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A taxometric analysis of Autism Spectrum Disorders in toddlers

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A TAXOMETRIC ANALYSIS OF AUTISM SPECTRUM DISORDERS IN TODDLERS

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Abstract

Autism Spectrum Disorders (ASD) are a group of neurodevelopmental disorders that are generally first diagnosed in childhood. With the advances in technology to identify this group of disorders, children are being identified at younger and younger ages. Early identification of ASD is critical due to the beneficial effects of early intensive behavioral interventions. While children are being diagnosed with the disorder at very high rates, etiology and definitions of the disorders are still being investigated. Great variability exists with regard to symptoms between individuals. Additionally, less is known about symptom expression in individuals without ASD and symptoms of ASD occurring as a discrete category or along a dimension across populations. Taxometric analysis was employed to determine the underlying latent structure of ASD in toddlers at risk for developmental disabilities. A dimensional latent structure of ASD in a population of toddlers at risk for developmental disabilities was found. This has important implications for the upcoming DSM-V, and the proposal of collapsing PDD-NOS and Asperger's Disorder into a single autism spectrum disorder category.

Introduction

The word "autism," first coined by Bleuler in 1911 (Kanner, 1965), has since become a well-recognized term. Once considered a rare form of psychopathology in childhood, autism spectrum disorders (ASD) are currently being diagnosed at very high rates. Consequently, there has been much attention to this particular disorder by researchers, clinicians, parents, government agencies, and the media. While there is an extensive amount of research on the disorder, there is still debate on its definition, etiology, and the underlying structure of symptoms.

History of ASD

Bleuler coined the term autism while referring to the extreme withdrawal of oneself characteristic of people with schizophrenia. However, the disorder known as autism is a distinct diagnostic entity from schizophrenia. Leo Kanner (1943) was the first to use the term when referring to children with ASD. Kanner, an Austrian psychiatrist, immigrated to the United States in the 1920s. While serving on the faculty at Johns Hopkins University, he wrote of a group of 11 children, all displaying a similar set of characteristics. The children, as described by Kanner, exhibited an "extreme autistic aloneness" (Kanner, 1943, p. 242). Since infancy, these children experienced difficulties with relating to other people in a typical manner. The children did not interact appropriately with peers and preferred objects to people. Furthermore, the children described by Kanner all had communication deficits. Three of the children never spoke and of those who did speak, their language lacked communicative intent. All of the children had an insistence on sameness: they would become quite disturbed with changes to their routine and the environment. Noises, food, social contact, and incomplete objects

often caused distress to these children. Kanner described the children's maladaptive behaviors as the result of intrusions upon the child from the environment. Kanner also noted that while these children were once thought to have intellectual disability (ID), he considered them to have "*good cognitive potentialities*" (p. 246). The children were also described as physically normal overall, with some children having larger head circumferences. Gait disturbances were noted; however, fine motor capabilities appeared intact. Lastly, Kanner noted that all of the children came from highly intelligent and educated families. Fathers were psychiatrists, chemists, and professors, and the majority of mothers were college graduates.

At the same time of Kanner's "discovery" of autism, another researcher independently wrote of a seemingly similar disorder. In 1944 Hans Asperger, an Austrian pediatrician described a small group of children as having "autistic psychopathy." Asperger's original 1944 work was not translated into English until 1991 by Uta Frith (Frith, 1991) and the term Asperger's syndrome was not introduced to the English language until 1981 by Lorna Wing (Wing, 1981). Similar to the children described by Kanner, Asperger's children also displayed unusual social relationships. Asperger reported that the children had abnormal eye gaze (e.g., just glancing past people when communicating) and displayed abnormal facial and gestural expressions. The children also exhibited abnormal speech and language use. Furthermore, the children had unusual interests and knowledge, displayed stereotypic movements, and exhibited abnormal responses to sensory stimuli. In addition to the abnormalities in socialization, communication, behavior, difficulties with hygiene maintenance, attention, eating, and sleeping were also noted in this group of children (Frith, 1991).

Prior to the identification of autism by Kanner, and for many years following, researchers and clinicians considered autism and schizophrenia related disorders. Both disorders were grouped together in classification systems prior to the 1980s (i.e., *Diagnostic and Statistical Manual*), as well as in journals such as the *Journal of Autism and Childhood Schizophrenia.* Nonetheless, some researchers believed the two disorders were discrete entities. An early distinction between these two disorders was made by Kolvin (1971). He distinguished between different forms of childhood psychosis with regard to age of onset. The earlier onset was termed *infantile psychosis* and was characterized by onset prior to 3 years of age, social isolation, and either extreme reactions to environmental changes or stereotypies. Conversely, *late onset psychosis* developed between the ages of 5 and 15 years, and the children displayed schizophrenic symptoms consistent with adult onset schizophrenia (Kolvin, 1971). While the disorders were both termed psychosis, the descriptions were consistent with autism and childhood onset schizophrenia.

An influential researcher, Michael Rutter, delineated key differences between autism and schizophrenia (Rutter, 1968). Rutter pointed out the discrepancy in male to female ratios (autism having higher male to female ratio), stability of symptom patterns across time in autism, higher prevalence of impaired intellectual functioning in autism, and a later age of onset in schizophrenia as support for the two disorders being distinct entities. Even though Rutter made this empirical distinction between autism and childhood schizophrenia in the 1960s, it was not until 1980 with the *Diagnostic and Statistical Manual - Third Edition* (DSM-III; (American Psychiatric Association, 1980) that autism was included as a discrete diagnosis, separate from schizophrenia. Research

has continued on delineating the differences between ASD and childhood onset schizophrenia (Reaven, Hepburn, & Ross, 2008).

Core Features of ASD

According to the current literature and criteria from the *Diagnostic and Statistical Manual - Fourth Edition* (DSM-IV-TR; American Psychiatric Association [APA], 2000) ASD are characterized and diagnosed based on three hallmark symptom clusters: deficits in socialization, deficits in communication, and behavioral excesses such as restricted interests, insistence on sameness, and stereotypic movements. For children with the more severe forms of ASD, such as autistic disorder, these symptoms may be evident well before the child's third birthday. Children with less severe forms of ASD, such as Asperger's disorder, the symptoms may not be apparent until middle childhood.

Socialization. The most salient characteristic of people with ASD is the deficit in socialization. Children and adults with ASD have difficulties in relating to other people, and for some individuals with the disorder, these deficits are evident since infancy (Volkmar, 1987; Volkmar, Chawarska, & Klin, 2004). As children with ASD age, deficits in socialization become more salient. Older children with ASD may not seek out playmates, preferring to play alone. Nonverbal behavior is often lacking in children with ASD. Eye contact and eye-to-eye gaze is either non-existent or abnormal, as are gestures to facilitate communication.

Socialization deficits persist throughout childhood. While deficits in social skills may improve as a child develops, these deficits continue to affect the individual with ASD and are still present in adulthood (Matson, et al., 1996). Furthermore, when ID is

present along with ASD, social skills may be even more impaired (Njardvik, Matson, & Cherry, 1999).

Language and Communication. Another defining feature of ASD is communication deficits. As pretend play is a precursor to language, young children with ASD often do not engage in make-believe or pretend play. Many people with ASD never develop speech, and for those who do develop speech, abnormalities in functional use and pragmatics are noted. Children with ASD may exhibit echolalia and pronoun reversal (Rutter & Bartak, 1971). Peculiarities in the rhythm, intonation, and volume of speech are characteristic of verbal people with ASD (Tager-Flusberg, Paul, & Lord, 2005). Language may be used in a more stereotypic manner, rather than to convey meaning. As children age, an inability to initiate and sustain conversations may be noted. Coupled with deficits in verbal language, children with ASD also have deficits in communicating basic wants and needs.

Behavior. In addition to deficits in socialization and communication, children with ASD also exhibit behavioral excesses. Although not specific to this disorder, people with ASD display behaviors such as restricted interests, insistence on sameness, and repetitive behaviors. Typically developing children exhibit some of these similar behaviors during normal development. That is, stereotyped behaviors such as rocking and head banging are not uncommon during infancy, and rituals are not uncommon to toddlers. However, these particular behaviors persist into childhood in individuals with ASD. Behaviors such as repetitive movements involving the body and objects, including tapping objects on a surface, rocking objects, spinning, moving, and clutching objects,

rubbing the body, and stiffening of the hands and arms are characteristic of children with ASD (Watt, Wetherby, Barber, & Morgan, 2008).

In children with ASD, repetitive and restrictive behaviors may decrease over time and change with regard to topography (Esbensen, Seltzer, Lam, & Bodfish, 2009). Stereotypies are more common in the youngest children with ASD, then rituals and compulsions for older children, and adults being less likely to display these behaviors (Esbensen, et al., 2009). Militerni and associates (2002) looked at a sample of young children. Significant correlations were observed between motor behaviors and age, and stereotypies and cognitive function. The authors suggested a developmental component to the expression of repetitive behaviors. Younger children displayed more stereotypic trunk and limb movements. Children with lower cognitive functioning levels displayed more sensory-type behaviors such as licking, sniffing, visual stimulation, and self-injurious behavior. Conversely, older children were significantly more likely to engage in behaviors such as filling, emptying, collecting, and constructional play such as using building blocks or puzzles. Those children with higher cognitive functioning were more likely to engage in repetitive behaviors consisting of constructional play as well as verbal expressive behaviors, which included repetitive word usage.

Infants and Toddlers with ASD

Numerous studies over the past 20 years have reported on the efficacy of early behavioral interventions on improving outcomes for children with ASD (Cohen, Amerine-Dickens, & Smith, 2006; Eikeseth, Smith, Jahr, & Eldevik, 2002; Remington, Hastings, Kovshoff, & degli Espinosa, 2007; T. Smith, Groen, & Wynn, 2000). Many of these programs, specifically early intensive behavioral intervention, suggest children

begin the intervention programs between 2 and 3 years of age, and preferably as young as possible (Ben-Itzchak & Zachor, 2007; Eikeseth, Smith, Jahr, & Eldevik, 2007; Matson & Smith, 2008). However, this call for very early intervention poses some difficulties: the average age of diagnosis of ASD in the United States is 5 years of age and only after the child has been evaluated by an average of 4.5 specialists (Siklos & Kerns, 2007). If children are not receiving a diagnosis of ASD until the age of 5, they may miss the window of opportunity to reap the most benefits from early intensive behavioral intervention.

Some of the easily recognized characteristics of ASD may not be developmentally appropriate or salient in the early years. Thus, ASD may go unrecognized during infancy. Furthermore, there appears to be a significant percentage of children who develop seemingly normal, and then during the second year of life, they experience a regression in development and the emergence of symptoms consistent with ASD (Bryson, et al., 2007; Lord, Shulman, & DiLavore, 2004; Luyster, et al., 2005). Therefore, identifying early symptoms may be quite difficult. Currently researchers are in a race to identify behaviors that may discriminate between infants and toddlers with ASD and those without. Data collection methods have included retrospective chart reviews, home videotapes, and assessment of children who are considered "at risk" for an ASD (i.e., siblings of children with ASD). Still, little is known about ASD symptoms in infants and toddlers (Chawarska, Volkmar, & Klin, 2008).

When identifying ASD in very young children, it is important to note the presence and absence of behaviors consistent with typical development, as well as, the presentation of atypical behaviors. Studies investigating symptoms of ASD in infants and toddlers

have identified behaviors that may be indicative of ASD at this young age. Children in the following studies likely have one of two more common ASDs: autism or PDDNOS. With regards to Asperger's Disorder, this disorder is much more difficult to identify during the first years of life. By current definition, these children do not experience communication delays or impairments in cognitive and adaptive functioning. Therefore, it is unlikely that they will be referred for services prior to 4 years of age (McConachie, Couteur, & Honey, 2005).

Social deficits can be observed before the first year of life. Typically developing infants look at the faces of caregivers, smile, engage in reciprocal vocalizations, and share attention with others, while some infants with ASD, according to parent report, do not smile socially or engage in eye contact (Volkmar, 1987). Other behaviors characteristics during the first year of life of children later diagnosed with autism include ignoring people, poor social interaction, and abnormal eye contact (Maestro, et al., 2005).

During the second and third year of life, symptoms of ASD become more salient. Behavioral deficits that can distinguish between two-year old children with ASD from those with a non-ASD developmental disability and typically developing children include lack of appropriate eye gaze; joyful expressions when gazing at others; sharing enjoyment or interests with others; coordination of gaze, facial expression, gestures and sound; and showing behaviors (Wetherby, et al., 2004). Similarly, other researchers have found that toddlers with ASD experience deficits in protodeclarative pointing, monitoring of gaze, pretend play, lack of interest in others, and failure to initiate contact (Baron-Cohen, 1996; Bryson, et al., 2007). Other behaviors noted as characteristic of children later diagnosed with ASD are less responsive to name and directing attention to another's

face (Bryson, et al., 2007; Maestro, et al., 2005; Osterling & Dawson, 1994). While cognitive ability is not a defining feature of ASD, more than half of the children may also experience a drop in cognitive functioning in the second year of life (Bryson, et al., 2007).

Behavioral excesses in 2-year-olds may include unusual prosody, repetitive movements with body and objects, and unusual posturing of the body or parts of the body (Wetherby, et al., 2004). Very young children may also engage in visual fixation, sensory and motor mannerisms, and stereotypies. Extremes in temperament and mood are also noted, such as excessive irritability or passivity (Bryson, et al., 2007; Maestro, et al., 2005). As can be noted from the review of behavioral symptoms of ASD in infants and toddlers, the social and communication deficits are more pronounced during this time period than the repetitive behaviors, insistence on sameness, and restricted interests. This observation may be due in part to different developmental trajectories with regard to the three ASD symptom clusters (Charman & Swerrenham, 2001).

Differential Diagnosis of ASD and Other Developmental Disabilities. The accurate identification of symptoms of ASD and discriminating these symptoms from other disorders is of great importance. As the majority of individuals with ASD also have intellectual disability (ID), some of the atypical development observed in ASD may be better attributed to the ID (Osterling, Dawson, & Munson, 2002). That is, some of the abnormal behaviors are not limited to ASD; rather the behaviors are related to a developmental delay in general. Additionally, certain gene-related developmental disorders are associated with higher rates of symptoms consistent with ASD (Descheemaeker, Govers, Vermeulen, & Fryns, 2006; Peters, Beaudet, Madduri, &

Bacino, 2004; I. Smith, Nichols, Issekutz, & Blake, 2005). Due to the overlap of some behaviors of ASD with ID and other developmental disorders, discriminating between these populations may at times be difficult. Thus, researchers and clinicians need to discern which behaviors exhibited by a child are related to ASD and which are better accounted for by an intellectual or developmental disability. According to the current literature, ASD and non-ASD developmental disorders can be distinguished from one another. However, it has not been established if these group differences are a distinction of type or degree.

Researchers have examined populations of children with ASD and non-ASD developmental disabilities to determine if differences were evident independent of intellectual functioning. Children with ASD in the second year of life may differ from children with non-ASD developmental disabilities in terms of verbal and non-verbal communication. Young children with ASD may have different rates of communicating with others, and verbalization with a communicative intent when compared to children with non-ASD developmental disabilities (Wetherby, Watt, Morgan, & Shumway, 2007; Wimpory, Hobson, Williams, & Nash, 2000).

Differences in socialization between young children with ASD and other developmental disabilities have also been noted and are more salient than deficits in communication at this young age (Matson, et al., in press). Such differences include, gaze shifts, gaze/point/follow responses, socialization with play and lap games, joint attention, greeting others, waving, anticipatory posture, eye contact, and socially directed feelings (e.g. anger) when compared to children with non-ASD developmental disabilities (Wetherby, et al., 2007; Wimpory, et al., 2000). In addition, 12-month-old children with

ASD and ID can be differentiated from those children with just ID. The children with ASD and ID oriented to their name and looked at other people less than children with just ID. The children with just ID looked at other's faces at similar rates to typically developing children (Osterling, et al., 2002). Dawson and colleagues (2004) examined children between the ages of 2 and 4 years with ASD, developmental delay, and typically developing and compared them on social abilities. What the authors found was that children with ASD failed to orient (head and/or eye turns) to social and non-social stimuli and were less likely to initiate joint attention or respond to other's attempts to share attention when compared to children with developmental disability and those who were typically developing. On these variables, the latter 2 groups did not show significant differences from each other.

In addition to differences in socialization and communication, young children with ASD differ from other children with developmental disabilities in the area of behavioral excesses. Toddlers with ASD engage in behaviors such as repetitive movements involving the body and objects, including tapping objects on a surface, rocking objects, spinning, moving, and clutching objects, rubbing the body, and stiffening of the hands and arms, differentiating them from toddlers with non-ASD developmental disabilities (Watt, et al., 2008). A retrospective study investigating autism symptoms in the first 2 years of life also found significant differences between children later diagnosed with ASD compared to children later diagnosed with ID (Dahlgren & Gillberg, 1989). Such differences included, dislike for being disturbed, content to be left alone, did not attract others to own activity, empty gaze, abnormal reaction to sounds, stare and look at objects and patterns in an atypical manner, and attachments to unusual objects.

Furthermore, children with ID did not differ significantly from typically developing children in these areas.

However, other studies found it difficult to distinguish between groups of children with less severe forms of ASD at very young ages. Children with a diagnosis of "other" PDD (PDDNOS or Asperger's Disorder) were indistinguishable from children with a language disorder at 20 months based on measures of the ADI-R (Cox, et al., 1999). However, as children aged, some symptoms became more salient and in the Cox and colleagues (1999) study, by the time these children were 42 months of age, imaginative play and offering comfort distinguished the two groups. Additionally, older children with ASD may be distinguished from children with non-ASD developmental disabilities on responses to sensory stimuli. Children, between the ages of 5 and 80 months, with autism were significantly more hyporesponsive to social and non-social stimuli than children without non-ASD developmental disabilities (Baranek, David, Poe, Stone, & Watson, 2006). While children with ASD and those with other developmental delays may exhibit similar behavioral characteristics, the current literature supports the ability to distinguish between these two groups even at very young ages.

ASD Symptoms in Other Populations

Symptoms of ASD are not limited to just children who have what is termed idiopathic autism. Idiopathic autism refers to cases of autism that are not secondary to another condition; there is no known cause for the person's autism. Deficits in socialization, communication, and repetitive behavior have also been noted in populations presenting with other conditions. Symptoms such as communication and socialization deficits can be observed in individuals with ID. Additionally, people with

severe and profound ID may engage in repetitive behaviors and stereotypic movements. Individuals who have been subjected to extensive sensory deprivation, may also engage in repetitive and socially isolative behaviors (Lovaas, Newsom, & Hickman, 1987). While these symptoms and behaviors are similar to people with ASD, these two groups can be distinguished from one another (Matson, Smiroldo, & Hastings, 1998).

The study of behavioral patterns of children with genetic conditions has also revealed a high over lap of ASD symptoms, with some estimates at about half of individuals with genetic and chromosomal abnormalities (Skuse, 2007). A large percentage of children with tuberous sclerosis exhibit symptoms consistent with a diagnosis of autistic disorder (Madsen, et al., 2002). Of children with a 22q11.3 deletion, 20% show symptoms consistent with ASD and up to 14% may meet criteria for autistic disorder (Fernell, Gillberg, & Wendt, 1991). Children with Down's syndrome also exhibit symptoms of ASD at higher rates than the general population. One study found that approximately 7% of the sample of children with Down's syndrome displayed symptoms warranting a diagnosis of ASD, and about one third of the children who did not meet criteria for an ASD displayed obsessive/ritualistic behavior (Kent, Evans, Paul, & Sharp, 1999). Just under 20% of people with Prader-Willi syndrome may also meet criteria for autistic disorder (Descheemaeker, et al., 2006). Approximately 42% of children with Angelman's syndrome may meet criteria for autistic disorder, and the children who do not meet criteria for a diagnosis still exhibited characteristics consistent with ASD, such as stereotypies and deficits in play skills (Peters, et al., 2004). Lastly, ASD symptoms also appear to occur at high rates in CHARGE syndrome (I. Smith, et al., 2005).

Other conditions in which ASD symptoms have been reported to occur at high rates are psychological and physical disorders. More than half of the children with attention-deficit/hyperactivity disorder (ADHD) exhibit symptoms similar to ASD, such as deficits in nonverbal communication, stereotyped motor movements, and preoccupation with parts of objects (T. Clark, Feehan, Tinline, & Vostanis, 1999). Furthermore a large percentage of individuals with obsessive compulsive disorder present with social and communication characteristics consistent with ASD, but may be diagnosed with a personality disorder instead (Bejerot, 2007). A Swedish study investigating ASD symptoms in a population of children who were previously diagnosed with infantile hydrocephaly, found that 23% of the sample met criteria for an ASD (Fernell, et al., 1991).

Autism traits are also reported in non-referred samples. Using a measure of autistic traits and cognitive characteristics of people with high functioning ASD (i.e., I.Q. > 70), a group of researchers found that males scored significantly higher than females (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). Furthermore, college students studying science (e.g. physics, engineering) scored significantly higher on autism traits than students studying humanities and the social sciences (Baron-Cohen, et al., 2001). People who have a male twin (monozygotic or dizygotic) display higher rates of behavioral characteristics consistent with ASD, even if they and their twin do not meet criteria for a diagnosis of ASD (Ho, Todd, & Constantino, 2005). Also, siblings of children with ASD may exhibit symptoms consistent with ASD, but do not meet criteria for diagnosis (Micali, Chakrabarti, & Fombonne, 2004).

From the studies investigating ASD symptoms in family members of people with ASD, individuals with chromosomal abnormalities, and in the general population, characteristics of ASD appear to be common (Constantino & Todd, 2003). Other studies have reported the same findings of ASD symptoms normally distributed in the general population; although positive skew or extension on the right tail may be observed (Constantino & Todd, 2003, 2005; Posserud, Lundervold, & Gillberg, 2006; Ronald, et al., 2006). Additionally, ASD symptoms appear dimensional along the general population (Spiker, Lotspeich, Dimiceli, Myers, & Risch, 2002).

Current Definitions

Various definitions and classification systems for ASD have presented since Kanner first wrote of the disorder more than 60 years ago. Prior to the inclusion of autism in the DSM-III (American Psychiatric Association, 1980), numerous other classification systems were used. Disagreement between researchers existed on what constitutes core symptoms of the disorder, and the various systems showed small diagnostic overlap (DeMyer, Churchill, Pontius, & Gilkey, 1971).

The DSM-IV-TR (American Psychiatric Association, 2000), includes the diagnostic category, Pervasive Developmental Disorders (PDD). Under this umbrella of PDD, five different diagnoses are included: autistic disorder, Asperger's disorder, Rett's disorder, childhood disintegrative disorder (CDD), and pervasive developmental disorder not otherwise specified (PDDNOS). Autistic disorder, Asperger's Disorder, and PDDNOS are the more common of the PDDs, with Rett's and CDD considered rare disorders.

According to the DSM-IV-TR (APA, 2000) in order for an individual to meet criteria for autistic disorder, socialization and communication impairments, as well as, behavioral excesses must be present. The person must exhibit at least two of the following impairments with regard to social interaction: impairment in the use of nonverbal behaviors (e.g., eye gaze, facial expressions, gestures); failure in the development of relationships with peers (appropriate for the developmental level); deficits in the sharing of enjoyment, interest, and achievements with other people; and deficits in social/emotional reciprocity. At least one of the following deficits in communication is displayed: delay in or lack of developmental of spoken language; impairment or inability to initiate or maintain a conversation (in those with adequate speech); repetitive and/or stereotyped use of language; and lack of make-believe play that is varied and spontaneous, as well as, imitative play. With regard to behavioral excess, the person must exhibit restricted, repetitive, and stereotyped behavior including one of the following: abnormally intense preoccupation with stereotyped/restricted interest; inflexible with adherence to certain routines or rituals that are nonfunctional; stereotypies; and preoccupation with parts of objects. In addition to exhibiting symptoms from each of the three domains, at least 6 symptoms total, must be present. Lastly, delays or abnormality in functioning must be present prior to 36 months of age in at least one of the following areas: social interaction; language use in the context of social communication; and imaginative play. Also, the symptoms cannot be better accounted for by Rett's disorder or CDD.

Similar to the diagnostic criteria for autistic disorder, criteria for Asperger's disorder also requires impairment in two areas of social interaction (i.e., nonverbal

behaviors; development of peer relationships; sharing enjoyment; and social/emotional reciprocity). Also similar to the diagnosis of autistic disorder, the child must exhibit at least one symptom related to restricted, repetitive, and stereotyped behaviors or interests (i.e., abnormal, intense interest and preoccupation; inflexible adherence to routines; stereotypies; and preoccupation with parts of objects). The deficits must also cause clinically significant impairment in the individual's functioning. No clinically significant delay in language, cognitive development, or adaptive behavior is noted. Lastly, the individual does not meet criteria for another PDD or schizophrenia.

Only one disorder included under the PDD umbrella of the DSM-IV-TR (APA, 2000), Rett's disorder, has identifiable genetic markers. However, at this time it is behaviorally defined and included as a PDD according to the DSM-IV-TR (APA, 2000). In order for a person to meet criteria for Rett's disorder, the following symptoms must be present: seemingly normal pre- and postnatal development; seemingly normal psychomotor development through the first 5 months of life; and typical head circumference at birth. Following the above mentioned period of normal development, the following symptoms must also be met: head growth deceleration between 5 and 48 months of age; between the ages of 5 and 30 months a loss of previously acquired hand skills along with the development of stereotyped hand movements; loss of social engagement; poorly coordinated trunk and gait movements; and severely impaired expressive and receptive language development with severe psychomotor retardation.

Like Rett's disorder, CDD, has separate criteria within the PDD category of the DSM-IV- TR (APA, 2000). Individuals meeting criteria for CDD must exhibit seemingly normal development for at least the first two years of life, consisting of age-appropriate

verbal and nonverbal communication, social relationships, play behavior, and adaptive skills, followed by clinically significant loss of previously acquired skills prior to the age of 10 years in at least two of the following areas: expressive or receptive language; social skills or adaptive behavior; bowel or bladder control; play; or motor skills. Furthermore, abnormalities of functioning in at least two of the following areas need to be present: qualitative impairment in social interaction; qualitative impairments in communication; and restricted, repetitive, and stereotyped patterns of behavior, interests, and activities, including motor stereotypies and mannerisms.

The most commonly diagnosed disorder of the PDDs is PDDNOS (Fombonne, 2003, 2005). According to the DSM-IV-TR (APA, 2000), a diagnosis of PDDNOS is indicated when deficits of social interaction are severe, along with deficits in communication and/or the presence of restricted interests or stereotyped behaviors. This 'NOS' diagnosis is used as a residual category when the person is exhibiting symptoms consistent with a PDD, but does not meet criteria for any of the other disorders in the PPD category.

Etiology

Over the past 70 years, since Kanner first described the disorder, there have been countless theories as to the etiological underpinnings of ASD. One of the first proposed causes of ASD was environmental in nature (i.e., poor parenting). While this theory has fallen out of favor, both environmental and genetic influences are believed to contribute to the development of ASD.

Environmental Influences. In Kanner's initial account of the disorder, he described the parents of the children with ASD as overly intellectual and lacking warmth

(Kanner, 1943). Researchers and clinicians propelled the psychoanalytic theory that ASD was caused by parents being unresponsive, distant, and preoccupied with academic pursuits. Bruno Bettleheim (Bettelheim, 1972) was one such researcher. He elaborated that ASD was the result of poor parenting and "cold" mothers (Bettelheim, 1972). While the poor parenting cause has been debunked, other environmental influences have been implicated in the development of the disorder. Such environmental contributory factors have included congenital rubella (Chess, 1971; Chess, Fernandez, & Korn, 1978), vaccines (Wakefield, et al., 1998), and exposure to prenatal stress (Beversdorf, et al., 2005; Kinney, Miller, Crowley, Huang, & Gerber, 2008).

An environmental influence associated with ASD in the 1970's was congenital rubella. The United States experienced an outbreak of rubella in 1964. Following the outbreak came a surge of infants born with vision, hearing, and heart problems (Sever, Nelson, & Gilkeson, 1965). Moreover, other behavioral symptoms were soon noted in these children. Further examination of the children affected by congenital rubella revealed an association between exposure to the virus during gestation and the development of ASD (Chess, 1971; Chess, et al., 1978; Treffert, 1970). Trefter (1970) examined a large sample of Wisconsin children for ASD characteristics. The prevalence estimates for autism (i.e., children with a more strict diagnosis of autism) at the time was .7 per 10,000. Of the children exposed to rubella during pregnancy, 412 per 10,000 met criteria for autism. Fortunately, the rubella vaccine was licensed in 1969, with wide range efforts for mass vaccination.

An influential study published by Wakefield and colleagues (1998) reported of 12 children who developed behavioral symptoms, gastrointestinal abnormalities, and

developmental regression following the administration of the Measles-Mumps-Rubella (MMR) vaccine. The report was conducted through a gastroenterology clinic for children and the authors did note that the findings might have been due to referral bias. However, the authors also stated in the interpretation of the article that the symptoms of ASD were temporally associated with the MMR vaccine. This paper was not experimental in nature, and was more of a hypothesis generating exercise. Wakefield's study was widely published and present in the media. Following the release of the "findings" from the study to the press in the UK, public alarm ensued and a decrease in MMR vaccinations in children was noted (Offit & Coffin, 2003).

Numerous international studies have failed to find an association between ASD and the MMR vaccine (Fombonne & Chakrabarti, 2001). Studies investigated trends of ASD cases in the UK following the introduction of the MMR in 1988 (Farrington, Miller, & Taylor, 2001; Taylor, et al., 1999), withdrawal of the MMR in Japan (Honda, Shimizu, & Rutter, 2005), and relative risk of ASD for a vaccinated and unvaccinated group in Denmark (Madsen, et al., 2002), without finding any association between the MMR and ASD. Furthermore, 10 of the 12 authors from the original Wakefield study have since retracted the interpretation of an association between MMR and ASD (Murch, et al., 2004) and the journal that published the study, *The Lancet*, has recently retracted the article (Lancet, 2010).

Prenatal exposure to stress has also been implicated in the development of ASD. Beversdorf and colleagues (2005) conducted a study on exposure to stressful life events during gestation. Children with ASD were compared to children with Down's syndrome and typical development. Stressful life events ranged in severity and included divorce,

marital separation, death of a family member, new mortgage, foreclosure on a loan, job changes, and death of a close friend. When compared to the other two groups, mothers of children with ASD reported higher levels of stressful life events, particularly during 21-32 weeks of gestation. Other studies have found similar results with respect to exposure to stress during a particular gestational timeframe. Kinney and colleagues (2008) conducted a study on the rates of ASD in children exposed to hurricanes and tropical storms during gestation. What the authors found was that there was a dose-response effect for proximity to the storm, with increased rates of ASD, and even more so during middle to late pregnancy.

Genetic Influences. While the theory of poor parenting was quite controversial, it did not receive much resistance until the work of Bernie Rimland. Rimland was a psychologist and the father of a child with ASD. In 1964, he authored a book entitled *Infantile Autism: The Syndrome and its Implications for a Neural Theory of Behavior* (Rimland, 1964). In this book, Rimland contested the theory that parents were to blame for their child's ASD and suggested a biological basis for the disorder.

Around the same time Rimland spoke out on the poor parenting theory of ASD, other researchers began to follow suit. One such researcher was Sir Michael Rutter. Disagreeing with the psychogenic theory of ASD, he sought to identify genetic factors related to ASD (Rutter, 1968). Rutter argued that the basis for an environmental cause for the disorder (i.e., low incidence of siblings affected with the disorder and the disorder not being passed down to children from parents with autism) was seriously flawed. He stated that the incidence of autism in siblings was in fact higher than it occurred in the general

population. Additionally, as the disorder was quite disabling, those affected with the condition oftentimes did not have children.

One of the first studies to look at genetic factors related to ASD was by Folstein and Rutter (1977). The study investigated the concordance rate of autism between monozygotic and dizygotic twin pairs. A significantly greater concordance rate was found between the monozygotic twin pairs as compared to the dizygotic pairs. Interestingly, the authors reported that symptom severity varied within pairs of monozygotic twins. Numerous other studies have since been conducted with similar findings: higher concordance rates in monozygotic twins and evidence for a broader spectrum of the disorder within twin pairs (Bailey, et al., 1995; Couteur, et al., 1996; Steffenburg, et al., 1989).

Studies have also suggested that twinning is a risk factor for ASD. One study investigated autism symptomatology in a population sample of twin and non-twin children. Children who were a twin displayed higher levels of ASD symptoms than did children without a twin sibling (Ho, et al., 2005). The authors proposed that twinning results in more complications with pregnancy, different nutrient allotment to the fetuses, and higher susceptibility to viruses, which could all be contributed to an environmental insult to an individual with a genetic susceptibility (Ho, et al., 2005). Other research teams have not found this link between twinning and ASD (Hallmayer, et al., 2002).

To further support the genetic influences on ASD, higher rates of the disorder occur between siblings, at about 3% (Bolton, et al., 1994). Additionally, studies have supported that a lesser variant of ASD, not meeting criteria for a diagnosis, occurs in families of people with ASD (Bailey, Palferman, Heavey, & Le Couteur, 1998; Piven,

Palmer, Jacobi, Childress, & Arndt, 1997). Furthermore, family members may exhibit symptoms consistent with just one of the impairments associated with ASD, and not all three (Bolton, et al., 1994). Autistic disorder reportedly has a 90% heritability rate, being one of the most hereditary disorders known (Freitag, 2007).

Phenotye/Genotype. Identification of the autism phenotype is a hot topic due to the current definitions of the disorder being inadequate or inconsistent across studies (Lecavalier, Gadow, DeVincent, Houts, & Edwards, 2009). Without adequately identifying the phenotype of a disorder, investigating the genotype proves difficult (Matson, 2007). A major hindrance in identification of specific genes that lead to the development of ASD is that it is likely that not one single gene contributes to the symptomotology. Instead, multiple genes and their interactions with one another are likely the culprit (Pickles, et al., 2000). Furthermore, the disparate findings and failure to replicate studies may be related to the heterogeneous nature of the disorder, leading to varied presentations across studies (Lam & Aman, 2007). As such, studies of more homogenous groups may lead to the identification of different genes, and possibly to different subtypes of the disorder. Increasing the exclusion criteria, and forming more of a homogenous group, may assist with the identification of autism-susceptibility genes (Buxbaum, et al., 2001). As previously discussed, family members of people with autism may experience just one characteristic of ASD. This fractioning of ASD impairments lends to the possibility of different causative factors for each of the facets of ASD (Bolton, et al., 1994). One consideration for identifying genes for ASD is to investigate the association with each of the three impairments independently, as these behaviors are

possibly independent of one another (Happé, Ronald, & Plomin, 2006), and moderately correlated at best (Ronald, et al., 2006).

Currently, studies are emerging that are investigating ASD and the different behavioral characteristics associated with the disorder and their relation to gene variants. Through these types of studies, particular genes and genotypes are being connected with certain behavioral phenotypes of ASD. Numerous genes have been implicated in this disorder, with the likely cause being a combination of multiple genes resulting in ASD. Abnormalities of chromosome 16 (Weiss, et al., 2008), chromosome 2 (Buxbaum, et al., 2001), and chromosome 7 (Idol, Addington, Long, Rapoport, & Green, 2008; Schellenberg, et al., 2006; Vincent, et al., 2008) have been implicated in ASD. Chromosome 15 has been implicated when investigating families with more than one person with ASD and cognitive functioning in the ID range (Liu, Paterson, & Szatmari, 2008). Duplications of chromosome 15 have also been reported in a small number of cases (Simic & Turk, 2004). However, other studies have found no association between chromosome 15 and a population with autism (Kato, et al., 2008). To further support the genetic link to ASD, there is a higher rate of symptoms of ASD in people with single gene disorders such as tuberous sclerosis, fragile-X, and phenylketonuria (Freitag, 2007).

Other studies have identified variants of the 5p14.1 gene as being associated with individuals with diagnoses of ASD. Additionally these authors also found associations with ASD diagnoses and genes nearby 5p14.1. The genes, CDH10 and CDHP which are molecules involved in neuronal cell-adhesion, were significantly associated with ASD (Wang, et al., 2009). The latter part of the study suggests the variants in these molecules

may affect the structure and connectivity of the neurons in the brain of an individual with ASD.

In an attempt to identify a genotype consistent with certain behavioral characteristics of ASD, Brune and colleagues (2006) investigated polymorphisms of the serotonin transporter genes, 5-HTTLPR. Using the standardized, behavioral measures of ASD symptoms, the ADI-R (Lord, Rutter, & Le Couteur, 1994) and ADOS (Lord, et al., 1989), the investigators found that one variant of the gene was consistent with increased deficits in the use of nonverbal communication to regulate social interaction, while the other gene variant was associated with increased scores on the "stereotyped and repetitive mannerisms" subdomain and increased impairment with directing facial expressions (Brune, et al., 2006).

Another group of researchers attempted to identify phenotypic characteristics associated with ASD and their relationship to particular genotypes. Goin-Kochel and associates (2009) looked at children with ASD and different variants of the *MTHFR* 677 T allele. Using behavioral ratings from the ADI-R (Lord, et al., 1994), the authors found a particular gene variant that was associated with higher rates of the following behaviors: direct eye-gaze, complex body movements, history of self-injurious behavior, and over activity (Goin-Kochel, et al., 2009).

Due to the autism phenotype likely having numerous genotypes, parceling out the different components of the disorder may aid in the identification of autism genotypes. A limited number of studies currently exist that incorporate behavioral ratings and their association with genetic variants. Due to the heterogeneity of ASD, future genetic studies

should continue with this model of identifying genes connected to specific behavioral characteristics, as opposed to the associating genes with an ASD diagnosis in general.

Prevalence

Within Kanner's first description of autism, he considered it to be a rare disorder of childhood (Kanner, 1943). Early estimates placed the prevalence of autism at about 4.5 per 10,000 children (Lotter, 1966). Following the identification of autism by Kanner, a surge of children were being diagnosed with this rare disorder (Kanner, 1965). Since that time, rates of children diagnosed with ASD have continued to increase. This increasing prevalence of ASD has been a cause for concern. Changes to criteria and expanding definitions of the disorder over time are likely to be responsible for the surge of diagnoses (Wing & Potter, 2002).

Croen and colleagues (2002) investigated the prevalence of autism in California. The authors first wanted to determine the prevalence for that geographic area as well as explore factors related to the increase in prevalence. The authors identified all children with a diagnosis of autistic disorder (children with other ASDs were not included in these analyses) enrolled in state developmental disabilities services. Children with ID but not ASD were also identified and selected for participation. According to this study the rate of autism in California averaged 11 per 10,000 for the years 1987 through 1994. Prevalence was 5.8 per 10,000 in 1987 and 14.9 per 10,000 in 1994. The rate of autism diagnoses showed a marked increase from 1990-1992 and leveled off from 1993-1994. Increases in diagnoses were not different when comparing males and females, twins and singles, ethnicity, or maternal age. Interestingly, the rate of ID of unknown origin decreased during this time period from 28.8 per 10,000 to 19.5 per 10,000. The authors

initially contributed the differences in prevalence in these two groups partially due to diagnostic substitution. However, the authors retracted this conclusion due to questioning of the methodology used in the study as referrals of children with ASD and those with idiopathic ID typically occur at different ages between these two groups (Croen & Grether, 2003). Nonetheless, the study showed a marked increase in autism diagnoses in a short time period.

While Croen and colleagues could not conclude that diagnostic substitution contributed to the rising prevalence in ASD, it is still hypothesized as a plausible explanation (Rutter, 2005; Wing & Potter, 2002). Using a Canadian special education population, Coo and colleagues (2008) investigated the hypothesis that the increase in ASD diagnoses was a result of diagnostic substitution. That is, children who were once classified as ID were subsequently identified as having an ASD. The authors reported that ASD diagnoses in children between the ages of 4 and 9 years increased 3.5 fold from 1996 to 2004. The authors attributed a large proportion of the increase (45%) to undetected cases. The authors also reported that approximately 51.9% of the increase in ASD diagnoses was attributed to diagnostic substitution. In addition to the better identification of these children, other children moved to the ASD category from other developmental disability classifications.

Recent studies of ASD prevalence rates indicate that these disorders are common in childhood. However, caution should be taken when extrapolating prevalence rates from epidemiological studies. Recruitment methods differ between studies, as does diagnostic methodology and criteria. Particularly, studies using data from education sources and those from psychology-based clinic studies may differ in classification

systems. Special education trends tend to under-estimate the prevalence of this group of disorders and should be cautioned for use as prevalence estimates at this time (Shattuck, 2006).

Using more recent studies, the prevalence of all ASD combined is approximately 60-65 per 10,000 people (Chakrabarti & Fombonne, 2001; Fombonne, 2005; Nicholas, et al., 2008). Fombonne (2005) conducted a review of epidemiological studies of ASD to date. Thirty-four studies were identified that studied the prevalence of autistic disorder. The mean prevalence was 8.7 per 10,000. However, more recent estimates place the disorder at much higher rates in the population, at about 13 children per 10,000 (Fombonne, 2005). Asperger's disorder is a relatively new diagnosis in the DSM; therefore prevalence estimates are few (Fombonne, 2005). Fombonne's review estimates that Asperger's disorder is more rare than autistic disorder, and may occur at rates one-fourth that of autistic disorder (Fombonne, 2005). Other estimates place the disorder between 0.3 to 48.4 per 10,000, with estimates likely to be about 2 per 10,000 (Fombonne, 2001). With regard to PDDNOS, estimates average at about 15 per 10,000 (Fombonne, 2003). Very few studies exist on the prevalence of CDD. Based on a limited number of studies, CDD is estimated to occur at 1-2 per 100,000 children (Fombonne, 2002, 2005). Rett's disorder is another very rare disorder with prevalence estimates at less than 1-2 per 10,000 girls (Leonard, Bower, & English, 1997; Skjeldal, von Tetzchner, Aspelund, Aas Herder, & Lofterød, 1997).

Assessment/Diagnosis

The first measure used to assess ASD can be credited to Bernie Rimland (Rimland, 1968). *Rimland's Diagnostic Checklist for Behavior-Disturbed Children* is a

76-item parent completed questionnaire that focused on the first five years of life and inquired about birth and the development of symptoms. While Rimland's measure was not well psychometrically established, the development of his measure initiated the expansion of other measures to assess ASD.

The *Autism Behavior Checklist* (Krug, Arick, & Almond, 1980) is an early instrument developed to screen symptoms of autism in children as part of an educational assessment battery. The 57-item parent or teacher report measure consists of five subscales: sensory, relating, body and object use, language, and self-help. Split-half reliability is reportedly good (Krug, et al., 1980). However, validity studies have conflicted. Using discriminant analysis and a measure of academic skills, Teal and Weiber (1986) were successful in identifying 100% of the children with autism in their study. However, Volkmar and colleagues (1988) reported only 57% of the children with autism in their study were correctly diagnosed. Due to its questionable psychometrics, the ABC has fallen out of favor (Matson & Minshawi, 2006).

A popular measure used to assess ASD in children is the *Childhood Autism Rating Scale* (CARS) (Schopler, Reichler, DeVellis, & Daly, 1980). The CARS was developed to differentiate between children with ASD and those with ID for use in an educational program for children with disabilities. The CARS consists of 15 subscales: relating to people; imitation; emotional response; body use; object use; adaptation to change; visual response; listening response; taste, smell, and touch response and use; fear or nervousness; verbal communication; nonverbal communication; activity level; level and consistency of intellectual response; and general impressions. The measure is scored according to information gathered from parent interview and observation of the child.

Each subscale is rated on a 4-point scale, ranging from "1" indicating typical for the child's age to "4" indicating severely abnormal for the child's age. A score of 30 and above indicates that the child falls in the autistic range. Reliability is reportedly good with inter-rater agreement of .71. Furthermore, test-retest at 12 months yielded non-significant changes in scores (Schopler, Reichler, Renner, & Services, 1988). Validity studies have also reported high rates of correct classification of children with ASD (Schopler, et al., 1988; Teal & Wiebe, 1986). The CARS recognizes a spectrum of severity with regard to autism; but reportedly does not allow for other diagnoses along the ASDs other than autistic disorder. However, more recent studies using the CARS has indicated that the measure is able to distinguish between children with autistic disorder and PDDNOS (Stella, Mundy, & Tuchman, 1999).

Considered the "gold-standard" in the diagnosis of ASD is the *Autism Diagnostic Interview-Revised* (ADI-R) (Lord, et al., 1994). The ADI-R is a revision to the *Autism Diagnostic Interview* (Couteur, et al., 1989), allowing for assessment of children under the age of 5 years and shortening length the interview, which were limitations of the original measure. Parents and caretakers serve as informants. The interview is aligned with the DSM-IV-TR (APA, 2000), and assesses socialization, communication, and repetitive behaviors and restricted interests. Inter-rater reliability is good with correlations ranging from .62 to .89 (Lord, et al., 1994). While the ADI-R is a comprehensive assessment of autism symptoms, it is quite lengthy and relies solely on parent/caretaker report (Matson, 2007).

A companion tool to the ADI-R is the *Autism Diagnostic Observation Schedule* (ADOS) (Lord, Rutter, DiLavore, & Risi, 2000). Lord and colleagues developed this

assessment tool to serve as an observation-based assessment of social-communication symptoms consistent with ASD. The measure consists of different modules based on the child's age and verbal ability. The child is presented with probes and placed in certain situations to evoke social and communicative behaviors. The examiner rates the child on each item as "within normal limits," "infrequent or possible abnormality," or "definite abnormality." Diagnostic classifications based on the ADOS are based on DSM IV/ICD-10 classifications. Reliability analyses reveal excellent internal consistency and adequate inter-rater and test-retest reliability (Lord, et al., 2000). One potential limitation of the ADOS is that it only focuses on social and communicative behaviors, and not repetitive behaviors and restricted interests. Therefore, it is possible for an individual to meet criteria for a diagnosis of autistic disorder according to the ADOS, yet not meet the criteria for diagnosis using strict DSM-IV-TR (APA, 2000) criteria.

A relatively new tool used to assess symptoms of ASD in children is the *Autism Spectrum Disorders-Diagnostic for Children* (ASD-DC) (Matson, Gonzalez, Wilkins, & Rivet, 2008). This measure is one component of a larger battery that also assesses comorbid psychopathology and problem behaviors in children ages 3 to 18 years. The 37-item, informant based measure is completed by a parent or caretaker who knows the child well and is able to report on their social interaction and communication skills, as well as, behavioral excesses. The informant is instructed to rate each item by comparing their child to typically developing children, according to a likert-type scale: '0' (not different; no impairment), '1' (somewhat different; mild impairment), or '2' (very different; severe impairment). Three empirically derived factors, consistent with current literature on ASD, were established (Matson, Boisjoli, & Dempsey, in press). Reliability

analyses have revealed excellent internal consistency, and coefficients of .67 and .77 for inter-rater and test-retest reliability, respectively (Matson, Gonzalez, et al., 2008). The ASD-DC also exhibits good sensitivity and specificity with regard to differentiating between children with ASD and those with atypical development (e.g., Down's syndrome, ADHD) and typical development. Furthermore, the measure also correctly classifies along the autism spectrum at a high rate (Matson, González, & Wilkins, 2009).

Other tools have been designed to assess symptoms of ASD in much younger populations. The aforementioned measures typically assess children above the chronological age of 2 years, at the youngest. With the vast amount of empirical support on the effectiveness of early intensive intervention, identifying ASD symptoms in very young children is paramount. Researchers have attempted to develop measures to screen and assess this very young group of children.

One such measure is the *Checklist for Autism in Toddlers* (CHAT) (Baron-Cohen, 1992). The tool was designed for use by the pediatricians at the child's 18-month well-baby visit. The screener is both informant and observation based. The pediatrician or home health nurse asks the parent to respond yes/no to nine questions and then the pediatrician answers five of the items based on observation. The items address pretend play, joint attention by pointing, and monitoring of gaze. Studies on the measure have revealed low sensitivity and high specificity (Baron-Cohen, 2000). Sensitivity and specificity were improved when used with older children (Scambler, Rogers, & Wehner, 2001).

The *Modified Checklist for Autism in Toddlers* (M-CHAT) (Robins, Fein, Barton, & Green, 2001) is a revision to the original CHAT and was designed for use in the United

States. An observational component of the CHAT was eliminated on the M-CHAT. For administration of the CHAT in England, a home health nurse visits the family's home, which is customary with British healthcare. However, as there is no equivalent to this service in the United States, the authors of the M-CHAT decided to eliminate the observation portion and add more items. The M-CHAT was originally designed as a Level I screener, which is intended to be used with the general population. However, the measure has been employed as a Level II screener, for use with a subset of a sample that may already be considered at risk for a developmental disability. The M-CHAT appears to be valid in identifying children with ASD in an at-risk sample (Kleinman, et al., 2008; Robins, et al., 2001; Snow & Lecavalier, 2008). Cut-off scores for the M-CHAT include failure of 2 of the 6 critical items or failure of any 3 items. However, in a sample of children with ASD or other developmental disabilities, the cut-off of any 3 items on the measure was optimal with regard to correct classification (Snow & Lecavalier, 2008). According to the study, 70% of those children identified as at-risk for ASD according to the M-CHAT were diagnosed with ASD at follow-up evaluations. Another study found that between 73.9% and 78.5% of a sample of toddlers considered high-risk for developmental disabilities (were already receiving early intervention services) failed the screener and went on to meet criteria for a diagnosis of ASD (Pandey, et al., 2008).

A new assessment battery designed to assess symptoms of ASD in very young populations is the *Baby and Infant Screen for Children of aUtistIc Traits* (BISCUIT) (Matson, Wilkins, et al., 2008). This battery is a companion to the ASD-DC but for younger children. While the ASD-DC assesses children over the age of 3 years, the BISCUIT assesses children from 17-37 months. In addition to assessing symptoms of

ASD (BISCUIT-Part 1), the battery also assesses for symptoms of commonly occurring comorbid psychopathology (BISCUIT-Part 2), and problem behaviors (BISCUIT-Part 3). The BISCUIT is read to a parent or guardian by a mental health services professional. Similar in format to the ASD-DC, the informant is instructed to rate each item by comparing their child to typically developing children, according to a likert-type scale: '0' (not different; no impairment), '1' (somewhat different; mild impairment), or '2' (very different; severe impairment). Psychometric studies are underway, with initial studies reporting excellent reliability (Matson, Wilkins, et al., 2008) and validity (Matson, et al., in press).

Classification Systems

The current system used in the mental health field to classify psychological disorders is the DSM-IV-TR (APA, 2000). This tool is categorical in nature, with diagnoses indicated when a certain number and/or combination of symptoms are present. While the DSM-IV-TR (APA) provides a caveat regarding its use of categories, acknowledging that psychological disorders may actually be dimensional constructs, the DSM-IV-TR (APA) takes a categorical approach to mental disorders. That is, the disorder is either present or absent, with no gradient between. This categorical approach to psychopathology is beneficial in some respects, such as it efficiently provides a large amount of information through the use of a single term, aids in clinical decision making, assists with identifying rare conditions, and is consistent with the tendency for humans to inherently categorize phenomenon and preference for this organizational strategy (Klein & Riso, 1993). However, this approach also has its weaknesses (Klein & Riso, 1993).

The DSM-IV-TR (APA, 2000) evolved through the efforts of researchers and clinicians in an attempt to increase the reliability of the classification system in earlier editions. Prior to the DSM-III (APA, 1980), editions of the DSM had a psychoanalytic focus to diagnosis. Manifestations of disorders were oftentimes ambiguous, leading to low reliability of diagnoses. In response, subsequent revisions employed observable symptoms as criteria for diagnoses, and thus, increased reliability was noted. With this increased reliability, validity of diagnoses may have suffered (Carson, 1991). That is, by only including symptoms that are reliably observed as criteria, important components of the disorder may be missed (Schmidt, Kotov, & Joiner, 2004).

Another criticism of the categorical approach of the DSM-IV-TR (APA, 2000) deals with sub-threshold impairments. For disorders of this classification system, a person must be experiencing a certain number of symptoms in order to meet criteria. However, if the person is experiencing just one less symptom than is necessary for a diagnosis, yet experiencing debilitating effects, psychopathology may not be indicated (Maser, et al., 2009). This classification issue has implications with treatment and research on etiology. Is the underlying structure of the disorder any different if one less symptom is present? Furthermore, delineation of criteria for the DSM is not entirely based on empirical studies, but instead from consensus of a committee (Schmidt, et al., 2004). The validity of some DSM-IV-TR (APA, 2000) diagnoses is questionable and requires further investigation (Schmidt, et al., 2004).

The validity of ASD diagnoses has also been questioned. Field trials of the autistic disorder diagnosis were conducted for the DSM-IV (Volkmar et. al, 1992). Through the use of multivariate statistical analyses, the structure of ASD may be different from the three symptom clusters outlined in the DSM-IV-TR (APA, 2000). Factor analytic studies have been conducted to investigate the underlying structure of ASD. While many of these studies form factors that are similar to criteria in the DSM-IV-TR (APA, 2000), there are differences. Such as a 3-factor solution with two of the factors representing social-communication and the third representing verbal communication, with repetitive behavior items weakly correlated (Bolte & Poustka, 2001). Factor analysis of the CARS (Schopler, et al., 1988) revealed the following factors: social communication, emotional reactivity, social orienting, cognitive and behavioral consistency, and odd sensory exploration (Stella, et al., 1999). Other factor analytic

studies have revealed factors representing nonverbal communication/socialization, verbal communication, social relationships, and insistence on sameness/restricted interests (Matson, et al., in press). When just looking at the behavior domain of the ADI-R, different factors for repetitive sensory motor actions and resistance to change emerged (Cuccaro, et al., 2003). In addition to the questionable validity of the symptom clusters of ASD, the validity of the different disorders subsumed under the Pervasive Developmental Disorder category of the DSM-IV is also debatable. That is, validity studies of Asperger's Disorder and PDD-NOS have revealed inconsistency with diagnoses and limited ability to discriminate across the disorders. Results of these studies lend to the need for further validity studies of the classification system of ASD in the DSM-IV-TR (APA, 2000). Currently, proposed revisions to the upcoming DSM-V include disbanding the Asperger's Disorder and PDD-NOS diagnoses (APA, 2010). Instead this category will only include one diagnosis label, *Autism Spectrum Disorder*, using qualitative specifiers of severity.

Conversely, another criticism of the DSM-IV (APA, 2000) and its categorical nature is that it may over-pathologize normal behavior. That is, some behaviors may fall on a continuum of normalcy and therefore a cutoff designating pathology is arbitrary. Studies on the genetic influence on behavior have noted that some disorders fall on the extreme end of normal behavior, on a continuous dimension (Andersson & Ghaderi, 2006). Rather than designate a person as disordered, symptoms may be better described in terms of severity, such as borderline, mild, or severe (Plomin, Owen, & McGuffin, 1994; Rounsaville, et al., 2002). Dimensional classification systems have received more

attention in recent years with the push for the upcoming fifth edition of the DSM to use a combination of a categorical and dimensional approach to diagnosis (Maser, et al., 2009).

One benefit to using a dimensional model, as opposed to a categorical model, for diagnosis is that clinical utility increases. A study by Samuel and Widiger (2006) assessed the clinical utility of assigning diagnoses according to a categorical system or a dimensional system. According to this study, a dimensional approach to diagnosis allowed for better communication of information to clients, encompassed more of the client's current difficulties, and was more beneficial in assisting the clinician with treatment formulation. The use of a dimensional approach to diagnosis allows for more specific and individualized information on the person's difficulties. Instead, the high usage of the 'Not Otherwise Specified' diagnoses may be due to the lack of clinical utility and coverage of diagnoses by the current classification system (L. Clark, Watson, & Reynolds, 1995).

The use of categorical or dimensional systems for diagnosis has strengths and weaknesses. The use of broad categories is conducive to service allocation and communication among clinicians and researchers, and less conducive to research related activities (Volkmar, 1998). Dimensional models of description are more conducive to genetic studies (Lecavalier, et al., 2009). As for now, the use of categories to classify disorders will most likely remain the dominant approach; however, it is important to understand disorders with regard to their variation and relationship to normal behavior (Cantwell & Rutter, 1994). Furthermore, the investigation of the underlying structure of a disorder is necessary for adequate definitions. Only through adequate description of a

phenomenon can laws be generated to explain, predict, and scientifically understand (Hempel, 1961).

Classification of ASD

For many years, ASD has been understood to be a discrete diagnosis, with boundaries between the disorder and normal functioning, as well as between the subtypes of the disorder (Rutter & Schopler, 1988). More recently, with the advances in technology in the identification of symptoms of ASD, genetic studies, and evidence supporting a broader autism phenotype, ASD is beginning to be conceptualized by some researchers and clinicians as a dimensional disorder, without clear boundaries (Baron-Cohen, et al., 2001; Constantino & Todd, 2003). Determining whether particular symptoms of ASD are categorical or dimensional is important for a number of reasons. That is, accurate definitions of a disorder have broad implications with regard to etiological identification, treatment planning, and treatment efficacy (Sevin, et al., 1995).

Categories. It is widely accepted that individuals affected with ASD are a heterogeneous group that vary with regard to cognitive functioning, co-occurring psychopathology, challenging behaviors, and severity of autistic symptoms. Studies have been performed to determine if there are different subtypes of ASD to which classifications can be made. Through empirical studies, the majority using cluster analysis, the most common subtyping methods are based on social/communication characteristics, intellectual/adaptive functioning, medical conditions, or a combination of the above subtypes (Borden & Ollendick, 1994; Donnelly, 1996; Eaves, Ho, & Eaves, 1994; Prior, et al., 1998; Stevens, et al., 2000; Tager-Flusberg & Joseph, 2003; Volkmar, Cohen, Bregman, Hooks, & Stevenson, 1989; Waterhouse, et al., 1996). Researchers

have reported that there are distinct groups of children with ASD based on language, and nonverbal and verbal discrepancies on tests of intelligence (Tager-Flusberg & Joseph, 2003). Other researchers have found two overlapping groups based on social functioning and cognitive and adaptive skills: 1) other PDD/active-but-odd, and 2) autistic/aloof. Children in the first group had higher cognitive and adaptive functioning with fewer ASD symptoms when compared to the second group (Waterhouse, et al., 1996).

A seminal study was conducted on December 31, 1970 in the London borough of Camberwell. Wing and Gould (1979) sought to identify all children 14 years of age and younger with social and communication deficits in this particular region on that day. Consistent with earlier studies on ASD, these researchers reported that children with deficits in socialization also experienced deficits in communication and imagination, and exhibited restricted interests. However, most notably, Wing and Gould (1979) found that the characteristic impairments of the children they studied occurred at varying severities: that is, the symptoms occurred on a continuum and were not discrete entities. The authors broke down social impairment into three categories. The first group was termed the "aloof group" and was characteristic of more severe forms of autism and what some refer to as "classic autism." These children tended to isolate themselves socially and reject approaches from others. Children in the aloof group also tended to have the most impaired verbal and non-verbal communication. Furthermore, imagination was non-existent in this group and play behavior was repetitive in nature. The "passive group," was comprised of children who tended to be diagnosed later in life compared to the aloof group. These children welcomed social approaches by others but failed to initiate social interaction themselves. Communication varied in this group, with some having

abnormalities with intonation, failure to use language for social purposes, or only talking about restricted topics. Like the aloof group, these children tended not to engage in imaginary play, but imitated the play of other children. These children engaged in rituals and insisted on sameness; however, not to the extent of the aloof group. Lastly the "active-but-odd" group was considered the least impaired of the three groups. These children initiated contact with others but in a socially odd and overly forward manner. Verbal abilities were often good, and sometimes even excellent; however, pragmatics were often lacking. These children may have exhibited stereotyped movements as young children but these behaviors faded with time. Repetitive behaviors and restricted interests in this group were often elaborate and abstract.

Sevin and colleagues (1995), using cluster analysis, identified four subgroups of ASD. The first was labeled 'atypical PDD' and included individuals that experienced the least severe impairments with regard to ASD symptoms, had normal to mild/moderate impairment in intellectual functioning, and exhibited an 'active-but-odd' style of socialization. The second group consisted of individuals that were experiencing mild autism. This group exhibited more impairment in ASD symptoms when compared to the atypical PDD group, had mild/moderate deficits in intellectual functioning, and had a passive style of social interaction. The third group consisted of individuals experiencing moderate autism. This group displayed more ASD symptoms and deficits in intellectual functioning in the severe range. The last cluster consisted of individuals with severe autism. They displayed extensive ASD symptoms, severe impairment in intellectual functioning, and were described as having an 'aloof' social interaction style. Based on the Wing and Gould's (1979) subtypes of autism and intellectual functioning, Sevin and

colleagues were able to identify subgroups within the autism spectrum. Wing and Gould's subtypes of ASD have received much attention and have been validated in numerous studies (Borden & Ollendick, 1994; O'Brien, 1996; Volkmar, et al., 1989; Waterhouse, et al., 1996).

In addition to subtypes of children based on social functioning, medical conditions, or cognitive/adaptive functioning, there appears to be two other groups of children within the diagnosis of ASD: those who exhibit abnormalities since infancy and another group who experiences regression in skills during the second year of life (Maestro, et al., 2005; Werner & Dawson, 2005). The infants who later experience regression, may engage in joint attention, word usage, and babbling at similar rates to typically developing children. However, by 24 months the children who regressed were more similar to other children with ASD in the areas of socialization and communication than to typically developing children. By 3-4 years of age, groups of children with ASD who experienced regression and those who displayed abnormalities very early on in life, did not differ in cognitive functioning or behavioral symptoms (Werner, Dawson, Munson, & Osterling, 2005). However, even in children who are thought to have regressed, there is some evidence of disturbances of regulatory behaviors, such as sleeping and sensitivity to sensory stimuli, early in life (Werner & Dawson, 2005).

The validity of the subtypes of ASD (i.e., autistic disorder, Asperger's disorder, and PDDNOS) has been called into question (Witwer & Lecavalier, 2008). Many studies that examine differences between the different subtypes of ASD use different criteria to classify the disorders. Secondly, many studies do not report on IQ, which could affect symptom expression. The studies that do exist on the validity of subtypes of ASD have

found differences on children's scores according to severity; however, categorical differences between these groups have not been implicated (Witwer & Lecavalier, 2008).

Dimensions. In addition to the introduction of subtyping, Wing and Gould (1979) were of the first to propose that symptoms of ASD occur on a continuum of severity. However, the term "spectrum" was used over "continuum" because a continuum refers to an even succession across the different disorders, while spectrum implies that the progression along the disorders may not be seamless (Wing, 2005). The authors felt a "spectrum" was more representative of the underlying structure of ASD.

The studies on twins by Rutter and Folstein (1977) pointed to a range of deficits associated with ASD. The early twin studies revealed a high concordance rate for autism in monozygotic twins and even a higher concordance rate when siblings with less severe impairments in social functioning and communication were included. Investigations of family members of people with ASD, in addition to twins, have also found deficits in socialization, communication, and repetitive behaviors but at less severe levels (Szatmari, et al., 2000).

Sub-threshold symptoms of ASD are referred to as the broader autism phenotype (Piven, et al., 1997). This is described as characteristics consistent with ASD, yet not of significant severity to cause impairment, and therefore no diagnosis is given. The broader autism phenotype has been found in family members of children with ASD, as well as widely distributed throughout the general population (Skuse, Mandy, & Scourfield, 2005). Studies are currently being undertaken to identify the characteristics of this broader phenotype; however, to a much lesser extent than studies attempting to identify the subtypes of ASD (Volkmar, State, & Klin, 2009). These qualitatively similar

characteristics of ASD (though in a milder form) are also observed in grandparents and aunts and uncles of children with ASD (Piven et al. 1997). Furthermore, parents of children with ASD have significantly higher rates of alexithymia (lack of understanding for own emotional responses) and experience difficulties with phonological processing (Szatmari, et al., 2008). The *Social Responsiveness Scale* (Constantino, 2002) is a measure of social deficits consistent with ASD. Using this measure Constantino and Todd (2006) found evidence of a familial transmission of autistic traits. The authors reported parents' elevated scores on the measure were related to higher scores of their children on the measure. Furthermore, when both of parents' scores fell in the upper quartile of scores, the child's scores were 1.5 standard deviations greater than children whose parents' scores fell in the normal range. As symptom severity varies within the range of normal behavior, the broader autism phenotype supports the position of ASD being a dimensional phenomenon (Rutter, 2005a).

Taxometrics

The use of empirically derived classification systems for mental disorders is relatively new in comparison to other sciences. Studies investigating the underlying structure of a psychological disorder have used statistical techniques such as cluster analysis, latent class analysis, and investigating distributions for bimodality. While each of these analyses is useful in the identification of the structure of a dataset, they may not be the optimal method for distinguishing categorical and dimensional phenomenon. That is, with both cluster analysis and latent class analysis, clusters or groups are formed with the data, therefore dimensionality will not be observed if present. As for bi-modality, unless there is a large difference between the two groups, the distribution may appear uni-modal, and wrongly leading the researcher to the conclusion that the data is composed of only one group (Schmidt, et al., 2004).

Increasing in popularity is a statistical procedure referred to as coherent cut-kinetics or taxometrics. Paul Meehl (1995) developed these techniques in attempt to identify a discrete category of schizophrenia. Although Meehl began working on these statistical procedures more than 50 years ago, the past 5 years has seen a dramatic increase in the number of studies published using taxometric analyses (Walters & Ruscio, 2009). In addition to identifying the latent structure of schizophrenia (Blanchard, Horan, & Collins, 2005; Cuesta, Ugarte, Goicoa, Eraso, & Peralta, 2007), these methods have also been employed to study the latent structure of various other constructs such as nicotine addiction (Goedeker & Tiffany, 2008), malingering (Walters, et al., 2008), eating disorder (Gleaves, Lowe, Green, Cororve, & Williams, 2000), separation anxiety (Silove, et al., 2007), and depression (J. Ruscio & Ruscio, 2000; Slade, 2007).

The purpose of Meehl's taxometric analyses is to investigate the underlying structure of a construct and determine if a taxon truly exists within the data. The taxon is composed of participants who experience characteristics of the construct under study. A true taxon is one that is natural and not arbitrary (Schmidt, et al., 2004). The complement is composed of participants who do not express symptoms consistent with the target construct. Additionally, taxometric analyses analyze whether the latent structure is continuous in nature.

One important reason to identify a taxon is for classification purposes. That is, the characteristics of the individuals who belong to that group can be studied, with the most salient characteristics having implications for the refinement of diagnostic criteria (J. Ruscio, Haslam, & Ruscio, 2006). Furthermore, identifying if a disorder is taxonic or dimensional may result in a reevaluation of the system currently in use for that disorder and diagnostic algorithms may be modified (J. Ruscio, et al., 2006). The use of these analyses will assist in potentially identifying subgroups of a sample, with implications for investigating different etiologies, and perhaps different interventions (Ingram, Takahashi, & Miles, 2008). By using a dimensional approach to diagnosis, individual and specific characteristics can be considered, particularly for the identification of treatment modalities (Widiger & Samuel, 2005).

Indicator Variables

When conducting taxometric studies, the researcher selects indicator variables that will be used in the analyses. Appropriate indicators for evaluating latent structure must be composed of the critical components of the construct, as well as only target the construct of interest and not another phenomenon. That is, the indicators must have good

content and discriminant validity. Indictors can be selected through consideration of theoretically and/or empirically derived conceptualizations of a construct. For example, indicators may represent DSM-IV-TR (APA, 2000) criteria or be derived from empirical studies such as exploratory factor analysis. Therefore, multiple indicator sets can be developed from a dataset.

Indicators should be evaluated prior to analyses to determine appropriateness (Ruscio et al., 2006). That is, to properly implement taxometric analyses, valid indicators are necessary. Indictor validity is determined by its ability to discriminate the taxon from the complement of a sample. The taxon and complement should be separated by an effect size of at least $d = 1.25$ (Meehl, 1995). Appropriateness of a particular set of indicators is also related to the correlations between the different indicators that will be used in the analysis. Ruscio, Haslam, and Ruscio (2006) suggest theoretically identifying the different components of a construct. From there, a composite of items can be generated to form an indicator variable of that particular component of the construct. When correlations between the indicators are large, this is referred to as nuisance variance and is not desirable at very high levels. Correlations of $r = .00$ are ideal. However, this criterion may be difficult to achieve, and $r < .30$ has been found to be acceptable (Meehl, 1995). Furthermore, correlations between indicators for the taxon group and complement group should be lower than correlations between the indicators for the entire sample. In order to ensure low correlations among indicators, the researcher is able to construct indicators through consideration of correlations. That is, indicators that represent the construct being studied are selected due to their independence from one another (J. Ruscio et al., 2006). In the selection of indicator variables, more than two indicators are

desirable. However, too many indicators may introduce redundancy and lead to unacceptable levels of within group correlations.

Measurements of normality are not a requirement for taxometric analyses; however, extensive skew may cause difficulty with the interpretation of results. Yet, psychological constructs commonly demonstrate a non-normal distribution (Micceri, 1989; Rojahn, Matson, Lott, Esbensen, & Smalls, 2001) and some skew is common, especially when the taxon group is small. Skew up to 2.0 should be accommodated by taxometric analyses with little caution for interpretation (Beauchaine, Lenzenweger, & Waller, 2008). Excessive skew, at or above 2.0, should be interpreted with caution and researchers may decide to employ comparison data for assistance with interpretation of graphs (A. Ruscio & Ruscio, 2002). Researchers need to be aware that skewed distributions may exert influence on the shape of the curves and therefore interpretation of results (J. Ruscio, et al., 2006).

Suitability Analyses

Ruscio and colleagues (2006) developed a procedure to evaluate the suitability of data for a taxometric analysis using empirical sampling distributions. These analyses assist the researcher in determining the taxometric method that is optimal for the given data. Analyses are conducted with simulated datasets that have the same parameters as the research data, with one of the simulated datasets having a taxonic structure and the other having a dimensional structure. The plots that result from the analyses are investigated and the analyses yielding the most interpretable plots are selected for use with the research data.

Statistical Analyses

A number of statistical methods exist for taxometric analysis of underlying latent structure of a construct. The methods are statistically different from the others as to avoid redundancy in the analyses and assist in the confirmation of structure. Mean Above Minus Below a Cut (MAMBAC) is a taxometric analysis method based on the premise that if there is more than one group within the sample, then there will be an optimal "cut" point between the groups. That is, there will be a point that will be able to distinguish if more than one group exists. Conversely, if an optimal score is not found, then latent structure may be dimensional. One of the indicators is designated as an input indicator and the other as an output indicator. Cuts along the input indicator are made at predetermined points, and mean scores of the output indicator are calculated for those above and below the cuts on the input indicator. The mean below and mean above are then subtracted and plotted on a graph along the y-axis, with cut scores presented along the x-axis. The larger the difference in scores and the higher the peak on the graph, the more likely it is to be a taxonic structure. When a non-taxonic structure is present, there will be no peak and the plots may be concave in shape.

Another commonly used taxometric technique is the MAXimum COVariance (MAXCOV). MAXCOV is based on the General Covariance Mixture Theorem (GCMT) (Waller & Meehl, 1998). This theorem is based on the partitioning of the covariances of a mixed sample (taxon and complement). Similar to MAMBAC, MAXCOV is also based on cut-kinetics. The covariance of two indicators is calculated along successive cuts of a third indicator variable. The graph consists of the covariance plotted along each of the cuts. Graphs representing a taxonic structure tend to peak. While, graphs representing a

dimensional structure are flat and do not display a peak. The shape of the graph is dependent on the covariance of the indicators. That is, with taxonic structure, indicators are not highly correlated in either the taxon or the complement, but are highly correlated at points where the two members are mixed equally, resulting in a peaked shape. Conversely, when a dimensional structure is present, the covariance should be relatively constant, yielding plots that are flat.

L-Mode is a statistical procedure that investigates the structure underlying a construct. L-Mode, unlike the other statistical analyses in taxometrics, is based on factor analysis. The analysis is guarded to just one factor. The factor scores, like the other analyses, are plotted and inspected. Plots with a bi-modal distribution are suggested to be taxonic, whereas those that are uni-modal are suggested to have a dimensional structure.

MAXimum EIGenvalue (MAXEIG) is an analysis similar to MAXCOV, but using multivariate analyses (Schmidt, et al., 2004). This method also cuts intervals along the indicator variable. The difference is that MAXEIG uses overlapping intervals, so participants can be included in more than one interval. Eigenvalues are computed for each window and plotted. Like MAXCOV, the plots of a taxonic structure are peaked and plots of a dimensional structure are flat. Conceptually, MAXEIG and MAXCOV are similar analyses; therefore, researchers should not conduct both procedures in a taxometric analysis due to redundancy. MAXEIG may be more suitable for use with positively skewed data than MAXCOV as positively skewed data may present as taxonic when the underlying structure is dimensional. This is due to the larger number of data points produced with MAXEIG than compared to MAXCOV. By increasing the data points, the curves become easier to interpret (J. Ruscio, Ruscio, & Keane, 2004).

Interpretation

The interpretation of results from taxometric analyses has one of three possible outcomes (Ruscio et al., 2006). The first, being the most ideal, is that the data produce results that are consistent with either a continuous or taxonic structure. The second and third possible outcomes involve ambiguity with interpretation. The second pattern of results exists when the plots are consistent with both a dimensional and taxonic structure. The third possible pattern for interpreting the data occurs when the results are not consistent with either a taxonic or dimensional structure. In both of these latter situations, the researcher abstains from interpreting the results as being either taxonic or dimensional. This lends to an important concept with taxometric analysis: the inferential framework is not best characterized as comparable to null hypothesis testing. Instead, taxonic and dimensional structures are perceived as being competing hypotheses (Schmidt, et al., 2004). With null-hypothesis testing, one rejects the null, which lends support for the alternative hypothesis. However, when investigating the latent structure of a construct, rejection of a taxonic structure would not imply the structure is dimensional, for example. As the pattern of results can be ambiguous, a competing hypothesis framework may be the best approach. That is, some data may appear to fall between a dimensional and taxonic structure, while other data may appear much different from both taxonic and dimensional structures (Riuscio, et al., 2006).

Consistency Testing

A hallmark to taxometric analysis is the use of consistency testing. The rational for consistency testing is to support findings through the convergence of results that the underlying latent structure is either taxonic or dimensional. Numerous, non-redundant

analyses exist to evaluate results. The best methods of interpretation of results are through the use of trained raters to interpret plots and the comparison curve fit index (CCFI) (J. Ruscio, Ruscio, & Meron, 2007). Trained raters are presented with plots along with comparison data. Raters are instructed to determine if the plots are most similar to either taxonic or dimensional comparison data. Percent agreement is calculated for ratings. The CCFI can be calculated to determine the degree of fit between the research data and comparison taxonic and dimensional data. Values of CCFI can range from 0 through 1 with scores approaching 0 indicating a dimensional structure and scores approaching 1 indicating a taxonic structure.

One consistency test is the nose-count test. This test counts the plots and determines the ratio of taxonic results to dimensional or ambiguous results. A ratio of 1:1, taxonic to dimensional data, is sufficient for indicating an underlying structure is taxonic, as false positives of taxonicity are uncommon (Schmidt, et al., 2004). The inchworm consistency test can be used with difficult to interpret results of MAXEIG. The number of cuts is increased; therefore, more overlapping windows that are smaller in size are produced. The results are plotted and taxonic structure takes the shape of a peak or an inchworm with a major crest to the right side (representing the head of the inchworm). Dimensional structures do not have a peak or crest (Schmidt, et al., 2004). Another consistency test involves the use of non-redundant taxometric methods and the plots inspected to determine underlying structure and convergence with other analyses. This allows for repeated and consistent results, as opposed to an isolated result that may be deceptive (Ruscio et al., 2006). The purpose of the base-rate variability test is to examine the consistency of base-rate estimates within the analysis. Schmidt and colleagues (2004)

proposed a cutoff score of .10 SD with base-rate estimates below this value implying a taxonic conjecture. The rationale is that taxonic structure would exhibit higher levels of consistency across the sub analyses, therefore exhibiting lower base rate estimates and standard deviations. Lastly, Goodness of Fit Index (GFI) is analyzed to determine the "fit" between the observed and predicted model. Schmidt and associates (2004) have provided estimates for determining fit consistent with latent structure. Values greater than .90 are consistent with taxonic structure and values less than .90 provide support for a dimensional structure.

Taxometric Analysis of Autism

To date there has only been two published studies employing taxometric methodology to ASD. Both studies only include participants with ASD and investigate subtypes based on different characteristics of the disorder. The first study was conducted by Munson and associates (2008). The study aimed at identifying subgroups of ASD based on cognitive functioning through latent class analysis and taxometric methods. Four hundred and fifty-six children between the ages of 24 and 66 months were included in the study. All children received an ASD diagnosis based on the *ADI-R* and *ADOS*. The *Mullen Scales of Learning* was used to identify intellectual functioning and form indicator variables. MAXCOV was conducted and the authors reported that 6 of the 12 plots had peaks, lending to the suggestion of taxonic conjecture. The authors of the study conducted a simplified version of taxometric analyses and therefore results should be interpreted with caution. That is, MAXCOV was the only analysis conducted and implementation decisions were not described. Additionally, plots were not presented in the paper for the reader to examine and the methods of interpretation were not described.

The second study, conducted by Ingram and associates (2008), investigated phenotypes in a group of children with ASD. Participants for the study were enrolled in a genetics research program for families with more than one person with ASD. The age of the participants averaged 8 years old and the sample was 78% male. Indicator variables were derived by theory and included social interaction/communication, insistence on sameness, repetitive motor activity, language acquisition, intelligence, adaptive behavior, and physical dysmorphology. The authors proposed ASD subtypes based on these seven subgroupings. MAMBAC and MAXCOV procedures were conducted, along with consistency testing. According to their results, subgroups of ASD exist according to social interaction/communication, intelligence, and physical dysmorphology. Conversely, insistence on sameness, repetitive motor actions, and language acquisition comprise a dimensional structure. A dimensional structure may also be characteristic of adaptive functioning; however, the results were conflicting for this variable. The authors state that it is taxometrically valid to subgroup ASD according to social interaction/communication, intelligence, and physical dysmorphology. While the authors were able to identify subgroups within the autism spectrum, they cautioned the generalization of results to other populations. Taxometric studies have yet to be conducted to determine if ASD is taxonic in non-ASD populations, such as populations with developmental disabilities. A large population sample is needed to determine if ASD is comprised of a taxonic or dimensional latent structure in various populations (Ingram, et al., 2008).

Purpose

The purpose of this study is to evaluate the latent structure of ASD in an at-risk population of toddlers. ASD are a group of disorders that are being diagnosed at very high rates. This increase in diagnosis may be due to a number of factors such as improved assessment techniques, increased awareness, broadening of criteria, and/or a true increase in the prevalence of the disorder (Wing & Potter, 2002). In order to begin to identify the reasons for the increase in ASD diagnoses, it is important to evaluate the underlying structure and current criteria for the disorder and develop assessment techniques that result in better precision with regard to diagnosis. Investigating the latent structure of ASD in all populations is an important undertaking. However, at this time understanding the symptom patterns in very young children at-risk for a developmental disability is of the high priority as this age group may garner the greatest benefit from intervention. Therefore, understanding the structure of this disorder in toddlers is warranted. Taxometric analyses can assist with defining the borders (if they exist) of a disorder and determining if the current classification system is over-inclusive or possibly under-inclusive (J. Ruscio, et al., 2006). More specifically, taxometric analyses may assist in determining if ASD symptoms form a discrete group in a population of individuals with developmental disabilities or represent a dimensional phenomenon in this population. Furthermore, by better defining the underlying structure of a disorder, more refined genetic studies may be possible.

Autism symptoms occur in various populations, and appear to be continuously distributed throughout the population (Hoekstra, Bartels, Verweij, & Boomsma, 2007).

Family members of people with ASD express characteristics consistent with ASD, but to a lesser extent. Individuals with certain genetic disorders (e.g. fragile X, tuberous sclerosis) are more likely to exhibit symptoms of ASD. Furthermore, ASD symptoms are present in the general population, which may vary in severity according to profession and gender (Baron-Cohen, et al., 2001; Hoekstra, et al., 2007). Based on the current research ASD, it is hypothesized that the latent structure of ASD symptoms in an at-risk sample of toddlers is consistent with a dimensional structure.

Method

Participants

One thousand, one hundred and forty nine toddlers participated in this study. For taxometric analysis, sample size should be at least 300 participants, with a minimum of 30 members from the taxon (Schmidt, et al., 2004). To conduct *a priori* analyses, a taxon group was required to be identified. Large-scale studies are not available at this time to estimate base rates of ASD in toddlers enrolled in early intervention programs. However, smaller studies (N < 700) have reported rates of ASD to be approximately 23-25% of a population of toddlers receiving early intervention services (Kleinman, et al., 2008; Pandey, et al., 2008). Using a conservative base rate estimate of ASD of 23% (Kleinman, et al., 2008), a sample of at least 132 participants is required; however, at least 300 participants is optimal. Twenty three percent of the participants scoring highest on the BISCUIT-Part 1 were identified as the taxon for *a priori* validity analysis purposes. The remaining 77% of the sample was designated as the complement.

Participants for this study were children enrolled in a state-funded early intervention program. Children ranged in age from 18 to 36 months. All children enrolled in the early intervention system have been identified as having a developmental disability or a medical condition likely to result in a developmental disability. Participants ranged in age from 18 to 36 months (M = 26.46, SD = 4.90) and 70.8% of the sample was male. Ethnic identification of the sample was as follows: 54.0% Caucasian, 39.8% African American, 1.9 % Hispanic, and 3.9% other ethnicity. Data on ethnicity were missing for 5.8% of the sample. Participants in the sample had medical conditions and developmental disabilities including asthma (6.27%), epilepsy or seizures (2.09%), allergies (3.22%),

prematurity (2.34%), Down's syndrome (1.91%), drug exposure in utero (0.70%), reflux (0.61%), chronic ear infections (6.88%), developmental delay (2.96%), failure to thrive (0.44%), stroke (0.44%), heart conditions (1.04%), and sickle cell anemia (0.53%). Nine percent of the sample had additional diagnoses including traumatic brain injury, hydrocephaly, genesis corpus callosum, chronic lung disease, cerebral palsy, eczema, and anemia. No additional diagnoses were reported for 61% of the sample.

Procedure

Parents/guardians served as informants for the interviews used in this study. Personnel qualified to provide services through the State of Louisiana's EarlySteps program conducted the interviews. EarySteps is Louisiana's Early Intervention System under the Individuals with Disabilities Education Act, Part C, which provides services to infants and toddlers and their families from birth to 36 months. Children qualify if they have a medical condition likely to result in a developmental delay, or have developmental delays. Assessors hold degrees ranging from bachelor to doctoral level and are licensed or certified in their respective discipline. Assessor disciplines include physical therapy, occupational therapy, speech language pathology, social work, education, and psychology. As part of the EarlySteps system, parents/caretakers are interviewed regarding their child's development from entrance into the program and every six months following, until discharge from the program at 36 months of age. In addition to measures on developmental milestones, children enrolled in the program also receive ASD screening. The BISCUIT battery and M-CHAT are administered to all children from age 18 months through 36 months. All assessors who administer the BISCUIT battery are

required to attend an 8-hour training on the administration of the measures and general information on ASD.

The BISCUIT-Part 1 is administered as part of the larger BISCUIT battery. In addition to the BISCUIT, the M-CHAT is also administered. Measures are read aloud by the assessor, while the informant reads along. Interviews are conducted in quiet areas, typically in the child's home or educational setting. Children are routinely present during the interviews, affording the opportunity for behavioral observations of the child by the assessor.

Measures

Baby and Infant Screen for Children with aUtIsm Traits (BISCUIT)-Part 1 is a 62-item measure that assesses symptoms of ASD in children between the ages of 17 and 37 months. The BISCUIT-Part 1 is part of a larger battery that also assesses for comorbid psychopathology and problem behaviors in very young children. The 62 items are rated on a 3-point Likert-type scale. Informants are instructed to rate each item by comparing their child to a typically developing child of the same age. Qualifiers for the ratings are: '0' (not different; no impairment), '1' (somewhat different; mild impairment), or '2' (very different; severe impairment). Additionally, an addendum of age appropriate qualifiers for each item is provided for the assessors, to aid in clarification of item meanings in a standardized manner. Three factors emerged through exploratory factor analysis: repetitive behavior/restricted interests, socialization/nonverbal, and verbal communication (Matson, Boisjoli, Hess, & Wilkins, 2010). Reliability analyses revealed excellent internal consistency ($\alpha = .97$) (Matson, Wilkins, et al., 2008). The BISCUIT-Part 1 distinguishes between children with ASD and children at-risk for a developmental

disability with an overall correct classification rate of 88.8 (sensitivity = 93.4; specificity = 86.6) using a cut-off score of 17 (Matson, et al., in press).

The *Modified Checklist for Autism in Toddlers* (M-CHAT) (Robins, et al., 2001) is a 25-item parent report questionnaire for children between the ages of 16 and 30 months. The measure was developed from the CHAT with intended use in the United States. Parents are instructed to rate each item as either 'yes' or 'no.' The items inquire about the presentation of certain behaviors, such as joint attention, reciprocal social play, sensory abnormalities, pretend play, and interest in socialization. Cut-off scores for the M-CHAT include failure of 2 of the 6 critical items or failure of any three items. However, in a sample of children with ASD or other developmental disabilities, the cut-off of any 3 items on the measure was optimal with regard to correct classification (Snow & Lecavalier, 2008).

Taxometric Analyses

Taxometric analyses were employed to identify the latent structure of ASD symptomatology across a young at-risk sample. The R-language (J. Ruscio, 2004) computer program was utilized to analyze data. Potential indicators were derived from items of the BISCUIT-Part 1 and the M-CHAT. From there, suitability analyses were conducted to determine the optimal procedures considering the parameters of the research data. Lastly, taxometric analyses and consistency testing were conducted.

Indicator Selection. Indicators were selected based on current empirical studies of ASD in young children. Items were selected from the BISCUIT-Part 1 and the M-CHAT. Initially, the three empirically derived factors of the BISCUIT-Part 1 and the total score of the M-CHAT were evaluated to determine validity of the indicators. Items from

BISCUIT-Part 1 factor analysis (Matson, et al., 2010) loadings of 0.50 or greater were included in this analysis. Due to the need for independent indicators for a taxometric analysis, items with higher loadings (better measure of the respective factor) were selected for use in the present study.

Items on each of the three BISCUIT-Part 1 factors were summed to form a composite score for each factor. To optimize power, *a priori* analyses were conducted to ensure indicators were valid. Prior to taxometric analyses, the assumption was made that a taxon exists. From there, base rate estimates were used to identify a homogenous group of the distribution of participants (A. Ruscio & Ruscio, 2002). That is, 23% (an estimate of the rate of ASD in an at-risk sample (Kleinman, et al., 2008)) of the participants scoring highest on the BISCUIT-Part 1 were identified as the taxon for validity analysis purposes. Using the taxon sample, within group correlations were conducted to determine the amount of "nuisance variance." The total score of the M-CHAT was included as a potential indicator for a total of 4 potential indicators and were included in these initial validity analyses. The socialization/nonverbal communication and repetitive behavior/restricted interests factors and the total score of the M-CHAT all had acceptable indicator validities with Cohen's *d* above the recommended 1.25 (Meehl, 1995). Cohen's *d* for the communication factor did not exceed the recommended cutoff and was therefore not included in the subsequent analyses.

The socialization/nonverbal communication and repetitive behavior/restricted interests factors and the total score of the M-CHAT were included as indicators of the current study. The indicator validities were acceptable with Cohen's *d* ranging from 2.14 to 3.15 ($M = 2.51$, $SD = 0.56$). All 3 indicators in the full sample were positively skewed

with M = 1.89 (range = 1.77 - 2.17). Excessive positive skew could result in plots that appear taxonic when the underlying structure is dimensional, and therefore should be considered during the interpretation of results.

Using the taxon sample, within group correlations were conducted to determine the amount of "nuisance variance." The indicator correlation for the entire sample was r = .69. Indicator correlations for the taxon group was a mean of r = .39 and the mean for the complement group was r = .28. While the correlations for the taxon group exceeded the recommended r = .30, the correlations for the separate groups were substantially less than the full sample (J. Ruscio, et al., 2006). See Table 1 for correlations.

Table 1

Full-sample and within group correlations

Full-sample (N=1149)			
	Socialization/ nonverbal	Repetitive behavior/ restricted interests	M-CHAT Total
Socialization/ Nonverbal	1.00	0.73	0.65
Repetitive behavior/ restricted interests	0.73	1.00	0.68
M-CHAT Total	0.65	0.68	1.00
Taxon (n=277)			
	Socialization/ nonverbal	Repetitive behavior/ restricted interests	M-CHAT Total

(table continues)

Socialization/Nonverbal	1.00	0.49	0.39
Repetitive behavior/ restricted interests	0.49	1.00	0.28
M-CHAT Total	0.39	0.28	1.00

Complement (n=872)

	Socialization/ nonverbal	Repetitive behavior/ restricted interests	M-CHAT Total
Socialization/ Nonverbal	1.00	0.27	0.27
Repetitive behavior/ restricted interests	0.27	1.00	0.29
M-CHAT Total	0.27	0.29	1.00

Suitability Analyses. Employing the identified indicators, suitability analyses were conducted to determine the best analyses for the characteristics of the research data. Simulated data were derived from R-language (J. Ruscio, 2004). The simulated data had the same distributions and correlations as the research data, with different latent structures: taxonic or dimensional. Taxometric analyses (e.g., MAMBAC, L-MODE, MAXEIG) were conducted with the simulated data and empirical sampling distributions were provided. The graphs were visually inspected by trained raters to determine if the plots represented a taxonic or dimensional structure, or were ambiguous and unable to interpret. Percent agreement among raters was calculated. The methods chosen were

those taxometric analyses that were most interpretable using the simulated data. Two techniques were chosen to analyze the research data.

Taxometric Analyses. Using the methods identified in the suitability analyses, taxometric analyses were conducted with the sample. Eighteen raters were used for interpretation of plots. Percent agreement was calculated among raters.

Next the CCFI was calculated for both taxometric analyses to determine the degree of fit between the research data and comparison taxonic and dimensional data. The CCFI is a method of determining model-fit by using the root mean square residual. The sum of the *y* ordinate values of the research and dimensional or taxonic data are employed to determine the fit. Values of CCFI can range from 0 through 1 with scores approaching 0 indicating a dimensional structure and scores approaching 1 indicating a taxonic structure. Values approaching 0.50 should be interpreted with caution as the fit does not appear to be better accounted for by either the taxonic or dimensional structures (J. Ruscio, et al., 2006).

Consistency Testing. Lastly, consistency testing was employed to determine the level of convergence of results. The nose-count test, inchworm test, base-rate variability analysis, Goodness of Fit Index, and comparison of multiple analyses were conducted. Convergence of results across multiple independent analyses provides support for conclusions based on taxometric analyses.

Results

Suitability Analyses

Simulated data were generated using similar parameters as the research data and with underlying taxonic and dimensional structures. Using the simulated data, MAXEIG, MAMBAC, and L-Mode were performed. Eighteen raters were then employed to evaluate the interpretability of the plots of both the taxonic and dimensional comparison data (J. Ruscio, et al., 2004; J. Ruscio, et al., 2007). Raters were instructed to compare plots of the simulated taxonic and dimensional data with the research data. Next they were instructed to decide which of the simulated comparison data (i.e., taxonic or dimensional) were most similar to the research data. Raters were provided with the option of rating "ambiguous" for plots that they were not able to discern whether the fit was more similar to the taxonic or dimensional data. See Figure 1 for suitability analysis plots. See Appendix for specific instructions for raters.

Percent agreement was calculated between raters for each of the analyses. Highest rater agreement occurred with the MAMBAC procedure. Eighty-eight percent of the raters (n = 16) agreed the plots were dimensional, and11.11% labeled the graphs as taxonic (n = 2). None of the raters determined the MAMBAC plots to be ambiguous. The next highest rater agreement occurred with the MAXEIG procedure. Eighty-three percent of the raters (n = 15) agreed the plots were dimensional, and 16.7% of the raters (n = 3) labeled the graphs as taxonic. None of the raters determined the MAXEIG plots to be ambiguous. Lastly, L-Mode was conducted. Seventy-two percent (n = 13) of the raters agreed that the L-Mode simulated data was ambiguous, 22.2% (n = 4) rated the plots as taxonic, and 5.6% (n = 1) rated the plots as dimensional.

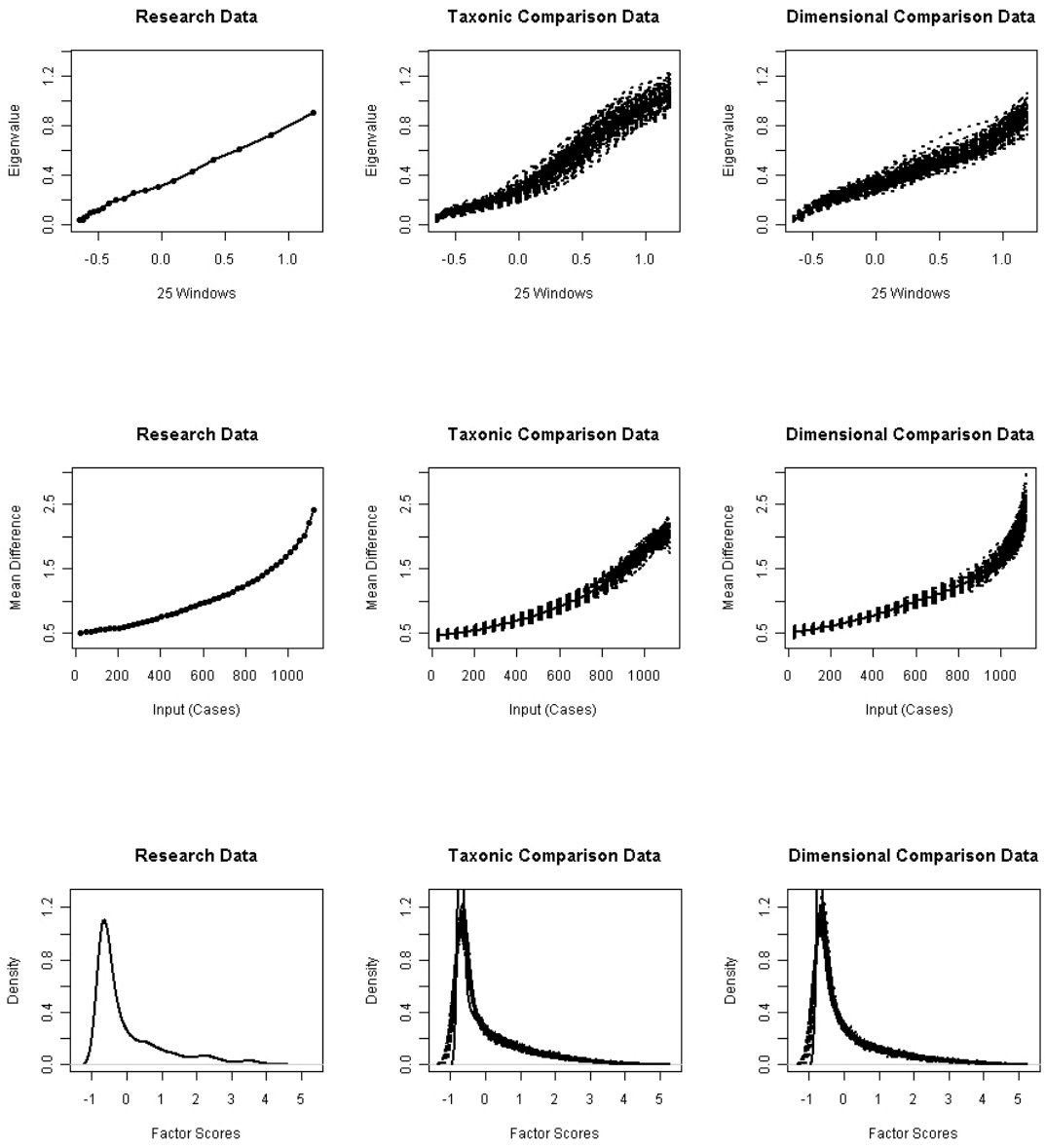

Figure 1: Suitability plots for MAXEIG (top panel), MAMBAC (middle panel), and L-Mode (bottom panel)

Taxometric Analyses

The taxometric analyses selected for this investigation were chosen based on suitability for use considering the specific parameters of the research data. MAXEIG and

MAMBAC were selected for use in the current study due to high rater agreement of suitability analyses on the structure of the plots, with no raters determining the plots to be ambiguous. Due to the higher than ideal nuisance correlations, MAXEIG was selected as the primary analysis, as MAMBAC tends to be less robust than MAXEIG under these conditions (Schmidt, et al., 2004). Due to the ambiguity of the plots produced by the L-Mode procedure using the simulated data, this procedure was not suitable for use with the data at hand.

MAXEIG. The MAXEIG procedure was conducted with the 3 indicators (socialization/nonverbal communication and repetitive behavior/restricted interests factors of the BISCUIT-Part 1, and the total score of the M-CHAT). Each of the 3 indicators served once as the input variable with all other indicators serving at the output variable. Twenty-five windows with 0.9 overlap were used, with a total of 338 participants per window. A total of 3 curves were produced. One hundred samples of taxonic and dimensional comparison data were generated. Nuisance correlation had a mean of r = -0.03 for within the taxon group and r = 0.46 for within the complement group. Nuisance correlation for the complement group exceeded the suggested limit of r = 0.30 (Meehl, 1995). Indicator validities were acceptable, with a range of d = 3.47-3.66.

Latent structure was investigated by examining the model fit. Model fit was determined by rater agreement of curve shape and calculating the Comparison Curve Fit Index (CCFI). Eighteen raters were selected to provide ratings on taxonicity of plots. All raters were clinical or school psychology graduate students unfamiliar with taxometric analyses. Due to the raters' unfamiliarity with taxometrics, comparison data were employed to aid in interpretation (Meehl, 1995). Raters were presented with 2 graphs

consisting of averaged MAXEIG curves with overlay comparison data. One graph had comparison data that were taxonic in structure and the second graph had comparison data that were dimensional in structure. Raters were instructed to examine both graphs and determine which plots were better fits with the research data. One hundred percent (n = 18) of the raters selected the dimensional comparison data as being a better fit with the research data. None of the raters selected the taxonic data as being a better fit with the research data. See Figure 2 for MAXEIG plots. See Appendix for specific instructions for raters.

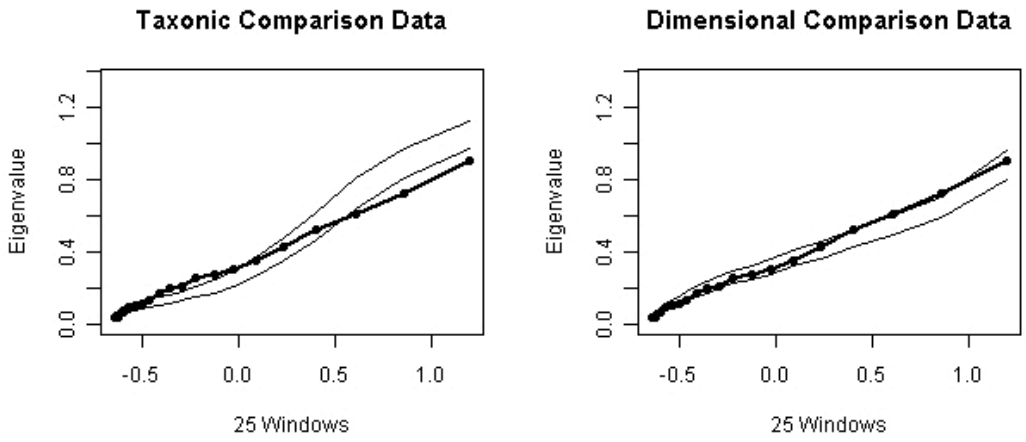

Figure 2: MAXEIG plots with simulated taxonic comparison data (left) and simulated dimensional comparison data (right). The lighter lines represent +/- 1 *SD* from the *M* of the comparison data sets. The dark plotted lines represent the research data.

The CCFI was calculated to determine the level of fit between the research data and the comparison data. The CCFI for the MAXEIG analysis was 0.29. As the measure of fit was less than 0.50, a dimensional structure is supported.

Consistency Testing. The inchworm consistency test was conducted to further evaluate the underlying structure of the research data. This powerful analysis is particularly useful in the current study due to the positive skew observed by the indicators

(J. Ruscio, et al., 2004). As mentioned previously, positively skewed data of a dimensional nature may exhibit a rising curve that could be mistaken for a small taxon. However, when using the inchworm consistency test to follow-up MAXEIG results, a small taxon will result in a peak or cusp, whereas an underlying dimensional structure with positive skew will not peak (J. Ruscio, et al., 2006). Fifty and 100 windows with 0.9 overlap were used, with a total of 195 and 105 participants per window respectively. Inspection of the graphs did not reveal peaks in the plots. Additionally, the comparison taxonic data resulted in a peak with increased windows, while the research data did not. See Figure 3 for inchworm consistency test plots.

The "nose-count" consistency test was conducted to evaluate the consistency of results for the MAXEIG analyses. The nose-count test entails counting the peaks on each of the plots and determining the ratio of taxonic results to dimensional or ambiguous results. While there is much debate on the appropriate ratio of taxonic to dimensional plots, a ratio of 1:1 is sufficient for indicating an underlying structure is taxonic, as false positives of taxonicity are uncommon (Schmidt, et al., 2004). Two independent raters examined each of the plots for the MAXEIG analysis. The raters were blind to the hypothesis of the current study. Raters were not experienced in taxometric analyses and received the following instructions for interpreting the plots: rate the separate plots as taxonic, dimensional, or ambiguous based on the following plot characteristics. Plots are rated taxonic when there is a uni-modal peak or a right cusping peak with a downward slope following the cusp. Plots are rated as dimensional if there are no peaks and are generally flat, and/or concave shaped. Lastly, plots are rated as ambiguous when a taxonic or dimensional curve cannot be determined. Raters agreed that all 3 plots looked

dimensional. See figure 4 for MAXEIG plots.

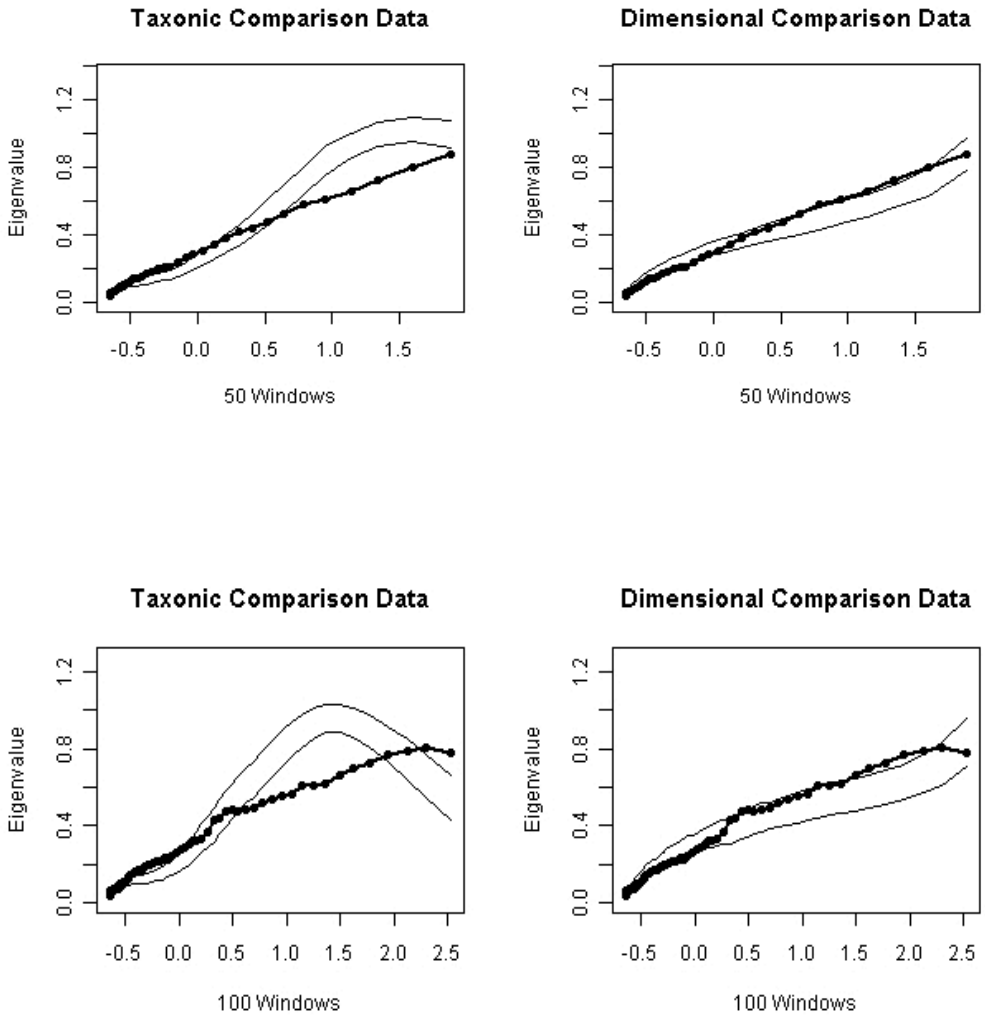

Figure 3: Inchworm consistency test for MAXEIG plots of 50 and 100 overlapping windows with simulated taxonic comparison data (left) and simulated dimensional comparison data (right). The lighter lines represent +/- 1 *SD* from the *M* of the comparison data sets. The dark plotted lines represent the research data.

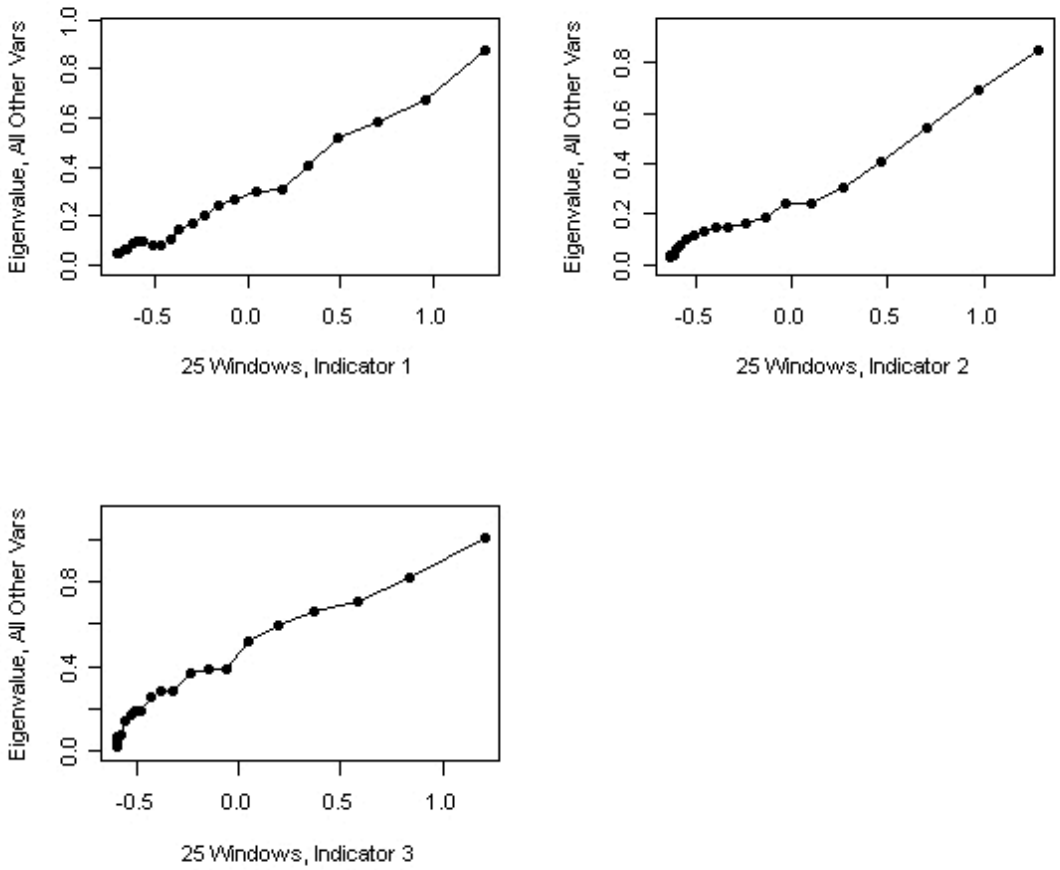

Figure 4: MAXEIG plots for nose-count test

The base-rate variability test was also conducted. The base-rate variability for the MAXEIG analysis had a mean of 0.08 and standard deviation of 0.01, which implies stability and a taxonic conjecture. Lastly the Goodness of Fit Index (GFI) was analyzed to determine the "fit" between the observed and predicted model. The GFI for the MAXEIG analysis for the current study was 0.84, which implies a dimensional structure.

MAMBAC. The MAMBAC procedure was conducted with the 3 indicators (socialization/nonverbal communication and repetitive behavior/restricted interests factors of the BISCUIT-Part 1, and the total score of the M-CHAT). The 3 indicator variables served in all possible input-output pairs with a total number of 6 curves. Cuts

were made at 50 evenly spaced intervals beginning at 25 cases from either end. One hundred samples of taxonic and dimensional comparison data were generated.

The 18 raters used for ratings of suitability analyses and the MAXEIG analysis also provided ratings on taxonicity of the MAMBAC plots. Methods for plot ratings of the MAMBAC plots were identical to the MAXEIG analysis. Ninety-four percent of the raters selected the dimensional comparison data as being a better fit with the research data. See Figure 5 for MAMBAC plots. See Appendix for specific instructions for raters.

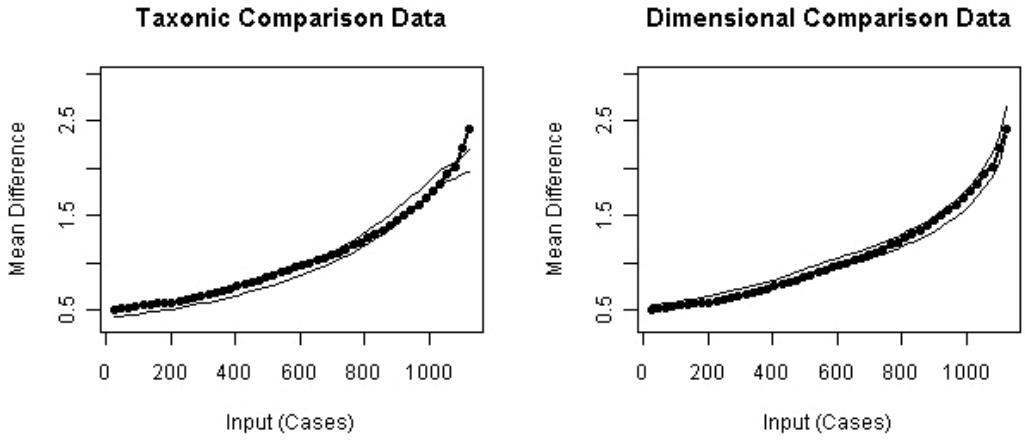

Figure 5: MAMBAC plots with simulated taxonic comparison data (left) and simulated dimensional comparison data (right). The lighter lines represent +/- 1 *SD* from the *M* of the comparison data sets. The dark plotted lines represent research data.

The CCFI was calculated to determine the level of fit between the research data and the comparison data. The CCFI for the MAMBAC analysis was 0.30. As the measure of fit was less than 0.50, dimensional structure is supported.

The "nose-count" consistency test was conducted to evaluate the consistency of results for the MAMBAC analyses. Just as with the MAXEIG nose-count test, two independent raters examined each of the plots for the MAMBAC analysis. The raters

were blind to the hypothesis of the current study. Raters were not experienced in taxometric analyses and received the following instructions for interpreting the plots: rate the separate plots as taxonic, dimensional, or ambiguous based on the following characteristics. Plots are rated taxonic when uni-modal peaks or a right cusping peak with a downward slope following the cusp. Plots are rated as dimensional if there are no peaks and are generally flat, and/or concave shaped. Lastly, plots are rated at ambiguous when a taxonic or dimensional curve cannot be determined. Raters agreed that all 6 plots appeared dimensional. See Figure 6 for MAMBAC plots.

The base-rate variability test was also conducted with the MAMBAC analysis. The base-rate variability for the MAMBAC analysis was a mean of 0.17 and standard deviation of 0.03, suggesting taxonic structure. Lastly the Goodness of Fit Index (GFI) was analyzed to determine the "fit" between the observed and predicted model. The GFI for the MAMBAC analysis for the current study was 0.94, which suggests a taxonic structure.

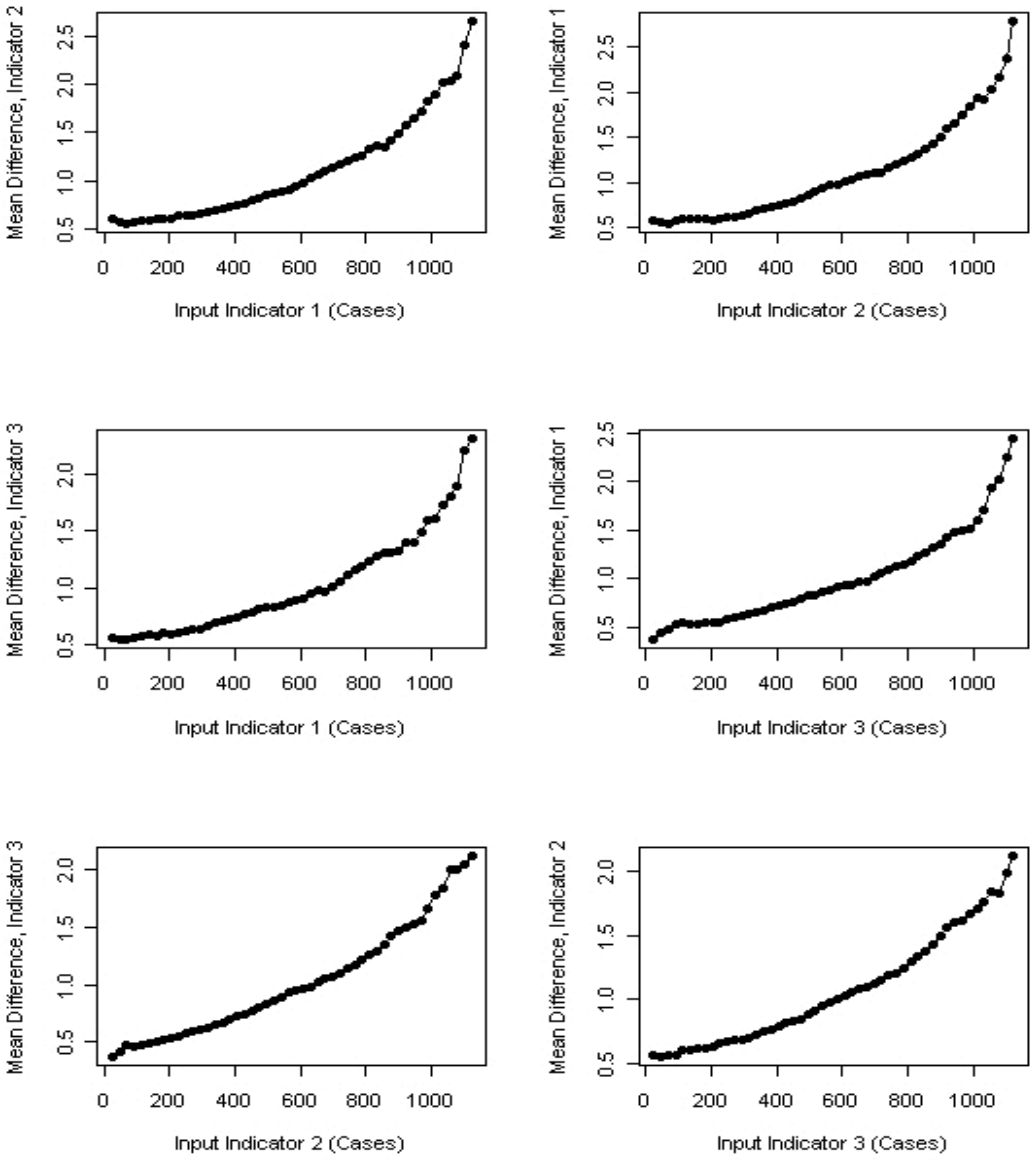

Figure 6: MAMBAC plots for nose-count test

Summary of Results

The purpose of this study was to investigate the underlying structure of ASD in a population of toddlers at-risk for developmental disabilities. Taxometric methodology was used to determine if symptoms consistent with ASD are taxonic or dimensional in this specific population. Consistent with the hypothesis, the results of the taxometric analyses conducted in this study support a dimensional structure of autism symptoms in an at-risk population of toddlers.

The MAXEIG and MAMBAC analyses were conducted to examine the latent structure of the data. The CCFI and raters' classification were used as the primary taxometric methods for interpretation. The CCFI for MAXEIG and MAMBAC both suggested a dimensional structure. Additionally, 100% of the raters in this study rated the MAXEIG research data plots as dimensional when compared to simulated data plots. Similarly, 94% of the raters scored the MAMBAC plots as dimensional when compared to simulated data plots. Both the MAXEIG and MAMBAC results suggest that the indicators employed in this study to represent ASD symptoms in at-risk toddlers are dimensional in structure. See Table 2 for taxometric analyses results.

Table 2: Taxometric Statistics

	MAMBAC	MAXEIG
Percent Rater Inspection (n)		
Taxonic	5.56 (2)	0.00 (0)
Dimensional	94.44 (16)*	100.00 (18)*
Ambiguous	0.00 (0)	0.00 (0)

(table continues)

CCFI	0.31*	0.29*
Nose Count		
Taxonic	0.00	0.00
Dimensional	100.00*	100.00*
Ambiguous	0.00	0.00
GFI	0.94**	0.84*
Base-rate estimates		
M	0.17	0.08
SD	0.03**	0.01**

Note: *=dimensional structure suggested; **=taxonic structure suggested

Hallmark to taxometric analyses is consistency testing. To corroborate the results of the primary analyses, additional yet unrelated analyses were conducted to examine further consistency. Examination of the GFI, nose count test, and base-rate variability analyses were conducted for both the MAXEIG and MAMBAC analyses. Additionally, the inchworm consistency test was conducted with the MAXEIG analysis.

The purpose of the base-rate variability test is to examine the consistency of base-rate estimates within the analysis. Schmidt and colleagues (2004) proposed a cutoff score of 0.10 SD with base-rate estimates below this value implying a taxonic conjecture. The rationale is that taxonic structure would exhibit higher levels of consistency across the sub analyses, therefore exhibiting lower base rate estimates and standard deviations. The base-rate variability for both the MAXEIG and MAMBAC analyses implied stability and

a taxonic conjecture. However, the validity of the base-rate variability test has been questioned due to low rate of accurate classification of taxonic data sets using this test (Schmidt, et al., 2004).

Goodness of Fit Index (GFI) was analyzed to determine the "fit" between the observed and predicted model. Schmidt and associates (2004) have provided estimates for determining fit consistent with latent structure. Values greater than 0.90 are consistent with taxonic structure and values less than 0.90 provide support for a dimensional structure. The GFI for the MAXEIG analysis was 0.84, and the GFI for the MAMBAC analysis was 0.94. Similar to the base-rate variability analysis, the GFI's validity has also been questioned and interpretation based solely on these analyses should be cautioned (J. Ruscio, et al., 2007).

The nose-count test was also conducted as an analysis of consistency. Raters agreed that the 3 plots from the MAXEIG analysis and the 6 plots from the MAMBAC analysis appeared dimensional. Due to no plots rated as taxonic, taxonic conjecture is not suggested based on the nose-count test.

The inchworm consistency test was conducted to further evaluate the underlying structure of the research data. This powerful analysis is particularly useful in the current study due to the positive skew observed by the indicators (J. Ruscio, et al., 2004). As mentioned previously, positively skewed data of a dimensional nature may exhibit a rising curve that could be mistaken for a small taxon. However, when using the inchworm consistency test to follow-up MAXEIG results, a small taxon will result in a peak, whereas an underlying dimensional structure with positive skew will not peak (J. Ruscio, et al., 2006). The comparison taxonic data resulted in a peak with increased

windows, while the research data did not. These results suggest a dimensional latent structure.

The results of the primary analyses for the MAXEIG analysis and the consistency tests largely support a dimensional structure for the symptoms of ASD in an at-risk toddler population. Only one consistency test, which is reported to have questionable validity (J. Ruscio, et al., 2007), out of six MAXEIG analyses suggested a taxonic conjecture. Similarly, the MAMBAC primary analyses supported a dimensional structure for the data used in this study. Just two of the MAMBAC follow-up consistency tests, both with questionable validity (J. Ruscio, et al., 2007), suggested taxonicity out of five analyses. Additionally, the MAXEIG and MAMBAC primary analyses converged on the same outcomes, providing further corroboration and support for a dimensional structure of ASD symptoms in a population of toddlers at-risk for developmental disabilities. Nine of the 12 analyses conducted in this study converged on a dimensional structure.

There were several limitations to the current study. While nuisance correlations for within group comparisons during the suitability analyses and subsequent taxometric analyses were relatively low, they were still on average slightly above the recommended 0.30. High nuisance correlations can result in difficult to interpret results. However, in the current study, difficulty in interpretation of plots did not appear to be a concern as there was high rater agreement with both the suitability and taxometric analyses. Furthermore, indicators that are highly correlated may indicate shared loadings of a dimensional nature on the underlying construct (J. Ruscio, et al., 2006).

Another weakness in the current study involves excessive skew. Each of the indicators exhibited potentially problematic skew. Excessive positive skew can result in

right-rising plots that may form a cusp and interfere with interpretation. These plots may be mistakenly interpreted as taxonic, when a latent dimension exists. However, in the current study, the inchworm consistency test followed the MAXEIG analysis to further investigate shape. Results of the inchworm test were also suggestive of a dimensional structure.

While the large majority of analyses conducted in this study provided results consistent with a dimensional structure, Ruscio and colleagues (2006) caution that pseudo-dimensionality is more likely than falsely identifying a taxon. That is, it is more likely to identify a latent structure as being dimensional when it is not, than wrongly identifying a latent structure as taxonic. However, due to the high consistency of results across analyses, support for a dimensional latent structure is provided.

Another potential limitation of this study is the validity of the indicators. While the *a priori* validity analyses suggested the indicators were independent from one another, and differentiated the proposed groups, it is possible that the indicators were identifying phenomenon other than ASD. However, both measures used in the analyses have been established as being valid (Matson, et al., in press; Snow & Lecavalier, 2008). Furthermore, *a priori* groups were based on the highest scoring participants on the BISCUIT-Part 1. Indicators were compiled from items of the BISCUIT-Part 1, so differences in scores on the indicators based on artificially forming a taxon, would be expected. However, these artificial groups were only used in the *a priori* analyses to determine suitability and not used with the taxometric analyses and do not interfere with results of this study.

Discussion

The debate on the conceptualization of mental disorders as being taxonic or dimensional within a population is growing in popularity. Paul Meehl (1995) can be credited with developing methodology to identify the latent structure of a construct and providing important reasons and implications for investigating the underlying structure of a disorder. These implications include methods of assessing disorders and treatment decisions (Meehl, 1992). Assessment measures of psychopathology should be developed specific to the structure of the disorder. That is, taxonic disorders may only require a small number of items to identify the taxon, while a dimensional structure may require a large number of scale items in order to determine the individual's placement along a dimension (Meehl, 1992).

Another implication deals with the loss of information in relation to treatment decisions that can result from the categorization of dimensional disorders. When a condition is determined to be categorical, a specific prognosis may be provided and a particular intervention is recommended. However, if a condition is dimensional the prognosis and efficacy of an intervention may differ between individuals and across severity and symptom presentation. The construct of BAP has been emerging in the literature, with support for the existence of milder forms of ASD that do not meet criteria for a diagnosis. Some individuals with BAP may not meet criteria for a diagnosis, but still experience some difficulty in certain areas of functioning. Without a diagnosis, these individuals may not be eligible for treatment services. Individuals with sub-threshold symptoms may benefit from treatments similar to those meeting criteria for a diagnosis of ASD. Likewise, individuals with milder forms of ASD, meeting criteria for a diagnosis,

may not require the same level of intensity of intervention as a person with a more severe form of the disorder. Acknowledging a dimension of severity aids in the clinical utility when providing a diagnosis (Widiger & Samuel, 2005).

The findings from the current study of a dimensional structure for ASD in a population of at-risk toddlers supports the proposed revisions to the DSM-V (APA, 2010). That is, the current workgroup revising the PDD category for the DSM-V are proposing a single diagnosis for the disorder that will vary according to severity. The proposed revision involves absolving the Asperger's Disorder and PDD-NOS diagnoses. Instead, a single diagnosis termed *Autism Spectrum Disorder* will be used to classify these specific symptom clusters. Rationale for the revisions to the ASD diagnoses is due to the questionable validity of the different PDD categories of the DSM-IV (Szatmari, 2000). That is, studies have not adequately differentiated between individuals with Asperger's Disorder, PDD-NOS, and individuals with 'High Functioning' autism (i.e. people with autism and without ID) (Miller & Ozonoff, 2000). Therefore, individuals with these diagnoses may differ in degree of severity rather than kind.

Implications of the current study include the need for further investigation of symptom patterns and what constitutes sufficient impairment to warrant a diagnosis in this particular population. The analyses from this study suggest that there is no clear categorization of ASD symptoms in this sample. Thus, identification of children who exhibit enough symptoms to warrant a diagnosis and therefore attain services may be arbitrary. One of the hypotheses for the increase in ASD is attributed to the expanded definition of the disorder (Wing & Potter, 2002), and with no clear categorization of ASD symptoms in the current study, children at-risk for developmental disabilities may differ

based on degree of autism symptoms, rather than the symptoms being present or absent. In the current study, the portion of toddlers that are not expected have an ASD diagnosis (i.e. the complement) still exhibited symptoms consistent with ASD. Coupled with other impairments, these children may meet current criteria for ASD. Therefore, the current diagnostic criteria may be over-inclusive in this population and may benefit from re-evaluation with consideration of the latent structure of the disorder.

As this study is the first of its kind with this particular population, replications are warranted. Results of the current study do suggest a latent dimension of autism symptoms in a sample of toddlers at-risk for developmental disabilities, which may guide future studies on conceptualization and classification of the disorder. As this study investigated symptoms of ASD in an at-risk sample, generalization to the general population should be cautioned. Future taxometric studies should utilize indicators constructed from other valid instruments with various modes and methods of assessment. Additionally, future studies should investigate different phenotypes of ASD using taxometric analyses and genetic studies to aid in identifying specific genotypes of the disorder. Future directions of taxometric analysis should also involve participants more representative of the general population.

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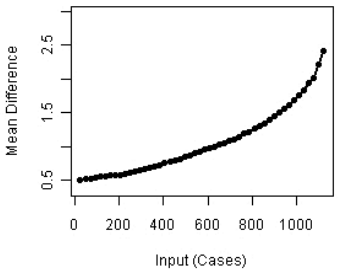
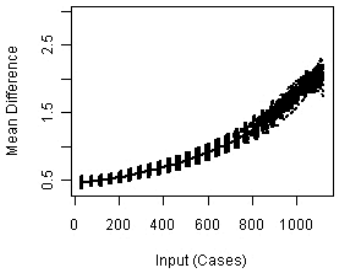
Appendix Instructions For Raters

There are a total of 3 pages with 5 sets of plots to look at. Please place your ratings on the lines.

A



1



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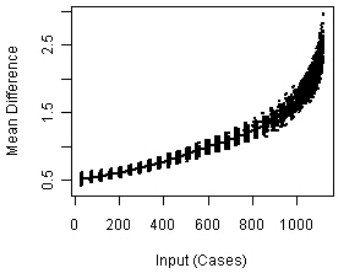
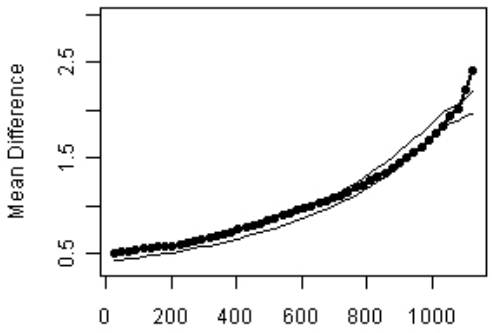


Look at the plot labeled "A" and compare to the plots labeled "1" and "2."

Which plot, 1 or 2, is most similar to A? _____

If you are unable to determine if plot 1 or 2 is more similar to A, put a "3" on the line above. *Please reserve a score of 3 for plots that you aren't able to determine which is most similar.*

1



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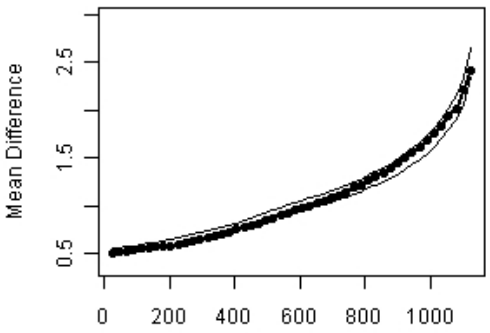
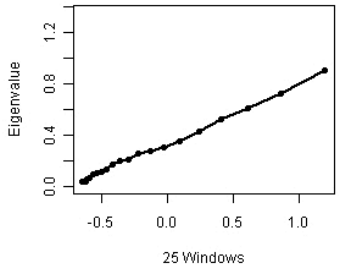


Look at plots 1 and 2. The dark plotted lines are research data. The 2 light colored lines are +/- 1 SD of data that I am comparing my data to for fit.

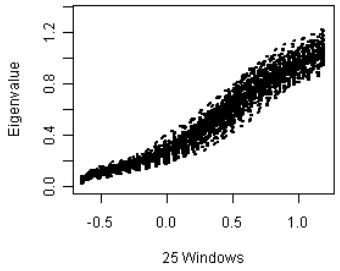
Which plot, 1 or 2, does my research data (dark plotted lines) fit better with the comparison data (light colored lines)? _____

If you are unable to determine if the light colored lines on plot 1 or 2 is more similar to the research data, put a "3" on the line above. *Please reserve a score of 3 for plots that you aren't able to determine which is a better fit.*

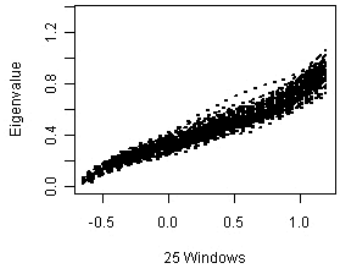
A



1



2



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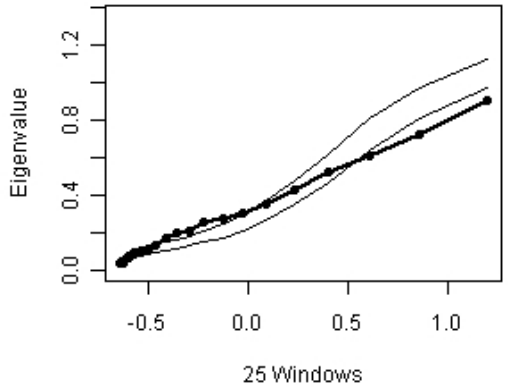
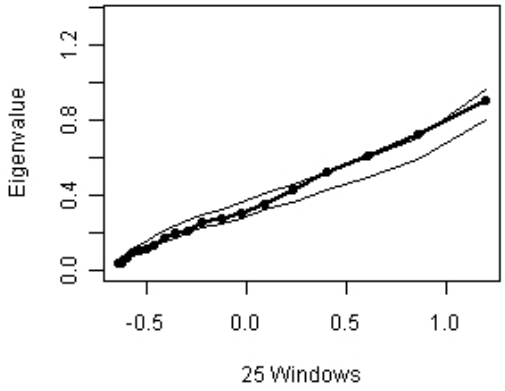


2



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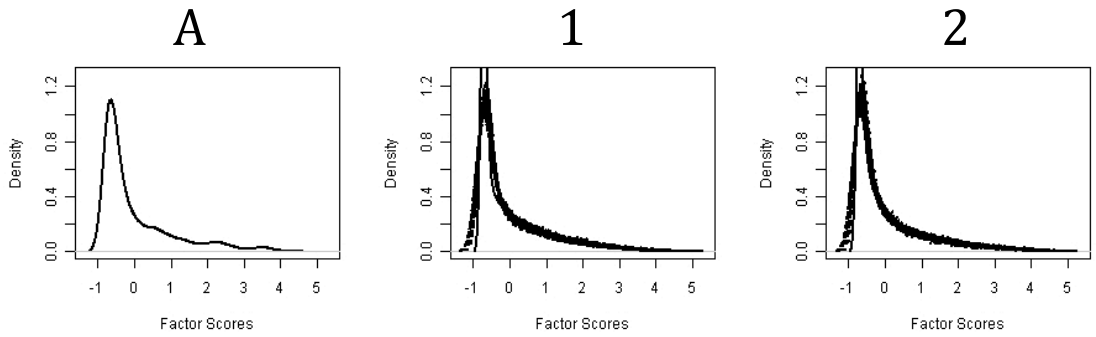

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If you are unable to determine if plot 1 or 2 is more similar to A, put a "3" on the line above. *Please reserve a score of 3 for plots that you aren't able to determine which is most similar.*

Vita

Jessica Ann Boisjoli was born April 1977 in Troy, New York. Jessica attended the State University of New York at Cobleskill from 1995 through 1997 where she earned an Associate in Arts and Sciences degree. She then attended the University at Albany from 1997 through 1999 where she earned her Bachelor of Arts in psychology. Jessica enrolled in the clinical psychology graduate training program at Louisiana State University in Baton Rouge, Louisiana, in 2005. Her research and clinical training focused on the assessment and treatment of individuals with intellectual disabilities, autism spectrum disorders, and comorbid psychopathology. Jessica completed her pre-doctoral internship at the Kennedy Krieger Institute, Johns Hopkins University.