiatric disorders. Diagnoses included Tourette's Disorder, Oppositional Defiant Disorder, mental retardation, autism, encopresis, depression, and anxiety. Assisted staff in supervising boys during all camp activities, including a Ropes course, canoeing, art, nature walks, and mealtimes.

REFERENCES

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