EXECUTIVE DYSFUNCTION IN AUTISM AND ASPERGER’S DISORDER: A META-ANALYTIC REVIEW OF COGNITIVE PLANNING

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I HEREBY RECOMMEND THAT THE DISSERTATION PREPARED UNDER MY SUPERVISION BY ASHLEY JONES RENO ENTITLED EXECUTIVE DYSFUNCTION IN AUTISM SPECTRUM DISORDERS: A META-ANALYTIC REVIEW OF COGNITIVE PLANNING BE ACCEPTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PSYCHOLOGY.

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Abstract

One of the most prominent theories of autism and Asperger’s Disorder suggests that their symptoms arise from a primary deficit in executive functions (EF). While many researchers have argued that the executive dysfunction profile may be used as diagnostic support, there have been studies to suggest that autism and Asperger’s Disorder may not be clearly differentiated from other clinical disorders on the basis of their executive functioning profiles (e.g., Booth, et al., 2003). Therefore, it is important to examine specific aspects of executive functioning (e.g., cognitive planning, etc.) among children diagnosed with autism and Asperger’s Disorder in order to determine whether a distinct executive dysfunction profile exists among these populations. In the current investigation, a meta-analysis of 33 studies that administered cognitive planning measures to groups diagnosed with autism and Asperger’s Disorder (total N = 1,020) and without autism or Asperger’s Disorder (N = 1,591) was conducted. While children diagnosed with autism and Asperger’s Disorder exhibited significant weaknesses in cognitive planning, there was a lack of universality of cognitive planning deficits among individuals with autism and Asperger’s Disorder relative to other clinical groups. Overall, these findings suggest that cognitive planning weaknesses are neither necessary nor sufficient to cause all cases of autism and Asperger’s Disorder. Rather, difficulties with cognitive planning appear to be one important component of the complex neuropsychology of ASDs.
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Dedication

This dissertation is dedicated to my husband and partner, Seth Reno, Ph.D., who made this possible through his unwavering support and love throughout my graduate career. I would also like to dedicate this dissertation to my mother, Ruth Luneborg, who instilled in me the patience, perseverance, and dedication needed complete this project, as well as for her endless encouragement, patience, and support.
Chapter I

Autism and Asperger’s Disorder are defined as Autism Spectrum Disorders (ASD) by the National Institute of Mental Health (2010) and the Center for Disease Control (2010). Within the United States of America, it is estimated that an average of 1 in 88 children have an ASD (Autism Society of America, 2012; CDC, 2010; NIMH, 2010). Furthermore, ASD diagnoses are suspected to be growing at a rate of 10-17% per year, posing a serious concern for the general public as well as practicing clinicians within the field of Psychology (Autism Society of America, 2009). With this growing concern for ASD, it is imperative that clinicians gain a fuller understanding of the characteristics associated with the disorders so that we are able to increase the occurrence of accurate diagnoses and appropriate care and treatment for this unique population.

Rationale

Many researchers have attempted to elucidate the underpinnings of autism and Asperger’s Disorder, often pointing to a diverse set of causal factors. Among such factors are neural theories (i.e., temporal lobe hypothesis, cerebellar hypothesis, and the frontal lobe hypothesis; Robbins, 1997; Schroder, Desrocher, Bebko, & Cappadocia, 2010) and cognitive impairment theories (i.e., weak central coherence, theory of mind, and the executive dysfunction hypothesis; Beaumont & Newcombe, 2006; Rajendran & Mitchell, 2007; Schultz, Charwarska, & Volkmar, 2006). In recent years, the executive dysfunction hypothesis has received much attention. Researchers have discovered that individuals diagnosed with autism or Asperger’s Disorder exhibit executive dysfunction on various
neuropsychological tests, including measurements of planning, inhibition, set-shifting, self-monitoring, organization, flexibility, and working memory (Ventola & Tsatsanis, 2011). In addition, individuals diagnosed with autism and Asperger’s Disorder may not be differentiated based on their performances on executive functioning tasks. Based upon such findings, many have argued that executive dysfunction may be a global impairment found among individuals on the autism spectrum and that the various symptoms commonly associated with autism spectrum disorders arise from this deficit (Manjiviona & Prior, 1999; Ozonoff, Rogers, & Pennington, 1991; Szatmari, et al., 1990). Thus, it is argued that executive dysfunction may serve as a diagnostic marker for this population.

In contrast, however, some researchers have discovered that individuals diagnosed with autism or Asperger’s Disorder may not be differentiated from other clinical groups (e.g., ADHD, OCD, TBI) on the basis of their performances on tasks of executive dysfunction (Hill, 2004). These findings suggest that while children diagnosed with autism or Asperger’s Disorder may exhibit executive dysfunction, such difficulties may not underlie the symptoms and characteristics unique to the autism spectrum diagnosis. Executive dysfunction, therefore, may not be an appropriate diagnostic marker for autism and Asperger’s Disorder.

To examine further the differences between children diagnosed with autism and Asperger’s Disorder and children diagnosed with other clinical disorders commonly associated with executive dysfunction, researchers have examined whether distinct executive dysfunction profiles may exist for the various clinical populations (e.g., Ozonoff & Jensen, 1999). While some studies support the notion that distinct executive functioning profiles do exist (e.g., Ozonoff & Jensen, 1999; Sergeant, et al., 2002), there
have also been studies to suggest a less clear difference between autism and Asperger’s Disorder and other clinical groups (e.g., Booth, et al., 2003). Therefore, it is important to continue to examine specific aspects of executive functioning among children diagnosed with autism and Asperger’s Disorder in order to determine whether a distinct executive dysfunction profile exists among these populations. Such information may help to identify the validity of the executive dysfunction hypothesis of ASDs, as well as to establish whether specific executive dysfunction profiles may be used to aide in the diagnosis of autism and Asperger’s Disorder.

**Planning.** Cognitive planning has been one of the most extensively studied components of executive functioning among children diagnosed with autism or Asperger’s Disorder. In fact, a literature search conducted in August, 2011 resulted in 33 papers between 1985 and 2011 that compared performances on tasks of planning ability between groups with and without autism and/or Asperger’s disorder. While much of this research has suggested that children diagnosed with autism or Asperger’s Disorder do exhibit planning deficits (e.g., Bennetto et al., 1996; Ozonoff & Jensen, 1999; Ozonoff et al., 1991), there have also been findings that suggest the contrary (e.g., Liss, et al., 2001). Thus, the research findings regarding planning ability among children diagnosed with autism and Asperger’s Disorder remain unclear.

**Aim and Purpose**

The aim of the current study is to conduct a meta-analytic review of available studies comparing executive functioning with regard to planning ability between groups with and without autism and/or Asperger’s Disorder. This research will help to elucidate the executive dysfunction hypothesis among autism and Asperger’s Disorder.
Specifically, this study will contribute to the scientific knowledge regarding the validity of the executive dysfunction hypothesis as an underlying mechanism of autism and Asperger’s disorder. In addition, this research will help to determine if aspects of planning may contribute to the development of an executive dysfunction profile as a diagnostic marker, differentiating autism and Asperger’s Disorder from other clinical disorders commonly associated with executive dysfunction.
Chapter II. Literature Review

Autism and Asperger’s Disorders

Autism and Asperger’s Disorder are classified as pervasive developmental disorders (PDD), which are regarded as neuropsychiatric disorders (Volkmar & Lord, 2007; Volkmar & Pauls, 2003). According to the Diagnostic and Statistical Manual of Mental Disorder, Fourth Edition, Text Revision (2000), PDDs are characterized by patterns of delay and deviance in multiple areas of development, including reciprocal social interaction skills (e.g., poor facial expression; few or no peer relationships; does not share enjoyment with others; etc.), communication skills (e.g., failure or late development of spoken language; difficulty initiating or sustaining conversations), or the presence of stereotyped behavior (e.g., hand or finger flapping), interests (e.g., extreme interest in facts about a favorite topic, such as insects or cars), or activities (e.g., lining up toy cars rather than playing with them in a make-believe manner). Furthermore, the onset of autism and Asperger’s Disorder is typically within the first months of life (APA, 2000; Volkmar & Lord, 2007; Volkmar & Pauls, 2003).

Historical development.

Autism. Autism was first discovered by Leo Kanner in 1943, and his findings continue to influence the definition of autism today (Volkmar & Klin, 2005; Volkmar & Lord, 2007). His published work included the study of 11 children brought to John Hopkins Hospital for severe social and communication abnormalities and restricted and narrow interests (Davison & Neale, 2000; Volkmar & Klin, 2005). Kanner (1943)
described these children as having “autistic disturbances of affective contact,” and he reported that his patients exhibited a disorder characterized by lacking social engagement, communication problems, and unusual responses to the inanimate environment (Volkmar & Lord, 2007). Furthermore, Kanner (1943) viewed the children’s inability to relate and interact with others in a socially appropriate manner as the essential feature of autism (Volkmar & Lord, 1998).

Based on his research, Kanner (1943) originally coined this condition early infantile Autistic Disorder, which was borrowed from Bleuler’s term “autism” for the idiosyncratic, self-centered thinking observed in schizophrenia (Volkmar & Lord, 1998). Kanner’s use of “autism” was intended to describe the autistic child as living in her or his own world; however, his use of the term differed from that of schizophrenia in that it represented a failure of development rather than a regression (Choo, 2007; Kanner, 1943; Volkmar & Lord, 2007). This use of the term “autism” led to confusion regarding the relationship between autism and schizophrenia and impeded research efforts (Volkmar & Lord, 1998; Volkmar & Klin, 2005).

It was not until the early 1970s that researchers were able to resolve the issue regarding the relationship between Schizophrenia and autism (Volkmar & Lord, 2008). Specifically, the work of Kolvin (1971) and Rutter (1970) showed that the two disorders could be differentiated based on their onset patterns, course, and family genetics (Volkmar & Klin, 2005; Volkmar & Lord, 1998). Due to this lapse in time, much of the early research on autism is difficult to interpret as it is based on incorrect assumptions about the relationship between autism and schizophrenia (Volkmar & Klin, 2005).
Some researchers, however, were able to refine the understanding of autism despite Kanner’s initial use of the term “autism,” and many began to disprove some of Kanner’s initial findings regarding characteristics associated with the disorder. For example, it was discovered that autism is often associated with medical conditions and that children with autism often scored in the mental retardation range on IQ tests, both of which ran contrary to Kanner’s report of autism (Volkmar & Lord, 1998). In addition, Kanner originally observed poor parent-child interactions and indicated that the autistic child’s parents tended to be high achieving (Volkmar & Lord, 1998). This observation led clinicians to attribute the disorder to poor child care and parent-child interactions; however, later controlled studies disproved both of Kanner’s original observations (Volkmar & Klin, 2005; Volkmar & Lord, 1998). While there has been much advancement within autism research and the understanding of the characteristics associated with the disorder, autism continues to be an area of focus within the research world. Further advancement continues to be necessary to improve diagnostic accuracy and treatment options.

Asperger’s Disorder. Asperger’s Disorder has been recognized in the literature almost as long as autism but was only recently officially included in the DSM (Volkmar, et al., 2000). Asperger’s Disorder was first introduced by Hans Asperger, a Viennese physician in 1944 (Volkmar, et al., 2000; Volkmar & Lord, 2007; Klin, McPartland, & Volkmar, 2005). In his work, Asperger (1944) described a group of boys with marked social impairments and good language and cognitive skills. In addition, Asperger described the group of boys as having unusual, circumscribed interests and awkward motor skills. He later coined the disorder “autistichen Psychopathen,” or “autistic
personality disorder,” which was somewhat similar to Kanner’s 1943 description of autistic children (Volkmar, et al., 2000). However, neither man was familiar with the other’s work (Volkmar, et al., 2000; Volkmar & Lord, 2007). Despite their similarities, Asperger (1944) suggested many characteristics that were not reported in Kanner’s (1943) work. For example, Asperger noted that the condition he described was only found among males, that the individuals had good use of language, and that the disorder appeared to run in families (Volkmar & Lord, 2007).

Asperger’s work was published in German and received little attention outside of German-speaking countries. It was only after Lorna Wing’s (1981) highly influential review that Asperger’s Disorder began to be highly researched and considered as an unique diagnosis (Klin, Volkmar & Sparrow, 2000; Volkmar & Lord, 2007). Previous research, however, has been unable to fully differentiate Asperger’s Disorder from autism, and the continuity between the diagnoses continues to be a topic of debate (Klin & Volkmar, 1997; Klin, Volkmar & Sparrow, 2000; Volkmar, et al., 2000; Volkmar & Lord, 2007).

**Categorical diagnosis.** There have been many efforts to define autism and Asperger’s Disorder categorically in order to improve the accuracy of diagnosis. The first attempt was made by Rutter (1978), who defined autism as having four essential features. He indicated that a diagnosis of autism required early onset, impaired social development, impaired communication, and unusual behaviors (e.g., motor mannerisms and stereotypes). Rutter’s definition was later reflected in the DSM-III diagnostic criteria for autism (Volkmar & Klin, 2005).
Ritvo and Freeman (1978) offered a definition of autism that also addressed the neurological basis. Their definition included disturbances in development, responses to sensory stimuli, communication, and the ability to relate to others socially. The definition provided by Ritvo and Freeman (1978) was later adopted by the National Society for Autistic Children in the United States. This definition, however, was not as influential as the definition provided by Rutter, as it was more difficult to understand and was not as closely related to Kanner’s original work as Rutter’s definition (Volkmar & Klin, 2005).

The definition of Asperger’s Disorder has been developed over time in relation to the definition of autism, which has complicated the development of a clear diagnostic picture (Volkmar & Klin, 2000). In fact, Asperger’s Disorder first appeared in the ICD-10 with a disclaimer indicating that Asperger’s Disorder may be a variant of autism rather than warranting a separate diagnostic category (Volkmar & Klin, 2005). The overlap of Asperger’s Disorder with autism continues to be a topic of debate, and its definition continues to be examined and refined.

**Current conceptualization.**

*Autism.* The American Psychiatric Association (2000) used an empirical approach to define autism in the DSM-IV-TR. To establish their definition, APA conducted field trials in which nearly 1,000 cases were rated by multiple clinicians, and the interrater reliability was examined (Volkmar & Klin, 2005). The field trial data provided the empirical basis for establishing diagnostic criteria for autism (see Table 1).

For a DSM-IV-TR diagnosis of autism, the individual must exhibit social abnormalities (e.g., avoidance of eye contact, lack of emotional reciprocity, refusal to engage in social interaction), impaired communication (e.g., delayed or inadequate
language development, idiosyncratic language, lack of appropriate social imitative play), and a restricted range of interests and activities (e.g., repetitive head movement, hand flapping; APA, 2000). In addition, the onset of the condition must have been before the age of three. The severity of these core characteristics of autism varies among individuals. Furthermore, clinicians often make a distinction between “low functioning” and “high functioning” autistic individuals, ranging from high intelligence to mental retardation (Gilberg & Ehlers, 1998).

The International Classification of Diseases (ICD) offered diagnostic criteria for autism that is also often cited in the literature (World Health Organization, 1992). The criteria included in the 10th revision of the ICD are similar to the criteria included in the DSM-IV-TR (see Table 2). Specific criteria included the abnormal development in expressive or receptive language, reciprocal social interactions, or functional or symbolic play before three years of age. Additional criteria included impairment in social interaction (e.g., failure to use appropriate nonverbal communication), abnormalities in communication (e.g., delay in the development of spoken language), and restricted, repetitive, and stereotyped patterns of behavior, interests, and activities.

Individuals diagnosed with autism may also exhibit behaviors that are not included in DSM-IV-TR or ICD-10 diagnostic criteria (CDC, 2010), such as hyperactivity, impulsivity, aggression, self injury, or unusual eating habits. They may also show unusual emotional reactions or may have unusual reactions to the ways things smell, feel, or taste. These behaviors are often disruptive to the individual’s daily activities and may affect his/her ability to connect to people and to the world in general.
**Asperger’s Disorder.** To meet diagnostic criteria for Asperger’s Syndrome as listed in the DSM-IV-TR, the individual must exhibit marked impairments in social interaction (e.g., poor eye contact, lack of peer relationships, lack of emotional reciprocity) and a restricted range of interest and activities (e.g., rigid adherence to routines, hand flapping) (APA, 2000; see Table 3). However, they may not exhibit a significant delay in speech, cognitive development, or adaptive functioning. Thus, these individuals are often considered to be higher functioning than those diagnosed with autism (CDC, 2010; see below for a discussion on differential diagnosis of autism and Asperger’s Disorder).

ICD-10 diagnostic criteria for Asperger’s Syndrome required that there be a lack of clinically significant general delay in spoken or receptive language or cognitive functioning. However, the individual must exhibit abnormalities in reciprocal social interaction, as well as unusually intense circumscribed interest or restrictive, repetitive, and stereotyped patterns of behavior, interests, and activities (see Table 4).

In addition, individuals with Asperger’s Disorder are often clumsy, exhibit odd posture, and have poor coordination (CDC, 2010). Furthermore, these individuals tend to lack empathy and have difficulty understanding the subtle nuances of social relationships. As a result, these children are often socially isolated and may often experience bullying. Moreover, individuals with Asperger’s Disorder have marked difficulty in conversational skills and often carry on conversations on topics that are of interest to them but may not be of any interest to others (Volkmar, et al., 1996).

**Differential diagnosis.** Kanner (1943) and Asperger (1944) initially described autism and Asperger’s Disorder as two distinct diagnoses; however, there has long been
debate regarding the continuity of the two diagnostic categories. Indeed, many researchers and clinicians believe that Asperger’s Disorder is merely a milder form of autism and would be better defined as high functioning autism (Gillberg & Ehlers, 1998; Miller & Ozonoff, 2000).

Wing (1991) offered an extensive overview of the similarities between autism and Asperger’s Disorder. For example, both groups show impairments in communication, such as difficulty with social communication, impairment in the use on nonverbal communication, idiosyncratic phrases, and repetitive questioning. Furthermore, both groups exhibit social isolation, difficulty relating to others, lack of emotional reciprocity, and a lack of imaginative play. Finally, both groups engage in stereotypic or odd motor behaviors, as well as exhibit narrow interests or abilities and desire for sameness.

Despite the many arguments that autism and Asperger’s Disorder represent variants of the same disorder, there have been many research efforts to distinguish Asperger’s Disorder from high functioning autism (Klin, McPartland, & Volkmar, 2005). There are five areas on which many researchers have focused to establish differences. These include: (1) motor skills, (2) cognitive ability, (3) language development, (4) social interaction, and (5) right versus left hemisphere dysfunction (Kugler, 1998; Miller & Ozonoff, 2000). This section will highlight the five areas concerning differential diagnosis.

**Motor skills.** Asperger (1944) observed motor skill difficulties in his work, which had not been previously described by Kanner (1943). Poor motor skills continues to be considered a potential feature of Asperger’s Disorder. For example, Gillberg and Ehlers (1998) found that children diagnosed with Asperger’s Disorder exhibited delayed motor
milestones, characterized by clumsiness and a lack of gross motor coordination. Furthermore, some argue that children with autism usually show relative strengths in motor functioning while children with Asperger’s Disorder are clumsy (e.g., Gilberg, 1989; Kugler, 1998). In contrast, however, other researchers have not identified significant differences between the two groups on motor functioning. Szatmari, Bartoluci, and Bremmer (1995) compared individuals diagnosed with Asperger’s Disorder and autism using a standardized measure of early motor skills history and failed to identify any group differences. Furthermore, many researchers have argued that the studies suggesting significant differences in motor functioning between the two groups are often based on the objective opinions of clinicians rather than standardized measures of motor functioning and when standardized tests of motor skills have been used the results have been inconsistent (Kugler, 1998; Miller & Ozonoff, 2000; Smith, 2000; Volkmar & Klin, 2000). As a result of these inconsistent research findings, motor skills deficits have not been included as diagnostic criteria for Asperger’s Disorder in either the DSM-IV-TR or the ICD-10 (Miller, 2000).

Cognitive ability. Asperger (1944) described his patients as being of average or high intelligence, and this continues to be consistent with diagnostic criteria for Asperger’s Disorder in the DSM-IV-TR and the ICD-10. Kanner (1943) also described his patients as being of average intelligence, but later research suggested that many individuals diagnosed with autism fell within the mental retardation range on tests of intelligence (Miller & Ozonoff, 2000). Therefore, cognitive ability is often cited as a variable that differentiates autism from Asperger’s Disorder. However, mental retardation or below average cognitive ability is not included in the diagnostic criteria for autism. For
example, previous research has suggested that certain interventions (e.g., applied behavioral analysis) may lead to improvements in the child’s performance on measures of intellectual functioning, resulting in higher IQ scores (e.g., Ferretti, et al., 2010). However, improvements in IQ does not warrant change in the child’s diagnosis from autism to Asperger’s Disorder since IQ is not included in diagnostic criteria.

Furthermore, there have now been many accounts of high functioning autism, which is defined as an individual who meets full criteria for autism with full scale IQ scores greater than 70 (Miller & Ozonoff, 2000). Therefore, there appears to be some overlap in cognitive abilities among autistic and Asperger’s Disorder groups as well.

**Language impairment.** Language development is another area of contention in which researchers have attempted to differentiate high functioning autism from Asperger’s Disorder (Gillberg & Ehlers, 1998; Kugler, 1998; Miller & Ozonoff, 2000). Again, diagnostic criteria require that individuals have normal speech development to be diagnosed with Asperger’s Disorder. There are, however, no requirements regarding the age of speech development to obtain a diagnosis of autism, which allows room for overlap in the diagnoses of autism and Asperger’s Disorder (Miller & Ozonoff, 2000).

On the other hand, Kanner (1943) and Asperger (1944) indicated that language impairment is a defining characteristic of both autism and Asperger’s disorder. Therefore, researchers have attempted to define difference in language impairment between autistic and Asperger’s Disorder groups. While the results have largely been mixed among studies, researchers have generally found that individuals diagnosed with autism are more deviant than those diagnosed with Asperger’s Disorder in language and communication (Kugler, 1998). More specifically, individuals with autism appear to have more difficulty
with both early behaviors (e.g., babble, echolalia, pronoun reversal, and repetitive speech) and behaviors assessed later in life (e.g., articulation, vocabulary, and verbal output).

**Social interaction.** Another distinction between individuals diagnosed with autism and Asperger’s Disorder relates to their interest in social interactions (Autism Society of America, 2010; Kugler, 1998; Van Krevelen, 1971). Individuals diagnosed with autism are often viewed as aloof and uninterested in others. Children diagnosed with Asperger’s Disorder, on the other hand, are interested in interacting with others and often want to fit in with their peers but lack the necessary skills to do so. Van Krevelen (1971) stated that the child diagnosed with autism “lives in a world of his own,” while the child diagnosed with Asperger’s Disorder “lives in our world in his own way” (p. 84).

**Right vs. left hemisphere dysfunction.** A final area of contention in the differential diagnosis debate includes the argument for differing patterns of hemispheric dysfunction among autism and Asperger’s Disorder. Specifically, previous researchers have postulated that while children with high functioning autism tend to exhibit patterns of left hemisphere dysfunction, children diagnosed with Asperger’s Disorder likely exhibit right hemisphere dysfunction (Volkmar & Klin, 1998). This argument has been fueled by the phenomenological similarities between Asperger’s Disorder and Nonverbal Learning Disorder, a disorder that occurs as result of developmental right brain dysfunction (Klin, Volkmar, Sparrow, Cicchetti, & Rourke, 1995). Rourke (1989, 1995) proposed that Nonverbal Learning Disorder develops from damage to the commissural fibers and right hemispheric associational white matter tracts, which causes dysfunction in communication to the right hemisphere. As a result of this right hemisphere
dysfunction, Nonverbal Learning Disorder is characterized by a cluster of deficits, such as nonverbal problem solving, visuospatial organization, psychomotor coordination, and tactile perception. In addition, Nonverbal Learning Disorder is characterized difficulty understanding and expressing pragmatic and prosodic aspects of language and difficulty in adapting to novel and complex situations. These deficits often result in problems of social perception, social judgment, and social interaction.

Given the striking similarities between Asperger’s Disorder and Nonverbal Learning Disorder characteristics, efforts have been made toward examining the neuropsychological profiles of children diagnosed with Asperger’s Disorder in hopes of gaining further insight into the continuum between the Asperger’s Disorder and Nonverbal Learning Disorder. For example, Gilberg (1991) found that individuals diagnosed with Asperger’s Disorder tend to show higher performance IQ scores than verbal IQ scores, which is consistent with the typical Nonverbal Learning Disorder IQ profile. Klin, et al. (1995) found that while the neuropsychological profile of children diagnosed with Asperger’s Disorder differed from that of children diagnosed with autism in 11 neuropsychological areas (i.e., fine motor skills, visual motor integration, visual spatial perception, nonverbal concept formation, gross motor skills, visual memory, articulation, verbal output, auditory perception, vocabulary, and verbal memory), the Asperger’s Disorder profile coincided closely with the neuropsychological profile captured by the term Nonverbal Learning Disorder. Overall, there has been an increasing number of studies that provide evidence for a convergence between NLD and AS syndromes (see Ellis & Gunter, 1999 for a review), suggesting that there may be at least two types of conditions at the higher end of the spectrum of severe social disabilities: one
type with predominately left hemisphere involvement (i.e., high functioning autism) and the other with right hemisphere involvement (i.e., Asperger’s Disorder/Nonverbal Learning Disorder; Klin, et al., 1995). On the other hand, Klin, et al. (1995) also noted that while high functioning autism and Asperger’s Disorder may be differentiated based upon the reported 11 neuropsychological tests, “the conditions may still share the same etiology or other pathogenetic processes while having phenotypic difference solely accounted for by neuropsychological differences” (p. 1138). Thus, Asperger’s Disorder and high functioning autism may be viewed as being the same diagnostic entity but differing in their neuropsychological profiles, much the same as the differences between lower functioning and higher functioning autism.

In order to explore further the right versus left hemisphere differentiation in high functioning autism and Asperger’s Disorder, researchers have sought evidence through brain imaging studies, though such research is limited. For example, McAlonan, et al. (2009) conducted a study in which they mapped white matter volumes among children diagnosed with high functioning autism, children diagnosed with Asperger’s Disorder, and a control sample through voxel-based morphometry. They found that compared to the control group, the high functioning autism sample had less frontal and corpus callosal white matter in the left hemisphere and the Asperger’s Disorder sample has less frontal and corpus callosal white matter in the right hemisphere, suggesting that the etiological factors between the groups may be distinct. McKelvey, Lambert, Mottron, and Shevell (1995) conducted a study of three children diagnosed with Asperger’s Disorder and found that all three children had abnormal right hemisphere functioning on single-photon emission computed tomography (SPECT) imaging. Volkmar, et al. (1996) found
evidence of more prominent cerebral abnormalities in the right side in an adolescent boy with Asperger’s Disorder. A magnetic resonance imaging study of seven children diagnosed with Asperger’s Disorder and co-morbid Tourette’s syndrome revealed mostly right-sided abnormalities (Berthier, Bayes, & Tolosa, 1993). Finally, neurological signs of right hemisphere impairments (i.e., left-sided difficulties) have been described in two individuals diagnosed with Asperger’s Disorder (Berthier, Starkstein, & Leiguarda, 1990). These studies thus provide evidence to suggest that patients with Asperger’s Disorder may have damage to, or dysfunction of, associational white matter tracts that are particularly deleterious to the functioning of the right cerebral hemisphere (Gunter, Ghazuiddin, & Ellis, 2002). In contrast, however, the left temporal lobe has also been implicated in Asperger’s Disorder (Jones & Kerwin, 1990).

**Summary.** While many researchers and clinicians have attempted to differentiate the high functioning autism and Asperger’s Disorder based upon a multitude of constructs and characteristics, there is currently no definitive answer on this matter. Further complicating this debate is the heterogeneous nature of the populations diagnosed with both disorders. Differential diagnosis, however, remains a significant aspect of understanding and treating individuals on the autism spectrum. Therefore, it is important that efforts be made at clarifying the continuum or differentiation of high functioning autism and Asperger’s Disorder.

**The roles of pleiotropy and epigenesis.** According to Courchesne, Yeung-Courchesne, and Pierce (1999), pleiotropy and epigenesis are “two biological mechanisms and conditions that are designed to create efficiency and adaptive neuroplasticity during development” that “open the door to many different possible
developmental outcomes” (p. 307). They argued, however, that these two mechanisms leave the cortical system vulnerable to abnormal interjections from various stimuli, such as abnormal genetic or environmental conditions.

Luria proposed the Hierarchical Model of Cortical Functioning (for review, see Kolb & Wishaw, 1996), which posits that there is a hierarchy of cortical zones that form an interdependent system. He suggested that any disruption in one cortical zone is likely to affect another cortical zone. For example, it is well known that a focal brain lesion will lead to initial loss in the associated area, but then later lead to loss of function in other interconnected areas of the system (i.e., diaschisis). Similarly, Courchesne, et al. (1999) suggested that any abnormal interjection at any site may in turn trigger further abnormal development in other sites of the brain. They argue that the two mechanisms, pleiotropy and epigenesis, are likely the cause for the heterogeneous nature of symptoms and behaviors across individuals cases of ASD.

**Pleiotropy of ASDs.** Pleiotropy is defined as the “diverse effects of a single gene or gene pair on several organ systems and functions” and has been found to be a mechanism for genetic coding efficiency (Courchesne, et al., 1999, p. 308). In contrast, this efficiency is also responsible for creating the possibility of any disruption in a single system to also cause multiple systems or developmental jobs to fail. Courchesne, et al. (1999) suggest that this phenomenon may also describe the heterogeneity found among individuals on the autism spectrum. Specifically, individuals on the autism spectrum may have a single or multiple gene mutations, which would be sufficient to account for the heterogeneous phenotypes seen in autism spectrum disorders (i.e., autism versus Asperger’s Disorder, etc.). This hypothesis has been supported in the literature through
accounts of single gene mutations that have led to heterogeneous behavioral abnormalities in humans and in animals, including phenylketonuria (PKU), fragile-X syndrome, and Lesch-Nyhan syndrome.

**Epigenesis of ASDs.** Epigenesis is defined as a “process by which an organism develops from an undifferentiated cell through successive formation and development of organs and parts that do not pre-exist in the fertilized egg” (Courchesne, et al., 1999, p. 313). At each phase of development, the organ is impacted by the environment surrounding the cells, which may alter the development outcome resulting in variant forms of the “normal” system. In autism spectrum disorders, when the epigenetic process is interjected by abnormal occurrences, the process will lead to many different possible developmental outcomes, further triggering abnormal development in other sites of the brain, as well as other areas of functioning. This process is further impacted by the differential environmental conditions among children on the autism spectrum, which causes further heterogeneous behavioral and symptom outcomes.

**Divergence and convergence.** Pleiotropy and Epigenesis, however, does not explain why two individuals on the autism spectrum with same genetic defects exhibit differences in the types or levels of severities of the abnormality. Courchesne, et al. (1999), however, argued that this is likely to arise from differences in their experienced contingent events (e.g., differing environments). This process is referred to as divergence heterogeneity.

Furthermore, while pleiotropy and epigenesis explain why each person on the autism spectrum has multiple abnormalities and why no two autistic people have exactly the same type or severity of autism (e.g., autism versus Asperger’s Disorder), it is also
possible that there is more than one etiology that can trigger the development of an ASD (Courchesne, et al., 1999). Specifically, it is possible that many of the most common outcomes associated with ASDs (e.g., cerebellar abnormalities, attention abnormalities, communication abnormalities, etc.) might be associated with different, unrelated etiologies that lead to overlapping or common biological or behavioral outcomes. Thus, individuals with autism and Asperger’s Disorder may in fact have differing underlying etiologies that lead to overlapping and similar symptom presentations.

**Conclusion.** While the pleiotropy and epigenesis hypotheses provide a different way to conceptualize the continuum of autism and Asperger’s Disorder, differential diagnosis remains unclear and a hotly debated argument among researchers. Thus, further research is needed to continue to clarify the etiology of the disorders.

**Proposed DSM-V changes for autism and Asperger’s Disorder.** Despite the ongoing debate regarding differential diagnosis of autism and Asperger’s Disorder, the DSM-V Committee proposed changes to the categorization and diagnostic criteria for autism spectrum disorders (APA, 2010; see Table 5). Specifically, the proposal suggested that the previously distinct diagnoses of autistic disorder, Asperger’s disorder, childhood disintegrative disorder, and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS) be subsumed under one single category labeled Autism Spectrum Disorder. According to the DSM-V Committee, this change has been suggested due to the inconsistencies in the ability to distinguish the disorders over time. Furthermore, they argued that often the variables that have been relied upon to distinguish the disorders on the autism spectrum have been associated with severity, language level or intelligence rather than features of the disorder. Finally, the DSM-V committee posited that because
autism disorders are defined by a common set of behaviors, a single diagnostic category that includes clinical specifiers (e.g., severity level, verbal abilities, etc.) and associated features (e.g., known genetic disorders, epilepsy, intellectual disability, etc.) would best represent the entire autism spectrum.

In addition to the change in diagnostic categorization, the DSM-V Committee proposed making changes to the domains previously included in diagnostic criteria. Specifically, the proposed change collapses the domains of social interaction and communication into a single domain, labeled “social/communication deficits,” while the domain of “fixed interests and repetitive behaviors” remains in the diagnostic criteria. The DSM-V Committee argues that this change will allow for a more accurate description of the disorder, as deficits in communication and social behavior are inseparable. They also argued that because language delays are not universal in ASDs, they should not define the ASD diagnosis. Rather, delays in language should be considered a factor that influences the overall clinical picture of the diagnosis.

Other changes to be included in the DSM-V include the merging of several social/communication criteria, requiring two symptom manifestation for repetitive behavior and fixated interests, including unusual sensory behaviors in the subdomain of stereotyped motor and verbal behaviors, and highlighting the importance of gaining multiple sources of information (e.g., clinical observation, parent/teacher report, etc.) to meet criteria.

Among the changes proposed by the DSM-V committee, the deletion of Asperger’s disorder as a distinctive diagnostic category is likely the most controversial change. For example, Ghaziuddin (2011) argued that the Asperger’s Disorder diagnosis
has become “clinically useful to describe certain types of individuals who may respond to a different set of interventions than those with typical autism” (p. 192). Others have also pointed out that the Asperger’s Disorder diagnosis has increased awareness of milder forms of autism and allowed for the creation of specialized services (Wing, 2005). Ghaziuddin (2011), therefore, suggested that until there are specific biological markers for autism, the diagnosis of autism spectrum disorders should also be based on clinical utility (Ghaziuddin, 2011).

Currently, the DSM-V is scheduled to be published in 2013. The proposed criteria for Autism Spectrum Disorder to be included in DSM-V are provided in Table 5. Included in Table 6 are the proposed severity specifiers for the “Social Communication” and “Restricted interests and repetitive behaviors” criteria included for a diagnosis of Autism Spectrum Disorder.

**Assessment and Identification of Autism and Asperger’s Disorder**

Assessing children with suspected autism or Asperger’s disorder requires a multilevel and multifaceted approach, as well as professionals who are well versed in the assessment of autism spectrum disorders (Filipek, et al., 1999; Klin & Volkmar, 1999; Volkmar, et al., 2005). A thorough assessment has the potential to serve many purposes for the child and the child’s family. Initially, the assessment allows for an accurate diagnosis and often establishes the child’s eligibility for educational and intervention services (Volkmar, et al., 2005). Additionally, proper assessment will provide an evaluation of the child’s strengths and weaknesses and guide needed interventions for the child, as well as assist the child’s family in understanding the child’s overall presentation and level of need (Marcus, Flagler, & Robinson, 2001). While this rigorous approach to
assessments are often time consuming and not without complication, it is in the best interest of the child to conduct an accurate and complete assessment.

**ASD screening measures.** Previous research suggested the importance of *screening* to identify autism spectrum disorders in young children. The screening process is characterized by the use of brief measures to identify children in need of further assessment to rule-in or rule-out an autism spectrum disorder (Coonrod & Stone, 2005). While an autism spectrum diagnosis cannot be made on the basis of screening measures, this process helps to aid in the early identification and intervention of autism spectrum disorders.

According to Lord, Risi, and DiLavore (1999), screening measures should be performed on all children at their regular wellness checks. Such measures help to distinguish children who may be at risk for any atypical development from typically developing children. For this process, the physician or other healthcare professional should always engage the child’s parents in a discussion regarding any concerns they may have about the child’s development (Filipek, et al., 1999). In addition, it is common practice to administer standardized measures of development, such as the Ages and Stages Questionnaire (Bricker & Squires, 1999), the BRIGANCE Screens (Glascoe, 1996), or The Child Development Inventories (Ireton & Glascoe, 1996) (for review, see Filipek, et al., 1999).

There are also screening measures that specifically assess for autism spectrum disorders. These measures include the Modified Checklist for Autism in Toddlers (M-CHAT) and the Pervasive Developmental Disorders Screening Test-Stage 1. The M-CHAT (Robins, et al., 2001) is a parent-report measure used to identify children 16-30
months old at risk for autism. Specifically, the M-CHAT assesses for behaviors commonly associated with autism (e.g., repetitive behaviors, pretend play, gaze monitoring). In addition, there is a structured, follow-up interview that may be conducted with the parents/caregivers of the child to gain more specific information. A study was conducted to assess the psychometric properties of the M-CHAT and revealed specificity values ranging from .95 to .99, sensitivity values ranging from .95 to .97, positive predictive values ranging from .36 to .79, and negative predictive value of .99. Overall, this measure appears to be promising in screening for autism.

The Pervasive Developmental Disorders Screening Test-Stage 1 (PDDST; Siegal & Hayer, 1999) is also a parent-report questionnaire and was designed to screen for autism spectrum disorders in children under six years of age. Items on this measure focus on various behaviors commonly associated with autism spectrum disorder (e.g., nonverbal communication, language, play, social interaction, etc.). An initial study on the PDDST psychometric properties revealed specificity values of .71 and sensitivity values of .85.

**ASD rating scales.** Rating scales are also commonly used in the assessment of children with a suspected autism spectrum disorder. These measures are often used to help to distinguish children who are at risk for autism spectrum disorders from those who are risk for other developmental disorders (Coonrod & Stone, 2005). There are many measures that are specific to autism and Asperger’s Disorder available for use. The most widely used and well researched measures include the Childhood Autism Rating Scale, Gilliam Autism Rating Scale, Social Responsiveness Scale, Social Communication
The Childhood Autism Rating Scale (CARS; Schopler, Reichler, DeVellis, & Daly, 1980) is the most widely used and well researched standardized instrument designed to assess for behaviors associated with autism in young children. The CARS is comprised of 15 items on which children and adults are rated after being observed by a professional. In addition, the CARS includes a severity rating that is often used in the assessment of long-term outcomes. The CARS total score has been repeatedly examined and has been shown to be reliable and internally consistent (Lord & Corsello, 2005).

The Gilliam Autism Rating Scale (GARS; Gilliam, 1995) is another parent-completed questionnaire used to assess for autism in individuals aged 3-22 years. In addition, the measure may be completed by teachers and professionals familiar with the child. The GARS contains 56 items distributed across four subscales: Social Interaction, Communication, Stereotyped Behaviors, and Developmental Disturbances. The GARS has been found to have adequate reliability and validity but has also been found to underidentify autism in young children (Chawarska, Klin, & Volkmar, 2008; Goldstein, Naglieri, & Ozonoff, 2009).

The Social Responsiveness Scale (SRS; Constantino, 2002) is a parent or teacher rated questionnaire developed to distinguish autism spectrum disorders from other child psychiatric disorders. More specifically, the SRS is a 65-item questionnaire that helps to identify the presence and extent of autistic social impairment through questions that focus on child’s ability to engage in emotionally appropriate reciprocal social interactions. The SRS is appropriate for use with children, aged 4-18 years. The SRS shows good
reliability and validity; however, there does appear to be some overlap in scores when comparing children on the autism spectrum to typically developing children. Therefore, the SRS is best used to assess for symptom severity and response to treatment rather than for use as a diagnostic measure (Lord & Corsello, 2005).

The Social Communication Questionnaire (SCQ; Eaves, et al., 2006) is a parent rated questionnaire containing 40 yes-or-no items regarding behaviors associated with autism. Questions on the SCQ were created based on a parent interview measure, the Autism Diagnostic Interview-Revised (see below), and correspond to a diagnosis of autism from the DSM-IV-TR (APA, 2000). The measure is appropriate for use with children aged four years and older with a mental age of at least two years. Sensitivity of the SCQ was measured at 0.85 and specificity was measured at 0.75.

The Gilliam Asperger’s Disorder Scale (GADS; Gilliam, 2001) is designed to evaluate children with suspected Asperger’s disorder. Specifically, the GADS is completed by a caregiver and assesses for behavioral disturbances that are required for a diagnosis of Asperger’s disorder. The measure is appropriate for individuals aged 3-22 years, and questionnaire items are divided into four subscales, including Social Interaction, Restricted Patterns of Behavior, Cognitive Patterns, and Pragmatic Skills. Both validity and reliability have been supported for the GADS.

The Australian Scale for Asperger’s Syndrome (ASAS; Attwood, 1997) is a widely used instrument in schools and among parents. The ASAS has not been included in peer reviewed published papers but has been made available on a public website (Lord & Corsello, 2005). It has gained much popularity due to its accessibility. The ASAS contains 19 items that are scored on a 7-point scale ranging from “rarely” to “frequently.”
The items on the scale focus on behaviors associated with Asperger’s disorder, including social and emotional difficulties, cognitive skills deficits, communication skills deficits, specific interests, and motor clumsiness. This measure has faced some scrutiny due to the lack of peer reviewed research on the reliability and validity of the measure.

“Gold standard” ASD measures. In children who are identified as being at risk for an autism spectrum disorder during an assessment using screening measures, it is important that a more thorough assessment be conducted to verify a diagnosis. Two widely used measures include the Autism Diagnostic Interview-Revised and the Autism Diagnostic Observation Schedule. These measures are considered to be the “gold standard” in the assessment of children with suspected autism spectrum disorders for research purposes (Filipek, 1999).

The Autism Diagnostic Interview-Revised (ADI-R; Lord, et al., 1994) is a structured parent interview that engages the parent or caregiver in questions regarding behaviors commonly associated with autism spectrum disorder. More specifically, the interview focuses on social relatedness, communication, and ritualistic or perseverative behaviors. The interview is consistent with diagnostic criteria for autism spectrum disorder according to the DSM-IV-TR (APA, 2000) and ICD-10 (WHO, 1992, 1993) and provides threshold scores for the diagnosis of Autistic Disorder. Administration of the ADI-R takes approximately one hour and requires specific training and validation procedures. Because of these requirements, the ADI-R is less frequently used in a clinical context.

The Autism Diagnostic Observation Schedule (ADOS; Lord, Rutter, DiLavore, & Risi, 1999) is a semi-structured observational assessment of children with suspected
autism spectrum disorder. The ADOS allows for the assessment of verbal and nonverbal children and adults and takes approximately 30-45 minutes to administer. There are four modules within the ADOS, which include investigator-directed activities to evaluate communication, reciprocal social interaction, play, stereotypic behavior, restricted interests, and other abnormal behaviors. Similar to the ADI-R, the ADOS allows the clinician to assess for characteristics consistent with diagnostic criteria in the DSM-IV-TR (APA, 2000) and ICD-10 (WHO, 1992, 1993). In addition, the ADOS also provides threshold scores for the diagnosis of Autistic Disorder. The administration of the ADOS requires specific training and validation procedures. Because administration of the ADOS takes less time than the ADI-R, many autism specialty professionals are using this instrument in their clinical practices.

The Autism Diagnostic Observation Schedule, Second Edition (ADOS-2; Lord, et al., 2012) is scheduled to be released in the United States in late 2012. It currently has only been released in Canada. Similar to the ADOS, the ADOS-2 is a semi-structured, standardized assessment measure used to examine a child’s communication, social interaction, play, and restricted and repetitive behaviors. The new edition will provide updated protocol booklets that offer more clear and explicit instructions for the examiner. The updated measure will also provide new algorithms for scoring that will allow that examiner to compare the child’s results across modules more uniformly. Furthermore, there will be a new Comparison Score that allows the clinician to compare the child’s level of autism symptoms to that of children diagnosed with ASD who are the same age and have similar language skills. Finally, the updated ADOS will provide a new module for toddlers, aged 12-30 months, who do not consistently use phrase speech.
Neuropsychological assessment. In addition to assessing the child for behaviors and symptomatology consistent with an autism spectrum disorder diagnosis, the neuropsychological evaluation involves the assessment of the child’s specific strengths and weaknesses to establish a baseline for the child; to document changes in behavior and overall functioning; and to better inform treatment (Corbett, Carmean, & Fein, 2009; Marcus, Flagler, & Robinson, 2001). Furthermore, neuropsychological assessment results may help to identify specific target behaviors that may be addressed at the intervention level, as well as help to identify useful strategies that assist at the intervention level.

Common areas of assessment among neuropsychologists include intellectual functioning, adaptive behavior, achievement, speech/language, attention, memory, sensory functioning, motor functioning, executive functioning, visual-spatial skills, and social-emotional skills (Corbett, Carmean, & Fein, 2009; Klin, Saulnier, Tsatsanis, & Volkmar, 2005).

In addition to understanding the individual child’s neuropsychological profile, assessment results also help to inform research and theories regarding the underlying mechanisms of autism spectrum disorders. There is currently no consensus on a specific neuropsychological profile of ASDs, nor is there consensus on the underlying mechanisms of ASDs. Rather, it has been argued that further research involving the assessment of children diagnosed with an ASD is in need to gain a better understanding of these disorders (Corbett, Carmean, & Fein, 2009).

Prevalence

The prevalence of autism was first studied in 1966, at which time a rate of 4 per 10,000 was found (Steiman, M., Simon, R., Reisinger, L., & Fombonne, E., 2010). More
recent studies, however, report a continued increase in the prevalence of autism
diagnoses. Specifically, Fombonne (2005a) reviewed studies conducted between the
years of 1994 and 2004 and found that prevalence rates had risen to approximately 12.7
per 10,000. Chakrabarti and Fombonne (2005) collected data between 1999 and 2003 and
found the prevalence of autism to be 22.0 per 10,000. Currently, conservative estimates
of autism prevalence are 13.0 per 10,000 (Fombonne, 2005b).

There have been far fewer studies on the epidemiology of Asperger’s Disorder
than on autism (Fombonne, 2005a; Gillberg & Ehlers, 1998). This is likely due to the late
inclusion of Asperger’s Disorder in the ICD and DSM. The available data suggests that
the prevalence of Asperger’s Disorder is lower than that of autism (Fombonne, 2005a;
Fombonne, 2005b). Current estimates of Asperger’s Disorder prevalence are
approximately 2.6 per 10,000.

The U.S. Centers for Disease Control and Prevention (CDC; 2012) recently
released updated prevalence rates of all autism spectrum disorders, including children
who had been given a DSM-IV-TR diagnosis of Autistic Disorder, Asperger’s Disorder,
or Pervasive Developmental Disorder-Not Otherwise Specified. The findings from the
study were based upon data collected from 14 sites within the Autism and Developmental
Disabilities Monitoring (ADDM) Network during the 2008 surveillance year. Each
ADDM site represents a specific geographic area within the U.S. that gathers data from
health and special education records regarding the number of ASD diagnoses within that
area. Findings from the 2008 surveillance year suggested that 11.3 in every 1,000 (or 1 in
88) children in the U.S. had been diagnosed with ASD. This number represented a 40-
fold increase in prevalence of such diagnoses over the past 40 years.
While the evidence suggests a significant increase in the incidence of autism and Asperger’s Disorder among children in the U.S., there are potential sources of bias that may impact the increases in prevalence being reported. For example, one factor that may influence the autism prevalence rates include an increased awareness regarding autism spectrum disorders among clinicians and other professionals, parents, and the general public (Williams, Mellis, & Peat, 2005). Other potential sources of bias include changing diagnostic criteria over time, broadening of the autism concept, as well as improved services (Fombonne, 2008). Despite these possible sources of biases included in the changing prevalence rates, it is apparent that incidence rates of autism and Asperger’s Disorder are relatively high. Therefore, it is important that autism and Asperger’s Disorder continue to be the focus of research and treatment efforts.

**Neural Theories of Autism Spectrum Disorders**

In an effort to better understand the neurological underpinnings of phenotypic characteristics of individuals diagnosed with autism spectrum disorders, researchers and theorists have developed various theories and models of the core ASD characteristics. One such group of theories includes neural theories, which describe differing regions of the brain that are supported by research as being involved in ASDs. The various regions of the brain implicated in ASDs most supported in the literature include the frontal lobe, cerebellum, and temporal lobe (Schroeder, J.H., Desrocher, M., Bebko, J.M., & Cappadocia, M.C., 2010).

**Frontal lobe.** A neural theory that has received much attention among researchers is the frontal lobe theory. This theory offers an explanation for many areas of deficit in children with autism or Asperger’s disorder and has been supported by MRI studies, as
well as studies involving neuropsychological assessment. This section will discuss the support for the frontal lobe theory from MRI studies. Support from neuropsychological assessment will be discussed later in the Cognitive Impairment Theories section.

According to Schroeder, et al. (2010), the frontal lobes are responsible for many functions that have been implicated in autism and Asperger’s disorder. Specifically, the prefrontal cortex is involved in executive functioning, which includes skills such as planning, organization, set shifting, and cognitive flexibility. All of these executive functioning skills have been found to be deficient in children diagnosed with autism or Asperger’s disorder.

The frontal lobe also contains several neural regions important in language, including Broca’s area which has been found to be responsible for language production. Dysfunction in this area of the brain have been linked to the language deficits commonly associated with autism spectrum disorders. In particular, children diagnosed with autism often exhibit language difficulties ranging from complete lack of speech to difficulties with pragmatic language. These difficulties suggest that frontal lobe dysfunction may be responsible for characteristics of ASDs.

Several neuroimaging studies support the frontal lobe hypothesis associated with autism and Asperger’s Disorder (Schroeder, et al., 2010). For example, Carper and Courchesne (2000) discovered that children diagnosed with autism were found to have decreased frontal lobe size, as well as increase cerebellum size. Another study by Dapretto, et al. (2006), found that boys diagnosed with high-functioning autism and Asperger’s Disorder showed reduced activity in the frontal lobes while observing and imitating emotional expressions of others compared to typically developing children.
Thus, results from neuroimaging studies further support the frontal lobe hypothesis of autism and Asperger’s Disorder.

**Cerebellum.** Historically, the cerebellum has been known to regulate sequenced movement (Schroeder, et al., 2010). However, recent research has also implicated the cerebellum in a variety of psychological functions, including procedural learning, emotion, thought, and attention. The cerebellum receives projections from the cortex and most sensory systems, integrates this information, and then projects to all major motor systems, the thalamus, and some sensory cortices.

There are several characteristics associated with autism and Asperger’s Disorder that suggest a connection between the cerebellum and the expression of ASDs (Schroeder, et al.). Specifically, children diagnosed with autism and Asperger’s Disorder are often described as being clumsy and uncoordinated, which is consistent with cerebellum dysfunction. Furthermore, individuals with autism and Asperger’s Disorder often exhibit language deficits, which may be viewed as sequenced movement mediated by the cerebellum. Finally, children with autism and Asperger’s Disorder often exhibit difficulty in their ability to shift attention (e.g., difficulties shifting attention may lead to repetitive and restrictive behaviors and difficulties transitioning activities, which are commonly found in ASDs), which has also been found to be associated with cerebellum functions.

The cerebellum hypothesis has been further supported by neuroimaging studies (Schroeder, et al., 2010). Studies using magnetic resonance spectroscopy (MRS) found the children with autism and Asperger’s Disorder had decreased neuronal function in the cerebellum (Chungani, Sundram, Behen, Lee, & Moore, 1999; Devito, et al., 2007).
Furthermore, an MRI study conducted on boys diagnosed with autism found that their large frontal lobes were associated with smaller cerebellar volume, suggesting an anatomic relationship accounting for shared pathologies involving the frontal lobe and cerebellum (Carper & Courchesne, 2000).

Thus, research involving the cerebellum and ASDs suggests that cerebellar function may play a role in impaired motor, attention, language and cognitive functioning commonly found in individuals with autism and Asperger’s Disorder. In addition, cerebellar dysfunction may interact with frontal lobe impairments in individuals with ASD (Schroeder, et al., 2010).

**Temporal lobe.** The temporal lobe has been found to be responsible for functions such as audition, memory, and object perceptions. It is also responsible for many areas of functioning found to be deficient in autism and Asperger’s Disorder (Schroeder, et al., 2010). The temporal lobe and, more specifically, Wernicke’s Area are responsible for receptive language, which has been found to be problematic in autism and Asperger’s Disorder. Furthermore, the temporal lobe is involved in functions associated with social cognition, including joint attention, action observation, and empathy, which may underlie the social deficits characteristic of autism and Asperger’s Disorder. Neuroimaging studies have also been used to support the cerebellum hypothesis associated with autism and Asperger’s Disorder. MRI studies have found that unlike typically developing children, individuals with autism showed no correlation between the volume of the superior temporal gyrus and scores on IQ and language measures, which may account for the language deficits associated with ASDs (Bigler, et al., 2007). Thus, the temporal lobe
may be involved in the language and social deficits commonly associated with autism and Asperger’s Disorder.

**Cognitive Impairment Theories of Autism Spectrum Disorders**

Since Kanner (1943) and Asperger (1944) first described autism and Asperger’s Disorder, many researchers and theorists have offered differing theories to explain the disorders (Rajendran & Mitchell, 2007). Beginning in the 1980s, researchers began to point to a primary cognitive deficit as the component underlying the core elements of autism and Asperger’s Disorder. Since then, research has been dominated by three main theories to explain autism (Beaumont & Newcombe, 2006; Rajendran & Mitchell, 2007; Schultz, Charwarska, & Volkmar, 2006). These theories include weak central coherence, Theory of Mind deficits, and the main focus of this project, theory of executive dysfunction (Adler, Nadler, Eviatar, & Shamay-Tsoori, 2010; Baron-Cohen, et al., 2005; Pellicano, 2010; Rajendran & Mitchell, 2007; Schultz, Charwarska, & Volkmar, 2006).

In this section, I will provide a brief overview of the weak central coherence and Theory of Mind hypotheses, as well as an in depth overview of executive dysfunction in autism and Asperger’s Disorder.

**Weak central coherence.** Frith (1989) and Frith and Happe (1994) presented the weak central coherence hypothesis, which refers to the autistic individual’s preferential attention to parts rather than the whole. In addition, the weak central coherence hypothesis posits that these individuals tend to be unable to interpret information within context. In contrast, typically developing individuals process information by understanding the overall message or the gist (Frith, 1989; Happe, 1996; Rajendran & Mitchell, 2007). For example, Frith (1989) argued that children with autism have an
extraordinary ability to focus on the detail or parts, but have difficulty “seeing the forest for the trees.”

There have been several studies that have exhibited this tendency among individuals on the autism spectrum. For example, Hermelin and O’Connor and Tager-Flusberg found that children with autism were almost equally successful at recalling meaningful sentences and random word strings, whereas typically developing children and children diagnosed with mental retardation recalled the meaningful sentences better than the random word strings (as cited in Happe & Frith, 1996, p. 1388).

Furthermore, while most theories of autism tend to focus only on deficiencies, the weak central coherence hypothesis also offers insight into the assets that can be found in autism (Happe, 1996; Happe & Frith, 1996). Shah and Frith (1993) conducted a study in which autistic subjects performed better than the control sample on block design tasks regardless of age and ability. Happe (1996) offered another example of the weak central coherence hypothesis in a study that examined perceptions of illusory figures. Happe (1996) found that individuals with autism made more accurate judgments of illusory figures, or visual illusions, than control subjects and attributed this to the autistic individual’s failure to extract the overall gist from the figures. That is, because the autistic individual did not grasp the gist, they were not susceptible to the illusion and were better able to pick out the parts.

Such research findings have offered explanations for characteristics of autism spectrum disorders. Specifically, the weak central coherence hypothesis explains the social deficits found among individuals diagnosed with autism spectrum disorders as stemming from difficulties in grasping the social context that typically modulates social
behavior (Schultz, Chawrwarska, & Volkmar, 2006). This hypothesis may also account for savant skills (Happe, 2005). For example, Pring, Hermelin, and Heavey (1995) found that autistic children with savant art skills likely see wholes in terms of their parts, which appears to be a general characteristic of individuals with an aptitude for drawing.

The weak central coherence hypothesis is a very influential cognitive model for autism, but there is currently little research evidence to support this hypothesis. Moreover, the available research studies provide inconsistent results, making it difficult to determine the validity of the weak central coherence hypothesis (Beaumont & Newcombe, 2006; Rajendran & Mitchell, 2007; Schultz, Chawrwarska, & Volkmar, 2006).

**Theory of mind.** Premack and Woodruff (1978) developed the Theory of Mind hypothesis in 1978, which described an individual’s “ability to attribute mental states to oneself and others and to predict the behavior of others based on their mental states” (Spek, Scholte, & Berckelaer-Onnes, 2009, p. 280). Later, Baron-Cohen, Leslie, and Frith (1985) applied the Theory of Mind hypothesis to autism spectrum disorders. In their study, participants completed the unexpected transfer test of false belief, which measures the child’s ability to understand their own and other’s minds, as well as the fact that people act on the basis of their own beliefs and that these beliefs can represent or misrepresent reality. The findings from the study suggested that 80% of autistic participants failed the task. Many researchers have replicated the findings of Baron-Cohen, et al., and it has been hypothesized that most individuals on the autism spectrum experience Theory of Mind deficits. Moreover, many researchers have suggested that the
deficits in Theory of Mind underlie the social deficits central to autism spectrum disorders (Ozonoff & McMahon Griffith, 2000). There are two levels within this hypothesis, first and second-order Theory of Mind (Ozonoff & McMahon Griffith, 2000; Spek, Scholte, & Berckelaer-Onnes, 2009). First-order Theory of Mind refers to an individual’s ability to infer another’s mental state (e.g., “I think she thinks…”). Second-order Theory of Mind refers to an individual’s ability to infer one person’s mental state about another person’s mental state (e.g., “I think she thinks he thinks…”).

Because some individuals passed the first-order Theory of Mind task in Baron-Cohen, et al. (1985) study, Happe argued that the hypothesis could not fully explain the deficits of autism spectrum disorders and could not be viewed as a global deficit (as cited in Rajendran & Mitchell, 2007). Furthermore, later researchers also found that while individuals diagnosed with autism typically failed second-order Theory of Mind tasks (Baron-Cohen, 1989), studies involving participants diagnosed with Asperger’s Disorder failed to show impairments in Theory of Mind (Bowler, 1992; Ozonoff, Pennington, & Rogers, 1991, Spek, et al., 2010). These findings led many to assert that Theory of Mind deficits are not global to autism, and the hypothesis continues to be a controversial topic within autism research (Rajendran & Mitchell, 2007; Tager-Flusber, 2007). Other explanations for the deficits in Theory of Mind tasks among individuals on autism spectrum include underlying deficits in verbal skills, IQ, and executive functioning (Ozonoff & McMahon Griffith, 2000).

Executive functioning. “Executive function” is an umbrella term that refers to skills required to prepare for and execute complex behavior. Such behaviors include
planning, impulse control, inhibition, working memory, shifting set, and mental representations of tasks and goals (Ozonoff & McMahon Griffith, 2000). In general, all executive functions allow the individual to disengage from the immediate environment in order to guide behavior (Hill, 2004; Ozonoff, 1995).

**Functional neuroanatomy.** Deficits in executive functioning are often found among individuals with damage to the frontal lobes and also among individuals diagnosed with developmental disorders that likely involve congenital deficits in the frontal lobes (e.g., schizophrenia, attention deficit/hyperactivity disorder, obsessive compulsive disorder, Tourette’s Syndrome, and autism; Hill, 2004). Specifically, executive function is a cognitive construct used to describe behaviors linked to the prefrontal cortex of the brain (Duncan, 1986; Ozonoff & McMahon Griffith, 2000; Suchy, 2009). The prefrontal cortex area of the brain includes all areas anterior to the motor and premotor cortices, as well as the supplementary motor area (Suchy, 2009). Many researchers suggest that the prefrontal cortex may be divided into three distinct areas that are involved in cognitive, emotional, and motivational processes (Alexander & Stuss, 2000; Alvarez & Emory, 2006; Pinel & Edwards, 2005). These areas include the dorsolateral, ventromedial, and orbitofrontal circuits, each of which is responsible for distinct processes.

The dorsolateral prefrontal cortex is located directly in front of the motor cortex on the lateral surface of the prefrontal cortex (Pinel & Edwards, 2005). This area has been linked to the cognitive control of behavior, including executive functions such as verbal fluency, planning, response inhibition, working memory, organizational skills, reasoning, problem solving, and abstract thinking (Alexander & Stuss, 2000; Alvarez &
Emory, 2006; Pinel & Edwards, 2005). Thus, individuals with damage to this area of the brain will exhibit deficits in these areas of functioning. For example, Pinel and Edwards (2005) suggested that individuals with damage to the dorsolateral prefrontal cortex exhibit deficits in creative thinking, inhibiting incorrect responses, and in developing and carrying out plans of action.

The ventromedial prefrontal cortex includes the anterior portions of the cingulate gyrus, located in the medial portion of the prefrontal cortex (Pinel & Edwards, 2005). Processes associated with the ventromedial prefrontal cortex include motivation, and sensitivity to rewards and punishments (Alexander & Stuss, 2000; Alvarez & Emory, 2006; Pinel & Edwards, 2005; Sucy, 2009). Thus, individuals with damage to the ventromedial prefrontal cortex will exhibit apathy, decreased initiative, decreased social interaction, and psychomotor retardation.

Finally, the orbitofrontal cortex is located at the frontal pole (i.e., on the anterior tip of the brain) on the inferior surface of the prefrontal lobes just next to the orbits (i.e., eye sockets; Pinel & Edwards, 2005). This area of the brain is thought to be responsible for socially appropriate behaviors (Alvarez & Emory, 2006). Lesions or damage in this area are linked to disinhibition, impulsivity, and antisocial behaviors (Alexander & Stuss, 2000; Alvarez & Emory, 2006).

Suchy (2009) suggested that the prefrontal cortex may be divided further into the two cerebral hemispheres. Specifically, the two hemispheres have also been found to be responsible for distinct processes. The left prefrontal cortex is associated with the initiation of responses, as well as the processing of information that is verbal, concrete, or detail-oriented. On the other hand, the right prefrontal cortex is associated with processes,
such as the inhibition of responses, visual-spatial information processing, managing abstract information, and is thought to be gestalt oriented.

While executive functions have been attributed to the cortices within the prefrontal cortex, there is also research that suggested other areas of the brain are also involved (Suchy, 2009). Specifically, the frontal lobes are connected to a variety of other regions of the brain, causing the executive functions to also rely upon the integrity of such networks. For example, working memory is not only dependent upon the dorsolateral prefrontal cortex, but also relies on the portions of the parietal lobe. Therefore, the executive functions are complex sets of processes that rely on much more than just the prefrontal cortex.

**Empirical support in autism and Asperger’s Disorder.** The executive dysfunction theory in autism is analogous with neuropsychological patients who suffered damage in the frontal lobes that resulted in impaired executive functions (Hill, 2004). Thus, the executive functioning impairment commonly found in children diagnosed with autism and Asperger’s Disorder appears to lead to behaviors that are commonly found among individuals with executive dysfunction that has resulted from frontal lobe damage (Ozonoff, 1995). For example, behaviors commonly observed among individuals diagnosed with autism and Asperger’s Disorder include rigidity, inflexibility, and adherence to strict routines in their daily activities, which are behaviors commonly found among individuals exhibiting executive dysfunction related to frontal lobe damage. Moreover, children diagnosed with autism and Asperger’s Disorder tend to have narrow interests and repetitive or stereotyped activities, which is consistent with the executive functioning behavior, perseveration. Finally, individuals diagnosed with autism and
Asperger’s Disorder are also frequently described as being impulsive and having difficulty inhibiting responses, which again is commonly found among individuals with frontal lobe damage.

Early studies supported the hypothesis that children diagnosed with high functioning autism would exhibit deficits in executive functioning (for a review, see Ozonoff, 1995; Prior & Ozonoff, 1998). One of the first empirical studies of executive functioning in autism was conducted by Rumsey (1985) in the 1980s. For this study, Rumsey (1985) administered the Wisconsin Card Sort Task (WCST), a measure of cognitive flexibility, to nine “non-retarded,” highly verbal adults diagnosed with autism. Results suggested that the autistic participants performed significantly worse with regard to the formulation of rules and perseveration on the WCST when compared to normal control participants. Similarly, Rumsey and Hamburger (1990) found that when compared to control participants and severely dyslexic men, “non-retarded” autistic men performed significantly worse on the WCST with regard to the number of categories achieved. Prior and Hoffmann (1990) were the first to test executive functioning in autistic children, and they found that the autistic participants performed less well on three tests of executive functioning than control participants (i.e., WCST, Milner Maze, Complex Figure Test). The executive dysfunction hypothesis of autism spectrum disorders was later supported by a meta-analysis conducted by Pennington and Ozonoff (1996). In their study, they completed a systematic review of studies conducted between 1964 and 1994 examining the relationship between executive functioning and autism in children. Their findings suggested that 13 of the 14 available studies on executive functioning in autism demonstrated greater executive function deficits among autistic
participants relative to study controls, further supporting the executive dysfunction hypothesis.

Researchers have also examined whether children diagnosed with autism and Asperger’s Disorder may be differentiated based upon their performances on tasks of executive functioning. While individuals with Asperger’s Disorder appear to perform better than those diagnosed with autism on executive functioning tasks, both groups appear to perform less well than control participants on such tasks (Ozonoff, Rogers, & Pennington, 1991; Verte, Geurts, Roeyers, Oosterlan, & Sergeant, 2006). Furthermore, many have argued that these two groups cannot be differentiated based on tasks of executive functioning, as many studies have shown insignificant differences between their performances (Manjiviona & Prior, 1999; Ozonoff, Rogers, & Pennington, 1991; Szatmari, et al., 1990). Such findings appear to support the executive dysfunction hypothesis of ASDs, as well as the argument that Asperger’s Disorder is a mild form of autism and therefore does not warrant a separate label.

In order to validate further the executive dysfunction hypothesis of ASDs, researchers have also examined the executive functioning profiles of children diagnosed with autism or Asperger’s Disorder relative to children diagnosed with other clinical disorders associated with executive dysfunction. Many of these studies have in fact supported the hypothesis, as their findings suggest that individuals with autism show more severe executive function deficits than those diagnosed with other disorders (e.g., Attention Deficit/Hyperactivity Disorder, conduct disorder; for a review, see Ozonoff, South, & Provencal, 2007). For example, Ozonoff and Jensen (1999) assessed three groups of children diagnosed with clinical disorders associated with executive
dysfunction (i.e., ADHD, Tourette’s Syndrome, and autism) and discovered that a
differential pattern of performance emerged for each group. Specifically, children with
autism exhibited greater impairment on test of planning and cognitive flexibility, whereas
the children diagnosed with ADHD exhibited greater impairment on tasks of inhibition.
Thus, it is possible that specific executive functioning profile may exist that differentiate
autism and Asperger’s Disorder from other clinical groups.

In contrast, however, there have also been studies to suggest that children
diagnosed with autism or Asperger’s disorder may not be differentiated from other
clinical disorders commonly associated with executive dysfunction (e.g., ADHD) on the
basis of their performances on measures of executive functioning (Booth, et al., 2003; for
review, see Hill, 2004). Thus, there continues to be debate regarding the specificity of the
executive dysfunction hypothesis in ASDs.

Hill (2004) argued that such findings make it difficult to identify executive
dysfunction as a causative factor underlying ASDs, as well as to use such information to
aid in the diagnosis of autism or Asperger’s Disorder. Further research is needed to
determine whether or not children diagnosed with autism and Asperger’s Disorder may
be clearly differentiated from other clinical groups on the basis of a unique executive
dysfunction profile. Therefore, it is important that researchers begin to examine specific
aspects of executive functioning to clearly identify areas of weakness and/or strengths
among children diagnosed with autism and Asperger’s Disorder relative to other clinical
groups.

Summary. There have been many studies that support the existence of executive
function deficits among individuals on the autism spectrum across a wide range of ages.
Such findings offer support for the frontal lobe theory of ASDs, which suggests that executive function impairment may be a central deficit in autism and Asperger’s Disorder. There, however, continues to be a debate regarding the accuracy and specificity of the executive dysfunction theory due to the many studies offering conflicting findings and an unclear pattern of differentiation between children diagnosed with ASDs and other clinical groups associated with executive dysfunction.

**Cognitive Planning**

In order to develop further understanding of specific aspects of executive dysfunction among children diagnosed with autism or Asperger’s Disorder, the current study aims to examine cognitive planning. According to Hayes-Roth and Hayes-Roth (1979; as cited in Das, Kar, & Parrila, 1996), planning consists of “anticipating a goal-directed course of action” (p.54). Luria (1978) defined planning ability as “a process that involves the ability to organize behavior in relation to a specific goal that must be achieved through a series of intermediate steps.” Scholneck and Friedman (1987) provided a more comprehensive definition of planning, stating that planning involves six steps: (1) forming a representation of the problem; (2) choosing a goal; (3) deciding to plan; (4) formulating a plan; (5) executing and monitoring the plan; and (6) learning from the plan. Furthermore, they suggested that these six steps of planning occur within three levels of functioning: (1) the individual engages in planning in the reality of a problem; (2) the individual engages in planning in accordance with an imagined scheme of a problem; and (3) the individual engages in planning in the role of mediator between the scheme and actual behavior.
Das, Kar, and Parrila (1996) further suggested conceptualizing planning in terms of three levels of analysis: activity, action, and operation. At the level of activity, planning is conceptualized as “a method of realizing or aiming toward one’s general life goals and motives, such as self-fulfillment, self-improvement, education, career development, or planning for retired life” (p. 55). Thus, activity planning is considered to be future-oriented and equivalent to problem solving. It is involved in mediating between one’s life goals and the external world. For example, if an individual’s goal involves obtaining a job as a psychologist, he or she must plan ways to manage the external world to obtain that goal (e.g., complete high school, major in psychology at the college level, attend graduate school, etc.).

The planning level of action is conceptualized as being equivalent to problem solving and aimed toward solving particular goals or solving a particular problem (Das, Kar, & Parrila, 1996). Action planning may be further conceptualized as everyday planning, or the planning of everyday goals, such as scheduling daily meetings or planning a meal for a dinner party. Whereas activity planning involves planning steps toward major life goals, such as the activities toward becoming a professional psychologist, action planning involves planning the steps toward the activities required for the major life goal. Thus, the individual would need to consider which college or institution to attend, how he or she will finance the education, and planning the area in psychology in which he or she would like to work.

Finally, the planning level of operations is conceptualized as being “equivalent to strategies and tactics, and consists of working toward the solution of a problem in accordance with task-imposed constraints” (Das, et al., 1996, p. 57). In contrast to the
other two levels of planning, operations planning is considered to be oriented toward the present. Everyday examples of operations planning include locating a book in the library or operating a blender or other kitchen machinery toward making a meal.

**Neuroanatomy of planning.** Many studies have been conducted to examine the neurological underpinnings of planning dysfunction. Specifically, planning impairments have been tied to frontal lobe damage in studies using various laboratory tests, such as the Tower of London (Shallice, 1982). The findings from such studies have shown that damage to the frontal cortex leads to deficits in the individual’s ability to form and carry out complex plans in novel and complex situations. Furthermore, studies involving children diagnosed with disorders that have been found to impact the prefrontal lobes and subcortical regions (e.g., autism, ADHD, schizophrenia) have also show impairment on such tasks, suggesting that deficits in planning ability are likely tied to the frontal lobe dysfunction (Eslinger, Biddle, & Grattan, 1997).

Researchers have sought to examine further the neurological underpinnings of cognitive planning through neuroimaging studies. Specifically, several studies have examined cognitive planning by conducting functional magnetic resonance imaging (fMRI) while subjects complete various planning measures, such as the Tower of London (e.g., Rowe, Owen, Johnsrude, & Passingham, 2001). Findings from these studies suggested that the mid-dorsolateral prefrontal cortex plays a critical role in cognitive planning (Owen, 2005; Rowe, et al., 2001; Tanji, Shima, & Mushiake, 2007). For example, Rowe, et al. (2005), found that the activity in the mid-dorsolateral prefrontal cortex on the Tower of London represented the completion of mental operations, such as
generating, selecting, and remembering mental moves but was not involved in actually carrying out the moves on the Tower of London task.

While many neuroimaging studies have implicated bilateral involvement of the mid-dorsolateral prefrontal cortex in cognitive planning (for a review, see Tanji, et al., 2007), Kaller, Rahm, Spreer, Weiller, and Unterrainer (2011) examined whether various aspects of cognitive planning differentially engage either the left or right hemispheres of the mid-dorsolateral prefrontal cortex. In order to examine the possibility of lateralization in cognitive planning, the researchers conducted an fMRI study using the Tower of London to assess differential hemispheric involvement between search depth and goal hierarchy. They described goal hierarchy as “concern[ing] the obviousness of priorities for individual goal moves that can be deduced from the structure of the goal state” (p. 309). As an example, the authors provided the following scenario:

Imagine you just arrived home when the phone rings and an old friend announces that he will drop in at short notice within the next hour. You abruptly realize that you have to manage several goals before your friend arrives: you should offer at least some pasta for dinner. However, your kitchen is in a mess and you have hence to do a huge pile of dirty dishes. Since kitchen utensils such as pots, knife, spoon, and board are minimum prerequisites for preparing pasta with a basic tomato sauce, you will have to start with cleaning up. So, the order in which to work on your goals is not arbitrary but is completely identifiable from your overall goal. (p. 307)
In contrast, search depth was described as the “need to mentally accomplish intermediate moves and associated interdependencies” (p. 309). The authors built upon the previous to further explain search depth:

You have two alternatives, either washing dishes and kitchenware by hand or using the dishwasher. Both will equivalently serve the goal of having a clean kitchen. However, as a washing cycle in the dishwasher takes more than half an hour, this might get you in conflict with achieving your second goal in time since you cannot prepare and boil down a tomato sauce for the pasta while your pot, kitchen knife, spoon, and cutting board are in the machine. That is, you have to take into account the interdependencies between the different alternative steps to achieve your goals—and consequently, you have to wash all much-needed kitchen utensils by hand, whereas, for sake of time, you may put all remaining equipment in the dishwasher. (p. 307-308)

Results from this study suggested that while activations were stronger in the left mid-dorsolateral prefrontal cortex for higher demands on goal hierarchy than on search depth, activations were stronger in the right mid-dorsolateral prefrontal cortex for higher demand on search depth. These finding suggest, that specific cognitive planning operations may have opposing lateralizations.

**Everyday significance of cognitive planning.** Planning ability allows the individual to set and maintain goals in his or her everyday life (Garner, 2009). It affects both short-term and long-term activities. For example, planning ability allows one to complete errands and daily tasks more efficiently in the short-term. In contrast, one
would be likely to simply react to pressures of the moment rather than considering the implications of their actions in the long-term without sufficient planning ability.

One major area of concern regarding planning ability is academic performance (Garner, 2009). Specifically, increased planning skills have been found to be associated with enhanced academic performance. Planning allows the individual to prioritize learning tasks effectively, complete assignments in a timely manner, and plan a long-term course of study toward graduation. Meltzer, Pollica, and Barzillai (2007) further argued that a child who lacks planning ability is more likely to approach an academic task impulsively, causing the child to “get stuck” when the next step or end goal of the task is unclear. A child’s planning ability is therefore critical to understand the objective educational tasks, visualize the steps of the task, organize the timing of task, obtain the necessary resources, and to effectively carry out the task.

Similarly, planning has been associated with reading skills. For example, researchers have discovered that children who perform worse on planning measures relative to typically developing children tend to struggle with reading comprehension despite having intact single word reading and comprehension (e.g., Sesma, Mahone, Levine, Eason, & Cutting, 2008). Furthermore, children with poor planning skills have also been found to play a role in children’s ability to take notes while reading and write a report from those notes (Altemeier, Jones, Abbott, & Berninger, 2006). Thus, children with poor planning skills will tend to struggle in areas of academic performance, such as reading, taking notes, completing assignments, and long-term course planning. These findings suggest that it will be important to intervene at the academic level for children who exhibit deficits in their planning ability.
It has been further argued that one’s planning ability may contribute to his or her personality attributes (Das & Naglieri, 1995). From this perspective, planning is a relatively enduring trait that influences behavior, and, much like other personality traits, is susceptible to being adversely affected by frontal lobe damage. Das and Naglieri (1995) pointed out that the individual’s personality is often described by their planning ability. For example, one may be described as being organized, deliberate, and under control, or as being disorganized, confused, and indecisive, all of which are closely related to planning ability.

The notion of planning ability as a personality trait has been supported by various research studies. For example, Hunt, DeLacey, and Randhawa (1987) found that children who exhibited poor planning skills tended to be high on personality variables, such as impulsiveness, venturesomeness, and extraversion, as measured by the Junior Eysenck Personality Questionnaire. In another study, extroversion was again found to be significantly related to lower planning performance on a perceptual maze test (Weinman, Elithorn, & Cooper, 1985). Such findings suggest that planning ability likely contributed to one’s personality attributes.

Severtson, Mitchell, Mancha, and Latimer (2009) examined the relationship between planning ability and injection drug use. They discovered that study subjects who exhibited poorer performance on a measure of planning (i.e., Tower of London) tended to exhibit increased frequency of injection drug use, as well as increased sharing of injection drug use equipment. They concluded that these finding suggest that planning ability moderated the association between frequency of injection use and risky injection
practices and should be considered when developing harm reduction strategies of injection drug users.

Smith, Hay, Campbell, and Trollor (2011) conducted a study to assess the relationship between cognitive functions, including planning ability, and obesity in older adults. They discovered that executive dysfunction, including impaired planning, was related to greater weight gain in such individuals. As a result of their findings, they suggested that cognitive remediation strategies may be a useful tool in the treatment and prevention of obesity.

Finally, planning ability has also been studied in the context of adolescent sex practices (Gebhardt, Kuyper, & Dusseldorp, 2006). The researchers for this study were interested in assessing extent to which cognitive planning and motivations for sex may explain condom use. The results suggested that better cognitive planning was related increased condom use for those adolescents who did not endorse love as a motive for having sex. Thus, interventions should be aimed at increased planning with regard to condom use for adolescents who cite love as their motive for having sex.

**Planning in Autism Spectrum Disorder.** Planning ability has been widely assessed in children diagnosed with autism or Asperger’s disorder (for a review, see Hill, 2004). Many researchers have found that these children exhibit planning dysfunction, and these findings have offered much support to the executive dysfunction hypothesis in autism spectrum disorders. However, there have also been studies in which children diagnosed with autism or Asperger’s disorder did not exhibit planning dysfunction relative to a comparison group. Thus, planning ability in autism and Asperger’s disorder remains unclear. Given the importance of planning ability in everyday functioning, it is
imperative that we gain a greater understanding of planning in autism spectrum disorders. Furthermore, such knowledge may contribute to a clearer picture regarding the unique executive dysfunction profile associated with autism and Asperger’s Disorder, as well as offer important information regarding intervention planning.

The Current Investigation

The increasing rate of autism and Asperger’s Disorder diagnoses have contributed to a growing need for better understanding of the characteristics associated with these populations. As noted throughout this study, there is substantial evidence to suggest that individuals diagnosed with autism and Asperger’s Disorder often exhibit executive dysfunction. However, there continues to be debate regarding the universality and specificity of such deficits among these populations. Therefore, it is important that researchers begin to explore specific aspects of executive dysfunction, such as cognitive planning, in order to gain a clearer picture regarding the relative strengths and weaknesses within executive functioning among this unique population. The current investigation aims to review and analyze systematically the available literature regarding the executive functioning component, cognitive planning, through a meta-analysis. Such information will help to validate the executive dysfunction hypothesis; to illuminate specific areas of strength and weakness of executive functioning; and to guide the use of executive dysfunction profiles as diagnostic markers and the focus of intervention efforts.
Chapter III. Method

Identification of Studies

In an effort to gather extensive data for the meta-analytic review, computer searches of the PsycInfo and Medline bibliographic databases were conducted in August, 2011. The terms “autism,” “autistic,” “Asperger’s,” “autism spectrum disorders,” and “pervasive developmental disorder” were cross-referenced separately with “executive,” “executive function,” “frontal,” “planning,” “tower,” “mazes,” “stockings of Cambridge,” “Rey,” and “Delis-Kaplan.” The search terms were selected based on the most widely and commonly used terminology in the research literature on planning ability in autism spectrum disorders at the time of this study. Literature abstracts were reviewed to identify relevant studies using the search terms described above. Additional studies were located by searching the reference lists of relevant research studies.

The initial search produced 972 papers. After some articles were deleted due to duplication and incompatibility with the study, 224 abstracts were reviewed to identify if the article potentially met the inclusion and exclusion criteria defined above. If the abstract appeared promising, the entire article was then reviewed to verify whether it contained the necessary information for coding.

Inclusion/exclusion criteria. To be included in the meta-analytic review, studies must have met the following inclusion criteria: (1) dissertation theses and studies published in refereed journals between 1985 and August 2011 were included in the review (the cutoff of 1985 was chosen, as the first empirical investigation of executive
functioning in autism was published in 1985 (i.e., Rumsey, 1985); (2) included a sample of study participants diagnosed with autism and/or Asperger’s disorder without comorbid diagnoses; (3) study sample diagnosed according to either DSM or ICD criteria; (4) included a comparison/control group; (5) the comparison group was matched to the ASD group by either mental age or intelligence quotient; (6) study sample included only children aged 18 years or younger; (7) use of one or more measures that explicitly assesses planning as an executive function; (8) included statistical information required to conduct the meta-analysis (e.g., means, standard deviations, sample size, etc.); and (9) be in English. Unpublished studies and studies of adults were not included. After completing the literature search and reviewing each available study for inclusion criteria, 33 studies were identified to be included in the present study.

**Data Collection**

After obtaining the complete set of studies, data from each study was extracted by two methods. Specifically, information was obtained from each study and coded (see Appendix A for coding protocol). Additionally, study metrics were converted to a standardized metric for further analysis.

**Coding.** All articles and dissertations were coded by the primary investigator. The coding procedures were mechanical and objective; thus, one coder was determined to be sufficient. All identifying information was coded directly into an Excel Spreadsheet and all other gathered data was coded directly into a database within Comprehensive Meta-Analysis by Biostat.

**Identifying information.** Identifying information was coded for each study. Specifically, each study was assigned a study identification number. All studies were
coded for each of the authors’ last names and initials of first and middle names, and the study publication date and full title was coded. The pages of the published study and the volume and issue number of each journal article were also included on the coding form. Finally, the page number where the statistical data needed for the meta-analysis was located was also coded.

**Dependent variables.** Information regarding the dependent variables was also coded for each study. This included the type of planning measure used in each study and statistical data (i.e., means, standard deviations, and sample size for each comparison group). The type of planning measure was coded by the following descriptors: 1 Tower of London; 2 Tower of Hanoi; 3 NEPSY Tower; 4 DKEFS Tower; 5 Rey Complex Figure; 6 Stocking of Cambridge; 8 BRIEF Plan/Organize subtest; 9 other.

**Study characteristics.** In addition to identifying information and dependent variable information, several variables of interest were also coded for each study. Diagnostic information was coded for each group. The autism spectrum group was coded as 1 for autistic only, 2 Asperger’s disorder only, or 3 mixed ASD sample. The comparison group for each study was coded by the following descriptors: 1 ADHD-inattentive type; 2 Tourette’s Syndrome; 3 Conduct Disorder; 4 Typically Developing; 5 ADHD-combined type; 6 Reading Disorder; 7 Moderate traumatic brain injury; 8 Severe brain injury; 9 cannot tell; 10 ADHD mixed sample; 11 Mixed clinical control; 12 Developmental Language disorder; 13 Obsessive Compulsive Disorder; 14 Developmental Delay; and 15 Language and Learning disorder combined. Additionally, each comparison group was coded as either a clinical sample or typically developing control sample for simplification.
Mean age and IQ was coded for each comparison group, as well as the IQ inclusion criteria for each study. Other descriptors included on the coding form were predominant race and sex of the study samples and the criteria by which the comparison groups were matched. Finally, the basis for the autism spectrum disorder diagnosis of study participants was also coded. Specifically, any study measures used to confirm diagnosis (e.g., ADI-R, ADOS-G, SCQ, etc.) were coded and the criteria used for diagnosis (e.g., DSM-III, DSM-IV, ICD-10, etc.) were coded. See Appendix A for the coding protocol.

Statistical data. The most common metric used to synthesize the findings from multiple studies that include group contrasts is Cohen’s d (Cohen, 1988) or the standardized difference between groups (see below for a full description of the meta-analytic procedures). In order to compute d, the following statistics were coded for all study samples: sample size, group means, and group standard deviations.

Data Analysis

Software. Microsoft Excel was used to run descriptive statistics for study samples. The software used to conduct the meta-analysis for this investigation was Comprehensive Meta-Analysis 2.0 (Borenstein, Hedges, Higgins, & Rothstein, 2005). Contributing authors of this program included statisticians from the United States and United Kingdom and development was funded by a grant provided by the National Institute of Health in the United States. The software is based upon statistics proposed by Hunter and Schmidt (1990) and allows for the examination of multiple groups and multiple outcomes.
**Meta-analysis.** In order to evaluate psychological theories and phenomena, it is important to accumulate available empirical evidence to compare and contrast research findings (Johnson & Eagly, 2000). Historically, researchers have attempted to evaluate multiple research studies by completing narrative reviews, in which scholars draw conclusions based upon their impressions of the overall trend among study findings. This technique is often guided by a vote count of the number of studies that produced either statistically significant or insignificant findings regarding study hypotheses (Hunter & Schmidt, 1990; Johnson & Eagly, 2000; Mullen, 1989).

While narrative reviews are helpful in some contexts (e.g., introductions to journal articles), the method is inadequate for reaching conclusive findings regarding the degree of empirical support for a psychological phenomena or theory (John & Eagly, 2000). Specifically, critics of this method argue that there are four general errors within narrative reviews: (1) narrative reviews often only contain samples known by the author, thereby excluding many of the available studies; (2) narrative reviews do not typically state the procedures used for coding study variables or the ways in which study quality was assessed, causing it difficult for readers to judge the accuracy of the review; (3) narrative reviews are inadequate in describing how differences in study methods contribute to contrasting study results; and (4) narrative reviews rely on statistical significance of each study to judge the findings, which is a poor basis for comparing studies that have different sample sizes (for review, see John & Eagly, 2000).

Given the difficulties and inadequacies associated with narrative reviewing, efforts have been made toward developing research methods that make the review process more reliable and valid. The result of these efforts has been the development of
meta-analysis, which allows the researcher to statistically combine the results of several studies that address a shared research hypothesis (Lipsey & Wilson, 2000). Specifically, the meta-analytic approach increases statistical power, permits the estimation of a population effect size, and allows an examination of moderating variables (i.e., evaluation of the variables that may be moderating the relationship between study sample diagnosis and performance on planning measures; Alvarez & Emory, 2006). Therefore, a meta-analysis was conducted for the current project in order to accumulate and evaluate validly and reliably available research findings regarding cognitive planning dysfunction among children diagnosed with autism and/or Asperger’s Disorder.

**Effect size.** Outcome data for research studies are typically reported in various forms and are often based on differing measurements of the dependent variable. Study results are therefore not easily compared (Lipsey & Wilson, 2000). Thus, it is necessary to convert all available data into a common metric in order to effectively synthesize and analyze data from a wide range of available studies. There are a wide variety of effect size indicators available from which to choose. The most commonly used effect size indicators belong to either the \( r \) family (correlational coefficient; e.g., \( r, \rho, \phi \)), or the \( d \) family (standardized difference; e.g., Cohen’s \( d \), Hedge’s \( g \)). One must consider both theoretical and practical matters when determining the appropriate indicator for their study.

Cohen’s \( d \) was chosen as the effect size indicator for the current study. The reasoning behind this choice is three-fold. First, researchers suggest that effect size indicators in the \( d \) family are better suited than those in the \( r \) family for studies examining the strength and direction of mean differences between groups (Hedges & Olkin, 1985;
Zakzanis, 2001). Furthermore, Johnson and Eagly (2000) suggested that the convention is to use \( r \) as the effect size indicator if studies involved in the meta-analysis report correlations between two continuous variables, whereas it is appropriate to use the \( d \) family when the studies involved in the meta-analysis report ANOVAS, \( t \)-tests, or chi-squares for comparisons between two groups. Thus, it was decided that the \( d \) family was the most appropriate choice for determining the effect size indicator, as the current study involves (a) the examination of the strength and directions of mean differences between an ASD sample and control/typically developing sample on measures of cognitive planning and (b) the studies used in this analysis reported ANOVAS, \( t \)-tests, or chi-squares for comparisons between two groups.

Second, there are two effect size indicator options within the \( d \) family that are appropriate for comparisons of group means (Zakzanis, 2001). These indicators include Cohen’s \( d \) and Hedge’s \( g \), for which the equations are:

\[
\text{Hedge’s } g = \frac{[M_1 - M_2]}{SD_{\text{control}}}
\]

\[
\text{Cohen’s } d = \frac{[M_1 - M_2]}{SD_{\text{pooled}}}
\]

where

\[
SD_{\text{pooled}} = \sqrt{\left(\frac{N_1 SD_1^2 + N_2 SD_2^2}{N_1 + N_2 - 2}\right) + \left(\frac{1}{N_1} + \frac{1}{N_2}\right)}
\]

Zakzanis (2001) argued that Cohen’s \( d \) is the best effect size indicator for use in neuropsychological research, and thus should be chosen over Hedge’s \( g \). Specifically, the computation of Hedge’s measure assumes that the standard deviation of the control sample is not much different from the standard deviation of the sample of interest (i.e.,
assumes variance homogeneity). However, Zakzanis (2001) pointed out that it is uncommon to find proportional standard deviations between samples in neuropsychological assessment, and the basic assumption underlying hedge’s g would therefore be inappropriate for such analyses. In contrast, the computation for Cohen’s $d$ includes the pooled standard deviation of both groups being compared, and therefore does not assume variance homogeneity. Thus, Cohen’s $d$ was felt to be the most appropriate effect size indicator for the current investigation, as this study is interested in differences between performances of ASD and control groups on neuropsychological measure of cognitive planning.

Finally, Cohen’s $d$ was also chosen for practical reasons. All studies involved in the meta-analysis reported sample size, group means, and standard deviations, so there was no need to transform data to another effect size indicator, such as $r$. Furthermore, Cohen (1988) offered a frame of reference for interpreting effect size, making it less difficult to interpret findings. Specifically, Cohen’s (1988) suggested the following for labeling the magnitude of effect sizes: a standardized mean difference less than or equal to .20 is small, .21-.79 is medium, and .80 and greater is large.

For purposes of the current investigation, positive effect size values indicated that the comparison group performed better than the autism spectrum group on the outcome measure. For example, a positive effect size indicated that the ASD group performed worse than the clinical or typically developing control group on the cognitive planning measure. For those measures in which a higher score reflected better performance, the sign of the effect size was reversed.
Independence vs. non-independence. Another issue that must be considered in meta-analysis is whether study data points should be considered to be either independent or non-independent. For this meta-analysis, there were several studies that reported more than one outcome measure and/or more than one comparison group within their study (e.g., an ADHD group and a typically developing group). Thus, it was important to determine whether or not to assume independence between each comparison in a given study. There are four methods (Mullen, 1989) by which the researcher may handle this problem: (1) use each effect size as if came from an independent sample; (2) use the results from only one control group or outcome measure within each study; (3) conduct individual meta-analyses for each control group and outcome measure; or (4) average the effect sizes of the control groups and outcome measures in a given study to form one estimate. The second and third options were discarded for this study for three reasons. First, there is no way to determine which outcome measure would be the most appropriate measure to use within a given study. Second, we would lose valuable data regarding various comparison groups should we discount such findings. Third, there are too few studies to conduct a meta-analysis for each comparison group and each outcome measure (i.e., this would result in too little power).

The method chosen to manage the problem of independence vs. non-independence in this study was to conduct a two-stage meta-analysis, involving both option one and option four (Johnson & Eagly, 2000). This method allows the investigator to first address the combined effect size across studies by averaging all effect sizes within each study to form one value (i.e., only one effect size from each study is included in the overall or combined effect size). The second stage of this method then allows the
investigator to divide study variables into various groupings (e.g., NEPSY Tower vs. Tower of Hanoi vs. Key Search, etc.) by assuming independence between such variables. This two stage process allows the investigator to assess the available data on multiple levels.

**Fixed effect vs. random effects models.** To combine effect sizes from each study into the average effect size across all studies, the investigator must determine whether or not to use a fixed or random effects model. In a fixed effect model, it is assumed that there is one true effect size that underlies all of the studies included in the analysis and that any differences in the observed effects are due to sampling error (Borenstein, Hedges, Higgins, & Rothstein, 2009). In contrast, the random effects model allows for the effect size to vary among studies due to differences in variables, such as the mixes of participants and outcome measures used. Such variability among study design is likely to lead to differences among study effect sizes that may not be accounted for by sampling error alone. Given the differences in population diagnoses among ASD groups and control groups, as well the differences between cognitive planning measures used among studies, the random effects model was used for the current investigation.

**Tests of homogeneity.** One of the main goals of a meta-analysis is to determine whether the summary effect size across studies is homogenous (Huedo-Medina, Sanchez-Meca, Marin-Martinez, & Botella, 2006). If the summary effect size is determined to be homogenous, then it is assumed that any observed differences among effect sizes are due to sampling error. However, if the summary effect size is determined to be heterogeneous, then it is assumed that the observed differences among effect sizes are due to other moderating variables (e.g., population diagnoses, etc.).
The homogeneity of effect sizes was assessed by the $Q$ test, defined by Cochran (1954). If the $Q$ test is significant, the homogeneity hypothesis is rejected, and it is assumed that variance among study effect sizes is due to study characteristics aside from sampling error (Huedo-Medina, et al., 2006). On the other hand, if the $Q$ statistic is not significant, the homogeneity hypothesis is not rejected and it may be assumed that any observed variance among study effect sizes is due to sampling error alone (Huedo-Medina, et al., 2006).

**Examination of moderator variables.** Another goal of meta-analysis is to examine moderating variables should heterogeneity exist among study effect sizes (Huedo-Medina, et al., 2006). Analysis of categorical moderating variables (e.g., ASD group diagnosis, control group diagnosis, cognitive planning measure, etc.) was conducted by assessing the $Q$ statistic between and within study variances and testing for statistical significance among the $Q$ statistics by conducting $\chi^2$ analyses for each moderator. A significant $Q$ between study variance indicates that there is significant variability between the groups that comprise the categorical moderator variable greater than would be expected simply by chance (Alvarez & Emory, 2006). In contrast, a significant $Q$ within study variance indicates that there is still significant variability within each effect size that is not being explained by the moderating variable (Alvarez & Emory, 2006). Thus, a categorical moderator variable explains all of the heterogeneity present in the summary mean effect size only when the variance is significant between groups and not significant within groups.

In order to test continuous moderators, including age and IQ, a weighted least squares regression procedure was used (Hedges, 1994). Effect sizes were weighted by
their sample size and then regressed onto the relevant predictor variable (i.e., age). A Z-test of the unstandardized regression coefficient was used to determine the statistical significance of the moderator, using 95% confidence intervals.

**Fail-safe N.** With any meta-analysis there is a risk that the studies being included represent only a subset of studies actually previously conducted, as studies reporting insignificant results are much less likely to be published in the literature (Alvarez & Emory, 2006). Thus, the “fail-safe N” statistic was examined to determine the number of unpublished, non-significant studies it would take to nullify the results of the current meta-analysis \(p < .05\). This statistics allows the examiner to determine whether the studies included in the current meta-analysis adequately represent the entire population of studies on this topic. Rosenthal (1991) argued that it is unlikely that the number of studies filed away is five times as many studies as the reviewer. Therefore, it is unlikely that there are more than 165 studies filed away for the current meta-analysis.

**Research Questions**

The following research questions were generated based upon recent findings and trends in the literature. The specific research questions investigated in this study were:

1. Do children diagnosed with autism and Asperger’s Disorder exhibit greater cognitive planning deficits relative to comparison groups?
2. Can children diagnosed with autism be differentiated from children diagnosed with Asperger’s Disorder based upon planning measure performance?
3. Does the comparison group diagnosis impact the extent to which children diagnosed with autism and Asperger’s Disorder exhibit greater cognitive planning deficits relative to comparison groups?
4. Does the type of planning measure used impact the level of cognitive planning dysfunction exhibited by children diagnosed with autism and Asperger’s Disorder?

5. Does age impact the level of cognitive planning dysfunction exhibited by children?

6. Does IQ impact the level of cognitive planning dysfunction exhibited by children?
Chapter IV. Results

Sample Characteristics

Out of the 224 abstracts reviewed, 33 studies met the inclusion criteria and were included in the present study (see Appendix B). The 33 studies included in the meta-analysis yielded a total sample of 2,611 participants, with 1,020 participants diagnosed with autism or Asperger’s Disorder and 1,591 participants in a comparison group. The average age for the entire sample ranged from 3.54 to 16 (with a mean age of the entire sample of 10.26, $SD = 2.54$). The average IQ of the entire sample ranged from 88 to 113.87 (with a mean IQ of the entire sample of 102.31, $SD = 7.12$; see Table 7).

**Autism and Asperger’s Disorder samples.** Study ASD samples were comprised of children diagnosed with Autism only ($n = 445$), Asperger’s Disorder only ($n = 88$), or mixed samples of children diagnosed with autism or Asperger’s Disorder ($n = 487$).

The mean age of the ASD samples ranged from 5.6 to 15.95 (with a mean age of the entire ASD sample of 10.72, $SD = 2.67$). Ages of each ASD subgroup was examined. The mean age of the autism only samples ranged from 5.2 to 15.7 (with a mean age of autism only samples of 9.55, $SD = 2.91$). The mean age of the Asperger’s Disorder only samples ranged from 8.5 to 10.6 (with a mean age of Asperger’s Disorder only samples of 9.34, $SD = 0.89$). Finally, the mean age of the mixed autism and Asperger’s Disorder ranged from 5.6 to 15.95 (with a mean age of 10.98, $SD = 2.45$).
The mean IQ of the ASD samples range from 88 to 113.87 (with a mean IQ of the entire ASD sample of 99.28, SD = 7.19). IQ for each subgroup was examined. The mean IQ of the autism only samples ranged from 88 to 113.87 (with a mean age of autism only samples of 99.45, SD = 7.83). The mean IQ of the Asperger’s Disorder only samples ranged from 100.8 to 105.2 (with a mean age of Asperger’s Disorder only samples of 103.25, SD = 2.24). Finally, the mean IQ of the mixed autism and Asperger’s Disorder ranged from 88.89 to 113.87 (with a mean age of 98.34, SD = 7.34). Of note, not all studies reported IQ scores for study subjects (i.e., 4 studies did not report ASD sample IQ).

**Comparison groups.** Comparison groups were comprised of children diagnosed with a clinical disorder commonly associated with executive dysfunction (n = 632), including ADHD-inattentive type (n = 55), ADHD-combined type (n = 47), a mixed sample of children diagnosed with ADHD-inattentive type and ADHD-combined type (n = 185), Tourette’s Syndrome (n = 54), reading disorder (n = 34), comorbid language and learning disorders (n = 29), moderate traumatic brain injury (n = 33), severe traumatic brain injury (n = 34), developmental delay (n = 19), developmental language disorder (n = 105), obsessive compulsive disorder (n = 17), a clinical control group (n = 20), and typically developing children (n = 959).

The mean age of the comparison groups ranged from 3.54 to 15.23 (with a mean age of the entire comparison group sample of 10.75, SD = 2.47). The mean age for children diagnosed with a clinical disorder commonly associated with executive dysfunction (clinical comparison) and typically developing children was also examined. The mean age of the clinical comparison samples ranged from 4.83 to 14.9 (with a mean
age of clinical comparison samples of 10.37, \( SD = 2.32 \). The mean age of the typically developing samples ranged from 3.54 to 16 (with a mean age of clinical comparison samples of 10.24, \( SD = 2.71 \)).

The mean IQ of the comparison groups ranged from 91 to 113.8 (with a mean IQ of the entire comparison group sample of 104.44, \( SD = 6.42 \)). The mean IQ for children diagnosed with a clinical disorder commonly associated with executive dysfunction (clinical comparison) and typically developing children was also examined. The mean IQ of the clinical comparison samples ranged from 91.3 to 113.8 (with a mean IQ of clinical comparison samples of 101.6, \( SD = 5.57 \)). The mean IQ of the typically developing samples ranged from 91 to 113.4 (with a mean IQ of clinical comparison samples of 106.53, \( SD = 6.24 \)). Of note, not all studies reported IQ scores for study subjects (i.e., 4 studies did not report comparison group sample IQ).

**Additional descriptors.** Most studies used diagnostic criteria from the DSM-IV to define groups with and without autism or Asperger’s disorder \((k = 30)\), while the remaining studies used diagnostic criteria from the ICD-10 \((k = 3)\). In addition, 23 studies used additional questionnaires or interviews to confirm the autism or Asperger’s Disorder diagnosis, while the remaining 10 studies did not use any supporting evidence from questionnaires or interviews for the ASD diagnosis. Specifically, 19 studies used the ADI-R, 16 studies used the ADOS-G, five studies used the Social Communication Questionnaire, 3 studies used the CARS, and one study used the GADS to confirm autism or Asperger’s Disorder diagnoses. All studies were included in the overall meta-analysis, and results from studies that defined autism spectrum diagnoses based on these various diagnostic criteria were later compared.
Planning Assessment Measures

Among the studies, planning ability was operationalized by 15 assessment measures. These measures included the Tower of London, Tower of Hanoi, NEPSY Tower, Delis-Kaplan Executive Function Scale (DKEFS) Tower, CANTAB Stockings of Cambridge, Behavior Rating Inventory of Executive Functioning (BRIEF) Plan/Organize subtest, WISC-R Mazes subtest, WPPSI Mazes, Milner Mazes, Key Search, Six Parts Test, Zoo Map, Plan, and Planning Task. An overview of each planning assessment measure will be provided in the following sections.

**Tower of Hanoi.** The Tower of Hanoi (TOH) is a measure of planning ability requiring the formulation and execution of a strategy in accordance with a set of rules to achieve an externally imposed goal (Simon, 1975). The materials of the TOH consist of three vertical pegs and 3–5 discs of different sizes, positioned at peg number 1 at the start. Goal state is the identical configuration in peg numbers 2 or 3. It is a pyramidal configuration. There are two rules: (a) a larger disc must not be placed on the top of a smaller one; and (b) only one disc is allowed to move at a time. Impaired performance of the TOH or a similar task has been shown by frontally damaged adults.

**Tower of London.** The Tower of London (TOL) was developed based upon the TOH by Shallice (1982) to measure executive planning ability and is another widely used measure in clinical and research settings. The TOL is comprised of a board with three pegs differing in length and three balls colored blue, red, and green. To complete the task, the examinee is provided two such boards and informed that one board is arranged with the balls in a start position and one board is arranged with the balls in a goal position. For each TOL problem, the examinee is instructed that the balls in the start position have to
be moved into the goal position, and they are given three ruled by which the task must be completed: (1) the balls may only be moved one at a time; (2) the balls cannot be placed outside the pegs; and (3) a maximum of three balls are allowed to be placed on the tallest peg, a maximum of two on the middle peg, and a maximum of one on the shortest peg. The primary score used to assess planning ability on the TOL is the total move score, which is defined by the number of moves the examinee makes beyond the minimum number of moves required to reach the goal position summed over all problems (Koppenol-Gonzalez, G.V., Bouwmeester, S., & Boonstra, A.M., 2010).

**NEPSY Tower.** The NEPSY Tower is a subtest of a larger instrument, NEPSY: A Developmental Neuropsychological Assessment (Korkman, Kirk, & Kemp, 1998). This subtest is based on the TOL and was also developed to assess planning ability. To complete this task, the child is asked to move three colored balls to target positions on three pegs in a prescribed number of moves. In addition, there are particular rules to which the child must adhere on this timed task. Overall, the set of instructions and task is very similar to TOL. NEPSY Tower is appropriate for use with children, aged 5-12 years.

**Delis-Kaplan Executive Function System (D-KEFS) Tower Test.** The Tower Test is a subtest of the Delis-Kaplan Executive Function System (Delis, et al., 2001) and was also developed as a test of planning and problem solving abilities based on the TOH/TOL tasks. For this task, examinees are provided two boards with three pegs and a set of discs arranged on the pegs. They are then asked to arrange the discs according to the provided model. The examinee’s ability to use the fewest possible moves to achieve the tower depicted in the model is scored, and lower scores denote worse performance.
**CANTAB Stockings of Cambridge.** The Stockings of Cambridge is a subtest of the Cambridge Neuropsychological Test Automated Battery (CANTAB) and is also based on the TOL test. This measure was developed as a spatial planning task and is conducted on the computer. The examinee is provided two views of the task board, each with three colored balls which appear to be in “stockings” or stacks. The individual must repeat the pattern shown in the example view by touching the ball and then touching where the ball is to be moved. The individual's planning ability is based on how quickly and accurately the pattern is imitated.

**Behavior Rating Inventory of Executive Function (BRIEF).** The Behavior Rating Inventory of Executive Function (BRIEF) is a questionnaire developed by neuropsychologists to collect information from caregivers and teachers regarding the executive functions of children, aged 5-18 years (Gioia, Isquith, Guy, & Kenworthy, 2000). The BRIEF was created to allow caregivers and teachers to provide useful information about the executive functions of youth by reporting on their everyday behavior in the home and school environments. Furthermore, researchers have previously argued that neuropsychological measures of executive functioning completed in the laboratory or clinical setting do not reflect the child’s everyday behavioral difficulties (Liss, et al., 2001). Therefore, the BRIEF was also created to establish a measure of executive functioning with more ecological validity. Within the BRIEF, the Plan/Organize scale is used to assess the child’s ability to think prospectively and follow a plan.

**WISC-R/WPPSI Mazes subtests.** The Mazes subtest of the Wechsler Intellectual Scale for Children-Revised (WISC-R) and the Wechsler Preschool and
Primary Scale of Intelligence requires examinees to complete a series of increasingly complex mazes. These subtests were used as a measure of planning ability, as they involves the ability to plan ahead to successfully complete the task (Liss, et al., 2001).

**Milner Maze.** The Milner Maze (Milner, 1965) is a task requiring examinees to discover and remember the correct path to complete a maze. The examinee is provided the following rules: (1) if an error is made, the examinee must go back to the last correct response and proceed; (2) the examinee cannot move diagonally; (3) and the examinee may not retrace portions of the correct path. Similar to the Wechsler mazes, this task requires that the examinee plan ahead to successfully complete the task.

**Key Search.** The Key Search task is a subtest of the Behavioral Assessment of the Dysexecutive Syndrome (BADS) battery. This battery was designed as an ecologically valid measure of executive function. For this task, examinees are required to search for an imaginary key they have lost on a field (a square on a piece of paper). Planning ability is assessed by examining the individual’s drawing of their search route.

**Six Parts Test.** The Six Parts Test requires the examinee to plan a strategy in order to carry out six activities to complete an overall task. The examinee is also required to monitor their progress on each activity in order to ensure they keep to their plan. The activities consist of two picture naming tasks, two counting tasks and two sorting tasks, and the examinee is given five minutes on a visible timer. Finally, the examinee is required to complete something from each task without performing two tasks of the same type consecutively. Points were awarded for attempting all six activities (12 points), were deducted for breaking the order rule (three points), were awarded for using specific
strategies to achieve these two aims (four points), and were deducted if the child inefficiently returned to a particular task three or more times (one point).

**Zoo Map.** The Zoo Map test is another measure of planning. It is split into two parts. Part 1 of the task requires the examinee to plan a solution to a problem. The problem requires that the examinee follow a set of rules: (1) they may only visit certain animals and places in a zoo; (2) they must stay on the paths and walk along certain paths only once. Points are awarded for visiting the animals in an optimal sequence and were subtracted for breaking any of the rules. Part 2 of the Zoo Map Test removes most of the planning requirements, so for the purposes of this meta-analysis, only scores from Part 1 were included.

**Plan.** Mackinlay, Charman, and Karmiloff-Smith (2006) developed a measure of planning for their study called “plan.” For this measure, study participants were provided directions for a task. After they understood the task, the participants were required to verbally describe how they planned to carry out the task. The description of their plan to complete the task was recorded verbatim and scored by study investigators based on scoring criteria which developed around planning ability.

**Planning task.** Gillotty (2001) used “planning task” in her investigation of planning ability. For this task, children were seated in front of 12 containers and were told that a piece of candy was hiding in one of the containers. The child was also provided stickers. They were then informed that they could have the candy once they located it in the correct container. Children were given several trials to obtain the piece of candy and received points if they chose to use the stickers to help find the piece of the
candy (i.e., the sticker was used to help rule out incorrect containers). Video tapes of the children completing the task were scored by independent raters for this study.

**Meta-analysis Results**

Interpretation of the magnitude of effect sizes was based on Cohen’s (1988) suggestion for labeling: a standardized mean difference less than or equal to .20 is small, .21-.79 is medium, and .80 and greater is large. Positive effect size values obtained for the present study indicate that the comparison group performed better than the autism spectrum group on the outcome measure.

**Primary analysis.** Overall, there were 68 comparisons between an autism and/or Asperger’s Disorder group and a comparison group on planning measures. Of the 68 comparisons, there were 60 comparisons in which the autism and/or Asperger’s Disorder group performed better than the comparison group (see Table 8). 24 comparisons resulted in a large effect size ($d = 0.82$-3.45; 35%), 28 of the comparisons resulted in a medium effect size ($d = 0.22$-$0.79$; 41%), and eight comparisons resulted in a small effect size ($d = 0.02$-$0.19$; 12%).

Of the eight remaining comparisons, one comparison resulted in no difference between the autism and/or Asperger’s Disorder group and the comparison group ($d = 0.00$; 1%), and seven comparisons resulted in negative $d$ values, as the autism and/or Asperger’s Disorder group outperformed the comparison groups on the planning measures. One of these comparisons resulted in a small effect size ($d = -0.05$; 1%), and six of these comparisons resulted in a medium effect size ($d = -0.64$ - -0.22; 9%).

The effect sizes were averaged across executive function measures to produce a weighted summary mean effect size of medium magnitude (Cohen, 1992), $d = 0.67$, with a
95 percent confidence interval of 0.51 to .84 (see Table 9). The failsafe N statistic (Orwin, 1983) indicated that 1,511 additional studies with null results (i.e., effect sizes equal to zero) would be necessary to reduce the weighted mean effect size to a non-significant level. This finding is well above the critical number of studies (i.e., 165); therefore, the theoretical possibility of a falsification of our results by unpublished non-significant studies is therefore highly unlikely.

A test of homogeneity using the weighted effect sizes was significant, $Q(32) = 93.9, p < .0001$, indicating that the effect sizes come from two or more populations. Thus, there are potential moderator variables that may be impacting the extent to which children diagnosed with autism and Asperger’s Disorder exhibit greater planning dysfunction relative to comparison groups.

**Analysis of moderator variables.**

**ASD diagnosis.** The autism spectrum group diagnoses were analyzed to assess whether they were a significant moderator of the summary effect size. First, effect size data was gathered for each diagnostic category (see Table 10). The autism only group revealed a medium effect, $d = 0.715$, with a 95 percent confidence interval of 0.49 to 0.95. The Asperger’s Disorder only groups revealed a medium effect, $d = 0.33$, with a 95 percent confidence interval of -0.07 to 0.73. Finally, the mixed autism and Asperger’s Disorder groups also revealed a medium effect, $d = 0.531$, with a 95 percent confidence interval of 0.34 to 0.73. Overall results suggested that ASD group diagnosis was not a significant moderator, $Q_{between} = 3.029, p = 0.220$. These findings suggested that no differences exist between ASD groups with regard to performance on cognitive planning measures (see Table 10).
**Comparison group diagnosis.** The comparison group diagnoses were analyzed to assess whether they were a significant moderator of the summary effect size. Comparison group diagnoses were first divided into two categories: (a) children diagnosed with clinical disorders commonly associated with executive dysfunction or a clinical comparison group and (b) typically developing children (see Table 11). Effect size data was gathered for each diagnostic category. The clinical comparison groups revealed a medium effect, $d = 0.233$, with a 95 percent confidence interval of 0.05 to 0.42. The typically developing groups revealed a large effect, $d = 0.81$, with a 95 percent confidence interval of 0.65 to 0.97. Overall results suggested that comparison group diagnosis was a significant moderator, $Q_{between} = 21.73, p<0.0001$. These findings suggested that the comparison groups does impact the extent to which the autism and Asperger’s Disorder groups exhibit greater cognitive planning deficits. Specifically, an analysis of the results suggested that studies comparing the ASD group to a typically developing group revealed a larger summary mean effect size than studies comparing the ASD group to a clinical comparison group, $d = 0.81$ and $d = 0.23$, respectively.

A test of homogeneity using the weighted effect sizes was significant for the clinical comparison group, $Q (22) = 57.35, p < .0001$, indicating that the effect sizes for the clinical comparison groups come from two or more populations. Thus, there are potential moderator variables that may be impacting the relationship between cognitive planning and sample diagnoses. To distinguish further among clinical comparison groups, clinical diagnoses were assessed to determine whether they were a significant moderator of the summary effect size (see Table 12). Evaluation of each clinical sample revealed small effect sizes for developmental language disorder ($d = 0.14$), language/learning
disorder ($d = 0.02$) and obsessive compulsive disorder ($d = 0.17$); medium effect sizes for attention deficit/hyperactivity disorder (ADHD), inattentive type ($d = 0.45$), ADHD, hyperactive type ($d = 0.28$), ADHD, unspecified ($d = 0.25$), reading disorder ($d = 0.39$), and Tourette’s Syndrome ($d = 0.44$); and large effect sizes for a mixed clinical diagnosis sample ($d = 1.19$). A medium, negative effect was found for three clinical comparison groups, including developmental delay ($d = -0.36$), moderate traumatic brain injury ($d = -0.45$), and severe traumatic brain injury ($d = -0.23$), suggesting that the ASD group outperformed each of these three groups on cognitive planning measures. Overall results suggested that clinical comparison group diagnosis was a significant moderator, $Q_{between} = 41.44, p < 0.0001$. While there was significant within-group homogeneity at each level of the variable, there were too few studies at each level to draw meaningful conclusions.

A test of homogeneity using the weighted effect sizes was also significant for the typically developing comparison group, $Q (32) = 85.19, p < .0001$, indicating that the effect sizes for the typically developing comparison groups come from two or more populations. Thus, there are potential moderator variables that may be impacting the relationship between cognitive planning and typically developing comparison groups diagnoses.

**Cognitive planning measure.** Cognitive planning measures were analyzed to assess whether they were a significant moderator of the summary effect size. First, effect size data was gathered for each planning measure (see Table 13). Results revealed a small effect for the Rey Complex Figure Test ($d = 0.09$); a medium effect for BRIEF ($d = 0.55$), DKEFS Tower ($d = 0.69$), Key Search ($d = 0.67$), NEPSY Tower ($d = 0.62$), Planning Task ($d = 0.34$), Six Parts Test ($d = 0.69$), Stockings of Cambridge ($d = 0.43$),
Tower of London \((d = 0.62)\), WISC-R Mazes \((d = 0.56)\), WPPSI Mazes \((d = 0.24)\), and Zoo Map \((d = 0.34)\); and a large effect for Tower of Hanoi \((d = 1.2)\), Plan \((d = 1.1)\), and Milner Mazes \((d = 1.24)\). Overall results suggested that cognitive planning measure was not a significant moderator, \(Q_{\text{between}} = 25.36, \ p = 0.064\).

**Meta-regression of moderator variables.**

*Age.* To investigate further the causes for heterogeneity, a meta-regression analysis was performed for age of ASD and comparison groups. The meta-regression analysis did not reveal a significant association between age and effect size. Specifically, results suggested that mean age of the ASD groups \((b = 0.002, z = 0.12, p = 0.91)\) and mean age of the comparison groups \((b = -0.02, z = -1.45, p = 0.15)\) were non-significant moderators.

*IQ.* A meta-regression of ASD and comparison groups IQ was also performed to further investigate causes for heterogeneity. The meta-regression analysis did not reveal a significant association between comparison group IQ and effect size. Specifically, results suggested that comparison group IQ \((b < 0.001, z = 0.06, p = 0.95)\) was a non-significant moderator. In contrast, the meta-regression analysis did reveal a significant association between ASD group IQ and effect size. Specifically, results suggested that the ASD group IQ \((b = -0.01, z = -2.52, p = 0.01)\) was a significant moderator. Larger effects occurred in samples with lower IQ, indicating a stronger association between poorer performance on cognitive planning measures relative to comparison groups in lower IQ children diagnosed with autism or Asperger’s Disorder.
Chapter V. Discussion

The aim of this meta-analytic study was to integrate findings from a wide selection of quantitative research on cognitive planning in children diagnosed with autism and Asperger’s Disorder in order to give a clearer perspective on the executive dysfunction profile of these populations. The purpose of this study was five-fold: (a) to examine whether children diagnosed with autism and Asperger’s Disorder exhibit greater planning deficits relative to all comparison groups; (b) to determine whether children diagnosed with autism may be differentiated from children diagnosed with Asperger’s Disorder based upon their performances on measures of cognitive planning; (c) to explore if comparison group diagnosis has any impact on the extent to which the autism and Asperger’s Disorder groups exhibit greater planning dysfunction; and (d) to determine whether the type of cognitive planning measure used impacts the extent to which the autism and Asperger’s Disorder groups exhibit greater planning dysfunction relative to comparison groups; and (e) to determine whether age and IQ impact cognitive planning deficits in children diagnosed with autism and Asperger’s Disorder. Findings from the current meta-analysis will be discussed with reference to specific research questions introduced in Chapter III.

Research question #1. In order to examine whether children diagnosed with autism and Asperger’s Disorder exhibit greater cognitive planning deficits relative to comparison groups, the weighted mean effect size across all available studies was analyzed. The results suggested that children diagnosed with autism and Asperger’s
Disorder exhibit greater weaknesses in terms of cognitive planning relative to comparison groups. Furthermore, an examination of the failsafe $N$ statistic indicated that it would take 1,511 studies to nullify the current meta-analytic results, suggesting that the possibility of a falsification of the results by unpublished non-significant studies is highly unlikely.

However, upon further examination, it was discovered that there was a significant level heterogeneity among studies, suggesting that the variability found among studies may not be attributed to sampling error alone. Thus, the findings suggested that there are moderating variables that confound the overall weighted mean effect size. As a results of this finding, moderator variables were further examined.

**Research question #2.** ASD group diagnosis was examined as a moderator variable to determine if there were significant differences between the autism only, Asperger’s Disorder only, and mixed autism and Asperger’s Disorder groups that may account for the variance found among the total sample. An examination of the moderating variable, ASD diagnoses, revealed medium effects across each level (i.e., autism only, Asperger’s Disorder only, and mixed autism and Asperger’s Disorder groups). The analysis indicated that there was not a significant difference between ASD diagnostic groups, suggesting that ASD diagnosis was not a significant moderator of the overall mean effect.

These findings suggested that children diagnosed with autism may not be differentiated from children diagnosed with Asperger’s Disorder based upon cognitive planning measure performance. Rather, cognitive planning dysfunction appeared to be a commonly shared characteristic among these two populations. While one might conclude that this data provides support for the argument that Asperger’s Disorder is a variant of
the autism diagnosis rather than a distinct and separate diagnostic category (Manjiviona & Prior, 1999; Ozonoff, Rogers, & Pennington, 1991; Szatmari, et al., 1990), one must also consider the convergence hypothesis (Courchesne, et al., 1999), that suggests variable etiologies among disorders may lead to overlapping behavior and symptom profiles among disorders.

**Research question #3.** Comparison group diagnosis was also examined as a moderator variable to determine whether there were significant differences between studies that used typically developing children as the comparison group and studies that children diagnosed with clinical diagnoses commonly associated with executive dysfunction as the comparison group. Results revealed a large effect for studies comparing the ASD group to typically developing children and a medium effect for studies comparing the ASD group to children diagnosed with a clinical disorder commonly associated with executive dysfunction. In examining study heterogeneity, a significant difference was discovered between these two groups of studies, suggesting that comparison group diagnosis was a significant moderator of the overall weighted mean effect. Thus, while children diagnosed with autism and Asperger’s Disorder exhibited greater planning deficits relative to all comparison groups included in the meta-analysis, they tended to exhibit a greater level of difference in cognitive planning when compared to typically developing children than when compared to other clinical groups.

Further examination of the results, however, suggested that there continued to be significant levels of variance after controlling for comparison group diagnostic categories. An attempt to further examine the clinical comparison group was made by dividing the category into specific comparison group diagnoses. While there were too
few samples available for each diagnostic category to draw statistically significant conclusions, a preliminary observation of the available data was made. Results suggested a medium effect for studies involving children diagnosed with ADHD inattentive type, ADHD hyperactive type, ADHD unspecified, reading disorder, and Tourette’s Syndrome; a small effect for studies involving developmental language disorder, language/learning disorder, and obsessive compulsive disorder; and children diagnosed with autism and Asperger’s disorder outperformed children diagnosed with developmental delay, moderate traumatic brain injury, and severe traumatic brain injury.

These preliminary results suggest that children diagnosed with autism and Asperger’s Disorder did not exhibit greater planning deficits than all clinical comparison groups and that there is variation in the extent to which they exhibit greater planning deficits relative to the additional clinical groups. While these findings must be interpreted with caution until more studies are available for each clinical comparison groups, the findings suggested that cognitive planning deficits are not a unique characteristic to children diagnosed with autism and Asperger’s Disorder. Furthermore, the findings suggested that there remains no clear pattern of differentiation from other comparison groups on the basis of cognitive planning skills.

**Research question #4.** Type of cognitive planning measure was also examined as a moderator variable to determine whether significant differences would exist between studies using differing methods of cognitive planning skills measurement. An examination of this moderating variable did not reveal significant between group differences, suggesting that the type of cognitive planning measure used did not impact the extent to which children diagnosed with autism and Asperger’s Disorder exhibited
greater cognitive planning deficits relative to comparison groups. Thus, it appears that the cognitive planning measures included in the current meta-analysis were to some extent measuring the same cognitive construct despite the inclusion of seemingly different measures (e.g., clinical vs. parent-rated measures; well established and validated vs. newly developed and study specific measures).

**Research question #5.** In order to examine whether age impacted the level of cognitive planning dysfunction exhibited by children, a meta regression of age and planning skills was run. The results did not suggest age was a significant moderator of cognitive planning skills in either the autism and Asperger’s Disorder groups or the comparison groups. These findings suggested that age did not have an impact on the level of cognitive planning dysfunction across studies, and thus does not contribute to the overall heterogeneity found within the weighted mean effect.

While researchers have found that cognitive planning is a skill that continues to develop into adolescence and beyond (Das, Kar, & Parilla, 1996), the current results suggested that children with autism and Asperger’s Disorder exhibited greater planning deficits relative to comparison groups regardless of age. Thus, age did not play a major role in the differentiation between the autism and Asperger’s Disorder groups and comparison groups based upon cognitive planning measure performance.

**Research question #6.** Finally, IQ was examined as a moderator variable to determine whether IQ impacted the level of cognitive planning dysfunction among study samples. Results revealed that while IQ was not a significant moderator variable for children in the comparison groups, IQ was a significant moderator variable for children diagnosed with autism and Asperger’s Disorder. Thus, variations among IQ in the autism
and Asperger’s disorder groups appeared to have contributed to the overall heterogeneity of the weighted mean effect and may, therefore, account for a portion of the observed differences in cognitive planning skills between ASD and comparison groups.

More specifically, the results suggested that the children in the ASD group with lower IQ scores were more easily differentiated from the comparison groups than children in the ASD group with higher IQ scores. In contrast, as IQ scores improved for the ASD group, there tended to be lesser differentiation between the ASD and comparison groups in terms of cognitive planning performance. These findings indicated that IQ played a role in the overall weight mean effect size across studies, which suggested that cognitive planning skills alone do not distinguish children in the ASD groups from children in the comparison groups.

**Summary and conclusions.** While results from the current meta-analysis suggest that children diagnosed with autism and Asperger’s Disorder exhibit cognitive planning weaknesses, the results do not suggest that cognitive planning is a causative factor for autism or Asperger’s Disorder. Rather cognitive planning dysfunction is likely one of many factors underlying the expression of autism and Asperger’s Disorder and therefore may not be used as a diagnostic marker.

In order to examine other factors that may contribute to the differentiation between the ASD and comparison groups included in the meta-analysis, several variables were explored (i.e., ASD diagnosis, age, IQ, type of cognitive planning measure, and comparison group diagnosis). The results suggested that age, type of cognitive planning measure, and ASD diagnoses did not affect the extent to which children diagnosed with autism and Asperger’s Disorder exhibited greater planning dysfunction relative to
comparison groups. Conversely, the effect sizes obtained for intellectual functioning and comparison group diagnoses suggested that these areas might be just as, if not more, likely to account for the relative level of cognitive planning dysfunction than true cognitive planning differences between groups.

More specifically, it appears that IQ affected effect sizes, in that lower IQs among children diagnosed with autism and Asperger’s disorder accounted for greater cognitive planning dysfunction relative to the comparison groups, which is consistent with previous research findings that suggest executive functioning and intellectual functioning tend to be consistent (Foley, Garcia, Shaw, & Golden, 2009). Given that the level of cognitive planning dysfunction did not persist among children diagnosed with autism and Asperger’s Disorder despite differences in intellectual functioning, the overall weighted mean effect size does not represent true differences in cognitive planning skills between the ASD and comparison group alone, but rather also represent differences in intellectual functioning.

It was not surprising to discover a significantly larger effect size when the comparison groups were comprised of typically developing children rather than children diagnosed with a clinical disorder commonly associated with executive functioning. Furthermore, when specific diagnoses were examined within the clinical comparison group, there was a great level of variability in the extent to which the ASD group exhibited greater planning dysfunction than the comparison group, including cases in which the ASD group outperformed the comparison group. Consistent with previous research findings (for review, see Hill, 2004), these results suggest that children diagnosed with autism and Asperger’s Disorder may not be clearly differentiated from
children diagnosed with other clinical disorders associated with executive dysfunction.

Overall, the findings from the current meta-analysis provide a clearer picture regarding the executive functioning profile of children diagnosed with autism and Asperger’s Disorder. Specifically, while there is a consistent weakness in cognitive planning among children diagnosed with autism and Asperger’s Disorder, the findings suggest that cognitive planning is not a causative factor in autism and Asperger’s disorder and, therefore, may not be reliably used as a diagnostic marker of autism or Asperger’s Disorder.

Implications for Clinicians

Although the current meta-analytic results do not suggest that cognitive planning dysfunction underlies the expression of autism or Asperger’s disorder, it is apparent that these children tend to exhibit cognitive planning weaknesses. Cognitive planning dysfunction, therefore, appears to be an area in need of assessment and intervention among these populations.

Assessment. Findings from the current investigation support the need for neuropsychological assessment of children who have either suspected or documented diagnoses of autism or Asperger’s Disorder. While neuropsychological assessment is not a necessary component of the diagnostic assessment of children with suspected ASDs, such evaluations will help clinicians establish a baseline for the child, assess areas in need of treatment or intervention, and inform treatment progress (Corbett, Carmean, & Fein, 2009; Marcus, Flagler, & Robinson, 2001).

While there were no significant differences between studies using differing measurements of cognitive planning, various cognitive planning measures appeared more
sensitive to cognitive planning deficits in autism and Asperger’s Disorder than others. Specifically, the cognitive planning measures yielding large effect sizes included Tower of Hanoi, Milner Mazes, and Plan. However, it should be noted that only the Tower of Hanoi \((k = 6)\) had enough studies to draw generalizable conclusions. In contrast, the measures that appeared least sensitive (i.e., small effect sizes) to cognitive planning dysfunction in children with autism and Asperger’s Disorder was the Rey Complex Figure Test. All other measures were adequately sensitive to cognitive planning dysfunction, as their effect sizes fell in the medium range. Thus, the findings from this study suggest that perhaps the Tower of Hanoi may be the best measure of cognitive planning when conducting a neuropsychological assessment of children with autism and Asperger’s Disorder.

**Intervention.** Cognitive planning is a skill that is important in many areas of everyday functioning, including areas such as completing daily errands efficiently (Garner, 2009), reading (Altemeier, et al., 2006; Sesman, et al., 2008), and overall academic performance (Garner, 2009; Meltzer, et al., 2007). Furthermore, cognitive planning dysfunction has been associated with increased risky behaviors, such as injection drug use (Severtson, et al., 2009) and risky sex practices (Gebhardt, et al., 2006). Given the possible difficulties associated with cognitive planning deficits, children diagnosed with autism and Asperger’s Disorder would likely benefit from intervention strategies aimed at building improved cognitive planning skills.

One strategy aimed at improving planning skills, as well as other cognitive skills, is referred to as, “Metacognitive Strategy Instruction” (Kennedy, et al., 2008). This approach intervenes specifically on planning ability through the use of direct instruction
to teach children to regulate their behavior by breaking down complex tasks into simpler steps. For example, in order to improve planning in academic settings, students may be instructed and prepared to think about what their learning goals are and how they will go about accomplishing them. These intervention strategies may be employed by psychologists, other intervention specialists, and educators to improve planning skills in a variety of contexts and settings. Overall, research has found this strategy to be a helpful and effective technique in the improvement of planning skills (Kennedy, et al., 2008).

Other intervention strategies that implement reading skills through improved planning include training in the use of specific reading strategies, such as “comprehension monitoring” or “use of linguistic context” (Clay, 1985; Pinnell, 1989; RAND Reading Study Group, 2002; Vellutino & Scanlon, 2002). These processes may be implemented in the school environment, as well as through various therapeutic intervention settings, and have been found to be effective in proving reading comprehension among children with planning deficits.

While there are many varying strategies aimed at improving planning skills, it is important that psychologists and other intervention specialists become aware of the need for interventions aimed at cognitive planning dysfunction among children diagnosed with autism and Asperger’s Disorder. Only when planning deficits are recognized will the psychologist or other intervention specialist be able to choose strategies most appropriate for the child’s needs and ability level.

**Study Limitations and Future Directions**
Some of the limitations of this analysis are inherent in meta-analysis as a research tool, while other limitations are the result of limitations of the original studies included in the meta-analysis.

**Meta-analysis.** As in any meta-analysis using observational data, the effect estimates varied a great deal and there was a significant amount of heterogeneity across the studies. Though heterogeneity was minimized by using random effects models and by analyzing data by different categories or moderators, there may be residual factors that affected heterogeneity that have not been accounted for in the current analyses. While one could criticize the rationality of conducting a meta-analysis given the heterogeneity, others have argued against not conducting a meta-analysis. Specifically, they have suggested that leaving the reader to make all the decision about summative effect measures, when adequate data is available to summarize, leads to increased erroneous assumptions by the readers due to “vote counting” (Borenstein, Hedges, Higgins, & Rothstein, 2009; Hunter & Schmidt, 1990).

While an effort was made to make this review comprehensive, it is possible that some studies have not been included either due to publication in databases outside those included in this meta-analysis, or being published in non-scientific literature. However, the failsafe N statistic was run to control for this problem, and it was determined that the number of studies reporting null results required to nullify the current findings was well over what would be reasonably expected.

Finally, completion of a meta-analysis requires that each original study include specific information in order to make accurate comparisons and calculations. Unfortunately, not all of the studies obtained were able to be included in the current
meta-analysis, as they lacked information needed for statistical analysis. For example, some of these studies did not provide sufficient data to calculate an effect size and therefore could not be included. Similarly, not all studies provided adequate information to meet the inclusion criteria for the study, and were therefore excluded. Thus, it is recommended that future studies ensure that they provide data and information sufficient for replication and meta-analysis, so that they may be included in future meta-analytic reviews.

**Original studies.** One limitation of the original studies involved in the current meta-analysis is the relatively low number of studies involving each unique diagnostic category in the clinical comparison groups. Most clinical groups were included in only one study in which they were compared to the ASD group, limiting the generalizability of their results. Thus, it is recommended that future researchers continue to conduct studies involving the comparison of children diagnosed with autism and Asperger’s Disorder and children diagnosed with other clinical disorders commonly associated with executive dysfunction to more clearly elucidate the relative cognitive planning profiles among these populations.

Furthermore, given that IQ was a significant moderator of cognitive planning in the current meta-analysis, future researchers should only include samples that have been matched on the basis of intellectual functioning. Such information would further clarify the nature of cognitive planning in children diagnosed with autism and Asperger’s Disorder without this important confounding variable.

Although there were no significant differences between studies on the basis of type of cognitive planning measure used, researchers have argued that most
neuropsychological instruments measure more than one aspect of cognitive functioning (Hill, 2004). Therefore, the interpretation of the current meta-analysis results is complicated by the use of measures that also tap neurocognitive skills and abilities other than cognitive planning. For example, in addition to planning ability, tower tasks have been found to measure inhibition and working memory (Hill, 2004). Such findings make it difficult to interpret any deficits on planning measures as being purely a difficulty in the executive functioning domain of planning. Future studies of executive functioning of autism and Asperger’s disorder should attempt to use tasks that better assess and isolate planning ability from other neurocognitive skills.

Planning tasks have also been shown to have low test-retest reliability within the normal population (Bishop, Aamodt-Leeper, Creswell, McGurk, & Skuse, 2001; Lowe & Rabbitt, 1998). Therefore, the results from such planning measures must be interpreted with caution. In an effort to improve data interpretation, future research should be conducted with planning tasks that have existing reliability data and/or independently assess the reliability of chosen planning measures.

Finally, while these findings help to clarify the cognitive planning profile of children diagnosed with autism and Asperger’s Disorder, future research of other executive functioning domains is needed in order to further clarify the executive functioning profile among these populations. Such studies would help to further elucidate the executive functioning hypothesis for ASDs. Furthermore, although the current meta-analysis suggests that cognitive planning may not serve as a diagnostic marker, further research involving other executive functioning domains will help determine whether any
other areas of executive dysfunction may serve as a diagnostic marker of autism and Asperger’s Disorder.

Conclusion

Executive dysfunction in the planning ability domain plays an important role in the complex neuropsychology of autism spectrum disorders. Such dysfunction is likely to contribute to difficulties in everyday life. However, while there does appear to be deficits in planning among children on the autism spectrum, the results do not suggest that planning deficits are an underlying feature of all cases of autism or Asperger’s disorder. Additional research is needed to clarify the relationship between various diagnoses, intellectual functioning, and the relationship between cognitive planning and other neurocognitive domains.
Appendix A - Tables

Table A1

DSM-IV-TR Criteria for 299.00 Autistic Disorder

A. A total of six (or more) items from (1), (2), and (3), with at least two from (1), and one each from (2) and (3):
   (1) qualitative impairment in social interaction, as manifested by at least two of the following:
      a. marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
      b. failure to develop peer relationships appropriate to developmental level
      c. a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest)
      d. lack of social or emotional reciprocity
   (2) qualitative impairments in communication as manifested by at least one of the following:
      a. delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime)
      b. in individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others
      c. stereotyped and repetitive use of language or idiosyncratic language
      d. lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level
   (3) restricted repetitive and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:
      a. encompassing preoccupation with one of more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus
      b. apparently inflexible adherence to specific, nonfunctional routines or rituals
      c. stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole-body movements)
      d. persistent preoccupation with parts of objects
B. Delays or abnormal functioning in at least one of the following areas, with onset prior to age 3 years: (1) social interaction, (2) language as used in social communication, or (3) symbolic or imaginative play.

C. The disturbance is not better accounted for by Rett’s Disorder or Childhood Disintegrative Disorder.

*Note.* Adapted from *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (American Psychiatric Association, 2000).
**ICD-10 Criteria for Childhood Autism**

A. Abnormal or impaired development is evident before the age of 3 years in at least one of the following areas:
   1. receptive or expressive language as used in social communication;
   2. the development of selective social attachments or of reciprocal social interaction;
   3. functional or symbolic play.

B. A total of at least six symptoms from (1), (2) and (3) must be present, with at least two from (1) and at least one from each of (2) and (3):

   1. Qualitative impairment in social interaction are manifest in at least two of the following areas:
      a. failure adequately to use eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction;
      b. failure to develop (in a manner appropriate to mental age, and despite ample opportunities) peer relationships that involve a mutual sharing of interests, activities and emotions;
      c. lack of socio-emotional reciprocity as shown by an impaired or deviant response to other people’s emotions; or lack of modulation of behavior according to social context; or a weak integration of social, emotional, and communicative behaviors;
      d. lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g. a lack of showing, bringing, or pointing out to other people objects of interest to the individual).

   2. Qualitative abnormalities in communication as manifest in at least one of the following areas:
      a. delay in or total lack of, development of spoken language that is not accompanied by an attempt to compensate through the use of gestures or mime as an alternative mode of communication (often preceded by a lack of communicative babbling);
      b. relative failure to initiate or sustain conversational interchange (at whatever level of language skill is present), in which there is reciprocal responsiveness to the communications of the other person;
      c. stereotyped and repetitive use of language or idiosyncratic use of words or phrases;
      d. lack of varied spontaneous make-believe play or (when young) social imitative play.

   3. Restricted, repetitive, and stereotyped patterns of behavior, interests, and activities are manifested in at least one of the following:
a. An encompassing preoccupation with one or more stereotyped and restricted patterns of interest that are abnormal in content or focus; or one or more interests that are abnormal in their intensity and circumscribed nature though not in their content or focus;
b. Apparently compulsive adherence to specific, nonfunctional routines or rituals;
c. Stereotyped and repetitive motor mannerisms that involve either hand or finger flapping or twisting or complex whole body movements;
d. Preoccupations with part-objects of non-functional elements of play materials (such as their odor, the feel of their surface, or the noise or vibration they generate).

C. The clinical picture is not attributable to the other varieties of pervasive developmental disorders; specific development disorder of receptive language (F80.2) with secondary socio-emotional problems, reactive attachment disorder (F94.1) or disinhibited attachment disorder (F94.2); mental retardation (F70-F72) with some associated emotional or behavioral disorders; schizophrenia (F20.-) of unusually early onset; and Rett’s Syndrome (F84.12).

Table A3

DSM-IV-TR Criteria for 299.80 Asperger’s Disorder

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

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

C. The disturbance causes clinically significant impairment in social, occupational, or other important areas of functioning.

D. There is no clinically significant general delay in language (e.g., single words used by age 2 years, communicative phrases used by age 3 years).

E. There is no clinically significant delay in cognitive development or in the development of age-appropriate self-help skills, adaptive behavior (other than in social interaction), and curiosity about the environment in childhood.

F. Criteria are not met for another specific Pervasive Developmental Disorder or Schizophrenia.

Note. Adapted from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (American Psychiatric Association, 2000).
A. A lack of any clinically significant general delay in spoken or receptive language or cognitive development. Diagnosis requires that single words should have developed by two years of age or earlier and that communicative phrases be used by three years of age or earlier. Self-help skills, adaptive behavior and curiosity about the environment during the first three years should be at a level consistent with intellectual development. However, motor milestones may be somewhat delayed and motor clumsiness is usual (although not a necessary diagnostic feature). Isolated special skills, often related to abnormal preoccupations, are common, but are not required for diagnosis.

B. Qualitative abnormalities in reciprocal social interaction (criteria as for autism).

C. An unusually intense circumscribed interest or restrictive, repetitive, and stereotyped patterns of behaviour, interests and activities (criteria as for autism; however, it would be less usual for these to include either motor mannerisms or preoccupations with part-objects or non-functional elements of play materials).

D. The disorder is not attributable to other varieties of pervasive developmental disorder; schizotypal disorder (F21); simple schizophrenia (F20.6); reactive and disinhibited attachment disorder of childhood (F94.1 and .2); obsessional personality disorder (F60.5); obsessive-compulsive disorder (F42).

Note. Adapted from International classification of diseases: Diagnostic criteria for research, 10th edition (World Health Organization, 1992).
Proposed DSM-V Criteria for Autism Spectrum Disorder

Must meet criteria A, B, C, and D:
A. Persistent deficits in social communication and social interaction across contexts, not accounted for by general developmental delays, and manifest by all 3 of the following:
   1. Deficits in social-emotional reciprocity; ranging from abnormal social approach and failure of normal back and forth conversation through reduced sharing of interests, emotions, and affect and response to total lack of initiation of social interaction.
   2. Deficits in nonverbal communicative behaviors used for social interaction; ranging from poorly integrated verbal and nonverbal communication, through abnormalities in eye contact and body-language, or deficits in understanding and use of nonverbal communication, to total lack of facial expression or gestures.
   3. Deficits in developing and maintaining relationships, appropriate to developmental level (beyond those with caregivers); ranging from difficulties adjusting behavior to suit different social contexts through difficulties in sharing imaginative play and in making friends to an apparent absence of interest in people.
B. Restricted, repetitive patterns of behavior, interests, or activities as manifested by at least two of the following:
   1. Stereotyped or repetitive speech, motor movements, or use of objects; (such as simple motor stereotypies, echolalia, repetitive use of objects, or idiosyncratic phrases).
   2. Excessive adherence to routines, ritualized patterns of verbal or nonverbal behavior, or excessive resistance to change; (such as motoric rituals, insistence on same route or food, repetitive questioning or extreme distress at small changes).
   3. Highly restricted, fixated interests that are abnormal in intensity or focus; (such as strong attachment to or preoccupation with unusual objects, excessively circumscribed or perseverative interests).
   4. Hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of environment; (such as apparent indifference to pain/heat/cold, adverse response to specific sounds or textures, excessive smelling or touching of objects, fascination with lights or spinning objects).
C. Symptoms must be present in early childhood (but may not become fully manifest until social demands exceed limited capacities).
D. Symptoms together limit and impair everyday functioning.

Note. Adapted from www.DSM5.org (American Psychiatric Association, 2010).
Table A6

*Proposed Severity Specifiers for Autism Spectrum Disorder Criteria in DSM-V*

<table>
<thead>
<tr>
<th>Severity Level for ASD</th>
<th>Social Communication</th>
<th>Restricted Interests &amp; Repetitive Behaviors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>‘Requiring very substantial support’</td>
<td>Severe deficits in verbal and nonverbal social communication skills cause severe impairments in functioning; very limited initiation of social interactions and minimal response to social overtures from others.</td>
<td>Preoccupations, fixated rituals and/or repetitive behaviors markedly interfere with functioning in all spheres. Marked distress when rituals or routines are interrupted; very difficulty to redirect from fixated interest or returns to it quickly.</td>
</tr>
<tr>
<td>Level 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>‘Requiring substantial support’</td>
<td>Marked deficits in verbal and nonverbal social communication skills; social impairments apparent even with supports in place; limited initiation of social interactions and reduced or Abnormal response to social overtures from others.</td>
<td>RRBs and/or preoccupations or fixated interests appear frequently enough to be obvious to the casual observer and interfere with functioning in a variety of contexts. Distress or frustration is apparent when RRB’s are interrupted; difficult to redirect from fixated interest.</td>
</tr>
<tr>
<td>Level 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>‘Requiring support’</td>
<td>Without supports in place, deficits in social communication cause impairments. Has difficulty initiating social interactions and demonstrates atypical or unsuccessful responses to social overtures of others may appear to have decreased interest in social interactions.</td>
<td>Rituals and repetitive behaviors cause significant significant interference with functioning in one or more contexts. Resists attempts by others to interrupt RRB’s or to be redirected from fixated interest.</td>
</tr>
</tbody>
</table>

*Note.* Adapted from www.DSM5.org, American Psychiatric Association (2010).
### Table A7

**Descriptive Statistics by Group Diagnosis**

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<th>Study Group Diagnosis</th>
<th>n</th>
<th>Age (SD)</th>
<th>IQ  (SD)</th>
</tr>
</thead>
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<tr>
<td>ASD</td>
<td>1,020</td>
<td>10.72 (2.67)</td>
<td>99.28 (7.19)</td>
</tr>
<tr>
<td>Autism</td>
<td>445</td>
<td>9.55 (2.91)</td>
<td>99.45 (7.83)</td>
</tr>
<tr>
<td>Asperger’s Disorder</td>
<td>88</td>
<td>9.34 (0.89)</td>
<td>103.25 (2.24)</td>
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<tr>
<td>Mixed Autism and AD</td>
<td>487</td>
<td>10.98 (2.45)</td>
<td>98.34 (7.34)</td>
</tr>
<tr>
<td>Comparison Group</td>
<td>1,591</td>
<td>10.75 (2.47)</td>
<td>104.44 (6.42)</td>
</tr>
<tr>
<td>Clinical Comparison</td>
<td>632</td>
<td>10.37 (2.32)</td>
<td>101.6 (5.57)</td>
</tr>
<tr>
<td>Typically Developing</td>
<td>959</td>
<td>10.24 (2.71)</td>
<td>106.53 (6.24)</td>
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<tr>
<td>Total</td>
<td>2,611</td>
<td>10.26 (2.54)</td>
<td>102.31 (7.12)</td>
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Table A8

d-Values for Study Comparisons

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample</th>
<th>Measure</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cane, D.J. (2007)</td>
<td>M-ASD LLD</td>
<td>BRIEF (Plan/Organize)</td>
<td>0.02</td>
</tr>
<tr>
<td>Gillotty, L. (2001)</td>
<td>Autism DD TD</td>
<td>Planning Task</td>
<td>-0.36 1.04</td>
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<tr>
<td>Gioia, G.A., Isquith, P.K., Kenworthy, L., &amp; Barton, R.M. (2002)</td>
<td>M-ASD ADHD-C ADHD-I Mod TBI Sev TBI Reading D/o TD</td>
<td>BRIEF (Plan/Organize)</td>
<td>0.36 0.74 -0.45 -0.23 0.39 1.29</td>
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<tr>
<td>Glasier, P. (2009)</td>
<td>Asperger’s M-ADHD TD</td>
<td>DKEFS Tower</td>
<td>0.34 0.51</td>
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<tr>
<td>Study</td>
<td>Sample</td>
<td>Measure</td>
<td>$d$</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>---------</td>
<td>-----------------</td>
<td>------</td>
</tr>
<tr>
<td>Happe, F., Booth, R., Charlton, R., &amp; Hughes, C. (2006)</td>
<td>M-ASD M-ADHD TD</td>
<td>Stockings of Cambridge</td>
<td>-0.18 0.05</td>
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<td>Kuschner, E.S., Bodner, K.E., &amp; Minshew, N.J. (2009)</td>
<td>Autism TD</td>
<td>Rey-O</td>
<td>-0.15</td>
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<td>Landa, R.J., &amp; Goldber, M.C. (2005)</td>
<td>Autism TD</td>
<td>Stockings of Cambridge</td>
<td>1.01</td>
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<tr>
<td>Matson, M.A.</td>
<td>Asperger DLD TD Autism DLD TD</td>
<td>NEPSY Tower</td>
<td>-0.64 1.05 0.32 1.21</td>
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<tr>
<td>Low, R.</td>
<td>Autism TD</td>
<td>DKEFS Tower BRIEF (Plan/Organize)</td>
<td>1.35 1.55</td>
</tr>
<tr>
<td>Ozonoff, S., et al. (2004)</td>
<td>Autism TD</td>
<td>Stockings of Cambridge</td>
<td>0.87</td>
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<td>Study</td>
<td>Sample</td>
<td>Measure</td>
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<tr>
<td>----------------------------------------------------------------------</td>
<td>----------------------</td>
<td>---------------------</td>
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<tr>
<td></td>
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<td></td>
<td>TD</td>
<td>WPPSI Mazes</td>
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<td></td>
<td>TD</td>
<td>WPPSI Mazes</td>
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<td></td>
<td>TD-Age Matched</td>
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<td></td>
<td>TD-MA Matched</td>
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<td>Robinson, S., Goddard, L., Dritschel, B., Wisley, M., &amp; Howlin, P.</td>
<td>M-ASD</td>
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<tr>
<td>(2009)</td>
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<tr>
<td>Semrud-Clikeman, M., Walkowia, J., Wilkinson, A., &amp; Butcher, B.</td>
<td>Asperger’s ADHD-C</td>
<td>DKEFS Tower</td>
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<tr>
<td>(2010)</td>
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<td></td>
<td>TD</td>
<td></td>
<td>0.82</td>
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<td></td>
<td>Asperger’s ADHD-C</td>
<td>BRIEF (P/O)</td>
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<td></td>
<td>ADHD-I</td>
<td></td>
<td>-0.41</td>
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<td></td>
<td>TD</td>
<td></td>
<td>1.05</td>
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<td>Sinzig, J., Morsch, D., Bruning, N., Schmidt, M.H., &amp; Lehmkuhl, G.</td>
<td>M-ASD</td>
<td>Stockings of</td>
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<td>0.68</td>
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<tr>
<td></td>
<td></td>
<td>Zoo Maps</td>
<td>0.34</td>
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<tr>
<td>Zandt, F., Prior, M., &amp; Kyrios, M. (2009)</td>
<td>M-ASD OCD TD</td>
<td>Rey-O</td>
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<tr>
<td></td>
<td></td>
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<td>0.19</td>
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</table>

Key. M-ASD: Mixed Autism Spectrum Disorder Sample
TD: Typically Developing Sample
LLD: Comorbid Language and Learning Disorder Sample
M-ADHD: Mixed ADHD Sample
DD: Developmental Delay Sample
ADHD-C: ADHD-Combined Type Sample
ADHD-I: ADHD-Inattentive Type Sample
Mod TBI: Moderate Traumatic Brain Injury Sample
Sev TBI: Severe Traumatic Brain Injury Sample
Reading D/o: Reading Disorder Sample
DLD: Developmental Language Disorder Sample
TS: Tourette Syndrome Sample
CC: Clinical Control Sample
OCD: Obsessive Compulsive Disorder Sample
Table A9

*Average Effect Size Across Studies, 95% Confidence Interval, and Test of Heterogeneity*

<table>
<thead>
<tr>
<th>Number of Studies (k)</th>
<th>Weighted Mean Effect (d)</th>
<th>Standard Error</th>
<th>Variance</th>
<th>Lower Limit</th>
<th>Upper Limit</th>
<th>Q-value</th>
<th>df (Q)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>33</td>
<td>0.674</td>
<td>0.087</td>
<td>0.008</td>
<td>0.51</td>
<td>0.84</td>
<td>93.895</td>
<td>32</td>
<td>0.000</td>
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</table>
Table A10

*Effect Size and Homogeneity Indices for the Meta-Analysis of ASD Group Diagnoses*

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of effect sizes</th>
<th>d</th>
<th>Q-value</th>
<th>df (Q)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asperger’s Disorder</td>
<td>7</td>
<td>0.33</td>
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<td>Autism</td>
<td>21</td>
<td>0.72</td>
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<td></td>
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<tr>
<td>Mixed ASD</td>
<td>28</td>
<td>0.53</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Total between</td>
<td>3.029</td>
<td>2</td>
<td>0.22</td>
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<td></td>
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</table>

*Note.* *Significant indicates rejection of homogeneity (p < .05).*
Table A11

**Effect Size and Homogeneity Indices for the Meta-Analysis of Typical vs. Clinical Comparison Groups**

<table>
<thead>
<tr>
<th>Grouping</th>
<th>Number of effect sizes</th>
<th>$d$</th>
<th>$Q$-value</th>
<th>$df (Q)$</th>
<th>$p$</th>
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<tr>
<td>Clinical</td>
<td>23</td>
<td>0.23</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Typically Developing</td>
<td>33</td>
<td>0.81</td>
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<tr>
<td>Total between</td>
<td></td>
<td>21.73</td>
<td>1</td>
<td>&lt;0.001*</td>
<td></td>
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</table>

*Note.* *Significant indicates rejection of homogeneity ($p < .05$).
Table A12

*Effect Size and Homogeneity Indices for the Meta-Analysis of Clinical Comparison Group Diagnoses*

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of effect sizes</th>
<th>( d )</th>
<th>( Q )-value</th>
<th>( df (Q) )</th>
<th>( p )</th>
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<tbody>
<tr>
<td>Moderate TBI</td>
<td>1</td>
<td>-0.45</td>
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</tr>
<tr>
<td>Developmental Delay</td>
<td>1</td>
<td>-0.36</td>
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</tr>
<tr>
<td>Severe TBI</td>
<td>1</td>
<td>-0.23</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Language/Learning D/o</td>
<td>1</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obsessive Compulsive D/o</td>
<td>1</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Develop Language D/o</td>
<td>3</td>
<td>0.14</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>ADHD-Mixed</td>
<td>7</td>
<td>0.25</td>
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</tr>
<tr>
<td>ADHD-Combined</td>
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<td></td>
</tr>
<tr>
<td>Reading Disorder</td>
<td>1</td>
<td>0.39</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tourette’s Syndrome</td>
<td>2</td>
<td>0.44</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHD-Inattentive Type</td>
<td>2</td>
<td>0.48</td>
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<td></td>
</tr>
</tbody>
</table>

Overall between 41.44 10 <0.001*

*Note. *Significant indicates rejection of homogeneity (\( p < .05 \)).

TBI= Traumatic brain injury
D/o= Disorder
Develop= Developmental
ADHD= Attention Deficit/Hyperactivity Disorder
ADHD Mixed= group comprised of individuals diagnosed with any variant of ADHD
ADHD Combined= ADHD inattentive/hyperactive type
### Table A13

*Effect Size and Homogeneity Indices for the Meta-Analysis of Cognitive Planning Measure*

<table>
<thead>
<tr>
<th>Measure</th>
<th>Number of effect sizes</th>
<th>$d$</th>
<th>$Q$-value</th>
<th>$df (Q)$</th>
<th>$p$</th>
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</thead>
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<tr>
<td>Rey Complex Figure Test</td>
<td>3</td>
<td>0.09</td>
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<td></td>
</tr>
<tr>
<td>WPPSI Mazes</td>
<td>2</td>
<td>0.24</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zoo Map</td>
<td>1</td>
<td>0.34</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Planning Task</td>
<td>2</td>
<td>0.34</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stockings of Cambridge</td>
<td>10</td>
<td>0.43</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WISC-R Mazes</td>
<td>1</td>
<td>0.56</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tower of London</td>
<td>10</td>
<td>0.62</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEPSY Tower</td>
<td>7</td>
<td>0.62</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Key Search</td>
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<td>0.67</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Six Parts Test</td>
<td>1</td>
<td>0.69</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DKEFS Tower</td>
<td>9</td>
<td>0.69</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tower of Hanoi</td>
<td>6</td>
<td>1.20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milner Mazes</td>
<td>2</td>
<td>1.24</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total between</strong></td>
<td><strong>25.36</strong></td>
<td><strong>16</strong></td>
<td><strong>0.064</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* *Significant indicates rejection of homogeneity ($p < .05$).

WPPSI Mazes = Wechsler Preschool and Primary Scale of Intelligence, Mazes subtest  
WISC-R = Wechsler Intelligence Scale for Children-Revised, Mazes subtest  
DKEFS Tower = Delis Kaplan Executive Function System, Tower subtest
Appendix B

Meta-analysis Coding Protocol

1. Study Number (100-XX) [Study name]

2. Comparison Group

   Provides information regarding comparison group (i.e., typically developing, ADHD, etc.)

3. Planning Measure

   1 Tower of London   2 Tower of Hanoi   3 NEPSY Tower   4 DKEFS Tower
   5 Rey Complex Figure   6 Stockings of Cambridge   7 Trail Making Test
   8 BRIEF   9 other (specify)

4. ASD Mean Age

   If cannot tell, enter 99.99

5. Control Mean Age

   If cannot tell, enter 99.99

6. ASD DX

   1 Autism only   2 Asperger only   3 Mixed ASD sample

7. Control DX

   1 ADHD-I (inattentive type)   2 Tourette’s   3 Conduct Disorder
   4 Typically Developing   5 ADHD-C (combined type)
   6 Reading Disorder   7 Moderate TBI   8 Severe TBI
   9 cannot tell   10 ADHD Mixed sample   11 Mixed Clinical Control
   12 Developmental Language d/o   13 OCD   14 Developmental Delay
   15 Language & Learning d/o

8. ADI DX (ADI-R used to confirm ASD Diagnosis?)

   1 Yes   2 No
9. ADOS DX (ADOS-G used to confirm ASD Diagnosis?)
   1 Yes  2 No

10. SCQ DX (SCQ used to confirm ASD Diagnosis?)
    1 Yes  2 No

11. CARS DX (CARS used to confirm ASD Diagnosis?)
    1 Yes  2 No

12. GADS DX (GADS used to confirm ASD Diagnosis?)
    1 Yes  2 No

13. DX Basis (Diagnostic criteria used to confirm ASD Diagnosis?)
    1 DSM-IV/DSM-IV-TR  2 DSM-III/DSM-II-R  3 Prior DSM
    5 ICD 10  9 cannot tell

14. IQ ASD
    Enter mean IQ for ASD group (if available)

15. IQ Control
    Enter mean IQ for Control group (if available)

16. Predominant race
    1 >60% White  2 >60% Black  3 >60% Hispanic  4 >60% Other
    5 Mixed (none more than 60%)  9 Can’t tell

17. Predominant sex
    1 less than 5% Male  2 between 5 and 50% male  3 50% male
    4 between 50 and 95% male  5 greater than 95% male  9 cannot tell

18. CA Match (Were groups matched by age?)
    1 Yes  2 No

19. MA Match (Were groups matched by mental age?)
    1 Yes  2 No
20. IQ Match (Were groups matched by IQ?)
   \begin{enumerate}
     \item Yes
     \item No
   \end{enumerate}

21. Sex Match (Were groups matched by sex?)
   \begin{enumerate}
     \item Yes
     \item No
   \end{enumerate}

22. Clinical (Was the ASD group compared with a clinical sample?)
   \begin{enumerate}
     \item Yes
     \item No
   \end{enumerate}

22. IQ inclusion criteria
   \begin{enumerate}
     \item >69/70
     \item >80
     \item >65
     \item >75-78
     \item cannot tell
   \end{enumerate}

**Dependent Variable Information**

1. ASD sample mean
2. ASD standard deviation
3. ASD sample size
4. Comparison group mean
5. Comparison group standard deviation
6. Comparison group sample size

**Study Identification Information**

1. Study ID (100-1XX)
2. Study Authors
3. Publication Date (Year only)
4. Title of article/book/dissertation, etc.
5. Journal/Book Title
6. Pages of work
7. Volume #
8. Issue #

9. Publication Type

    1 Journal article/Book chapter  2 Dissertation/Thesis  3 Other

10. Page number where data found
### Appendix C

**Summary of Included Studies**

<table>
<thead>
<tr>
<th>Authors</th>
<th>Source</th>
<th>Groups Examined</th>
<th>Planning Tasks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cane, D.J.</td>
<td>(2007). Executive function performance and ecological teacher ratings: High functioning autism and language learning disability profiles and impact on academic achievement. <em>Dissertation Abstracts.</em></td>
<td>Mixed ASD (n=36); Comorbid Language and Learning Disorders (n=29)</td>
<td>BRIEF (Plan/Organize) Subscale</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Year</td>
<td>Title</td>
<td>Participants</td>
</tr>
<tr>
<td>-----------------------------------</td>
<td>------</td>
<td>-----------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Gillotty, L.</td>
<td>2001</td>
<td>Understanding of pretense and mental planning ability in children with autism, children with developmental delays, and children with typical development. <em>Dissertation Abstracts.</em></td>
<td>Autism (n=22); Developmental Delay (n=19); Typically Developing (n=20)</td>
</tr>
<tr>
<td>Gioia, G.A., Isquith, P.K., Kenworthy, L., &amp; Barton, R.M.</td>
<td>2002</td>
<td>Profiles of Everyday Executive Function in Acquired and Developmental Disorders. <em>Child Neuropsychology,</em> 8(2), 121-137.</td>
<td>Mixed ASD (n=54); ADHD-C (n=26); ADHD-I (n=27); Moderate TBI (n=33); Severe TBI (n=34); Reading Disorder (n=34); Typically Developing (n=108)</td>
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<tr>
<td>Glasier, P.</td>
<td>2009</td>
<td>The clinical and ecological utility of executive function assessment in children with attention deficit/hyperactivity disorder or Asperger's syndrome. <em>Dissertation Abstracts.</em></td>
<td>Asperger’s Disorder (n=20); Mixed ADHD (n=18); Typically Developing (n=38)</td>
</tr>
<tr>
<td>Happe, F., Booth, R., Charlton, R., &amp; Hughes, C.</td>
<td>2006</td>
<td>Executive function deficits in autism spectrum disorders and attention-deficit/hyperactivity disorder: Examining profiles across domains and ages. <em>Br &amp; Cog,</em> 61, 25-39.</td>
<td>Mixed ASD (n=32); Mixed ADHD (n=30); Typically Developing (n=32)</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Year</td>
<td>Title</td>
<td>Journal</td>
</tr>
<tr>
<td>----------</td>
<td>------</td>
<td>-------</td>
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<tr>
<td>Hooper, S.R., Poon, K.K., Marcus, L., &amp; Fine, C.</td>
<td>2006</td>
<td>Neuropsychological characteristics of school-age children with high functioning autism: Performance on the NEPSY.</td>
<td>Child Neuropsychology, 12, 299-305.</td>
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<tr>
<td>Kuschner, E.S., Bodner, K.E., &amp; Minshew, N.J.</td>
<td>2009</td>
<td>Local vs. global approaches to reproducing the Rey Osterrieth complex figure by children, adolescents, and adults with high-functioning autism.</td>
<td>Autism Research, 2, 348-358.</td>
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<table>
<thead>
<tr>
<th>Authors</th>
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<th>Participants</th>
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<tr>
<td>Ozonoff, S., &amp; Jensen, J.</td>
<td>1999</td>
<td>Brief report: Specific executive function profiles in three neurodevelopmental disorders. <em>Journal of Autism and Developmental Disorders, 29</em>(2), 171-177.</td>
<td>Mixed ASD (n=40); Mixed ADHD (n=24); Typically Developing (n=29); Tourette Syndrome (n=30)</td>
<td>Tower of Hanoi</td>
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<tr>
<td>Ozonoff, S., Pennington, B.F., &amp; Rogers, S.J.</td>
<td>1991</td>
<td>Executive function deficits in high-functioning autistic individuals: Relationship to theory of mind. <em>Journal of Child Psychology and Psychiatry, 32</em>(7), 1081-1105.</td>
<td>Mixed ASD (n=23); Clinical Control (n=20)</td>
<td>Tower of Hanoi</td>
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<tr>
<td>Semrud-Clikeman, M., Walkowia, J., Wilkinson, A., &amp; Butcher, B.</td>
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<td>DKEFS Tower; BRIEF Organize/Plan Subscale</td>
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