

Seattle Pacific University Digital Commons @ SPU

Clinical Psychology Dissertations

Psychology, Family, and Community, School of

Spring May 4th, 2016

Internalizing Symptoms: Relations to Executive Functions in Young Children with Autism Spectrum Disorder

Jessica L. Berg Seattle Pacific University

Follow this and additional works at: https://digitalcommons.spu.edu/cpy_etd Part of the <u>Child Psychology Commons</u>, and the <u>Clinical Psychology Commons</u>

Recommended Citation

Berg, Jessica L., "Internalizing Symptoms: Relations to Executive Functions in Young Children with Autism Spectrum Disorder" (2016). *Clinical Psychology Dissertations*. 14. https://digitalcommons.spu.edu/cpy_etd/14

This Dissertation is brought to you for free and open access by the Psychology, Family, and Community, School of at Digital Commons @ SPU. It has been accepted for inclusion in Clinical Psychology Dissertations by an authorized administrator of Digital Commons @ SPU.

Internalizing Symptoms: Relations to Executive Functions in Young Children with

Autism Spectrum Disorder

Jessica L. Peterson

A dissertation submitted in partial fulfillment of the requirements for the degree of

Doctor of Philosophy

In

Clinical Psychology

Seattle Pacific University

School of Psychology, Family and Community

May 2016

Approved by: Beverly J. Wilson, Ph.D. Professor of Clinical Psychology Seattle Pacific University Dissertation Chair Reviewed by: David Stewart, Ph.D. Chair of Clinical Psychology Seattle Pacific University

Amy Mezulis, Ph.D.HAssociate Professor of Clinical PsychologyDSeattle Pacific UniversityHDissertation Committee MemberS

Karen Toth, Ph.D. Research Scientist Seattle Children's Research Institute Dissertation Committee Member Katy Tangenberg, Ph.D. Dean, School of Psychology, Family & Community Seattle Pacific University

Dedication

I dedicate this manuscript to my husband, Aaron, for your incredible support, patience, love, and commitment throughout my graduate training. I also dedicate this manuscript to my parents, Dean and Jeanine, for your support, love, strength, and generosity my entire life.

Acknowledgement

I would like to extend my sincerest gratitude to the children and families who participated in this study for their time and effort in support of this research. I would also like to thank my advisor and committee chair, Beverly J. Wilson, Ph.D. for her guidance, compassion, expertise, and unwavering support from the beginning of my graduate training six years ago to the present. I wish to acknowledge and thank Amy Mezulis, Ph.D. and Karen Toth, Ph.D. for being part of my dissertation committee and contributing their expertise and thoughtful feedback. I extend my appreciation to Jeffrey Drayer, Ph.D. for his generosity in loaning the Tower of Hanoi-Revised materials to complete this research and his time and consultation regarding executive functioning research involving young children with autism spectrum disorder. I am grateful for the help and diligence of Julia Kim who assisted with coding procedures. To the Developmental Psychology research team, thank you for your time and contributions to this project.

Table of Contents

Dedication	ii
Acknowledgement	iii
List of Tables	vi
List of Figures	vii
Abstract	viii
Chapter I: Introduction and Literature Review	1
Autism Spectrum Disorder	4
Overview	4
Epidemiology	7
Etiology	8
Neuropathology of Austim	9
Executive Functioning	11
Overview	11
Theory of Executive Functioning	13
Development of Executive Functioning	14
Assessment of Executive Functioning in Young Children	17
Executive Functions in ASD	19
Internalizing Symptoms	30
Overview	30
Assessment of Internalizing Symptoms in Young Children	33
Internalizing Symptoms in ASD	35
Executive Functioning and Internalizing Symptoms	40
Neuroscience Research linking EF and Internalizing Symptoms	43
Executive Funciton, Internalizing Symptoms, and ASD	45
Present Study	49
Hypotheses	51
Hypothesis 1	51
Hypothesis 2	52
Hypothesis 3	53
Hypothesis 4	53
Hypothesis 5	55

Chapter II: Method	56
Participants	56
Demographic Information	57
Procedures	59
Measures	61
Chapter III. Results	69
Power Analysis	69
Data Entry	69
Data Screening Prior to Analysis	70
Data Analytic Plan	72
Descriptive Analyses	77
Tests of Hypotheses	81
Post-hoc Analyses	92
Summary of Results	103
Chapter IV. Discussion	107
Interpretation of Results	107
Clinical Implications	117
Strengths and Limitations	120
Conclusions and Future Directions References	

List of Tables

Table 1	Descriptive Information by Group	58
Table 2	Normality Among Continuous Variables in TD Group	
Table 3	Normality Among Continuous Variables in ASD Group	74
Table 4	Descriptive Statistics for Variables: Means, Standard Deviations, t-test	s,
	Chi-Square and Effect Sizes by Group	
Table 5	Bivariate Correlations among Study Variables	
Table 6	Hierarchical Regression: ToH-R Regressed on Verbal Mental Age and	
	Developmental Status	
Table 7	Hierarchical Regression: Shift Regressed on Verbal Ability, Maternal	
	History of Depression, and Developmental Status	.83
Table 8	Hierarchical Regression: Plan Regressed on Verbal Ability, Maternal	
	History of Depression, and Developmental Status	.83
Table 9	Hierarchical Regression: Parent BASC-2 Internalizing Symptoms	
	Composite Scores Regressed on BRIEF Shift and Plan	.84
Table 10	Hierarchical Regression: Teacher BASC-2 Internalizing Symptoms	
	Composite Scores Regressed on BRIEF Shift and Plan	.85
Table 11	Summary of Indirect Effects from Multiple Mediation Analysis of	
	Developmental Status on Parent Report of Internalizing Symptoms	
	through Executive Functioning Skills	.88
Table 12	Summary of Indirect Effects from Multiple Mediation Analysis of	
	Developmental Status on Teacher Report of Internalizing Symptoms	
	through Executive Functioning Skills	.90
Table 13	Summary of Indirect Effects from Multiple Mediation Analysis of Mater	rnal
	History of Depression on Parent Report of Internalizing Symptoms	
	through Executive Functioning Skills	.96
Table 14	Summary of Indirect Effects from Multiple Mediation Analysis of Mater	
	History of Depression on Teacher Report of Internalizing Symptoms	
		97
Table 15	Conditional Direct and Indirect Effects of Maternal History of Depress	
	and Executive Functioning on Parent Reported Internalizing Symptoms	
		100
Table 16	Conditional Direct and Indirect Effects of Maternal History of Depress	ion
	and Executive Functioning on Teacher Reported Internalizing Sympton	
	at Values of Developmental Status as a Moderator	

List of Figures

Figure 1. Proposed mediation model of the effects of executive functioning on the
relation between developmental status and internalizing symptoms
<i>Figure 2</i> . Mediation model of the association of developmental status on internalizing symptoms through exective functioning skills
<i>Figure 3.</i> Multiple mediation model depicting relations between developmental status, executive functioning skills, and internalizing symptoms displaying unstandardized regression coefficients
<i>Figure 4.</i> Multiple mediation model depicting relations between developmental status, executive functioning skills, and teacher report of internalizing symptoms displaying unstandardized regression coefficients
<i>Figure 5.</i> A mediational model of the association between developmental status and BASC-2 Teacher reported somatization symptoms via children's performance on the Tower of Hanoi-Revised
<i>Figure 6.</i> Multiple mediation model depicting relations between maternal history of depression, executive functioning skills, and parent reported internalizing symptoms displaying unstandardized regression coefficients
<i>Figure 7.</i> Multiple mediation model depicting relations between maternal history of depression, executive functioning skills, and teacher reported internalizing symptoms displaying unstandardized regression coefficients
<i>Figure 8.</i> Interaction between centered Shift and developmental status controlling for verbal mental age and maternal history of depression predicting parent reported internalizing symptoms for conditional indirect effects model101
<i>Figure 9.</i> Interaction between centered Shift as moderator and developmental status controlling for verbal mental age and maternal history of depression predicting parent reported internalizing behaviors for conditional indirect effects model102

Jessica Lynn Peterson Word Count: 343

Abstract

Children with ASD exhibit significantly higher rates of internalizing symptoms than typically developing (TD) peers and co-occurring anxiety and depression are associated with greater negative outcomes. The current study explored possible neurocognitive correlates underlying increased risk by examining relations between developmental status, executive functioning (EF), and internalizing symptoms in young children. Participants included 66 children between 36 and 85 months with 40 TD children (57.5% male) and 26 children with ASD (84.6% male). EF measures included the BRIEF (Goia, Isquith, Guy, & Kenworthy, 2000) Plan and Shift subscales and a neuropsychological task (TOH-R; Welsh, Pennington, & Groisser, 1991). Parents and teachers reported on children's internalizing symptoms on the BASC-2 (Reynolds & Kamphaus, 2004). Parents completed a demographic questionnaire which included assessment of maternal history of depression. Analyses utilized Hayes and Preacher's (2013) PROCESS macro to test a multiple mediation model in which developmental status is associated with internalizing symptoms through EF. Bootstrapping results supported the model ($R^2 = .48$, F(5, 60) = 11.30, p < 0.001) which accounted for 48% of the variance in parent report of internalizing symptoms. Significant indirect effects were found for Shift (point estimate = 14.31, SE = 4.19, 95% CIs [7.85, 24.74] and Plan (point estimate = 6.50, SE = 2.24, 95% CIs [2.43, 11.18]). A significant indirect effect was found for Plan (point estimate = 6.01, SE = 2.47, 95% CIs [1.61, 11.57]) with teacher reported internalizing symptoms as the outcome. Post hoc analyses explored relations between maternal history of depression given significant correlations with EF variables. A significant indirect effect was found for Shift (point estimate = 5.31, SE = 2.68, 95% CIs [1.42, 12.57]) on the relation between maternal history of depression and parent reported internalizing symptoms that was equivalent in both ASD and TD groups. An sigificant indirect effect was found for Plan (point estimate = 2.95, SE = 1.52, 95% CIs [.79, 7.14]) in the relation between maternal history of depression and teacher reported internalizing symptoms. Results suggest targeting EF skills may be important for addressing internalizing symptoms in young children with ASD.

Keywords: autism spectrum disorder; internalizing symptoms; executive functioning; cognitive flexibility, planning

Chapter I: Introduction and Literature Review

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder characterized by persistent impairments in social communication and social interaction and the presence of restricted, repetitive patterns of behavior, interests or activities (APA, 2013). Research indicates children with ASD exhibit significantly higher rates of internalizing symptoms and diagnoses such as anxiety and depression than typically developing children (Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; MacNeil, Lopes, & Minnes, 2009; Russell & Sofronoff, 2005; Solomon, Miller, Taylor, Hinshaw, & Carter, 2012). The increased risk of internalizing symptoms in children with ASD highlights the need for research examining potential underlying factors to improve mental health outcomes for these children. Research with typically developing children and children with other neurodevelopmental disabilities such as ADHD indicates that deficits in executive functioning (EF) skills are associated with internalizing symptoms (Ciairano, Visu-Petra, & Settanni, 2007; Nigg, Quamma, Greenberg, & Kusche, 1999; Riggs, Blair & Greenberg, 2003; Rinksy & Hinshaw, 2011; Jonsdottir, Bouma, Sergeant, & Scherder, 2006). In addition, interventions that promote neurocognitive functioning and EF skills in young typically developing children predict lower rates of internalizing symptoms and psychopathology in prospective investigations (Riggs, Greenberg, Kusche, & Pentz, 2006). An extensive body of research indicates that children with ASD exhibit elevated rates of executive functioning impairments particularly in the areas of cognitive flexibility and planning (Gioia, Isquith, Kenworthy, & Barton, 2002; Hill, 2004; Kenworthy, Yerys, Anthony, & Wallace, 2008; Pennington & Ozonoff, 1996). Structural and functional neuroimaging studies of children with ASD have found abnormal

development of prefrontal areas involved in executive functioning (Courchesne, et al., 2011; McAlonan et al., 2009; Zikopoulos & Barbas, 2010). Specifically, researchers have reported neuropathology in the dorsolateral prefrontal cortex associated with planning and set shifting abilities in individuals with ASD (Morgan et al., 2010) as well as widespread dysfunction of executive circuitry necessary for frontal lobe functioning and complex information processing (Courchesne & Pierce, 2005; Kumar et al., 2010; Shafritz, Dichter, Baranek, & Belger, 2008). Research investigating how variability in EF skills and prefrontal functioning contribute to the development of elevated internalizing symptoms and disorders in young children with ASD may inform interventions and potentially decrease the incidence of comorbid internalizing symptoms in individuals with ASD across the lifespan.

The current study investigated the possibility that an indirect link exists between developmental status (ASD vs. typically developing) and internalizing symptoms through executive functioning in young children. This hypothesis is supported by research suggesting that neuropathology of brain regions largely involved in the modulation of executive functions, including the dorsolateral prefrontal cortex (dlPFC), are associated with mood and anxiety disorders (Biver et al., 1994; Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Koenigs & Grafman, 2009; Price & Drevets, 2010, 2012). Empirical work also supports the presence of EF deficits and abnormal development of prefrontal areas such as the dlPFC in children and adults with ASD (Courchesne et al., 2011; Morgan et al., 2010). Therefore, EF abilities and their underlying neural substrates may explain the higher incidence of internalizing psychopathology in children with ASD. In addition, neuropathology of the frontal lobes and EF deficits create significant challenges in navigating daily life and adapting to the environment for children. EF impairments are associated with significant interference in everyday living for children with ASD according to parent observations (Drayer, 2009; Boyd, McBee, Holtzclasw, Baranek, & Bodfish, 2009). Therefore, neuropsychological deficits may undermine children's adaptive responses to stress and impart greater vulnerability to comorbid psychiatric disorders such as anxiety and depression. The current study will utilize an ecologically valid rating measure (BRIEF; Goia, Isquith, Guy, & Kenworthy, 2000) in addition to a performance-based neuropsychological task to investigate how everyday manifestations of executive dysfunction may explain the incidence of internalizing symptoms in children with ASD. Although research examining neurocognitive correlates of internalizing symptoms in youth with ASD is extremely limited, recent investigations with adolescents and school aged children with ASD support a mediational role of EF specifically cognitive flexibility in the relation between ASD diagnostic status and internalizing symptoms (Hollocks et al., 2014; Lawson et al., 2015).

The current investigation extends previous research through examination of these constructs in preschool and early school aged children with autism spectrum disorder and typically developing peers, exploration of multiple EF domains, and utilization of multiinformant methods for obtaining information on child internalizing symptoms. The age of children in this investigation is particularly important given EF show a protracted developmental trajectory mediated by prefrontal lobe maturation making it particularly susceptible to exogenous influences. Findings supporting a mediational relationship between executive functioning and internalizing symptoms in children with ASD could inform interventions for young children during a critical period of development when the brain is particularly plastic and amenable to environmental modulation. The following sections provide an overview of ASD, followed by definitions of the constructs of executive functions and internalizing symptoms, along with reviews of the theoretical and empirical literature pertinent to the current study.

Autism Spectrum Disorder

Overview

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by persistent impairments in social communication and interaction and the presence of restricted, repetitive patterns of behavior, interests or activities (APA, 2013). ASD encompasses conditions previously referred to as "pervasive developmental disorders" in The Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR; APA; 2000) including autistic disorder, Asperger's disorder, and pervasive developmental disorder not otherwise specified (PDD-NOS), childhood disintegrative disorder, and Rett's disorder. The DSM-5 (APA, 2013) adopted a single diagnostic dimension (ASD) due to concerns regarding limited reliability of DSM-IV-TR subtype assignment, poor predictive validity with multi-categorical system, data linking common genetic factors across subtypes, and research indicating a single spectrum better reflects symptom presentation, course, and response to treatment (Daniels et al., 2011; Lord, Luyster, Guthrie, & Pickles, 2012; Lord et al., 2012). Validation studies indicate DSM-5 ASD criteria evidences superior specificity compared to DSM-IV-TR criteria and suggest most children with PDD NOS and Asperger's diagnosis are eligible for ASD diagnosis under the new diagnostic system (Frazier et al., 2012; Huerta, Bishop, Buncan, Hus, & Lord, 2014).

Children with ASD show persistent impairments in social communication and social interaction including deficits in social-emotional reciprocity, non-verbal social communication used for social interactions, and developing, maintaining and understanding relationships (APA, 2013). To qualify for a diagnosis of ASD children must also demonstrate at least two symptoms of restricted, repetitive interests/behaviors (RRBs) which include stereotyped or repetitive motor movements, use of objects or speech, insistence on sameness/inflexible adherence to routines or ritualized patterns of nonverbal and verbal behavior, highly restricted, fixated interests that are abnormal in intensity or focus, and unusual sensory interests/reactions (APA, 2013). Although ASD represents a single diagnostic dimension, significant phenotypic heterogeneity exists among individuals with ASD including variability in severity of symptoms, etiologic factors, cognitive and language abilities, pattern of onset and clinical course, and associated conditions (Munson, Faja, Meltzoff, Abbott, & Dawson, 2008; Wing & Potter, 2002). Therefore, clinicians are encouraged to describe these variables with diagnostic specifiers available in DSM 5 (APA, 2013) pertaining to intellectual impairment, language impairment, association with known medical or genetic conditions, environmental factors, another neurodevelopmental, mental, or behavioral disorder as well as severity of symptoms in the domains of social communication and restricted, repetitive behaviors. Associated features with autism may include motor deficits or abnormalities (e.g., clumsiness, abnormal gait, walking on tiptoes), self-injury, and disruptive behaviors. Psychiatric comorbidity is also common with many individuals with ASD having one or more comorbid mental health disorders such as ADHD, specific learning difficulties, developmental coordinator disorder, anxiety, or depressive disorders

(APA. 2013). Medical conditions commonly associated with ASD include epilepsy, gastrointestinal and sleep problems.

ASD symptoms are present in the early developmental period although symptom manifestation may be related to course of changing social demands or mitigated by learned strategies developed with age (APA, 2013). Research indicates the majority of parents of children with ASD identify concerns by approximately 12 to 18 months of age typically in the area of language development (Barbaro, & Dissanayake, 2009). Retrospective studies indicate that early signs of ASD in infancy may include diminished visual attention to people and exploration of objects, delayed orientation to name, aversion to touch, and limited smiling and vocalization (Zwaigenbaum et al., 2005; Werner, Dawson, Osterling, & Dinno, 2000). The pattern of onset may include early developmental delays, developmental plateaus or regression in the form of loss of social or language skills with gradual or rapid deterioration occurring primarily between the first and second year of life (APA. 2013). Factors shown to have a significant impact on prognosis are intellectual functioning, language skills, and additional psychiatric problems. Early intervention during periods of substantial neurological plasticity is important for improved outcomes (Dawson, 2009). The current study included subjects with DSM-IV-TR diagnoses of autistic disorder, Asperger's disorder, and PDD-NOS due to the fact that recruitment occurred prior to publication of DSM 5 (2013). However, according to DSM 5 specifications all subjects in the current study would meet criteria for ASD due to a history of well-established DSM-IV-TR diagnoses. The following sections will provide an overview of the epidemiology, etiology, and neurological underpinnings of ASD

Epidemiology

The prevalence of ASD is approximately 1 in 68 school-aged children according to current estimates from The Autism and Developmental Disabilities Monitoring (ADDM) Network of the Center for Disease Control and Prevention (Baio, 2014). A recent National Health Statistics Report released by the CDC (Zablotsky, Black, Maenner, Schieve, & Blumberg, 2015) suggests 1 in 45 children ages 3 to 17 years have been diagnosed with ASD based on parent surveys. ASD is more common among males across prevalence studies affecting approximately 1 in 42 males versus 1 in 189 females (Baio, 2014; Fombonne, 2009). Findings from the CDC's ADDM Network based on data from 11 sites in 2010 found that approximately 30% of children with ASD met criteria for intellectual disability (IQ < 70), 23% met criteria for borderline IQ range (IQ = 71-85) and 46% had average or above average intellectual ability. Non-Hispanic Caucasian children are significantly more likely to be identified with ASD than Non-Hispanic African American children and Hispanic children and African American and Hispanic children are more likely than Non-Hispanic Caucasian children to be diagnosed with intellectual disability. Prevalence rates have risen dramatically since the 1990s leading many to question the origins of such an increase. New estimates from the CDC (Blumberg et al., 2013) indicate a significant increase in ASD incidence among school aged children citing prevalence rates of 1.16% in 2007 to 2.00% in 2011-2012. Researchers suggest that a myriad of factors are related to the rise in prevalence including broadening of autism diagnostic criteria, better diagnostic tools and training, increased awareness by parents and clinicians, diagnostic substitution of ASD for other developmental disabilities, service resources, and increased parental age (Bishop,

Whitehouse, Watt, & Line, 2000; Grether et al., 2009; Russell, Kelly, Golding, 2010). While these aforementioned factors are widely recognized as contributors to increasing prevalence rates, whether or not there has been a true rise in the number of children born annually with ASD continues to be explored and debated.

Etiology

The phenotypic heterogeneity of ASD creates significant difficulties in establishing etiology. A definitive etiology of ASD is unknown, however, researchers agree that genetic susceptibilities and their interaction with environmental factors underlie the complex etiology and brain abnormalities associated with this condition (Anderson, 2012; Tordjman et al., 2014). Family and twin studies support the significant heritability of ASD. Concordance rates for monozygotic twins range from 60-90% while the corresponding values for dizygotic twins range from 0-20% (Bailey, Couteur, Gottesman, & Bolton, 1995; Ronald & Hoekstra, 2011). Hundreds of genes appear to contribute to ASD and related disorders (Berg & Geschwind, 2012; Iossifov et al., 2012; Chang et al., 2015). Genetic syndromes such as Fragile X syndrome, Down syndrome, and tuberous sclerosis, defined mutations, and de novo copy-number variation account for approximately 10-20% of children with autism (Cohen et al., 2005; Abrahams & Geschwind, 2008). Recent studies have identified a large number of de novo mutations associated with ASD with large copy number variations and truncating single nucleotide variants appearing to play a causal role in ASD (Iossifov et al, 2011; Levy et al., 2011; Sanders et al., 2012). O'Roake and colleagues (2012) found de novo mutations largely paternal in origin are associated with the incidence of ASD in families without a previous history of this condition. These authors reported that many of the de novo mutations

identified in their study led to mutations of proteins important for brain cell communication.

Interest in environmental factors imparting risk for ASD continues to grow. Several prenatal, perinatal, and postnatal factors associated with autism risk include low birthrate and prematurity (Lampi et al., 2012), fetal distress or injury at birth, hyperbilirubinema, advanced paternal and maternal age, gestational diabetes, maternal birth abroad (Gardener, Spiegleberg, & Buka, 2009), maternal depression during pregnancy (Rai et al., 2013), exposure to valproate, thalidomide, and misoprostol, maternal fever during pregnancy (Zerbo et al., 2013), exposure to environmental toxins including air pollution (McCanlies et al., 2012), dietary factors (Herbert, 2010), and maternal immune system functioning (Braunschweig et al., 2012). Genetic and environmental influences may also cause several systemic physiological and metabolic abnormalities associated with ASD such as immune dysregulation, oxidative stress, and mitochondrial dysfunction (Rossignol & Frye, 2011). Researchers continue to explore combinations of genetic and environmental factors as contributors to behavioral characteristics of ASD and its underlying brain pathology. ASD is a developmental condition associated with substantial neurological abnormalities. The following section will review the abnormalities in brain systems and structures associated with ASD.

Neuropathology of autism

The neuropathology manifest in ASD is characterized by a diverse set of structural and functional abnormalities. Neuroimaging studies indicate anatomical pathologies of the cerebellum, amygdala, cerebral cortex, hippocampal formation, and frontal lobes (Courchesne, Campbell, & Solso, 2011; Schumann, Bauman, & Amaral, 2011). A prominent theory of neurological disturbance in ASD argues that an unusual rate of brain development characterizes the disorder. Studies of head circumference suggest that a period of rapid brain development begins around 12 months leading to the abnormal brain enlargement observed in children with autism (Barnard-Brak, Sulak, & Hatz, 2011; Dawson et al., 2007). Researchers propose that increased white matter accounts for a disproportionate amount of this volume particularly in the frontal lobes (Hazlett et al., 2005; Ben Bashat, 2007). Structural and functional neuroimaging studies as well as neuropsychological research indicate the role of aberrant frontal lobe development in children with ASD. Both delayed frontal lobe development and structural abnormalities associated with decreased efficacy have been noted in young children with ASD (Courchesne et al., 2011; Sundaram et al., 2008). Courchesne and colleagues (2011) propose that excessive neurogenesis and defective neural pruning result in frontal brain pathology early in development in children with ASD. Additional theories of ASD neuropathology implicate synaptic dysfunction (Berkel et al., 2010) and atypical brain networks (Kennedy & Courchesne, 2008). Recent evidence supports the functional underconnectivity theory of autism which states that the connection between frontal and more posterior regions of the brain are partially disrupted during development in children with ASD (Just, Keller, Malave, Kana & Varma, 2012). Evidence from neuroscience investigations suggests that connectivity in the frontal cortex is excessive and disorganized while connections between the frontal cortex and posterior regions of the brain are inadequate and poorly coordinated (Courchesne et al., 2011; Dinstein et al., 2011). According to researchers, network dysfunction and underconnectivity exist to varying degrees and account for the developmental onset of symptoms and diverse range

of behavioral and neuropsychological deficits observed in ASD (Geschwind & Levitt, 2007).

In summary, ASD is a highly heterogeneous disorder characterized by numerous neurological abnormalities with genetic and environmental etiologies. Understanding areas of strengths and challenges of children with ASD during early childhood is particularly important for designing early interventions with the goal of facilitating neurocognitive functioning and alleviating symptoms. The following section will provide an overview of executive functions and their relation to ASD as an area of neuropsychological functioning that has significant potential for modification in early development.

Executive Functioning

Overview

Executive functions (EF) are higher-order cognitive processes involved in monitoring and regulating cognitions and behavior. There are numerous definitions and theories regarding the component processes of EF. However, no formal definition of this construct exists and debate remains regarding its subcomponents (Jurado & Rosselli, 2007). Traditional conceptualizations of EF describe these processes as mediators of goal-directed behavior that underlie the ability to form goals, plan and organize action, execute goal-directed plans, and monitor performance (Lezak, 1982). Currently, EF is often referred to as an umbrella construct comprised of cognitive control or supervisory functions that organize and regulate cognitive activity, affect, and the expression of behavior (Gioia, Isquith, & Kenealy, 2008; Hill, 2004). Novelty and complex task demands are often linked with activation of the executive system although most activities of daily life likely require utilization of executive control (Stuss & Alexander, 2000). More recently EFs have been commonly dichotomized as "cool" executive processes when tasks are purely cognitive and "hot" executive functions when situations involve affect and motivation (Zelazo & Carlson, 2012). However, hot and cold EFs are considered inevitably connected and rarely utilized in isolation (Anderson, Jacobs, & Anderson, 2008).

Researchers have identified several key subdomains of EF including attentional control, behavioral inhibition, working memory, cognitive flexibility or set-shifting, planning ability and organization, and monitoring of performance (Isquith, Crawford, Espy, & Gioia, 2005; Miller, Giesbrecht, Müller, McInerney, & Kerns, 2012; Pennington & Ozonoff, 1996). Numerous studies have examined the structure of executive functions in children and adults. Factor analytic research supports a hierarchical framework of EF that is both unitary and fractionated in structure (Garon, Bryson, & Smith, 2008; Miller et al., 2012). Specifically, researchers have found that different EFs correlate with one another suggesting a unitary construct but also demonstrate dissociability supporting the presence of separate components (Miyake & Friedman, 2012). There is some evidence that EF may represent a unitary construct in very young children (Miller et al., 2012; Wiebe, Espy, & Charak, 2008) although studies with preschool-aged children have also indicated the presence of distinct EF factors (Miyake & Friedman, 2012). Other evidence supporting separate EF components comes from research showing differential associations between specific EF domains and IQ. For example, working memory has been shown to correlate the most strongly with intelligence while other EF domains such as flexibility and inhibition demonstrate weak associations with IQ (Friedman et al., 2006).

Adequate EF skills are imperative for children's behavioral, cognitive, and socioemotional development and functioning in daily life. Numerous studies have linked EF to a variety of important outcomes in childhood and adulthood. One large scale birth cohort study found deficits in self-control and executive functions at ages 3 to 11 years related to poorer health outcomes, lower income, and a higher rate of crimes 30 years later even after controlling for IQ, gender, and socioeconomic status (Moffitt et al., 2011). EF has demonstrated an influential role in children's academic, social-emotional, and behavioral development (Bull, Epsy, & Weibe, 2009; Riggs et al., 2006; Welsh, Nix, Blair, Bierman, & Nelson, 2010). The important implications of executive functioning skills for children's successful development have created a productive context for theory building and research over the last several decades. The following section reviews a theoretical model of executive functioning that informed the current study design and conceptualization of EF skills in young children.

Theory of Executive Function

A number of theoretical models of executive function have been proposed but a definitive conceptualization has not been accepted. The executive control system theory (Anderson, 2002) is grounded in developmental neuropsychology and based on factor analytic and developmental research. Factor analytic studies indicate the presence of three to four interrelated yet dissociable factors comprising executive function across a wide range of procedures and samples. Anderson's model (2002) of EF includes four domains: attentional control, cognitive flexibility, goal setting, and information processing. These four separate yet associated components comprise the overall control system assumed to be associated with specific prefrontal networks. The amount of input

from each domain is determined by task demands. The attentional control domain includes the ability to selectively attend and sustain attention to designated stimuli. Impulse control and the capacity to monitor behavior are also important components of this domain. The cognitive flexibility domain includes the ability to shift between response sets, demonstrate flexible problem solving, learn from mistakes, and adapt to new demands. Principal components of this domain include working memory, defined as the ability to hold and mentally manipulate information, and the capacity to utilize feedback and process multiple sources of information simultaneously.

The third executive function domain included in Anderson's model (2002) is goal setting which is comprised of activity initiation, conceptual reasoning, and planning. Intimately tied with this domain is the ability to organize strategically in the service of goal attainment. The final EF domain includes information processing which addresses issues of fluency, proficiency, and speed of output tied to the efficiency of prefrontal neural networks. Information processing and the other three domains are bidirectional and reflect cognitive processes that support one another. Anderson's theory represents a conceptualization of the neurological underpinnings of EF and provides an outline for EF assessment. The following section reviews the progression of these cognitive skills during a critical period of skill development.

Development of Executive Functioning

The major components of EF develop during infancy and preschool years setting the stage for acquisition of higher level cognitive processes seen in adolescence and adulthood. Once defined as a unitary concept located in the frontal lobes, evidence suggests that EF represents a distinct set of interrelated executive capacities that require coordination of participating neural systems to be effective (De Luca & Levener, 2008). Anderson's executive control system model (2002) describes development of the four EF domains as distinct but interrelated. EF skills are often categorized into two categories including lower order and higher order processes. Lower order EFs are the first to develop and are considered basic processes including inhibition and working memory while higher order skills such as planning and organization require integration of multiple cognitive processes. This conceptualization of EF development is supported by research suggesting a nonlinear progression of EF skills across the lifespan (De Luca & Leventer, 2008).

Development of frontal networks parallels the acquisition of EF capabilities in children and adults. The prefrontal cortex (PFC) functions as an executive monitor due to associated circuitry throughout the brain with structures important for perception, cognition, and behavior (Shallice, 2002). At birth the anatomical structure and foundation of frontal lobe circuitry are developed although largely unmyelinated and immature (Volpe, 1995). Prenatal brain development is programmed and controlled by genetic factors while postnatal brain development is guided by a combination of genetic coding and environmental influences. During the first two years of life, cortical development includes rapid formation of synapses known as synaptogenesis and myelination of brain structures (Anderson, 1998). Synaptogenesis and myelination of the PFC occur relatively late in this developmental period and the PFC continues to myelinate well into early adulthood (Romine & Reynolds, 2005).

Fledgling EF abilities are seen very early in development. At 12 weeks infants are able to detect the goal structure of an event (i.e., trying to obtain an object) (Sommerville, Woodward, & Needham, 2005) and at 7 to 8 months the first signs of inhibition systems and working memory are present and continue to show signs of improvement through the second year in typical development. During the preschool years, the frontal lobes continue to grow due to increases in gray and white matter. Typically developing children display significant improvement in inhibitory control and sustained attention between three and five years of age (De Luca & Leventer, 2008). Cognitive flexibility, working memory, and strategic planning show improvement between ages 4 and 8 years of age (Luciana & Nelson, 1998). Planning and goal-directed behaviors display increasing maturity during the preschool years and are supported by growing inhibitory and working memory capacities (Senn, Espy, & Kaufmann, 2004). The ability to coordinate EF domains also demonstrates significant improvement during the preschool years, with growth spurts in this ability at the end of the first year and between 3 and 6 years of age (Diamond, 2002). Research suggests that prior to age 3, the foundational skills necessary for EF domains begin to develop followed by increasing integration of these skills and domains (Garon, Bryson, & Smith, 2008).

Deficits in executive system formation are likely caused by disrupted development of the neural pathways and circuitry of the frontal systems (Luna et al., 2002). The neurological basis for executive functions is largely situated in the prefrontal cortices (Anderson et al., 2008). The prefrontal cortices include the dorsolateral, ventromedial, and orbitofrontal regions which are highly interconnected throughout the brain including posterior and subcortical cerebral regions (Alvarez & Emory, 2006). The dorsolateral prefrontal cortex (dIPFC) is involved in higher order cognitive processes and the integration of cognition and behavior (Alvarez & Emory, 2006). This region is associated with planning, set shifting, response inhibition, working memory, organizational skills, and problem solving (Anderson et al., 2008). The orbitofrontal cortex (OFC) is involved in sensory integration, decision making, and evaluation of rewards and punishments (Alvarez & Emory, 2006). The ventromedial prefrontal cortex is largely involved in emotion regulation and also decision making (Alvarez & Emory, 2006). Young children's EF and neural development are highly influenced by environmental stimuli in the form of family and other social factors. The protracted maturation of the frontal cortex and related brain circuitry suggests that these abilities and brain regions are highly dependent on environmental input (Lenroot et al., 2009). This highlights the important role of environmental stimulation in facilitating frontal lobe and brain network development and the potential for early environmentally based interventions to prevent or treat EF impairments particularly for children with developmental disabilities. Adequate assessment is an essential component of wellinformed interventions. While previously measured almost exclusively in adults, EF assessment in children is now a popular area of research and test development. The following sections review the current assessments of EF in young children.

Assessment of Executive Function in Young Children

Assessment of EF in young children is essential for understanding EF deficits in childhood disorders. An abundance of developmentally sensitive measures to assess preschool and early school-aged children's executive functions have been created or adapted from adult tasks in recent years. Research indicates that EF can be reliably measured with developmentally appropriate tasks in children as young as three (Espy, 2004). Laboratory based performance measures are frequently utilized in developmental and neuropsychological studies. However, the sole standardized and validated measure

of EF for preschool-aged children is the Developmental Neuropsychological Assessment (NEPSY; Korkman, Kirk & Kemp, 1998) attention-executive function subtests, highlighting the need for child based norms and psychometric examination of widely used laboratory measures of child EF. Researchers have developed performance tasks designed to assess a variety of EF domains such as cognitive set-shifting (*Shape School*; Espy, 1997), inhibition (*The Day-Night Task*; Gerstadt, Hong, & Diamond, 1994), problem solving planning ability (*Tower of Hanoi-Revised*; Welsh, Pennington, & Groisser, 1991), and working memory (*A-not-B task*; Diamond, 1988). Carlson (2005) assessed 602 typically developing preschool children (ages 2-6) using a battery of research-based assessments to examine EF development and task difficulty within this age range. Carlson found that among tasks administered across age groups, most showed age-related improvement independent of verbal skills and indistinguishable based on hot/cold features of the tasks.

Historically EF tasks have utilized summary scores to reflect executive function performance, making it difficult to distinguish between EF domains of interest and other cognitive abilities (i.e., language, perception) involved in completing the task. Anderson (1998) highlights the need for procedures that isolate and quantify specific domains in EF assessments. Garth, Anderson, and Wrennell (1997) suggested incorporating measures of processing speed and strategy with summary scores to delineate higher order processes from scores reflecting the range of cognitive skills necessary for the task. Another potential obstacle for detecting EF is the wide use of highly structured assessments administered in quiet and low-stimulus laboratory and clinic settings. While these performance –based tasks are designed to provide high internal validity they lack the

ability to capture behavioral manifestations of executive function in real world activities. Recently, emphasis has been placed on utilizing EF measures that provide ecological validity or predictive value of executive functioning in the everyday environment. Gioia and colleagues (2000) developed a rating scale to capture the behavioral manifestations of executive functions in children ages 5-18 called the behavior rating inventory of executive function (BRIEF; Goia, Isquith, Guy, & Kenworthy, 2000). A preschool version of this measure is also available for children ages 2-5 years of age, the BRIEF-Preschool version (Gioia, Espy, & Isquith, 2003). The rating scale assesses children's everyday self-regulatory behaviors in a wide range of EF domains using parent and teacher observations. This method allows clinicians and researchers to evaluate the everyday impact of executive functions or dysfunction on behavior. This instrument may also be particularly important for intervention planning because scores reveal how performance based EF deficits manifest in daily living. Gioia and colleagues (2008) advocate for the use of internally valid performance measures combined with ecologically valid measures of behavior for a comprehensive assessment of EF. The current study follows this approach by utilizing performance based tasks and behavior ratings in a population associated with executive dysfunction, children with autism. The following section examines EF in children with ASD.

Executive Function in ASD

Research demonstrating executive functioning deficits in children, adolescents, and adults with ASD is robust (Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Geurts, Verte, Osterlann, Roeyers, & Sergent, 2004; Gioia, Isquith, Kenworthy, & Barton, 2002; Hill, 2004; Kenworthy, Yerys, Anthony, & Wallace, 2008; Pennington & Ozonoff, 1996). Executive dysfunction has been found in individuals with autism and their family members of varying ages and levels of functionality using a variety of methods to assess executive functions although inconsistencies exist and methodology has varied widely. Several studies suggest individuals with ASD are more impaired on open-ended EF tests in which several possible strategies for performing the task exist and no explicit instructions on how to accomplish the task are given versus highly structured tasks (Van Eylen et al., 2015; White, Burgess, & Hill, 2009). This finding has been used to explain discrepancies in the literature in which individuals with ASD perform adequately on highly structured laboratory tasks but display obvious EF deficits in daily living particularly for older individuals with ASD. The executive dysfunction theory of autism has received substantial attention in autism literature over the last several decades as researchers search for a primary cognitive deficit to explain the triad of impairments present in ASD (Hughes, Russell, & Robbins, 1994; Ozonoff, Pennington, & Rogers, 1991). The theory proposes that a primary impairment in EF explains many social and non-social behaviors in ASD including perseverative and rigid responding, impaired switching between tasks, and difficulties initiating new non-routine activities (Hill, 2004). These behaviors are not accounted for by other prominent ASD theories including the Theory of Mind hypothesis (Baron-Cohen, Leslie, & Frith, 1985) and the Weak Central Coherence theory (Happé & Frith, 2006).

Theoretical work and empirical investigations surrounding ASD and executive dysfunction began when Damasio and Maurer (1978) compared the symptoms of ASD to those of persons with frontal lobe damage. Similar impairments noted between the two groups included switching between tasks, planning, and acquiring and utilizing social rules (Damasio & Maurer, 1978). Empirical evidence supports the presence of atypical frontal lobe development and structural abnormalities in children with ASD (Courchesne et al., 2011; Sundaram et al., 2008). More specifically, abnormalities in the prefrontal cortex which mediates EF have been observed in individuals with autism (Morgan et al., 2010). Reduced activation of the dorsolateral prefrontal cortex, associated with higher order EFs, has also been reported in individuals with ASD during neuropsychological tasks (Luna et al., 2002). Performance deficits on executive functioning tasks support the presence of neurocortical abnormalities in individuals with ASD.

Executive dysfunction among school aged children with ASD is well established (for reviews see Hill, 2004; Kenworthy, Yerys, Anthony, & Wallace, 2008; Pennington & Ozonoff, 1996; Sergeant et al., 2002) with EF deficits noted across the range of EF domains (Geurts, Verte, Osterlaan, Roeyers, & Sergent, 2004). However, children with autism demonstrate the most consistent and profound EF impairments in cognitive flexibility and planning on both neuropsychological assessments and informant ratings of everyday functioning (Granader et al., 2014; Pennington & Ozonofff, 1996; Sinzig, Morsch, Bruning, Schmidt, & Lehmkuhl, 2008; Van Eylen et al., 2015). Planning refers to the dynamic process of formulating a plan and sub-goals which are monitored, reevaluated, and updated in pursuit of a goal. The Tower of Hanoi (ToH) and Tower of London (ToL) are two tasks frequently utilized to assess planning and problem solving abilities. Children with high functioning autism have displayed impaired performance on these tasks relative to age and IQ matched controls with dyslexia, ADHD, and Tourette syndrome (Bennetto et al., 1996; Geurts et al., 2004; Ozonoff & Jensen, 1999; Ozonoff et al., 1991) and typically developing children (Ozonoff & Jenson, 1999; Robinson,

Goddard, Dritschel, Wisely, & Howlin, 2009). Cognitive flexibility often referred to as "set-shifting" is the ability to shift flexibly between thoughts and actions in response to environmental changes. The Wisconsin Card Sorting Task (WCST) is a common assessment of cognitive flexibility that requires individuals to switch cognitive set in response to verbal feedback as they sort cards according to three rules. Perseverative responses are an index of difficulties with cognitive flexibility. Impaired performance and significantly higher perseverative responding on cognitive flexibility tasks have been found in high functioning children with ASD compared to typically developing controls and children with ADHD, language disorders, Tourettes Syndrome, and dyslexia (Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Guerts et al., 2004; Ozonoff & Jensen, 1999, Sergeant et al., 2002). Hill's seminal review (2004) generally supported significant planning and set-shifting impairments in children with ASD and presented evidence for additional difficulties in inhibition of a prepotent response and generation of novel ideas and behaviors (i.e., generativity). Findings are generally mixed regarding deficits in working memory in children with ASD compared to age and IQ matched typically developing controls and matched comparison groups with Tourette Syndrome (Lopez, Lincoln, Ozonoff, & Lai, 2005; Ozonoff & Strayer, 2001; Yers et al., 2011).

Research indicates that children with ASD may display more profound EF deficits than children with other neurodevelopmental disorders (Corbett et al., 2009; Geurts et al., 2004; Ozonoff & Jenson, 1999). One study utilized a comprehensive neuropsychological battery to compare children between 6 and 12 years of age with ASD and ADHD (Geurts et al., 2004). The authors found that children with ADHD displayed EF impairment in inhibition and verbal fluency while children with HFA exhibited deficits across most EF domains suggesting more widespread executive dysfunction. However, several studies have found insignificant differences in EF skill across neurodevelopmental conditions. Goldberg and colleagues (2004) reported similar response inhibition, planning, and set shifting abilities in school aged children with HFA, ADHD, and typical development using a computerized battery of tasks. Research also suggests EF deficits in ASD may increase with age relative to IQ and environmental demands. Rosenthal and colleagues (2013) collected behavioral parent report ratings of EF using the BRIEF (Gioia et al., 2000) for 185 children with ASD without intellectual disability between the ages of 5 and 18 years of age and found significant age effects in which older children with ASD demonstrated greater EF difficulties compared to the normative sample and younger children with ASD. The authors noted a widening divergence of EF capabilities from the normative sample in individuals with ASD and more stable impairments in flexibility across age cohorts.

Whereas research examining executive functioning deficits in school aged children with autism is robust, research on EF in younger children with ASD is lacking. The few studies measuring EF skills in preschool and early school-aged children with ASD have generally reported mixed results regarding areas of deficit and specificity of EF dysfunction to autism. The first study conducted by McEvoy, Rogers and Pennington (1993) compared children with ASD (n = 17), children with developmental delays (n = 13) and typically developing children (n = 16) matched on verbal mental skills. The mean age of the typically developing group was 4. 2 years and the mean age of children with ASD was 5.1 years. McEvoy and colleagues (2013) used four simple measures of prefrontal function to measure EF: *A-not B task, Delayed Response Task, Spatial*

Reversal Task, and Alternative Task. On the Spatial Reversal task, children with ASD exhibited significantly more perseverative errors than children in the comparison groups indicating selected deficits in set shifting, problem solving, and self-monitoring. Significant group differences were not found for the three other tasks assessing inhibition and visual motor skills. However, McEvoy and colleagues (2013) noted that all tasks except Spatial Reversal exhibited floor or ceiling effects. A second study conducted by Dawson and colleagues (1998) examined the neuropsychological correlates of early symptoms of autism in preschool aged children. Subjects included 20 children with ASD (M = 5.4 years), 19 children with Down syndrome (M = 5.4 years), and 20 children with typical development (M = 2.5 years) matched on verbal mental age and verbal IQ and chronological age for the two groups with developmental disabilities. Early symptoms of autism were measured (i.e., social orienting, deferred imitation, shared attention, symbolic play) along with two neuropsychological tasks: one tapping the limbic system including the medial temporal lobe and orbital prefrontal cortex (Delayed Non Matching Sample) and one tapping the dorsolateral prefrontal cortex (Delayed Response). Based on the theory that ASD is etiologically linked to dysfunction of the limbic system including the amygdala and hippocampus, the authors hypothesized that early core symptoms of autism would be more related to performance on the task mediated by the limbic system versus the dorsolateral prefrontal region. The results indicated that children with ASD performed significantly worse on both EF tasks utilizing the limbic system and dorsolateral prefrontal region relative to comparison subjects. The severity of autism symptoms was strongly correlated to the task activating the limbic system but not the task activating the dorsolateral prefrontal cortex supporting a potential etiological role of limbic dysfunction versus dlPFC abnormalities in ASD. The authors interpreted the findings as contradicting the executive function theory that prefrontal cortex dysfunction and related EF play a causal role in ASD symptomology.

Griffith et al., (1999) studied EF in preschool children with autism using an even younger sample to clarify findings in this area of research. Two sub-studies were completed using 18 children with ASD (M = 4.3 years) and 17 children without autism but with a variety of developmental delays (M = 4.3 years) including cognitive delays, speech and language delays, and Down syndrome. Eight EF tasks linked to prefrontal functioning were utilized including A-not-B and Spatial Reversal tasks. The authors noted that all 8 EF tasks required inhibition and visual working memory, 3 tasks required set shifting, and one task required action monitoring. The results indicated a lack of significant group differences between children with ASD and children without ASD except for two findings that indicated superior performance for the ASD group on the Spatial Reversal and Boxes Scrambled tasks. However, results also indicated that both children with ASD and children with developmental disabilities performed below age expectations. Performance on several tasks was associated with verbal and non-verbal ability across groups. The findings of Griffith et al. (1999) appear to contradict Dawson's (1998) findings that children with ASD perform significantly worse on EF tasks than controls with developmental disabilities. Scores on the spatial reversal task did not change significantly following a one year longitudinal follow up indicating that EF deficits may persist over time.

Dawson and colleagues (2002) conducted the largest study of executive functioning in preschool aged children with ASD. Participants included three groups

matched on mental age including 79 children with ASD (M = 3.6 years), 34 children with developmental disabilities (M = 3.7 years), and 39 typically developing children (M = 2.3years). Executive functioning tasks included the *A-not-B task* and the *Delayed* Alternation Task linked to the ventromedial prefrontal cortex and the Spatial Reversal and Object Discrimination Reversal task associated with dorsolateral prefrontal cortex functioning. EF domains assessed in this study included shifting set, inhibition, and working memory. Results indicated a lack of group differences in performance on measures activating the ventromedial prefrontal cortex. However, significant floor effects were noted on the Spatial Reversal task. Performance on tasks activating the dorsolateral prefrontal cortex also resulted in a lack of group differences although there was a statistical trend for children with ASD to exhibit more perseverative errors and overall errors on the *Object Discrimination Task*. Children with ASD and developmental disabilities performed similarly on all tasks and displayed deficits in EF relative to children without developmental disorders. Based on these results, the authors concluded that EF deficits are not unique to preschool-aged children with ASD but that other children with developmental disabilities or delays also show similar deficits in these skills.

A recent study conducted by Drayer (2009) compared 29 children with ASD (M = 5 years 8 months; range = 4 years 0 months – 6 years 11 months) and 30 typically developing children (M = 5 years 9 months; range = 4 years 0 months – 6 years 11 months). Children in the ASD group were diagnosed with autistic disorder (n = 10) and PDD-NOS (n = 19). Intelligence testing indicated that 48% of participants with ASD qualified for an intellectual disability (IQ < 70) and 52% did not qualify for an

intellectual disability (IQ > 70). The study included a comprehensive battery of neuropsychological tasks measuring five domains of EF: shifting set (Dimensional Card Sorting Test), inhibition (Day-Night Test), planning/organization (Tower of Hanoi-R), self-monitoring (*Self-Control task*), and working memory (*Noisy Book task*). The author also collected an ecologically valid measure of EF using the BRIEF-P rating scale (Gioia, Espy, & Isquith, 2003) which was completed by parents and teachers. Results indicated pervasive global executive dysfunction in the sample of young children with ASD with greatest impairments in the shifting set domain followed by self-monitoring, plan/organize, inhibition, and working memory. Working memory was an area of relative strength for children with ASD based on parent and teacher report. When examining age effects, the results indicated that children without autism displayed increased performance as age increased while the performance of children with autism was largely static particularly for the domains of working memory, self-monitoring, and planning/organizing. Scores on performance-based tasks were strongly correlated with companion EF domains on the BRIEF-P indicating similar EF profiles based on EF laboratory tasks and ecologically valid measures of EF manifestations of behavior in home and classroom settings. IQ was strongly and positively correlated with increased EF task performance. Performance on working memory and planning/organization variables was the most impacted by IQ while set shifting and self-monitoring performance showed little change in outcome when accounting for intelligence. These results suggest that while EF abilities are related to IQ in young children with ASD, deficits relative to controls are still evident in children with ASD without intellectual disability. Drayer's findings (2009) also support the stability of EF deficits in children

with ASD across the preschool and early school-age periods suggesting that children with ASD may not experience the age-related increases in EF skills seen in typically developing children.

EF literature involving young children with ASD has been utilized to discount the theory that EF is the primary cognitive deficit playing a causal role in autism. Several studies indicate that preschool and early school-aged children with ASD do not display autism specific difficulties relative to other neurodevelopmental disorders (Griffith, Pennington, Wehner, & Rogers, 1999; Yerys et al., 2007). In addition, research suggests that EF deficits in young children with ASD may not be ubiquitous. For example, Pellicano (2007) examined EF in children 4 to 7 years of age without intellectual disability and found that not all individuals displayed deficits in EF skills. Approximately half of the children with autism demonstrated EF impairments in their study. Calculating individual differences by executive domain, the authors reported that 33% of children with intellectual disability displayed deficits in working memory and inhibition, 43% in planning, and 50% in cognitive flexibility. It appears that while EF dysfunction is common in young children with ASD it may not be universal. In addition, although EF deficits are widely reported in older children and adults with ASD, these findings are typically reported on a group level ignoring observed heterogeneity within ASD groups and differential patterns of findings across studies that suggest variation in cognitive deficits between individuals with ASD (Guerts, Sinzig, Booth, & Happe, 2014). Guerts and colleagues (2014) suggest the possible use of a DSM specifier for EF abilities like IQ and language may be important for guiding future research and treatment.

Despite evidence indicating that EF dysfunction may be a secondary deficit in autism, EF skills are associated with a number of important outcomes for children with ASD including adaptive behavior (Gillotty, Kenworthy, Black, Wagner, & Sirian, 2002; Ozonoff et al., 2004), theory of mind skills (Pellicano, 2010), social competence (Griffith et al., 1999; Pellicano, 2007) ASD symptomology including communicative symptoms (Joseph & Tager-Flusberg, 2004) and repetitive behaviors (South, Ozonoff, & McMahon, 2007; Yerys et al., 2011). A recent study by Vries and colleagues (2015) found that higher levels of EF deficits were associated with lower quality of life for school aged children with ASD. Etiological theories of autism are now considering multiple and coexisting cognitive deficits to explain the immense variability in behavioral phenotype of children with ASD. It is hypothesized that differences in cognitive abilities in three domains: executive functioning, theory of mind skills, and central coherence, may explain the variability in behavioral symptoms and functional outcomes of individuals with ASD (Happe, Ronald, & Plomin, 2006; Pellicano, 2010). Pellicano (2012) examined whether variability in emerging EF skills of preschool aged children with ASD could explain a portion of the phenotypical heterogeneity found in individuals with ASD (Pellicano, 2012). Results indicated that early EF in high functioning preschool aged children with ASD predicts social communication and repetitive interests during a three year follow up. This finding and aforementioned research indicates that consideration of individual differences in EF for individuals with ASD may provide valuable information regarding ASD symptom presentation, prognosis, and treatment implications (Geurts et al., 2014).

The current study will examine whether patterns of EF in young children with ASD can account for variability in the incidence of depression and anxiety symptoms. The sample will include a group of children with ASD without intellectual disability or significant language deficits as recommended by Hill and Bird (2006) to reduce the risk of attributing cognitive and language impairments to EF difficulties. The present study focused on preschool and early school-aged children given research suggesting early childhood is a critical period when EF skills may be particularly responsive to intervention (Diamond, Barnett, Thomas, & Munro, 2007). The following sections will provide an overview of internalizing symptoms followed by a review of anxiety and depression research and correlates in ASD.

Internalizing Symptoms

Overview

Internalizing symptoms are characterized by "intropunitive" moods including sadness, irritability, anxiety, fears and symptoms such as withdrawal behaviors and somatization. Internalizing symptoms and disorders are broad terms representing a dimensional conceptualization of anxiety and depression supported by research showing significant symptom overlap, comorbidity, and evidence suggesting similar underlying etiological processes (Achenbach & McConaughy, 1992; Barlow et al., 2011; Bayer et al., 2011; Clark & Watson, 2006; Eaton et al., 2013; Kovacs & Devlin, 1998; Zahn-Waxler e al., 2000). Given the substantial correlation between anxiety and depressive disorders some argue they may be considered one internalizing disorder in childhood (Karevold, Roysamb, Ystrom, & Mathiesen, 2009). However, empirical support for a categorical distinction between mood and anxiety symptoms and disorders in young children does exist (Luby, Belden, & Pautsh, 2009; Sterba, Egger, & Angold, 2007;

Tandon, Cardeli, & Luby, 2009) and further study is needed in this area. Internalizing disorders are among the most common form of child psychopathology (Carter et al., 2010). The prevalence rate of internalizing disorders in preschool aged children has been estimated at 10-15% (Carter et al., 2010). Research suggests approximately 25% of children will present with internalizing symptoms by age 18 years (Lewinsohn et al., 1993; McLeod et al., 2007). Males and females display similar rates of internalizing symptoms in preschool and elementary school periods (Carter et al., 2010). However, gender differences emerge during adolescence with females but not males exhibiting marked increases in internalizing symptoms and disorders during this time (Lewinsohn, Petit, Joiner & Seeley, 2003; Nolen-Hoeksema, & Girgus, 1994). Internalizing symptoms can present as early as toddlerhood and symptom presentation in preschool aged children including neurovegetative features is similar to that of older children (Luby et al., 2003). Research supports the stability of internalizing symptoms with significant associations between preschool internalizing symptoms and internalizing symptoms in school aged children (Slemming et al., 2010). Anxiety and depression symptoms tend to increase with age from infancy to childhood and even subthreshold symptoms are a risk factor for later internalizing symptoms and disorders (Gilliom & Shaw, 2004). Sterba and colleagues (2007) identified a heterogeneous longitudinal course of internalizing symptom development. They examined maternal based ratings of internalizing symptoms in a large sample from ages 2 to 11 years with approximately two-thirds of children exhibiting low and stable internalizing symptom trajectories, a smaller proportion exhibiting high and stable internalizing symptom trajectories, and children with moderate symptoms either increasing or decreasing across time. Evidence generally supports a more protracted and severe course given earlier onset of elevated internalizing symptoms (Barlow, 1988; Sterba et al., 2007).

Research exploring factors that contribute to the onset and maintenance of internalizing symptoms and disorders in children are largely focused on the categories of child traits, family traits (specifically maternal traits and behaviors), and environmental conditions. Empirically supported models of risk for early internalizing symptoms and diagnoses indicate an interplay between temperamental and environment variables (Crawford, Schrock, & Woodruff-Borden, 2011). A robust predictor of internalizing risk is difficult child temperament characterized by behavior inhibition, high emotional reactivity, and low levels of self-regulatory abilities (Anthony, Lonigan, & Phillips, 2002; Eisenberg et al., 2001; Guthrie, 2001). In addition, lower levels of attentional control have been associated with high rates of internalizing symptoms in children (Bishop, 2007; Eisenberg et al., 2001; Derakshan, Smyth, & Eysenck, 2009).

Family factors linked with increased risk of internalizing symptoms and disorders include high conflict and low family cohesion (Lucia & Breslau, 2006), parental high control, and harsh discipline (Bayer, Sanson, & Hemphill, 2006; Chorpita & Barlow, 1998). Maternal depression is a well-established predictor of childhood internalizing symptoms and diagnoses (Goodman, 2007 for review). A history of both mild and severe and chronic maternal depression have been associated with internalizing symptoms in children (West & Newman, 2003; Bayer et al., 2006; Campell et al., 2009; Luby et al., 2006). Connell and Goodman (2002) found small effect sizes for the relation between maternal depression and child internalizing symptoms (r = .16) in a meta-analytic review. Studies show mothers with depression tend to exhibit less positive emotion and more negative affect (Lovejoy et al., 2000), lower responsiveness to children's distress (Shaw et al., 2006), and provide less effective scaffolding for children's emotional coping (Hoffman et al., 2000) although the relation between maternal depression and child emotion dysregulation was not mediated by maternal scaffolding. Maternal negative affect has been found to mediate the relation between child high negative emotional reactivity, low self-regulation and internalizing symptoms (Crawford, Schrock, & Woodruff-Bordern, 2011). Exploring possible mechanisms of maternal depression risk, Roman and colleagues (2015) found that child executive function (but not verbal ability) at three years of age mediated the relation between maternal depression symptoms at the first longitudinal time point and children's internalizing symptoms at six years of age. They concluded that improving children's EF may serve a protective function against the impact of maternal depression. Additional and commonly cited risk factors for child internalizing symptoms include negative life events and low family socioeconomic status (Bayer et al., 2011; Duggal et al., 2001; Zahn-Waxler e al., 2000).

Internalizing symptoms and disorders place children at greater risk for additional mental health conditions, social and academic maladjustment and poor health outcomes in subsequent developmental periods (Folks & Kinney, 1992; Gilliom & Shaw, 2004; Kessler et al., 2011). Therefore, investigations examining underlying factors involved in the onset or maintenance of internalizing psychopathology can inform both prevention and intervention programs designed to facilitate healthy emotional adjustment in youth.

Assessment of Internalizing Symptoms in Young Children

A variety of methods exist for measuring internalizing symptoms in children. However, assessment of internalizing symptoms is inherently difficult given the largely internal and subjective nature of these problems. Behavioral observations have been employed to measure internalizing symptoms that can be observed directly. However, given that internalizing symptoms are dominated by internal processes and affective states that cannot be readily observed, internalizing symptoms are primarily assessed through clinical interview, self-report questionnaires or behavior rating scales. Structured clinical interviews such as the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Puig-Antich & Chambers, 1978) and the Diagnostic Interview Schedule for Children (DISC-IV; Shaffer et al., 2000) include sections for assessing anxiety and mood disorders in children ages 6 to 18 years of age. Parents and children are interviewed separately and their scores are combined to determine overall diagnostic category and symptom severity. Structured clinical interviews are very time consuming and often unfeasible in certain settings. Therefore, self-report questionnaires and behavior rating scales completed by third-parties are frequently utilized in the assessment of internalizing symptoms. Both general behavior rating instruments as well as instruments specifically targeting internalizing symptoms (i.e., narrow-band instruments) exist. Two widely used narrow-band instruments for assessing internalizing symptoms include the Children's Depression Inventory-Second Edition (CDI2; Kovacs, 2004) and the Multidimensional Anxiety Scale for Children (MASC; March, 2004). However, popular narrow-band instruments for measuring anxiety and depression generally assess children that are school age (i.e., 6 years old) or older. Therefore, clinicians and researchers assessing internalizing symptoms in preschool-aged children frequently employ broad behavior ratings scales applicable to this age range. The Child Behavior Checklist (CBCL; Achenbach, 1991) and the Behavior Assessment System for Children-Second Edition (BASC-2; Reynolds & Kamphaus, 2004) are two widely used

rating scales applicable to toddlers and preschool-aged children with subscales and composite scores measuring internalizing symptoms. The current study will utilized the parent and teacher version of BASC-2 to measure children's internalizing symptoms (i.e., depression, anxiety, and somatization).

Internalizing Symptoms in ASD

Internalizing symptoms including anxiety and depression are common in children with ASD. Several studies have indicated that children and adolescents with ASD exhibit significantly higher rates of internalizing symptoms and disorders than typically developing children (Kim et al. 2000, MacNeil et al., 2009; Russell & Sofronoff, 2005; Solomon et al., 2012; Suhkodolsky et al., 2008). Gurney et al. (2006) reported a 15-fold increase in the probability of a comorbid diagnosis of anxiety or depression among children and adolescents with ASD compared to children without an ASD diagnosis. Although studies involving preschool aged children are limited, several investigations examining comorbidity in children with ASD in this age range have demonstrated increased rates of internalizing symptoms relative to norms and typically developing control groups (Brereton, Tonge, & Einfeld, 2006; Gadow, DeVincent, Pomeroy, & Azizian, 2004; Mayes et al., 2011). Prevalence rate estimates for comorbid depression in ASD range from 4 to 34% (Stewart, Barnard, Pearson, Hasan & O'Brien, 2006 for review) in samples with ages ranging from two years to 18 years. Empirical reviews suggest that between 11% and 84% of children with ASD exhibit anxiety that causes impairment to some degree (van Steensel et al., 2010; White et al., 2009) in studies with mean ages ranging from 4.2 years to 16.3 years. The most commonly reported anxiety disorders in ASD include simple phobias, generalized anxiety disorder, separation

anxiety disorder, obsessive-compulsive disorder, and social phobia (White et al. 2010). Depressed mood is the most frequency cited marker of depression in research examining presentation of depression in ASD (Stewart et al., 2006). Variation in prevalence estimates of internalizing symptomology and diagnoses is likely influenced by differences in sample ascertainment, sample size, and the manner in which internalizing symptoms are operationalized and assessed. Despite the largely subjective and internal nature of anxiety and depressive symptoms, most studies rely on third party ratings to collect information on internalizing psychopathology (Solomon et al., 2012). Several large studies using the Behavior Assessment System for Children-2nd Edition (BASC-2; Reynolds & Kamphaus, 2004) found school aged children with ASD were rated significantly higher on the BASC-2 Anxiety, Depression, and Internalizing Composite scales compared to age-matched controls (Goldin, Matson, Konst, & Adams, 2014; Solomon et al., 2012).

Despite reports of increased rates of internalizing symptoms in ASD, there is controversy regarding differential diagnosis of ASD symptoms and internalizing symptoms given symptom overlap that may obscure presentation (Leyfer et al., 2006; Noordhof, Krueger, Ormel, Oldehinkel, & Hartman, 2015). For example, it can be difficult to determine whether symptoms of depression and anxiety such as social withdrawal/avoidance, abnormal speech patterns, flat affect, low energy, reduced motivation, sleep disturbance, reduced appetite, and compulsive behaviors represent distinct co-occurring symptoms or manifest as part of ASD. There is recent research exploring construct discrimination in regards to internalizing symptoms and ASD with extant studies focused on anxiety symptoms. A study conducted by Hallet and colleagues

(2013) indicated the CASI-Anxiety scale measures a unique construct separate from ASD severity while White and colleagues (2015) found the Multidimensional Anxiety Scale for Children may not measure identical constructs in children experiencing anxiety with and without ASD. Renno and Wood (2013) conducted a study with 88 children (ages 7-11 years) with ASD identified for concerns regarding anxiety using multiple methods for assessment including diagnostic interviews and parent and child based measures. Findings from structural equation modeling supported discrimination between anxiety and ASD severity and suggested anxiety symptoms and diagnoses in children with ASD may be similar to those in typically developing children. Noordhof and colleagues (2015) examined co-occurrence patterns of ASD symptoms with other domains of psychopathology using the general dimensional framework of internalizing and externalizing symptoms. Study methods used a large general population sample between ages of 10 years and 17 years with three longitudinal time points. Results of factor analysis indicated ASD symptoms represented a specific domain of psychopathology that was distinct from internalizing and externalizing domains. Hallet and colleagues (2010) explored the association between ASD traits and internalizing traits across middle to late childhood using a sample of 6,000 twins across two time points at approximately seven years of age and 12 years of age. Results indicated both traits were moderately to highly heritable but evidenced a low level of genetic overlap. A bidirectional relationship between ASD traits and internalizing was observed with a stronger influence of early ASD traits on subsequent internalizing traits particularly communication difficulties. Despite the aforementioned empirical work largely supporting construct discrimination of ASD symptoms and internalizing symptoms, further research in this area is greatly

needed; particularly studies exploring neurobiological bases or biological markers associated with comorbid internalizing symptoms in ASD that may aide distinction between features of ASD and separate co-occurring disorders (Leyfer et al., 2006).

Numerous studies have examined the relation between ASD severity and internalizing symptoms. Several studies suggest that increased anxiety and depression symptoms are associated with greater ASD severity (Kanne, Abbacchi, & Constantino, 2009; Suhkodolsky et al., 2008). However, empirical work also supports an association between ASD and increased internalizing psychopathology for individuals with fewer ASD symptoms, such as individuals with high functioning autism or Asperger's disorder (Estes, Dawson, Sterling, & Munson, 2007). Utilizing data from the large scale Simons Simplex Collection Project, Mazurek and Kanne (2010) reported a negative correlation between ASD symptoms and Anxiety, Depression, and Internalizing composite scores on the CBCL in children with ASD ages 4 to 17 years (M = 9.1 years SD = 3.5). Kim et al. (2000) found similar rates of internalizing symptoms in children with high functioning autism and Asperger's disorder. Researchers theorize that individuals with ASD without intellectual disability or significant language impairments possess greater insight regarding personal difficulties leading to greater distress and potentially internalizing disorders (Hedley & Young, 2006). In addition, individuals with ASD with average or higher IQ and language abilities may have a greater capacity to communicate their distress which could lead to the higher rates of internalizing scores on self, parent, and teacher report measures.

The association between age and internalizing symptoms in children with ASD is well-established based on extant research. Chronological age is positively correlated with increasing depressive symptomology (Brereton et al., 2006; Ghaziuddin,

Ghaziuddin, & Greden, 2002; Vickerstaff, Heriot, Wong, Lopes & Dossetor, 2007) and anxiety (Weisbrot, Gadow, DeVincent & Pomeroy, 2005). Greater intelligence, particularly verbal IQ, is associated with increased anxiety and depression ratings in children with ASD (Kim et al., 2000; Mayes et al., 2011; Mazurek and Kanne 2010; Sukhodolsky et al., 2008). Research has generally indicated a lack of differences in internalizing symptoms for children with ASD according to gender, race, and SES (Brereton et al., 2006; Sukhodolsky et al., 2008). In contrast, several studies suggest that girls with ASD may be at greater risk for internalizing symptoms than boys (Hartley & Sikora, 2009; Solomon et al., 2012). A recent study (Gotham, Brunwasser, & Lord; 2015) examined growth in anxiety and depression symptoms from late school age to young adulthood in 165 participants with ASD (n = 109) and nonspectrum developmental delay (n = 56) between ages 6 and 24 years. Results indicated anxiety and depressive symptoms were greater in subjects with ASD compared to individuals with nonspectrum developmental delays. Anxiety was positively related to verbal IQ and internalizing symptoms were associated with poorer emotion regulation in school age. Males with ASD exhibited elevated internalizing symptoms that were maintained into young adulthood and females demonstrated elevated internalizing symptoms that increased at a faster rate throughout adolescence compared to male counterparts.

Depressive and anxious symptomology in children with ASD are associated with increased maladaptive behaviors and are thought to exacerbate core symptoms of ASD including social difficulties, repetitive and stereotyped behaviors, as well as aggression (Ghaziuddin, Ghaziuddin, & Greden, 2002; Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006) and contribute to greater functional impairment (Chang, Quan, & Wood, 2012), lower life satisfaction, and greater social difficulties in adulthood (Gotham, Brunwasser, & Lord, 2015). Internalizing symptoms in ASD are associated with oppositional behavior, aggression (Kim et al., 2000), irritability, and hypersensitivity (Sukhodolsky, 2008). In addition, symptoms of ASD and comorbid anxiety and depression appear to increase with age as older children and adolescents exhibit more severe symptoms than younger children with these problems supporting the need to intervene earlier in the developmental course. The high comorbidity of internalizing symptoms in children with ASD indicates the need to examine correlates and risk factors for such conditions. The current study will investigate neuropsychological correlates of anxiety and depression symptoms in children ages 3:0 to 6:11 years of age.

Executive Functions and Internalizing Symptoms

Researchers have examined the role of executive functions in a range of psychopathological outcomes for young children surmising that abnormal development in this area is etiologically related to childhood psychiatric disorders. The majority of investigations have examined EF's role in the development of externalizing symptoms and disorders. Several studies have shown that young children with problem behaviors demonstrate deficits in EF (Hughes & Ensor, 2008; Pennington & Bennetto, 1993). Much less is known about the relation between EF and internalizing symptoms such as depression and anxiety. However, extant research has found significant relations between impaired EFs and internalizing symptoms (Ciairano et al., 2007; Nigg et al., 1999; Riggs et al., 2003). Riggs and colleagues (2003) conducted a longitudinal study investigating executive functions and behavior problem symptomology in 60 typically developing school aged children (age range = 6 years 9 months to 9 years 2 months). This study looked specifically at EF indicators, inhibitory control and sequencing ability, and their relation to socio-emotional outcomes. Inhibitory control predicted parent reported internalizing symptoms over a two year period and sequencing ability predicted parent report of internalizing symptoms over the same time period. The authors concluded that school age EF deficits place young children at risk for developing psychiatric problems including internalizing psychopathology and that early intervention for children with weak neurocognitive functioning may reduce future problems.

Several studies have also noted a negative association between verbal fluency and verbal working memory and internalizing symptoms in young children (Kusche, Cook, & Greenberg, 1993; Riggs et al., 1993). Eisenberg and colleagues have produced a substantial body of research documenting relations between effortful control and internalizing psychopathology (Eisenberg et al., 2001; Eisenberg et al., 2009; Valiente et al., 2004). Effortful control (EC) is a concept related to multiple EF domains. EC comprises the ability to voluntarily focus/shift attention and inhibit/ initiate behaviors (Eisenberg et al., 2001). Lengua (2006) has reported that a lack of growth in EC during the preschool period over two years predicted internalizing symptomology when children were 10 to 14 years of age. In a large community sample of at-risk boys and girls (N = 498), Martel and colleagues (2007) found that childhood low reactive control or poor inhibitory skills in response to emotionally arousing situations predicted the development of internalizing symptoms during adolescence.

Researchers have also examined the predictive linkages between EF and internalizing symptomology for individuals at high risk for EF deficits, including children

with ADHD. Rinsky and Hinshaw (2011) followed 140 females with ADHD and 88 comparison females across a five year period from childhood (i.e., 6-11 years of age) through early/mid-adolescence to examine if childhood EF abilities would predict social and emotional outcomes. Childhood planning deficits predicted comorbid internalizing disorders in adolescence while working memory marginally predicted the incidence of internalizing disorders in adolescence. The authors concluded that EF related to cognition such as planning and working memory may be more important for later psychological functioning than more behaviorally-oriented EFs such as inhibitory control. However, other studies measuring internalizing symptoms in a sample of children with ADHD have found an association between non-verbal working memory and inhibition. Using two measures which will be employed in the current study, the BASC-2 and BRIEF, Jarratt and colleagues (2005) found an association between all EF domains on the BRIEF and the BASC-2 internalizing composite in children between the ages of 9 and 15 years with ADHD. Jonsdottir and colleagues (2006) examined the relation between EFs and psychiatric comorbidity in a sample of 43 children aged 7 to 11 years of age referred for neuropsychological assessment with primarily diagnoses of ADHD. Results indicated that performance on the neuropsychological tower task, an index of planning ability, was negatively related to teacher ratings of atypicality and depression. In addition, performance on the Visual Attention task, an index of selective and sustained attention, was negatively related to teacher ratings of anxiety on the BASC-2.

Links between EF and internalizing symptoms have also been found in intervention studies designed to promote neurocognitive functioning. The Promoting Alternative Thinking Strategies (PATHS; Greenberg & Kusche, 1993) curriculum is designed to promote social and emotion competence in elementary–aged children. This curriculum follows a developmentally informed model targeting frontal lobe development during a period of great plasticity and rapid neuronal growth of this region. The PATHS program implemented in second and third grade led to children's improved inhibitory control and verbal fluency. EF improvements predicted lower rates of internalizing problem behaviors and EF skills were found to mediate the relation between intervention and reduced internalizing at one year follow up (Riggs et al., 2006). The aforementioned studies support the vital role of executive functions in children's internalizing symptomology and highlight the need for EF considerations in early interventions.

Neuroscience Research linking Executive Function and Internalizing Symptoms

Neuroscience research has attempted to locate brain regions and circuitry involved in the pathogenesis of internalizing symptoms. Several recent studies implicate neurological areas responsible for modulating executive functions, namely the prefrontal cortex, in the pathophysiology of affective disorders (Price & Drevets, 2010, 2012). Koenigs and Grafman (2009) reviewed neuroscience research utilizing various methodologies and found that two areas of the prefrontal cortex, the dorsolateral prefrontal cortex (dIPFC) and ventromedial prefrontal cortex (vmPFC), appear to play a distinct role in the neuroanatomy of mood disorders. As mentioned previously in this proposal, the dIPFC is primarily associated with higher order cognitive functions and EFs including working memory, planning, and organization. It also plays an important role in the integration of sensory information. The vmPFC is largely associated with emotion

regulation and affective decision making. The dIPFC is connected to a variety of cortical and subcortical regions including the orbitofrontal cortex, thalamus, basal ganglia, hippocampus, and parietal and occipital areas (Alvarez & Emory, 2006). The vmPFC is also widely connected to various areas including the hypothalamus, periaqueductal gray, and amygdala (Price & Drevets, 2010). Functional neuroimaging studies have reported elevated levels of vmPFC activity (Biver et al, 1994; Price & Drevets, 2012) and inefficient or dysfunctional activity of the dIPFC (Biver et al., 1994; Baxter et al., 1989) in depressed patients. In addition, patients who have recovered from depression have demonstrated increased activity in the dlPFC and decreased activity in the vmPFC (Brody et al., 2001). Lesion studies conducted by Koenigs and colleagues (2009) found that bilateral vmPFC lesions were associated with significantly less depression and bilateral dlPFC damage was associated with significantly higher levels of mood symptoms. Brain stimulation studies report a significant reduction in affective symptomology following dlPFC stimulation (Marangell, Martinez, Jurdi & Zboyan, 2007). Several investigations also implicate the dIPFC during the regulation of negative affect and normalization of mood states through reappraisal and suppression strategies via connections with limbic structures (Ochsner et al., 2004; Pizzagalli, 2011). This research strongly suggests that decreased dlPFC activity and increased vmPFC activity is associated with affective psychopathology (Koenigs & Grafman, 2009). In addition to depression, the lateral prefrontal cortices have also been implicated in anxiety disorders (Davidson, 2002; Kim, Gee, Loucks, Davies, & Whalen, 2011). Researchers in this area note that the higher order EF areas of the prefrontal cortex, such as the dorsolateral prefrontal cortex, lack significant connections with the amygdala and hypothesize that these regions are

connected to emotional disorders because they are responsible for integrating cognitive and affect input (Hikosaka & Watanabe, 2000).

Although the majority of studies examining neural correlates of internalizing disorders have been conducted with adults, recent research has also examined the prefrontal cortex in relation to pediatric psychopathology. Price and Drevets (2010) noted structural abnormalities in the prefrontal cortex of children with mood disorders suggesting abnormal PFC maturation. Neuroimaging studies have also reported lower activation of the dorsolateral prefrontal cortex during EF tasks mediated by this region in adolescents with MDD relative to healthy controls (Halari et al., 2009). Reduced activation of prefrontal areas during cognitive tasks has also been observed in youth at high risk for depression (Hulvershorn, Cullen, & Anand, 2011). In summary, this section reviewed the important role of prefrontal regions in anxiety and mood symptomology and disorders linking neurological abnormalities present in ASD with internalizing symptomology. The following section reviews the available research examining whether executive functions may be related to internalizing symptoms in ASD.

Executive Function, Internalizing Symptoms, and ASD

Research with typically developing children and children with ADHD and other neurodevelopmental conditions demonstrates the importance of executive functioning skills for children's healthy psychological adjustment (Kelly et al., 2012). Empirical work with these populations suggests that internalizing symptoms may be associated with deficits in EF skills for young children. For instance, Kelly and colleagues (2012) examined the relation between executive functioning and psychological adjustment in children with spina bifida/myelomeningocele (SMB). Similar to children with ASD, children and adolescents with SMB display impaired executive functioning abilities and have an increased risk for internalizing disorders such as depression, anxiety, and somatization symptoms (Kelly et al., 2012). The authors examined the mediational role of EF in the relation between developmental status and psychological outcomes. The sample consisted of 51 children and adolescents with SMB between the ages of 10 and 17 years of age (M = 13.0 years) and 45 typically developing children ages 10-16 years (M =11.8 years). Measures included the Behavior Rating Inventory of Executive Function (BRIEF; Gioia et al., 2000), Behavioral Assessment System for Children-Second Edition (BASC-2; Reynolds & Kamphaus, 2004), and the Child Depression Inventory (CDI; Kovacs, 1992). The results indicated that EF abilities including initiation, working memory, and planning/organizing fully explained the relation between neurodevelopmental group and maternal ratings of internalizing and depression symptoms. The authors concluded that interventions designed to facilitate EF development in an at-risk population could have implications for children's emotional functioning and psychological outcomes.

Several developmentally-based interventions for young children with ASD, such as TEACCH and the Early Start Denver model, target neuropsychological deficits in children with ASD. These approaches have demonstrated effectiveness in improving ASD symptomology, adaptive behavior, IQ, and language (Eikeseth, 2009). However, the association between these programs and psychological outcomes such as anxiety and depression for children with ASD has not been thoroughly explored. A few recent studies have reported on the psychological outcomes associated with interventions explicitly targeting EFs in children with ASD. Kouijzer et al. (2009) conducted 40 sessions of neurofeedback training with 17 children with ASD (intervention group n = 7; control group n = 7) between the ages of 8 and 12 years of age (M = 10.1 years). ASD symptoms and executive functioning abilities were assessed pre-intervention and three months following neurofeedback training completion. The authors reported significant improvement on a battery of EF tasks assessing attentional control, cognitive flexibility, and goal setting in children in the intervention group relative to controls. Children in the intervention group also displayed significant improvement in social and communicative abilities although measures of behavior problems including internalizing and externalizing problems were not included in this study. However, several studies utilizing similar neurofeedback training procedures in children with ASD have reported decreases in anxiety and mood post intervention (Jarusiewicz, 2002; Scolnick, 2005). These studies combined suggest that interventions associated with increased EF skills may be related to improvement in internalizing symptoms for children with ASD.

Research directly examining the relation between internalizing symptoms and EF in individuals with ASD is extremely limited and only recently published. To my knowledge only two recent studies exist in this area involving youth with ASD. Hollocks and colleagues (2014) investigated associations between neurocognitive functioning and anxiety and depression symptoms in adolescents with ASD. The study including 90 adolescents with ASD without intellectual disability from the populationderived Special Needs and Autism Project cohort (Baird et al, 2006) and used several performance based tasks to measure EF including the *Opposite Words task* measuring attention and interference inhibition, *Trail Making task* measuring attentional shifting, *Number Backwards task* measuring verbal working memory, and *Card Sorting Task* measuring cognitive set shifting. Internalizing symptoms were assessed using Strengths and Difficulties Questionnaire (SDQ; Goodman, Ford, Simmons, Gatward, & Meltzer, 2003) a mental health screening tool with 25 items and the Profile of Neuropsychiatric Symptoms (PONs; Santosh, Baird, Pityaratstian, Tavare & Gringras, 2006) with a focus on five items related to worries, fears, depressed thoughts, low mood, and labile mood. SEM analysis conducted to estimate the effects of EF latent variables on internalizing symptoms found that problems with EF were associated with higher levels of anxiety but not depression.

Lawson and colleagues (2015) examined whether specific EF deficits in ASD and ADHD serve as mechanisms underlying common psychiatric comorbidities with these disorders. Specifically, this research examined the hypothesis that parent reported difficulties with flexibility in ASD and inhibition in ADHD would mediate the association between diagnostic status and internalizing symptoms and oppositional/aggressive behavior. This study included a clinical sample of children diagnosed with ASD (n = 70) and ADHD Combined Presentation (n = 55) and used parent report measures including the BRIEF Shift (shift/flexibility), BRIEF Inhibit (behavioral inhibition) scale scores, and the CBCL Anxious/Depressed and Aggressive Behavior scales scores. Children with dual diagnoses of both ASD and ADHD were excluded from the sample. Results from a path analysis supported the authors' mediational hypotheses finding that ASD diagnostic status predicted greater flexibility problems which predicted higher anxiety/depression while ADHD diagnostic status predicted greater inhibition problems that were associated with increased rates of aggression. Flexibility problems associated with ASD also predicted aggression. Recent empirical work with adults examined the association between EF and co-morbid anxiety and depression symptomology as well as adaptive functioning using the Behavior Rating Inventory of Executive Functioning—Adult version (BRIEF-A; Roth et al. 2005) as a measure of everyday EF, Adult Behavior CheckList (ABCL; Achenbach & Rescorl, 2003) as a measure of internalizing symptoms (Wallace et al., 2015), and Adaptive Behavior Assessment System—Second Edition (ABAS-II; Harrison & Oakland 2003) as a measure of adaptive functioning. Analysis of data from 35 adults indicated flexibility problems were associated with anxiety while metacognition specifically planning/organization skills were associated with depression symptoms and problems with adaptive functioning. ADHD symptoms moderated the relationship between metacognition and adaptive functioning.

The current study extends this recent research in several important ways. Specifically, the present investigation utilized both performance and real world parent report EF measures, examined multiple domains of EF dysfunction associated with ASD namely planning and set shifting abilities which represent EF components of both behavior regulation and metacognition and explored the association between EF and cooccurring internalizing symptoms in a community based sample of younger children with ASD.

Present Study

The current research study investigated the possibility that an indirect link exists between developmental status (ASD vs. typically developing) and internalizing symptoms through executive functioning skills in young children with ASD. The factors impacting psychological outcomes in children with ASD are not fully understood and research indicates that psychiatric comorbidity increases with age in this population and is associated with more negative outcomes overall. Therefore, it is important for research to focus on variables which may help to alter these negative trajectories by investigating early indicators of current and future psychopathology. This investigation is supported by research indicating that abnormalities of the prefrontal cortex and deficits in executive functioning are associated with greater internalizing psychopathology in typically developing children and adults and recent investigations involving school aged children and adolescents with ASD. ASD is often associated with prefrontal pathology as well as executive functioning impairment with greater variability during the early childhood period when these skills are emerging. Therefore, variability in early EF skills in preschool and early school aged children with ASD may contribute to internalizing difficulties for this group. The current study examined whether differences in executive functioning on a performance based task (TOH-R; Welsh, Pennington, & Groisser, 1991) which requires prefrontal activation explains some of the variance in the incidence of internalizing symptoms in children with and without ASD. In addition, the current study utilized an ecologically valid measure of EF, the Behavior Rating Inventory of Executive Function (BRIEF, Gioia et al., 2000) parent report rating scale, to examine everyday manifestations of EF deficits and explore how they relate to internalizing symptoms. Deficits in everyday functioning mediated by executive skills may cause significant distress for individuals with ASD and impart greater risk for internalizing reactions. In addition, youth with ASD often have limited coping skills to effectively respond to stress in their environment and EF deficits create significant challenges for successful learning and generalization of new adaptive coping skills for these children. Information provided by this study will increase our understanding of how potential weaknesses in emerging EF abilities relate to internalizing symptoms in preschool and early school aged children with ASD. The information gathered during this investigation has the potential to inform interventions targeting the prevention and treatment of internalizing symptoms and disorders. It is particularly important to examine potential targets for prevention and early school age period EF skills are rapidly changing and are particularly malleable to environmental modification. Parents and teachers may also begin to see signs of internalizing psychopathology at this age. Understanding the mechanisms and neurocognitive profiles associated with internalizing symptoms in children with ASD and typical development will aid clinicians in developing interventions for young children that could potentially increase skills, modify underlying brain circuitry, and increase positive psychological outcomes for these children.

Hypotheses

I examined executive functions, specifically cognitive flexibility and planning skills, in relation to developmental status (ASD vs. typically developing) and internalizing symptoms in children ages 3:0 to 6:11 years. Based on previous research, the following hypotheses were made:

Hypothesis 1

Child developmental status (ASD vs TD) will predict children's internalizing symptomology. Prior research indicates that children with ASD exhibit significantly higher rates of internalizing symptoms than typically developing peers (Kim et al. 2000; MacNeil et al., 2009; Russell & Sofronoff, 2005; Solomon et al., 2012). Children with ASD of varying ages and levels of functionality score significantly higher on standardized rating scales of internalizing symptoms including depression and anxiety relative to norms and comparison groups of typically developing children (Brereton, Tonge, & Einfeld, 2006; Gadow, DeVincent, Pomeroy, & Azizian, 2004; Mayes et al., 2011). Research indicates increased risk for depression and anxiety symptoms relative to aged matched controls is present as early as preschool and early-school age periods for children with ASD without intellectual disability (Estes, Dawson, Sterling, & Munson, 2007; Kim et al., 2000; Mazurek & Kanne, 2010). The current study examined internalizing symptoms in a group of young children with ASD without intellectual disability. Therefore, it was hypothesized that children with ASD would exhibit significantly higher levels of internalizing symptoms per parent and teacher report than typically developing peers.

Hypothesis 2

Child developmental status will predict executive functioning skills. Research demonstrating executive functioning deficits in individuals with ASD of varying ages and levels of cognitive functioning is robust (Gioia, Isquith, Kenworthy, & Barton, 2002; Hill, 2004; Kenworthy, Yerys, Anthony, & Wallace, 2008; Pennington & Ozonoff, 1996). Studies examining the neuropsychological profile of children with ASD indicate significant impairments in cognitive flexibility and planning (Hill, 2004; Yers, Wallace, Jankowski, Bollich, & Kenworthy, 2011). Structural and functional neuroimaging studies of children with ASD also indicate the presence of abnormal development of prefrontal areas involved in executive functioning (Courchesne, et al., 2011; McAlonan et al., 2009; Zikopoulos & Barbas, 2010). Specifically, researchers have reported neuropathology in the dorsolateral prefrontal cortex associated with planning and set shifting abilities in individuals with ASD (Morgan et al., 2010). It was hypothesized that ASD status would be associated with lower EF performance on measures of planning and set shifting.

Hypothesis 3

Executive functioning performance will predict internalizing symptomology. Extant research demonstrates significant negative relations between EFs and internalizing symptoms in typically developing children (Ciairano et al., 2007; Lengua, 2006; Nigg et al., 1999; Riggs et al., 2003) and children with ADHD (Jarratt, Riccio & Siekierski, 2005; Rinksy & Hinshaw, 2011; Jonsdottir et al., 2006). In addition, outcome research examining interventions designed to promote neurocognitive functioning have found that EF improvements predict lower rates of internalizing symptoms and mediate the relation between intervention and internalizing symptoms (Riggs et al., 2006). Lastly, neuroscience investigations with clinical populations implicate neurological areas responsible for executive control in the pathophysiology of affective and anxiety disorders (Price & Drevets, 2010, 2012). Therefore, it was hypothesized that higher executive functioning scores would uniquely predict lower internalizing symptoms in both children with ASD and typical development.

Hypothesis 4

Executive functioning will mediate the relation between child developmental status and internalizing symptoms. This mediation model is displayed in Figure 1. I hypothesized that variability in executive functioning abilities would explain the association between developmental status and internalizing symptoms. Specifically, I hypothesized that ASD status would be associated with higher levels of internalizing symptoms through deficits in planning and cognitive flexibility. Despite limited research examining the relations between EF and internalizing symptoms in children with ASD, this hypothesis is supported by a recent study finding support for a mediational model in which ASD diagnostic status predicted deficits in cognitive flexibility which predicted increased anxiety/depression symptoms using the BRIEF Shift subscale and CBCL ratings for internalizing symptoms in children aged 6 to 16 years (Lawson et al., 2015). Furthermore, research indicates EF deficits and prefrontal pathology are present in children with ASD and prefrontal abnormalities in the dlPFC are implicated in affective and anxiety disorders in typically developing individuals (Biver et al., 1994; Davidson, 2002; Koenigs & Grafman, 2009; Price & Drevets, 2010). The aforementioned findings provide a sound neuroanatomical and theoretical basis for why EF skills may explain the incidence of internalizing symptomology in children with ASD. The study examined whether variance in EF skills per parent report and measured through a performance based task could account for significant variance in the association between developmental status and mood and anxiety symptoms and extend previous research by studying these relations in early childhood.

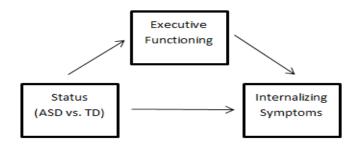


Figure 1. Proposed mediation model of the effects of executive functioning on the relation between developmental status and internalizing symptoms.

Hypothesis 5

Performance on the neuropsychological task requiring planning and set shifting abilities will be positively related to planning and set shifting abilities on the maternal behavior rating scale of executive functions. The rating scales utilized in this study, the Behavior Rating Inventory of Executive Function (BRIEF, Gioia et al., 2000) and the Behavior Rating Inventory of Executive Function-Preschool Version (BRIEF-P, Gioia, Espy, & Isquith, 2003), have demonstrated significant moderate correlations with performance based measures of executive functioning in children (Bishop, 2011; Collins, 2012; Oberg & Lukomski, 2011). In theory, elicited tasks measure components of EFs that are expressed in everyday functioning of children in their environment. Therefore, it was hypothesized that the elicited task and ecologically valid measure would be positively correlated in both groups of children

Chapter II: Method

Participants

This study was part of a larger investigation examining self-regulation in young children with ASD and typically developing children. The current study was approved by the Institutional Review Board at Seattle Pacific University. Eligibility inclusion criteria for the current study included the following; (a) children must be between the ages of 3-years-0-months and 6-years-11months, (b) children must demonstrate adequate verbal abilities required to complete study tasks, (c) children eligible for the ASD group must have a previous diagnosis of ASD from a licensed provider, (d) children eligible for the typically developing group could not receive a score in the "high risk" range on a parent report autism screening questionnaire, have a previous psychiatric or developmental diagnosis, and could not have a sibling diagnosed with ASD.

Participants included 66 children between the ages of 36 and 85 months. There were 40 children in the typically developing group and 26 children in the ASD group. One parent (N = 66) and teacher (N = 59) participated with children enrolled in the study. Demographic information is included in Table 1. Study groups did not differ significantly on chronological age (3-years (ASD = 6, TD = 15), 4-years (ASD = 5, TD = 8), 5-years (ASD = 7, TD = 12), and 6-years (ASD = 5, TD = 5)), verbal mental age, or family annual income (See Table 1). Child verbal mental age was calculated using the standard verbal scores from the Differential Abilities Scale, Version 2 (DAS-II, Elliot, 2007) and the child's chronological age within the following formula: (((Child_AgeYears*365.25) + (Child_AgeMonths*30))/365)*.01*STANDARD SCORE

(DAS-II Verbal Ability) = Verbal Mental Age expressed in years. However, groups

differed significantly in regards to child gender, total verbal ability, receptive language skills, expressive language skills, child ethnicity, and maternal history of depression (See Table 1). Children in the ASD group had significantly lower scores on measures of total verbal ability, t(64) = 4.75, p < .001, d = 1.19, receptive language skills, t(64) = 4.46, p < .001.001, d = 1.12, and expressive language skills, t(64) = 3.48, p = .001, d = .87, than the typically developing sample. Significant differences existed between groups in regards to gender, $X^2 = (1, N = 66) = 5.34$, p = 0.12, and ethnicity, Fisher's Exact test = 9.38, p = 0.12.012. The ASD group had a higher ratio of males to females (5.5:1) than the typically developing group (1.4:1 male to female). The ASD group gender ratio was also slightly higher than that found in the autism population (approximately 4:1 male to female). In regards to ethnicity, children in the typically developing group were predominately identified as Caucasian whereas the ASD group had a lower percentage of children identified as Caucasian and higher percentage of children identified as Hispanic and those identifying with two or more ethnic groups (See Table 1). A Chi-squared test indicated significant group differences in maternal history of depression, X^2 (1, N = 66) = 9.26, p =.002, with a higher frequency of maternal depression history in the ASD group (n = 17)compared to the TD group (n = 11).

Demographic Information. Parental guardians provided demographic information via questionnaire. The current sample included predominately well-educated and upper middle class families with the majority of the parent participants being mothers (78.4%). Sixty-five parent guardians (98.5%) identified as married or having a domestic partner and one caregiver (1.5%) identified as divorced. Parental level of education was reported as follows: 1.5% high school degree, 18.2% some college coursework, 37.9%

Table 1Descriptive Information by Group

	TD	ASD	-
Variable	(n = 40)	(n = 26)	t/X^2 /Fisher's
Child Variables			
Child Gender			5.34*
Female, N (%)	17 (42.5%)	4 (15.4%)	
Male, N (%)	23 (57.5%)	22 (84.6%)	
Chronological Age (months)			65
Mean (SD)	56.43 (13.42)	58.72 (15.00)	
Verbal Mental Age (months)			1.37
Mean (SD)	5.21 (1.35)	4.75 (1.30)	
Total Verbal Abilities	· · · ·		4.75***
Mean (SD)	111.63 (11.21)	98.15 (11.31)	
Receptive Language Abilities	· · · ·		4.46***
Mean (SD)	54.80 (6.78)	46.08 (9.08)	
Expressive Language Abilities			3.48**
Mean	58.30 (8.71)	51.23 (6.94)	
Child Ethnicity, N (%)			9.38*
Caucasian	32 (80%)	12 (46.2%)	
Hispanic	0 (0%)	2 (7.7%)	
Asian American	4 (10%)	4 (15.4%)	
Multiple Ethnicities	4 (10%)	8 (30.8%)	
Family Variables			
Maternal History of Depression	11 (28%)	17 (65%)	9.26**
Average Annual Income (SD)	\$153, 101	\$114, 364	1.45
	(\$114, 844)	(\$64, 360)	

Note. Total Verbal Abilities = DAS-II Verbal Reasoning Cluster Standard Score; Receptive Language Abilities = DAS-II Verbal Comprehension Subtest T Score; Expressive Language Abilities = DAS-II Naming Vocabulary Subtest T score; *p < .05;

bachelor's degree, 4.5% some master's coursework, 15.2% master's degree, 13.6% some professional schooling beyond master's degree, and 9.1% professional degree beyond master's degree. Annual household income ranged from \$25,000 to \$700,000 with an average of \$138,407 (SD =\$99, 920). There were no significant group differences on family demographic variables for parent gender, Fisher's Exact test, p = .247, annual household income, t(56) = 1.45, p = .154, parent relationship status, Fisher's Exact test, p = .606, and parent education, Fisher's Exact test, p = .196.

Procedures

Recruitment of participants. Families were recruited from local autism treatment clinics, research centers, and public and private elementary schools and preschools in the greater Seattle area. Recruitment handouts were provided to schools and clinics to allow interested parents to contact research coordinators for additional information about the study. Research staff also set up information tables at recruitment sites where parents could learn more about the study and sign up to receive a phone call from research coordinators. Additionally, pull-tab flyers were posted at schools, local libraries, community centers, and businesses that serve children and families and study announcements were placed in local parenting magazines and Autism listserv. Announcements, handouts, and pull-tab flyers provided general information about the study and contact information for research staff coordinating subject enrollment.

Enrollment visit. The Enrollment visit was conducted at the family's home, a local library, or Seattle Pacific University developmental laboratory based on the family's preference. Total enrollment visit duration is approximately 65 to 95 minutes. Parental informed consent and child assent were obtained at this visit. Parents of children with ASD were asked to sign a release form granting permission to contact the diagnosing provider for diagnostic records or provide a copy of the initial diagnostic report to confirm developmental status. A teacher release of information was also completed allowing teachers to fill out questionnaires assessing the child's behavior and social skills at school. Teachers were mailed a study packet including a copy of the release of information form, study questionnaires, and a prepaid envelope addressed to project offices to return completed measures.

During the enrollment visit, eligibility was assessed for case (i.e., ASD) or control (i.e., typically developing) status using several instruments. Parents completed a screener for symptoms of ASD: The Social-Communication Questionnaire-Current Form (SCQ; Rutter, Baily, & Lord, 2003). Children completed the Verbal Reasoning Cluster of the Differential Abilities Scale – Version II (DAS-II; Elliot, 2007) as a screener for verbal abilities. Participants were included in the current study if their range of scores on the confidence interval (95%) for the Verbal Reasoning Composite included a standard score of 85. In addition to eligibility screening instruments, children and parents completed several tasks and questionnaires as part of the larger study. For the purposes of the current study, children completed the Tower of Hanoi-Revised (ToH-R; Welsh, Pennington, & Groisser, 1991). During this task, children were presented with two models that contain 3 equal sized, large plastic pegs. One model was used by the examiner and the other model was used by the child. The child was instructed to move the rings across the three different pegs to match the goal state modeled by the examiner's model. Children have to follow several specific rules while completing this task. Examiners explained the task and rules to children using an instructional story that describes monkeys jumping from tree to tree. The ToH-R task was videotaped for later scoring and coding. Task duration is approximately 3 to 10 minutes. If determined eligible following the enrollment visit, families were scheduled for a final study visit at Seattle Pacific University.

University visit. Families were seen in the Developmental Laboratory at Seattle Pacific University for their second study visit lasting approximately 90 to 120 minutes. Children completed a battery of tasks assessing self-regulation, attention, problem solving, and inhibitory control. Tasks were videotaped for later coding. Parents completed an interview regarding their child's social/emotional behavior in an adjacent room where they were able to observe their child through a video monitor. At the end of the university visit, parents received \$50 and a \$5 coffee card and children received a small toy and stickers. Teachers received \$25 for their participation in the study. **Measures**

Demographic information and maternal history of depression. Parental guardians provided child and family demographic information including child age, gender, and ethnicity as well as parent gender, age, level of education, and annual household income. Information regarding maternal history of depression was collected via one item on the demographic questionnaire form. Families were asked whether mothers had ever been diagnosed or treated for depression with the option to indicate "yes" or "no".

Verbal ability. The *Differential Ability Scale—Version II* (DAS-II; Elliott, 2007) was used to assess children's verbal abilities. The DAS-II is a comprehensive assessment of cognitive abilities for children ages 2:6 through 17:11. The current study used the core Verbal Ability Cluster including the Verbal Comprehension and Naming Vocabulary subtests from the Early Years cognitive battery for children 2:6-6:11. The Verbal Comprehension subtest assesses receptive language abilities and consists of 42 items. The Naming Vocabulary subtest assesses expressive language skills and consists of 34 items. Items are scored as a "1" for correct responses or "0" for incorrect responses and totaled into a raw score. The DAS-II raw scores are converted to produce an ability score (similar to a standard score), T score for each subtest, Verbal Cluster standard score,

percentile rank, and age equivalents. Internal reliability coefficients of the Verbal Ability Cluster for the Early Years Battery are adequate ranging from .86 to .83 (Elliott, 2007). The DAS-II also has good test-retest reliability for the Verbal Reasoning Cluster Early Years Battery, scores ranged from .87 for ages 3:6-4:11 to .90 for ages 5:0-8:11 (Elliott, 2007).

ASD Symptomology. Parents completed the Social Communication Questionnaire – Current Form (SCQ; Rutter et al., 2003), a screener for symptoms of ASD, to ensure that children in the typically development group do not exhibit significant levels of ASD symptoms. The SCQ was based on the Autism Diagnostic Interview-Revised (ADI-R; Rutter et al., 2003) and formerly known as the Autism Screening Questionnaire (Berument, Rutter, Lord, Pickles, & Bailey, 1999). It is a 40-item binary scaled instrument (i.e., 0 for "no" and 1 for "yes") assessing the three domains of ASD symptomology: reciprocal social interaction, communication, and repetitive and stereotyped behaviors over the last three months. Sample items include, "Does she/he ever get her/his pronouns mixed up?", "Does she/he smile back if someone smiles at her/him?", and "Does she/he play any pretend or make-believe games?" The four scales of the SCQ are Social Interaction, Communication, Abnormal Language, and Stereotyped Behavior with scores ranging from 0 to 40. Administration time is approximately 10 minutes. Originally designed for individuals 4 years and older, the SCQ has been validated with preschool aged children three years of age and older (Allen, Silove, Williams, & Hutchins, 2007). The authors recommend a cut off score of 15 and that children who meet or exceed this criteria are displaying significant ASD symptomology that warrants further evaluation. The SCQ's sensitivity is 85% and specificity is 75%

using a cutoff score of 15 (Berument et al., 1999). The SCQ has strong discriminant validity between ASD and non-ASD cases (Chandler et al., 2007) and between ASD and ADHD (Ghziuddin, Welch, Mohiuddin, Lagrou, & Ghaziuddin, 2010). Internal consistency for the SCQ is also high with alphas ranging from .84 to .93 (Rutter et al., 2003).

Child planning neuropsychological task. *The Tower of Hanoi-Revised* (ToH; Welsh, Pennington, & Groisser, 1991). Children completed the ToH-R which is a task designed to assess planning ability in children 2.5 to 9 years of age. Previous research indicates that the ToH-R also likely taps problem solving abilities in very young children (Senn, Espy & Kaufmann, 2004). Bull, Espy, and Senn (2004) examined the relation between tower tasks and short-term memory, inhibition, and shifting ability in a large sample of preschool-aged children (M age 4 years 9 months, SD = 6). They found that ToH-R performance was associated with the ability to shift between mental sets (i.e., cognitive flexibility). This task has been utilized to assess planning capacity in typically developing preschool and school aged children and children with developmental disabilities such as autism and ADHD (Bull, Espsy, & Senn, 2004; Drayer, 2009; Fisher & Happe, 2005; Ozonoff, Pennington, & Rogers, 1991). During this task children are presented with two models that contain 3 equal size colored plastic discs that fit over three yellow plastic Fisher Price ® Rock-n Stack. Pegs are spaced 10cm apart from each other on a wooden testing board that is 44cm x 17.5cm x 3cm. One model is used by the examiner and the other model is used by the child. The peg diameters are graduated ensuring that the rings may only be stacked from largest to smallest. The child is instructed to move the rings across the three different pegs to match the goal state

modeled by the examiner's model. The child must follow several specific rules while completing this task. The goal is to use as few moves as possible to achieve the goal configuration of rings on pegs. Two practice trials and six trials are presented that require 2 to 7 moves to solve the problem. Trials are discontinued when the solution is achieved or when children make 20 moves. If the child makes a rule violation, the examiner immediately corrects the violation and moves the ring back to the previous positions (i.e., position prior to rule violation). The examiner then verbally reminds the child about the rule that was violated. The task is discontinued after 2 consecutive trial failures, with failure occurring when children makes 20 moves without solving the problem, refuse to make any additional moves or when they failed to make any legal moves for a given trial. The task is also discontinued if the child is not able to pass either the first or second practice problem. The ToH-R demonstrated a test-retest reliability of .72 in five- year-old children after a 25 minute interval (Bull, Espy, & Senn, 2004). Testretest reliability was .53 in a sample of 7 to 10 year old children after an interval of 30 to 40 days (Bishop, Aamodt-Leaper, Creswell, McGurk, & Skuse, 2001). Currently, there are no published norms for the preschool version for the *Tower of Hanoi* test. Scoring systems for the ToH-R are variable across studies (Espy et al., 2004; Bull, Espy, & Senn, 2004; Senn et al., 2004). The current study utilized the ToH-R scoring procedures for preschool-aged children reported by Senn, Espy, and Kaufmann (2004).

Scoring the ToH-R task. Examiners scored the ToH-R during administration of the task. The ToH-R task was also videotaped and reliability checks of examiner scores were conducted separately by a graduate student and an undergraduate research assistant. Reliability was determined based on ten independent coded training ToH-R

administrations. The average intra-class correlation with Cronback's alpha for the initial training ToH-R administration was .92. The average intra-class correlation for selection of 25% of the remaining ToH-R administrations was .96 indicating high reliability. A global measure of performance was calculated for the ToH-R along with several descriptive measures of performance. For each trial passed, children received points corresponding with the number of moves required to provide the solution (e.g., 2 pts for a trial requiring 2 moves to solve). Children also received a 25% bonus for using the fewest amount of moves possible to solve the trial. Examiners recorded the number of rule violations per trial and duration in seconds to complete each trial to measure accuracy and processing speed. Rule violations do not contribute to the total number of moves for a trial. A global measure of ToH-R performance is calculated using the sum of points (including bonus points) earned for all trials. Descriptive measures of performance focused on accuracy and speed of responding were collected. Specifically, an index of accuracy was calculated using a ratio of the total number of rule violations divided by the number of trials attempted. An index of responding speed included time to first move divided by total number of trials attempted. A summary score across all completed trials is calculated for total number of moves (plus bonus points).

Parent measure of child planning and set-shifting. *Behavior Rating Inventory of Executive Function* (BRIEF, Gioia, Isquith, Guy & Kenworthy 2000) and *Behavior Rating Inventory of Executive Function-Preschool Version* (BRIEF-P, Gioia, Espy, & Isquith, 2003). Parents completed the BRIEF or BRIEF-P at the enrollment visits. The BRIEF and BRIEF-P are questionnaires that assess preschool and school age children's executive functioning abilities in everyday activities according to parent report. The BRIEF is designed for children ages 5:0 to 18:0 years and contains 8 subscales which assess various aspects of children's executive functioning skills at home. The BRIEF-P is designed for children ages 2:5 to 5:11 years of age and contains 5 subscales assessing various executive functioning domains. The current study utilized the Shift and Plan/Organize subscales of the BRIEF and BRIEF-P. The Shift subscale contains 10 questions and assesses children's ability to demonstrate cognitive flexibility, transition easily, problem solve flexibility, and change focus or switch attention. Sample items from the BRIEF Shift subscale include "Resists or has trouble accepting a different way to solve a problem with schoolwork, friends, chores, etc.", "Acts upset by a change in plans", and "Thinks too much about the same topic". Sample items from the BRIEF-P Shift subscale include "Has trouble changing activities", "Is upset by a change in plans or routine", and "Has trouble adjusting to new people (such as a babysitter, teacher, fried, or daycare worker)". The Plan/Organize subscale also contains 10 items and measure children's ability to anticipate events, sequence, and implement instructions to achieve a goal. Sample items from the BRIEF Plan/Organize subscale include "Forgets to hand in homework even when completed", "Gets caught up in details and misses the big picture", and "Has good ideas but does not get job done (lacks follow through)". Sample items from the BRIEF-P Plan/Organize subscale include "When instructed to clean up, puts things away in a disorganized, random way", "Cannot find things in room or play area even when given specific instructions", and "Gets caught up in the small details of a task or situation and misses the main picture". The BRIEF evidences high internal consistency for the Shift subscale (.88) and Plan/Organize subscale (.91) (Gioia et al., 2000). The BRIEF-P also demonstrates high internal consistency for these two subscales

(Shift = .85, Plan/Organize = .80) (Gioia, Espy, & Esquith, 2003). Positive Correlations with the BASC-2, CONNERS, and the CBLC indicated good discriminant and convergent validity for the BRIEF (Gioia et al., 2000) and BRIEF-P (Gioia, Espy, & Esquith, 2003). The BRIEF demonstrated a test-retest reliability of .72 for the Shift subscale and .80 for the Plan/Organize subscale in the normative sample. The BRIEF-P demonstrated a test-retest reliability of .78 for the Plan/Organize subscale and .78 for the Plan/Organize subscale in the normative sample.

Internalizing symptoms. Behavior Assessment System for Children, Version 2 – Preschool and Elementary Versions, Parent Rating Scales and Teacher Rating Scales (BASC-2-PRS; BASC-2-TRS; Reynolds & Kamphaus, 2004). The BASC-2-PRS and BASC-2-TRS were used by the current study to assess internalizing symptoms. The parent rating scales and teacher rating scales included a preschool version (children ages 2:0-5:11) and an elementary version (children ages 6:00-11:11). The preschool version contains 134 questions and the elementary version contains 160 questions. Items are scored on a 4-pt Likert scale ranging from 0 (never) to 3 (almost always). Administration time is approximately 10 to 20 minutes for all versions. The BASC-2 questionnaires yield composite scores of externalizing problems, internalizing symptoms, behavior symptoms index, and adaptive skills. The current study utilized the Internalizing symptoms Composite and the clinical scales that make up this composite: Anxiety, Depression, and Somatization scale. Higher scores on the Internalizing symptoms composite and subscales indicate a higher frequency of internalizing symptoms. The Anxiety scale assesses generalized fears, anxiety and nervousness that is atypical. The Depression scale assesses for symptoms of depression such as anhedonia, depressed

mood, hopelessness, and pessimism. The Somatization scale assesses physical complaints associated with psychological difficulties. Sample items from the Internalizing Composite include "Says 'Nobody likes me", "Is sad", "Cries easily", "Complains about health", "afraid to make a mistake", "Is fearful", "Worries about what other children think", and "Is negative about things". BASC-2 T scores are calculated for each clinical scale and composite with scores 60 or higher considered *at risk* and scores 70 or higher considered *clinically significant*.

The BASC-2 Teacher Rating Scales evidence high internal consistency in the general norm group for composite scores (coefficient alphas range = .87-.97) and individuals scales (coefficient alphas range = 75-.95) (Reynolds & Kamphaus, 2004). Reliability coefficients in the clinical norm sample also indicate high internal consistency with alpha coefficients comparable to general norm group statistics (Reynolds & Kamphaus, 2004). The BASC-2 Parent Rating scales also demonstrate high internal consistency in the general norm group and clinical norm group for composite scores (coefficient alphas range = .85-.95) and individual scales (coefficient alphas range = .70-.90). The BASC-2 Teacher and Parent Rating Scales demonstrate good test-retest reliability (i.e., average alpha coefficient for subscales exceed .80; Reynolds & Kamphaus, 2004) and validity (Doyle, Ostrander, Skare, Crosby, & August, 1997).

CHAPTER III

Results

Power Analysis

An a priori analysis was conducted using G*Power software (Faul, Erdfelder, Buchner, & Lang, 2009) to calculate the sample size necessary for adequate power for the current analyses. I controlled for verbal mental age based on previous research demonstrating an association between verbal IQ, age, and executive functioning skills and internalizing symptoms in children with ASD (Hill; 2004; Kim et al., 2000; Mayes et al., 2011; Mazurek & Kanne 2010; Sukhodolsky et al., 2008). Three variables were entered as predictors in the power analysis: children's verbal mental age, children's developmental status, and children's executive functioning performance. Results indicated that 68 participants were needed to detect a conservative Cohen's F^2 effect size of .15 with a power equal to .80 and alpha level set at .05.

Data Entry

Data were entered using the Statistical Package for the Social Sciences (SPSS) Version 23 software and data was cross checked for accuracy. Developmental status was entered into SPSS and dummy coded (0 = typically developing, 1 = ASD). Child gender was entered and coded as 1 for female and 2 for male. Maternal history of depression was entered into SPSS and dummy coded (1 = "no" indicating no history, 2 = 'yes' indicating positive history). Parent and teacher ratings from the BASC-2-TRS and BASC-2-PRS were utilized as measures of child internalizing symptoms. The BASC-2 item raw scores, total scores, scaled scores, and percentiles were entered for the Internalizing symptoms composite, Anxiety scale, Depression scale, and Somatization scale. Parent ratings on the BRIEF-P and BRIEF were used to measure child executive functioning skills in real world settings. The item raw scores, total scores, scale scores, and percentiles were entered for the BRIEF and BRIEF-P Plan/Organize scale and the Shift scale. ToH-R summary scores were entered into SPSS including the total score (i.e., total number of points per trial plus bonus points for all trials passed) and descriptive performance scores (i.e., index of accuracy and speed to responding) as continuous variables. DAS-II Verbal Reasoning Cluster standard score, percentile, and child age in months and years were entered as continuous variables. Verbal mental age was also entered into SPSS as a continuous variable.

Data Screening Prior to Analysis

Data were screened for missing data, outliers, and examined for parametric multiple regression assumptions prior to analyses. All participants completed the DAS-II measure of verbal ability and parent report BRIEF questionnaire assessing executive functioning. One participant (1.5%). was missing parent report on the BASC-2. Six participants (9.1%) were missing teacher report on the BASC-2. Thirteen participants (19.7%) were missing data on history of maternal depression due to delayed inclusion of this measure in the study battery. Four participants (6.1%) were missing ToH-R performance scores including descriptive measure of accuracy and time to first move. Three participants were missing ToH-R data due to participant refusal or failure to complete any moves on the task and one participant's ToH-R scores were removed from data set due to major clinician administration error on review. Missing data can result in loss of statistical power, introduce bias, and limit generalizability of results given inappropriate management. Analysis of missing values, indicated that data were missing completely at random (MCAR) according to non-significant results on Littles MCAR Test, χ^2 (34) =27.05, p = .796. Current recommendations for addressing missing data include newer statistical techniques with fewer disadvantages than traditional techniques such as pairwise deletion or mean substitution. Given current recommendations (Rezvan & Simpson, 2015; Treiman, 2009) and to maximize statistical power, the current study utilized multiple imputation (MI) to address missing data. Multiple imputation, originally proposed by Rubin (1987), replaces missing data with a series of simulated values based on the observed data set and creates multiple data sets of imputated data that are analyzed independently. The original data set was imputated five times using SPSS-Version 20. Analysis of descriptions measures across imputations did not indicate significant variability. Therefore, the first imputated data set was used to complete all subsequent analyses based on current recommendations (personal communication Dr. Dana Kendall, October, 2015). Data were examined for outliers using histogram and boxplots. Several notable outliers were identified for internalizing scores on the BASC-2 parent and teacher report measures. A significant outlier was also identified for verbal ability on the DAS-II. All identified outliers were examined for accuracy and interpreted as clinically significant scores of internalizing symptoms and verbal ability consistent with population sampled.

Given the presence of a categorical predictor variable (i.e., developmental status), normality was assessed for all continuous study variables within each group (i.e., ASD and TD groups) separately based on recommendations from Field (2013). To assess normality the Kolmogorov-Smirnov test (K-S test) was conducted and interpreted in conjunction with histograms, Q-Q plots, and the values of skew and kurtosis. Results indicated four variables in the TD group and three variables within the ASD group failed tests of normality (see results in Table 2 for TD group and Table 3 for ASD group). Homogeneity of variance was examined using Levene's test. Variances were significantly unequal between groups (ASD vs. TD) for two variables: BRIEF Plan subscale standard scores, F(1,64) = 8.27, p = .005 and Tower of Hanoi global scores, F(1,64) = 7.90, p = .007. All other study variables yielded non-significant values for Levene's test. Data were also screened for multicollinearity by examining bivariate correlation matrices (see Table 5). Variables were considered to be multicollinear if they were highly correlated with r > .08 based on recommendations from Field (2013). The correlation between verbal mental age and chronological age was found to be multicollinear (r = .88). However, this was expected given the verbal mental age variable is created by combining estimates of chronological age and verbal ability. Following the recommendation of Field (2013) and Aquinis (2004), analyses utilized bootstrapping as a method that is robust to outliers and violations of assumptions and preferable to data transformations given increased likelihood of Type II errors with this approach.

Data analytic plan

Data were analyzed using the Statistical Package for the Social Sciences (SPSS) Version 23. Initial analyses included means, standard deviations, and ranges for all study variables. Correlation analyses were conducted between demographic, independent, mediator and dependent variables. Subsequent analyses controlled for variables significantly correlated with predictor or dependent variables. The primary hypothesis proposed a mediation model (see Figure 2). Mediational analyses examined whether there was an indirect effect between the independent variable (X) and the dependent variable (Y) through the mediator variable (M). Figure 2 illustrates a simple mediation

Variable	ble K-S Test of Normality			Kurtos	is	Skewness		
-	D	Df	Р	Kurtosis	z kurtosis	skewness	z skewness	
Age	.125	40	.118	-1.208	-1.65	0.087	0.233	
VMA	.119	40	.160	-0.923	-1.259	0.081	0.217	
DAS-II VA	.127	40	.100	1.522	2.076*	0.837	2.238*	
ToH-R	.196	40	.010	-1.550	-2.114*	-0.213	-0.569	
Shift SS	.162	40	.010	2.564	3.498*	1.500	4.011*	
Plan SS	.110	40	.200	0.294	0.401	0.043	0.115	
PR INTERN	.088	40	.200	0.283	0.386	0.522	1.396	
TR INTERN	.144	40	.035	0.203	0.277	0.899	2.403*	

Table 2Normality Among Continuous Variables in TD Group

Note: * indicates significant skewness or kurtosis. Age = chronological age, VMA = verbal mental age, DAS-II VA = Differential Abilities Scale, Version 2 Verbal Ability Standard Score, ToH-R = Tower of Hanoi-Revised Global Score, Plan SS = Behavior Rating Inventory of Executive Functioning- Preschool and School Age Version Plan Subscale Standard Score, Shift SS = Behavior Rating Inventory of Executive Functioning- Preschool and School Age Version Shift Subscale Standard Score. *D* is the K-S (Kolmogorov-Smirnov Test of Normality) test statistic. Z scores are calculated by dividing the respective standard error.

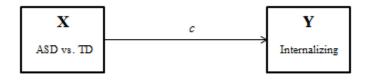
Normality Among	Continuous Va	ariables in ASD C	Group					
Variable	K-S Test of Normality			Kurtos	sis	Skewness		
	D	Df	Р	Kurtosis	z kurtosis	skewness	z skewness	
Age	.098	26	.200	-1.173	-1.322	0.125	0.274	
VMA	.073	26	.200	-0.420	-0.472	0.295	0.647	
DAS-II VA SS	.108	26	.200	-0.937	-1.056	-0.033	-0.072	
ToH-R	.216	26	.003	.290	.327	1.141	2.502*	
Shift SS	.129	26	.200	0.334	0.377	-0.438	-0.961	
Plan SS	.096	26	.200	-0.581	-0.655	-0.417	-0.915	
PR INTERN	.191	26	.015	1.748	1.971 *	1.305	2.861*	
TR INTERN	.250	26	.000	8.004	9.023*	2.432	5.667*	

Note: * indicates significant skewness or kurtosis. Age = chronological age, VMA = verbal mental age, DAS-II VA SS = Differential Abilities Scale, Version 2 Verbal Ability Standard Score, ToH-R = Tower of Hanoi-Revised Global Score, Plan SS = Behavior Rating Inventory of Executive Functioning- Preschool and School Age Version Plan Subscale Standard Score. *D* is the K-S (Kolmogorov-Smirnov Test of Normality) test statistic. Z scores are calculated by dividing the respective standard error.

Table 3

model utilizing the constructs of interest in the current study. Model paths are typically unstandardized regression coefficients. Path a in Figure 2B represents the effect of X on the proposed mediator M whereas path b represents the effect of M on Y accounting for the effect of X. The *total effect* of X on Y (path c) represented in *Figure 2* A can be divided into the *indirect effect* of X on Y through M and the *direct effect* of X on Y (path c'). The *indirect effect* of X on Y through M is a product of the a and b path regression coefficients (i.e., ab).

Panel A. Total effect path



Panel B. mediated effect path

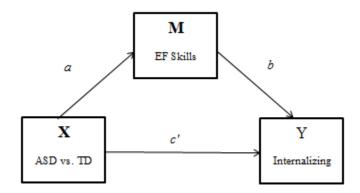


Figure 2. Mediation model of the association of developmental status on internalizing symptoms through exective functioning skills.

The current study utilized a non-parametric bootstrapping multivariate approach developed by Preacher and Hayes (2004; 2008) as a more statistically rigorous and powerful test of mediation hypotheses (Williams & McKinnon, 2008) that does not require assumptions of normality regarding the distribution of the sample distribution of the test statistic (*ab*) (Preacher & Hayes, 2004; 2008). This method can also be applied

with greater confidence to small samples because it is not based on large sample theory (Preacher & Hayes, 2004). The bootstrapping approach is a resampling technique that estimates the indirect effect by repeatedly sampling from the data set and calculating the indirect effect in each resampled data set. Inferences are made regarding the value of the indirect effect in the population using this sampling distribution and generated confidence intervals. The presence of a significant indirect effect is supported when the zero does not fall between the lower and upper bound confidence intervals. Hayes and Preacher's marco (2013), PROCESS, was used to examine a multiple mediational model in SPSS (Model 4). This method provides bootstrap estimates of path coefficients as specified in Baron and Kenny's method (1986) (a, b, c, c'), the indirect effect ab, an estimated standard error, and confidence intervals for the population value of *ab*. Based on the recommendations of Preacher and Hayes (2008), bootstrapping techniques for the current study utilized 5,000 resamples and 95% bias-corrected and accelerated confidence intervals for each analysis. Point estimates of the indirect effect are the mean of *ab* computed over the 5,000 resamples. All predictor variables were centered automatically by the PROCESS macro.

For all mediational analyses, developmental status (ASD vs. TD) was entered as the independent variable and verbal mental age calculated from the DAS-II was entered as a covariate. The study utilized one multiple mediation analysis to assess whether developmental status related to internalizing symptoms through executive functioning variables (ToH-R total score, BREIF/BRIEF-P Shift and Plan/Organize subscales) for both parent and teacher report of internalizing symptoms. Multiple linear regression analyses were used to test hypotheses regarding individual paths within the mediational model (Hypotheses 1-3), the association between maternal history of depression and internalizing symptoms, and the relation between EF performance based scores and BRIEF ratings per parent and teacher report.

Descriptive Analyses

Means, standard deviations, *t*-tests, and effect sizes are included for study variables in Table 4. There were no significant group differences for BASC-2 internalizing symptoms based on both parent and teacher report. Significant differences were found between groups for executive functioning skills. The ASD group had significantly higher scores on the BRIEF Shift and Plan subscales indicating greater difficulties with EF and significantly lower performance scores on the Tower of Hanoi-Revised.

The current study variable means and standard deviations were compared to available data with comparable samples. Smithson and colleagues (2013) collected BRIEF questionnaire parent report data for 44 children with ASD between the ages of 2.83-5.83 years of age (BREIF Shift M = 61.10, SD = 10.93; BRIEF Plan M = 61.10, SD = 11.84). Rosenthal and colleagues also collected BRIEF data via parent report for 34 children ASD between the ages of 5 and 7 year of age (BRIEF Shift M = 66.06, SD = 13.59; BRIEF Plan M = 62.62, SD = 14.03). The aforementioned samples evidenced lower means and comparable standard deviation values compared to BRIEF descriptive measures from the current study (See Table 4). However, like the current study scores were overall elevated with means falling in the "at risk" and "clinically significant" range. In terms of BASC-2 comparisons, Volker and colleagues (2010) collected BASC-2 data on 62 children between the ages of 6-16 years with ASD (BASC-2 Internalizing Problems M = 58.18, SD = 11.86) and 62 children with typical development (BASC-2

Internalizing Problems M = 48.10, SD = 9.19). Although this sample represented an older age cohort than the current study, means and standard deviations were comparable for both TD and ASD groups. Bradstreet and colleagues (2016) collected BASC-2 PRS-P questionnaires from 117 children diagnosed with ASD between the ages of 24 and 63 months and reported means and standard deviations on the BASC-2 Internalizing Problems by specific subtest (Anxiety M = 46.99, SD = 10.72; Depression M = 51.57, SD = 9.88; Somatization M = 49.08, SD = 9.10). These mean values were comparable to current study findings for the ASD group (Anxiety M = 55.90, SD = 17.11; Depression M = 58.50, SD = 14.86; Somatization M = 51.23, SD = 12.71) with slightly greater variability as indicated by larger standard deviations. A comparable sample of data for the Tower of Hanoi-Revised was collected by Senn, Epsy, and Kaufmann (2004) and included 117 preschool aged children ages 2 y ears 8 months to 6 years (All participants Mean = 13.66, SD = 8.78; Participants younger than 4 years Mean = 8.61, SD = 5.24; Participants older than age 4 years Mean = 16.93, SD = 9.09). These values were similar to those observed in the current study for children with TD (See Table 4). In regards to ASD samples, there is a lack of reported ToH-R descriptive statistics in previous studies or different ToH-R scoring procedures utilized limiting comparisons.

Correlation analyses were conducted between demographic, independent, mediator, and dependent variables (See Table 5). Several significant correlations were found between proposed mediators and outcomes including ToH-R with age, verbal ability and verbal mental age, BRIEF Shift with verbal ability, status, maternal history of depression, and BRIEF Plan, BRIEF Plan with verbal ability, status, maternal history of depression, and BRIEF Shift, parent report of internalizing symptoms with BRIEF Shift

Table 4

	Mean	s (SD)			
Variable	ASD (<i>n</i> = 26)	TD (n = 40)	t/X^2	d/\varPhi	
Shift-BRIEF	$\frac{(n-20)}{70.08}$	$\frac{(n-40)}{48.40}$	7.97***	1.99	
	(11.68)	(10.19)			
Plan-BRIEF	74.31	52.58	6.84***	2.26	
	(14.58)	(8.76)			
ТоН	9.71	19.08	-3.61**	.90	
	(9.36)	(11.58)			
Internalizing symptoms, PR	56.94	50.38	1.94	0.46	
	(17.11)	(10.41)			
Internalizing symptoms, TR	54.83	50.47	1.32	0.31	
	(17.20)	(9.41)			

Descriptive Statistics for Variables: Means, Standard Deviations, T-tests, Chi-Square and Effect Sizes by Group

Note: Shift-BRIEF = BRIEF shift subscale T scores, Plan-BRIEF = BRIEF Plan subscale T-scores, ToH = Tower of Hanoi Global Performance Score, Internalizing symptoms, PR = BASC-2 Internalizing composite scores parent report, Internalizing symptoms, TR = BASC-2 Internalizing composite scores teacher report. *p < .05, **p < .01, ***p < .001.

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Age											
2. VMA	.88**										
3. Verbal Ability	05	.42**									
4. Gender	.21	.04	29*								
5. Ethnicity	.05	02	14	05							
6. Status	.08	17	51**	.28*	.33**						
7. Mat Dep Hx	13	17	10	.13	.35**	.38**					
8. BRIEF-Shift	.18	05	46**	.13	.17	.71**	.36**				
9. BRIEF-Plan	.15	05	37**	.21	.24	.69**	.35*	.69**			
10. ToH-R	.54**	.66**	.33**	04	.03	40**	18	25*	24		
11. Internalizing, PR	05	15	19	.07	.11	.24	.20	.58**	.47**	09	
12. Internalizing, TR	.16	.02	22	.01	.10	.16	02	.22	.30*	01	.58**

Table 5Bivariate Correlations among Study Variables

N = 66; VMA = Verbal Mental Age; Verbal Ability = DAS-II Verbal Reasoning Cluster Standard Score; Status = Developmental Status (ASD vs. TD), Mat Dep Hx = History of Maternal Depression (dichotomous variable), BRIEF-Shift = BRIEF Shift T Score, BRIEF-Plan = BRIEF Plan T score, ToH-R = Tower of Hanoi Revised Global Performance score, Internalizing, PR = parent report from BASC-2; Internalizing, TR = teacher report from BASCS-2.

**p < .01, *p < .05.

and Plan, and lastly teacher report of internalizing symptoms with parent report of internalizing symptoms (See Table 5).

Hypothesis 1: Developmental status will predict children's internalizing symptomology. Two separate regressions analyses were conducted to examine the relation between developmental status and children's internalizing symptoms as measured by the BASC-2 Internalizing symptoms composite by parent and teacher report. Although trending towards significance, developmental status did not significantly predict parent reported internalizing symptoms, R^2 = .06, F(1,64) = 3.76, p =.068, 95% CIs [-.446, 13.63], f^2 = .06. Cohen's f2 statistic (Field, 2009) indicated a small effect size in the relation between developmental status and parent reported internalizing symptoms. For teacher report of BASC-2 Internalizing symptoms, developmental status also did not significantly predict internalizing behaviors, R^2 = .03, F(1,64) = 1.76, p = .248, 95% CIs [-2.28, 11.97], f^2 = .03, suggesting similar levels of internalizing symptoms per group.

Hypothesis 2: Developmental status will predict children's executive

functioning skills. Three hierarchical regression analyses were used to examine the relation between developmental status (ASD vs. TD) and executive functioning skills as assessed by three measures used in the current study: BRIEF Plan, BRIEF Shift, and ToH-R.

Tower of Hanoi-Revised. Children's ToH-R performance was regressed on developmental status, controlling for centered verbal mental age given a significant correlation with the outcome. Verbal mental age was entered on the first step followed by developmental status on the second step. Verbal mental age was a significant

predictor of ToH-R, accounting for 43% of the variance in ToH-R. Developmental status

significantly predicted ToH-R and accounted for an additional 9% of the variance in

ToH-R. See Table 6 for regression weights.

Table 6 *Hierarchical Regression: ToH-R Regressed on Verbal Mental Age and Developmental Status* (N = 66)

							95%	CI
Variable		R^2	F	В	SE	p	LL	UL
Model 1		.43	49.10***					
	VMA			5.72	.03	.001	4.45	6.99
Model 2		.52	33.79***					
	VMA			5.29	.03	.001	3.80	6.61
	Status			-6.93	10	.004	-11.34	-2.91

Note. *p < .05, **p < .01, ***p < .001. ToH-R = Tower of Hanoi Revised, VMA = verbal mental age, Status = developmental status, CI = 95% bias corrected and accelerated confidence intervals based on 1000 bootstrap samples; *LL* = lower limit, *UL* = upper limit.

Brief Shift. Shift was regressed on developmental status, controlling for centered verbal ability and history of maternal depression given significant correlations with the outcome. Verbal ability and maternal history of depression were entered on the first step followed by development status on the second step. Verbal ability and maternal history of depression were significant predictors of Shift, accounting for 31% of the variance. Developmental status was a significant predictor of Shift, accounting for an additional 22% of the variance. See Table 7 for regression weights.

BRIEF Plan. Plan was regressed on developmental status, controlling for centered verbal ability and history of maternal depression given significant correlations with the outcome. Verbal ability and maternal history of depression were entered on the first step followed by development status on the second step. Verbal Ability and maternal history of depression were significant predictors of Plan, accounting for 23% of

the variance. Developmental status was a significant predictor of Plan, accounting for an

additional 25% of the variance. See Table 8 for regression weights.

Table 7Hierarchical Regression: Shift Regressed on Verbal Ability, Maternal History of
Depression, and Developmental Status (N = 66)95% CI

							101	
Variable		R^2	F	В	SE	р	LL	UL
Model 1		.31	14.10***					
	VA			50	.11	.005	3.22	16.13
	MHD			9.73	3.23	.001	71	28
Model 2		.52	22.77***					
	VA			17	.11	.109	38	.04
	MHD			3.91	3.29	.251	-2.24	10.38
	Status			17.92	3.68	.001	10.34	24.93

Note. *p < .05, **p < .01, ***p < .001. Shift = Brief Shift subscale, VA = DAS-II verbal ability, Status = developmental status, MHD = Maternal history of depression (1 = yes, 0 = no), CI = 95% bias corrected and accelerated confidence intervals based on 1000 bootstrap samples; *LL* = lower limit, *UL* = upper limit.

Table 8

Hierarchical Regression: Plan Regressed on Verbal Ability, Maternal History of Depression, and Developmental Status (N = 66)

							95%	6 CI
Variable		R^2	F	В	SE	Р	LL	UL
Model 1		.23	9.54***					
	VA			40	.11	.001	62	16
	MHD			9.87	3.75	.017	2.34	17.10
Model 2		.48	19.30***					
	VA			04	.13	.738	26	.24
	MHD			3.40	3.26	.311	-2.93	9.65
	Status			19.90	4.06	.001	11.95	27.42

Note. *p < .05, **p < .01, ***p < .001. Plan = Brief Plan subscale, VA = DAS-II verbal ability, Status = developmental status, MHD = Maternal history of depression (1 = yes, 0 = no), CI = 95% bias corrected and accelerated confidence intervals based on 1000 bootstrap samples; *LL* = lower limit, *UL* = upper limit.

Hypothesis 3: Executive functioning will predict internalizing symptoms. In

order to examine the relation between executive functioning skills and children's internalizing symptoms, separate multiple regression analyses were conducted for the three measures of EF with both parent and teacher report of internalizing symptoms.

Continuous predictor variables were mean centered prior to analyses.

Parent report of internalizing symptoms. To examine whether executive functioning predicted parent report of children's internalizing symptoms, measures of EF was analyzed separately. Cognitive flexibility as measured by BRIEF Shift predicted children's internalizing symptoms and accounted for 33% of the variance within the model. See Table 9 for regression weights. Planning skills as measured by BRIEF Plan predicted children's internalizing symptoms and accounted for 22% of the variance within the model. See Table 9 for regression weights. Tower of Hanoi-Revised global scores did not significantly predict variance in children's internalizing symptoms, B = -.11, 95% CIs[-.33, .12], t = -.76, p = .452.

Table 9Hierarchical Regression: Parent BASC-2 Internalizing Symptoms Composite ScoresRegressed on BRIEF Shift and Plan

						95%	5 CI
Variable	R^2	F	В	SE	Р	LL	UP
Shift	.33	32.01	.52	.12	.001	.28	.76
Plan	.22	18.31	.42	.13	.001	.16	.66

Note. *p < .05, **p < .01, ***p < .001. Shift = BRIEF Shift subscale, Plan = BRIEF Plan subscale, CI = 95% bias corrected and accelerated confidence intervals based on 1000 bootstrap samples; *LL* = lower limit, *UL* =Upper limit.

Teacher report of internalizing symptoms. To examine whether different measures of executive functioning predicted teacher report of children's internalizing symptoms, each measure of EF was analyzed separately. Cognitive flexibility as measured by BRIEF Shift was not significantly related to the children's internalizing symptoms, F(1,64) = 3.13, p = .081, although there was a statistical trend toward significance. See Table 13 for regression weights. Planning skills as measured by BRIEF Plan predicted children's internalizing symptoms, F(1,64) = 6.25, p = .015, and

accounted for 9% of the variance within the model. See Table 10 for regression weights.

Tower of Hanoi-Revised global scores did not significantly predict variance in children's

internalizing symptoms, B = -.006, 95% Cls[-.23, .23], t = -.04, p = .965.

Table 10Hierarchical Regression: Teacher BASC-2 Internalizing Symptoms Composite ScoresRegressed on BRIEF Shift and Plan

					_	95% CI		
Variable	R^2	F	В	SE	Р	LL	UP	
Shift	.05	3.14	.19	.17	.310	13	.53	
Plan	.09	6.25	.25	.13	.058	.04	.53	

Note. *p < .05, **p < .01, ***p < .001. Shift = BRIEF Shift subscale, Plan = BRIEF Plan subscale, CI = 95% bias corrected and accelerated confidence intervals based on 1000 bootstrap samples; *LL* = lower limit, *UL* =Upper limit.

Hypothesis 4: Executive functioning skills will mediate the relation between developmental status and internalizing symptoms. A multiple mediation model was examined using a non-parametric bootstrapping multivariate approach developed by Preacher and Hayes (2004; 2008) and Hayes and Preacher's corresponding macro (2013), PROCESS. Based on the recommendations of Preacher and Hayes (2008), bootstrapping techniques for the current study utilized 5,000 resamples and 95% bias-corrected and accelerated confidence intervals for each analysis. All predictor variables were centered automatically by the PROCESS macro. Two separate multiple mediation analyses were conducted to examine this hypothesis including one with parent report of internalizing symptoms as the outcome and one with teacher report of internalizing symptoms as the outcome.

Parent report of internalizing symptoms. To examine whether developmental status is positively associated with parent report of internalizing symptoms through EF, a multiple-mediational model was conducted with ToH-R, Shift, and Plan as parallel mediators (Process Model 4). An integrated model with three mediators versus three

simple mediation models was chosen to allow multiple processes to operate simultaneously, compare the strength of specific indirect effects and increase power of analyses for tests of indirect effects given that all mediators are correlated with the outcome (Hayes, 2013). Verbal mental age and maternal history of depression were considered as covariates given significant correlations with both mediators and outcome in the model. When controlling for these variables in this analysis, no significant effects were found for maternal history of depression and omitting this covariate did not change the general pattern of results, thus analyses reported excluded this variable. Bootstrapping results (5,000 resamples and 95% bias-corrected and accelerated confidence intervals) supported our model ($R^2 = .48$, F(5,60) = 11.30, p < 0.001) and indicated the predictor variables jointly accounted for 48% of the variance in internalizing symptoms. As shown in Figure 3 and Table 11, results of the multiple mediation analysis revealed the total indirect effect of executive functioning on the association between developmental status and internalizing symptoms was significant as the bias and corrected and accelerated 95% confidence intervals did not include zero. Significant specific indirect effects were found for BRIEF Shift and Plan. No significant indirect effect was observed for ToH-R. Results indicated that BRIEF Plan and Shift are significant mediators of the association between developmental status and internalizing symptoms whereas ToH-R did not play a significant mediational role. Contrast analysis of significant indirect effects indicated no differences in magnitude such that the effect of BRIEF Shift was not statistically stronger than that of BRIEF Plan (contrast = 7.81, SE = 4.48, 95% CIs[-.06 to 17.63]). Based on Hayes' (2013) recommendation for analyses with dichotomous independent variables (i.e., developmental status), a partially

standardized effect size was used to evaluate the magnitude of indirect effects. The partially standardized effect size is interpreted as the number of standard deviations in the outcome that the two groups differ on average as a result of the indirect mechanism. The *total indirect partially standardized effect* = 1.43 (SE = .27, 95% CIs [.97, 2.05]), for *Shift indirect partially standardized effect* = 1.05, (SE = .24, 95% CIs [.63, 1.60]) and for *Plan indirect partially standardized effect* = .48 (SE = .16, 95% CIs [.17, .77]). Therefore, those in the ASD group received on average, internalizing symptoms scores that were 1.43 standard deviations or approximately 19 points higher compared to the typically developing group as a result of the *total indirect effect* through executive functioning skills. The results indicate a substantial indirect effect.

As show in Figure 3, the *total effect* of developmental status on internalizing symptoms (*path c*) was not significant (p = .0847) whereas the *direct effect* of developmental status on internalizing symptoms (*path c'*) was significant (p = .002). The *total effect* represents the unstandardized slope of the regression of Y on X whereas the *direct effect* represents the unstandardized slope of the regression of Y on X after controlling for the mediators. Hayes (2012) and Rucker, Preacher, Tormala, and Petty (2011) demonstrate that significant indirect effects can occur in the absence of a significant total or indirect effect. Given the change in magnitude and significance of *total* and *direct effects*, suppression effects were considered. Suppressor variables increase the predictive validity of another variable when included in the regression equation (MacKinnon et al., 2000). Furthermore, Rucker and colleagues (2011) define a suppressor variable as "one that undermines the *total effect* by its omission, meaning that accounting for it in a regression equation enhances the predictive utility of the other

variables in the equation. Evidence of suppression is found when including an intervening variable produces a value of c' (direct effect) that is greater in magnitude than c (total effect)" (Rucker et al., 2011, p. 366). Identical analytic methods are utilized to conduct tests of mediation versus tests of suppression (Mackinnon et al., 2000). However the difference in these effects can be seen in the relation between the *indirect effect* and the *total effect* with suppression occurring when the *indirect effect* has a sign that is opposite that of the *total effect* and mediation occurring with the *indirect* and *total effect* have the same sign (Rucker et al., 2011). Suppression and mediation effects can also occur in tandem. The results of the current study support a mediating effect of Plan and Shift variables due to matching sign (positive) with *total effect* as well as a suppressing effect of verbal mental age which has a sign opposite the *total effect* (negative) and serves as significant predictor of internalizing symptoms (b = -2.94, SE = 1.28, t = 2.30, p = .025, 95% CIs [-5.50, -.384]) in the regression equation including all mediators.

Table 11

Summary of Indirect Effects from Multiple Mediation Analysis Developmental Status on Parent Report of Internalizing Symptoms through Executive Functioning Skills Controlling for Verbal Mental Age

			_	BCa 95% CI		
	Point Estimate	SE	Ζ	Lower	Upper	
Indirect Effects						
Total	19.50	4.95		11.78	32.87	
Shift	14.31	4.19	4.22***	7.85	24.74	
Plan	6.50	2.24	2.27*	2.43	11.18	
ToH-R	-1.31	1.23	-1.09	-4.94	.35	

Note: Shift = Brief Shift Subscale, Plan = BRIEF Plan Subscale, ToH-R = Tower of Hanoi-Revised, BCa 95% CI = bias corrected and accelerated 95% confidence intervals. $p^* < .05$, $p^{**} < .01$, $p^{***} < .001$.

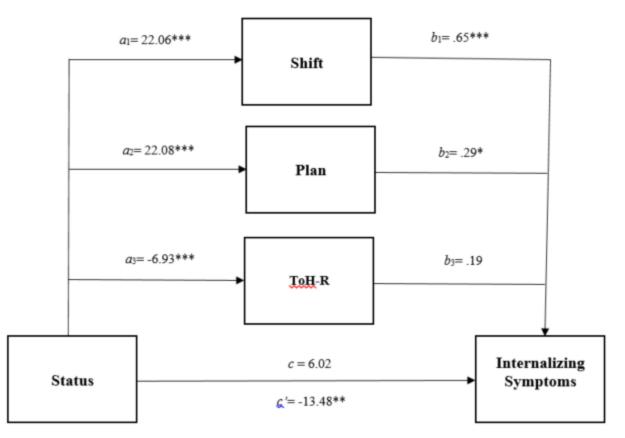


Figure 3. Multiple mediation model depicting relations between developmental status, executive functioning skills, and parent report of internalizing symptoms displaying unstandardized regression coefficients. Shift = Brief Shift Subscale, Plan = BRIEF Plan Subscale, ToH-R = Tower of Hanoi, c = total effect of developmental status on internalizing symptoms, c' = direct effect of developmental status on internalizing symptoms. $p^* < .05$, $p^{**} < .01$, $p^{***} < .001$

Teacher Report of Internalizing Symptoms. A multiple mediation model with

ToH-R, Shift, and Plan as parallel mediators (Process Model 4) was conducted to examine whether developmental status is positively associated with teacher report of internalizing symptoms through EF. Verbal mental age and maternal history of depression were considered as covariates given significant correlations with both mediators and outcome in the model. When controlling for these variables in this analysis, no significant effects were found for maternal history of depression and omitting this covariate did not change the general pattern of results, thus analyses reported excluded this variable. As shown in Figure 4 and Table 12, a significant specific indirect effect was found for BRIEF Plan. No significant indirect effects were observed for BRIEF Shift or ToH-R with teacher report of internalizing symptoms as the outcome. Results indicated that BRIEF Plan was a significant mediator of the association between developmental status and teacher report of internalizing symptoms whereas BRIEF Shift and ToH-R did not play a significant mediational role. The *Plan indirect partially* standardized effect = .46, (SE = .18, 95% CIs [0.08, 0.79]). Therefore, those in the ASD group received on average, internalizing symptoms scores that were .46 standard deviations or approximately 6 points higher on internalizing symptoms scale compared to the typically developing group as a result of the indirect effect through planning skills. The results indicate a moderate indirect effect. As shown in Figure 4, the total effect of developmental status on internalizing symptoms (*path c*) was not significant (p = .1787) and the *direct effect* of developmental status on internalizing symptoms (*path c'*) was also not significant (p = .6778). These results did not support presence of suppression suggested in previous analyses of parent report of internalizing symptoms given that the magnitude of c is greater than c' as expected with a mediational process.

Table 12

				BCa 95% CI		
	Point Estimate	SE	Ζ	Lower	Upper	
Indirect Effects						
Total	6.76	5.64		88	21.94	
Shift	1.31	4.84	.36	-5.94	13.54	
Plan	6.01	2.47	1.70	1.61	11.57	
ToH-R	56	1.35	38	-3.65	1.98	

Summary of Indirect Effects from Multiple Mediation Analysis Developmental Status on Teacher Report of Internalizing Symptoms through Executive Functioning Skills Controlling for Verbal Mental Age

Note: Shift = Brief Shift Subscale, Plan = BRIEF Plan Subscale, ToH-R = Tower of Hanoi-Revised, BCa 95% CI = bias corrected and accelerated 95% confidence intervals. $p^* < .05$, $p^{**} < .01$, $p^{***} < .001$.

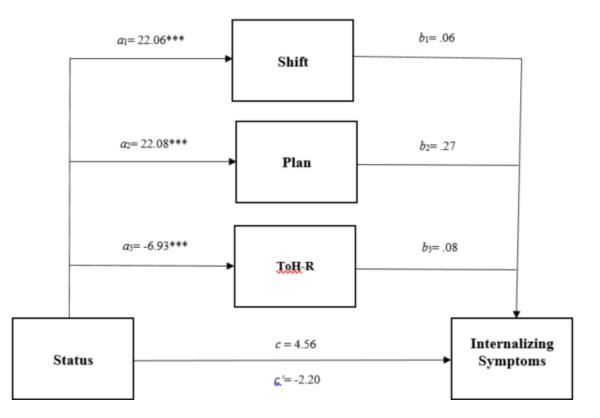


Figure 4. Multiple mediation model depicting relations between developmental status, executive functioning skills, and teacher report of internalizing symptoms displaying unstandardized regression coefficients. Shift = Brief Shift Subscale, Plan = BRIEF Plan Subscale, ToH-R = Tower of Hanoi, c = total effect of developmental status on internalizing symptoms, c' = direct effect of developmental status on internalizing symptoms. $p^* < .05$, $p^{**} < .01$, $p^{***} < .001$

Hypothesis 5: Performance measures of executive functioning will correlate

with rating scales of executive functioning. The fifth hypothesis that performance on the neuropsychological task (ToH-R) will be significantly related to planning and set shifting abilities on the behavior rating scale of executive functions completed by parents was examined using regression analyses. High scores on the BRIEF indicate greater problems with EF and low scores on the ToH-R indicate greater difficulties with EF. Therefore, it was hypothesized that the relation between BRIEF and ToH-R scores would be negative. The ToH-R total score was regressed on BRIEF Shift and Plan/Organize subscales separately with verbal mental age as covariate. BRIEF Shift evidenced a significant negative relation with ToH-R, B = -.17, 95% CIs[-.30, -.06], t = -2.41, p = .019. There was also a significant negative relation between Brief Plan and ToH-R, B = -.15, 95% CIs[-.27, -.02], p = .032.

Post-hoc Analyses

Results of the analyses indicated the performance-based measure of EF, ToH-R, did not predict parent or teacher report of internalizing symptoms or mediate the relation between developmental status and internalizing symptoms. To further explore the relation between developmental status, ToH-R, and possible relations with specific components of internalizing symptoms, namely the BASC-2 Anxiety scale, Depression scale, and Somatization scale that comprise the Internalizing symptoms Composite, a series of mediational analyses were conducted using Hayes and Preacher's macro (2013) and bootstrapping techniques with 5,000 resamples and 95% bias-corrected and accelerated confidence intervals for each analysis. Three separate mediational analyses were completed to examine whether developmental status is positively associated with parent report of anxiety, depression, or somatization symptoms through ToH-R, controlling for verbal mental age. Results did not support a significant indirect effect of ToH-R with parent report of anxiety (point estimate = -1.31, SE = 1.23, 95% CIs [-4.61, .44]), depression (point estimate = -1.24, SE = 1.30, 95% CIs [-4.53, .76]), or somatization (point estimate = -.12, SE = 1.11, 95% CIs [-2.49, 2.02]) as outcomes. Using BASC-2 teacher report, results did not support a significant indirect effect of ToH-R with teacher report of anxiety (point estimate = .22, SE = 1.44, 95% CIs [-2.49, 3.48]) or depression (point estimate = -.03, SE = 1.42, 95% CIs [-2.51, 3.37]) as outcomes. A

significant indirect effect was found for ToH-R with Somatization problems (point estimate = -1.66, SE = 1.06, 95% CIs [-4.6, -.20]), See Figure 5 for results. Both the *total effect* (t = .02, p = .9820) and the *direct effect* (t = .58, p = .56) were non-significant. Results indicated a small indirect effect size (*specific indirect partially standardized effect* = -.16, SE = .09, 95% CIs[-.38, -.01]) with the ASD group on average receiving internalizing symptoms scores that were .16 standard deviations or approximately 1.5 points higher on internalizing symptoms scale compared to the typically developing group as a result of the indirect effect through planning skills.

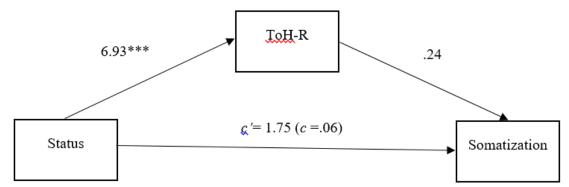


Figure 5. A mediational model of the association between developmental status and BASC-2 Teacher reported somatization problems via children's performance on the Tower of Hanoi-Revised (ToH-R). Standardized aggression coefficients from a bootstrap procedure are provided along the paths. c = total effect of developmental status on somatization symptoms, c' = direct effect of developmental status on somatization symptoms.* p<0.05, ** p<0.01, ***p<0.001.

To further explore relations between developmental status, children's

performance on the ToH-R, and internalizing symptoms additional analyses with the descriptive performance variables were examined in relation to these constructs. The two ToH-R performance descriptive measures include an index of accuracy calculated using a ratio of the total number of rule violations divided by the number of trials attempted and an index of responding speed calculated as a ratio of the sum of participants' time to first move across trials divided by attempted trials. First, the relation between descriptive

measures including the index of accuracy and speed of responding with global performance scores on the ToH-R was examined. Bivariate correlations indicated a negative relation between ToH-R global score and the accuracy index (r = -.44, p < .001) suggesting that as the ratio of rule violations to total number of trials attempted increases, global performance on the ToH-R decreases. The correlation between speed of responding and ToH-R global score also indicated a negative relation (r = -.29, p = .019) in which ToH-R global performance scores increased as speed of responding via time to the first move decreased. To explore whether ToH-R descriptive measures predict parent and teacher report of internalizing symptoms, I conducted two hierarchical regression analyses with centered verbal mental age entered in step one as a control variable and centered ToH-R accuracy index and ToH-R speed of responding index entered on step two. With parent report of internalizing symptoms as the outcome, results indicated that the overall the model was not significant, $R^2 = .03$, F(2,62) = .67, p = .575. With teacher report of internalizing symptoms as the outcome, results also indicated the overall model was not significant, $R^2 = .03$, F(2,62) = .58, p = .628. Overall these findings do not support a significant association between descriptive measures of ToH-R performance, specifically indexes of accuracy and speed of responding, and either parent or teacher reported internalizing symptoms.

Maternal history of depression, executive functioning skills, and internalizing symptoms. Given significant group differences in maternal history of depression and significant correlations between maternal history of depression and developmental status and executive functioning variables (BRIEF Shift & Plan), exploratory analyses examined whether an indirect effect exists between maternal history of depression and internalizing symptoms through executive functioning skills. These exploratory analyses were informed by recent investigations with typically developing children demonstrating maternal depression predicts children's executive functioning skills (Hughes, Roman, Hart, & Ensor, 2013) and an indirect effect of the maternal depression on internalizing symptoms through EF in young typically developing children (Roman, Ensor, & Hughes, 2016). Two separate multiple mediation analyses was conducted using a non-parametric bootstrapping multivariate approach (Preacher & Hayes; 2004; 2008) and Hayes and Preacher's corresponding macro (2013), PROCESS (Model 4); one for parent report of internalizing symptoms and one for teacher report of internalizing symptoms as the outcome controlling for verbal mental age.

Parent report of internalizing symptoms as outcome. As shown in Figure 6 and Table 13, a significant specific indirect effect was found for Shift but not for Plan or ToH-R. Results indicate that maternal history of depression is associated with depression and anxiety symptoms through Shift but not Plan or ToH-R. The *indirect partially standardized effect* for Shift = .39 (SE = .17, 95% CIs [.10, .80]). Therefore, subjects with a positive history of maternal depression received on average, internalizing symptoms scores via parent report that were .39 standard deviations or approximately 5.34 points higher compared to participants without a history of maternal depression as a result result of the *specific indirect effect* through cognitive flexibility (Shift) suggesting a small to moderate effect size.

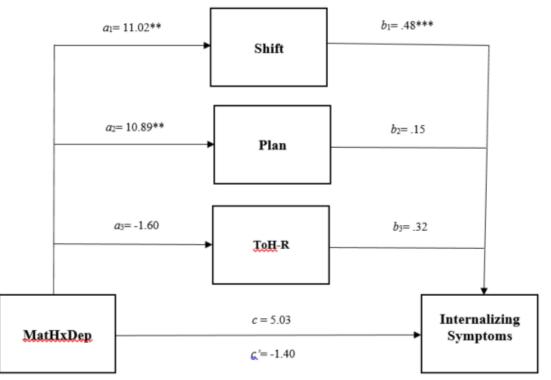


Figure 6. Multiple mediation model depicting relations between maternal history of depression, executive functioning skills, and parent reported internalizing symptoms displaying unstandardized regression coefficients. MatHxDep = Maternal history of depression, Shift = Brief Shift Subscale, Plan = BRIEF Plan Subscale, ToH-R = Tower of Hanoi, Internalizing Symptoms = Parent report of BASC-2 Internalizing symptoms, c = total effect of developmental status on internalizing symptoms. $p^* < .05$, $p^{**} < .01$, $p^{***} < .001$

Table 13

Summary of Indirect Effects from Multiple Mediation Analysis Maternal History of Depression on Parent Report of Internalizing Symptoms through Executive Functioning Skills Controlling for Verbal Mental Age

				BCa 95% CI	
	Point Estimate	SE	Ζ	Lower	Upper
Indirect Effects					
Total	6.44	2.98		1.53	13.42
Shift	5.31	2.68	2.31	1.42	12.57
Plan	1.64	1.47	1.07	46	5.65
ToH-R	51	.82	60	-3.00	.58

Note: Shift = Brief Shift Subscale, Plan = BRIEF Plan Subscale, ToH-R = Tower of Hanoi, BCa 95% CI = bias and corrected and accelerated 95% confidence intervals. Unstandardized regression coefficients are shown. $p^* < .05$, $p^{**} < .01$, $p^{***} < .001$.

Teacher report of internalizing symptoms as outcome. As shown in Figure 7 and Table 14, a significant specific indirect effect was found for Plan but not for Shift or ToH-R. Results indicated that Plan evidences a significant mediational role in the relation between maternal history of depression and internalizing problem but Shift and ToH-R do not demonstrate a significant indirect effect. The *indirect partially standardized effect* for Plan = .22 (SE = .12, 95% CIs [.05, .54]). Therefore, subjects with a positive history of maternal depression received on average, internalizing symptoms scores via teacher report that were .22 standard deviations or approximately 2.88 points higher compared to participants without a history of maternal depression as a result of the *specific indirect effect* through planning skills suggesting a small effect size.

Table 14

Summary of Indirect Effects from Multiple Mediation Analysis Maternal History of Depression on Teacher Report of Internalizing Symptoms through Executive Functioning Skills Controlling for Verbal Mental Age

			_	BCa 95% CI		
	Point Estimate	SE	Ζ	Lower	Upper	
Indirect Effects						
Total	3.46	2.65		66	10.16	
Shift	.68	2.19	.39	-2.99	6.36	
Plan	2.95	1.52	1.52*	.79	7.14	
ToH-R	17	.57	30	-2.55	.37	

Note: Shift = Brief Shift Subscale, Plan = BRIEF Plan Subscale, ToH-R = Tower of Hanoi, BCa 95% CI = bias and corrected and accelerated 95% confidence intervals. Unstandardized regression coefficients are shown. $p^* < .05$, $p^{**} < .01$, $p^{***} < .001$.

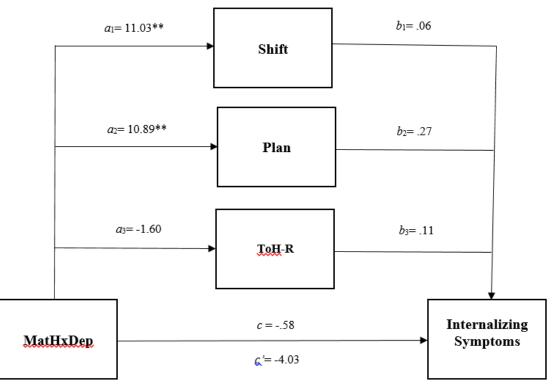


Figure 7. Multiple mediation model depicting relations between maternal history of depression, executive functioning skills, and teacher reported internalizing symptoms displaying unstandardized regression coefficients. MatHxDep = Maternal history of depression, Shift = Brief Shift Subscale, Plan = BRIEF Plan Subscale, ToH-R = Tower of Hanoi, Internalizing Symptoms = Teacher report of BASC-2 Internalizing symptoms, c = total effect of developm11.03ental status on internalizing symptoms, c' = direct effect of developmental status on internalizing symptoms. $p^* < .05$, $p^{**} < .01$, $p^{***} < .001$

Maternal history of depression, executive functioning skills, internalizing

symptoms, and developmental status. Given the results of post hoc analyses demonstrating a significant indirect effect between maternal history of depression and parent and teacher report of internalizing symptoms and components of EF (Shift & Plan), additional exploratory analyses investigated whether the magnitude of these specific indirect effects varied by developmental status. I hypothesized that the relation between maternal history of depression symptoms and internalizing symptoms through executive functioning would vary based on developmental status, specifically that developmental status would moderate the b path (relation between EF and internalizing symptoms). Several studies support the moderation component of this model hypothesizing increased strength of association between EF and internalizing symptoms (beta pathway) for children with ASD (Hollocks et al., 2014; Kelly et al., 2012; Lawson et al., 2015) versus TD.

A conditional indirect effects analysis was conducted using Preacher and Hayes' bootstrapping multivariate approach (2004; 2008) and Hayes and Preacher's corresponding SPSS macro (2013), PROCESS (Model 14). Two separate conditional indirect effects analyses were conducted based on post hoc results; one with Shift as mediator and parent report of internalizing symptoms as the outcome and one with Plan as mediator and teacher report of internalizing symptoms as the outcome. Verbal mental age was controlled for in both analyses. Results of this analysis with parent report as outcome and Shift as mediator are presented in Table 15. Analysis of the alpha pathway indicated that maternal history of depression significantly predicted cognitive flexibility (Shift) with 13% of variance in cognitive flexibility accounted for by this model. Analysis of the beta pathway indicated cognitive flexibility (Shift) significant predicted internalizing symptoms. The interaction effect of developmental status on the relation between Shift and internalizing symptoms was also statistically significant. The overall conditional indirect effect model was significant (p < .001) with predictors, interaction term, and covariates accounting for approximately 47% of the variance in parent reported internalizing symptoms.

Further examination of the conditional indirect effects at each level of the moderator revealed that both effects were statistically significant for the ASD and TD groups and the index of moderated mediation indicated equality of the conditional indirect effects in the two groups (Index = 6.28, SE = 4.32, 95% BaCIs [.29, 17.91]).

Therefore, a significant mediation of approximately equivalent strength is present for

both children with ASD and TD which does not support the presence of a conditional

indirect effect.

Table 15

Conditional Direct and Indirect Effects of Maternal History of Depression and Executive Functioning on Parent Reported Internalizing symptoms at Values of Developmental Status as a Moderator

		Mediator Variable Model				
Predictor	В	SE	Т	p		
Constant	40.49	9.32	4.34	.0001		
MHDep	11.03	3.62	3.04	.0034		
VMA (covariate)	.15	1.34	.11	.91		
		Dependent Variable Model				
Predictor	В	SE	Т	р		
Constant	35.10	10.01	3.51	.0009		
MHDep	1.76	2.90	.61	.0045		
Shift	.50	.17	2.95	.5474		
Status	-45.83	15.73	-2.91	.0050		
Shift X Status	.5	.25	2.28	.0260		
VMA (covariate)	-2.13	1.00	-2.12	.0378		
Direct Effect of Maternal History of Depression on Internalizing symptoms						
	В	SE	Т	Р		
	1.76	2.90	.61	.5474		
Conditional Indirect Effe symptoms at Values of the		l History of D	epression on Inte	ernalizing		
Moderator Value	В	Boot SE	Lower CI	Upper CI		
TD	5.49	2.58	1.50	11.97		
ASD	11.78	5.44	3.34	25.04		

Note. N = 66; CI = 95% confidence interval for indirect effect; if CI does not include zero indirect effect is considered statistically significant. MHDep = maternal history of depression. Shift = BRIEF Shift subscale. Status = Developmental Status (ASD =1, TD = 0).

The significant interaction effect between developmental status and Shift on

internalizing symptoms was examined further to better understand this relation.

Graphical representation of the interaction is presented in Figure 8 and suggests a

positive relation between Shift and internalizing symptoms for both ASD and TD groups,

whereby higher levels of Shift (indicating greater problems with cognitive flexibility) are associated with increased internalizing symptoms. Evaluation of simple slopes indicated the ASD group evidenced a larger slope (1.07, p < .01) than the TD group (0.50, p < .01) indicating the relation between Shift and internalizing symptoms changes more rapidly for children with ASD. To further aide interpretation, the interaction effect was also graphed with developmental status as the independent variable and Shift as the moderator (See Figure 9).

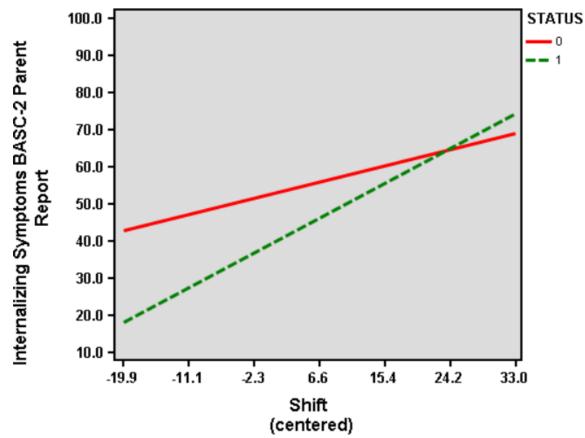


Figure 8. Interaction between centered Shift and developmental status controlling for verbal mental age and maternal history of depression predicting parent reported internalizing behaviors for conditional indirect effects model.

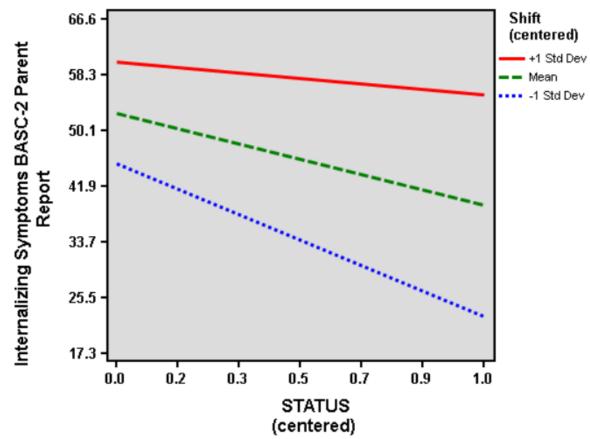


Figure 9. Interaction between centered Shift as moderator and developmental status controlling for verbal mental age and maternal history of depression predicting parent reported internalizing behaviors for conditional indirect effects model.

Evaluation of simple slopes with Shift as the moderator indicated that at mean Shift values the negative slope between developmental status (0 = TD, 1 = ASD) and internalizing symptoms was significantly different from zero (t = -3.38, SE = 3.96, p < .01) suggesting that ASD status is associated with lower internalizing symptoms at this level of the moderator. Evaluation of simple slopes at +1SD for Shift (t = -0.29, SE = 16.39, p =.39) and -1SD for Shift (t = -1.48, SE = 14.87, p =.07) were not significantly different from zero, although the p value for simple slope of -1SD was trending towards significance. These findings suggest that when levels of Shift are at the mean or -1SD below which indicates decreased difficulties with cognitive flexibility, ASD status (versus TD status) is associated with lower internalizing symptoms. Therefore, ratings of cognitive flexibility that are at the mean or below appear to have a greater buffering effect on internalizing symptoms for the ASD group versus the TD group. However, findings and interpretation are limited by unequal distribution of cases within categories of Shift scores. The majority of participants evidenced scores in the mean range (ASD = 13, TD = 26) while Shift scores 1+SD or higher were dominated by the ASD group (ASD = 12, TD =2) and Shift scores -1SD or lower were dominated by TD cases (ASD = 1, TD = 12).

Results of analysis with teacher report of internalizing symptoms as the outcome are presented in Table 16. Analysis of the alpha pathway indicated that maternal history of depression significantly predicted planning skills with 12% of variance in planning skills accounted for by this model. Analysis of the beta pathway indicated planning skills were not a significant predictor of internalizing symptoms. The interaction effect of developmental status on the relation between Plan and internalizing symptoms was not significant (p = .8958) and the overall conditional indirect effect model was not significant (p = .2125). Overall, conditional indirect effect model for teacher report of internalizing symptoms as the outcome was not supported.

Summary of Results

The current study found support for several main hypotheses and demonstrated important findings for future consideration and exploration. ASD status was found to predict all executive functioning variables in the study including measures of cognitive flexibility and planning skills on the BRIEF and performance on the ToH-R. The relation between developmental status and EF was negative with ASD status predicted increased

Table 16

Conditional Direct and Indirect Effects of Maternal History of Depression and Executive Functioning on Teacher Reported Internalizing symptoms at Values of Developmental Status as a Moderator

		Mediator Variable Model				
Predictor	В	SE	t	р		
Constant	45.40	9.64	4.71	.0000		
MHDep	10.89	3.74	2.91	.005		
VMA (covariate)	.05	1.39	.03	.97		
		Dependent Variable Model				
Predictor	В	SE	t	р		
Constant	40.87	14.95	2.73	.0082		
MHDep	-3.61	3.51	-1.03	.31		
Plan	.27	.24	1.14	.26		
Status	-5.55	18.62	30	.77		
Shift X Status	.07	.30	.25	.81		
VMA (covariate)	.01	1.23	.01	.99		
Direct Effect of Maternal History of Depression on Internalizing symptoms						
	В	SE	Т	Р		
	-3.61	3.51	-1.03	.31		
Conditional Indirect Effects of Maternal History of Depression on Internalizing symptoms at Values of the Moderator						
<u>v 1</u>		D (CE	1 01			
Moderator Value	B	Boot SE	Lower CI	Upper CI		
TD	2.92	2.03	.28	8.55		
ASD	3.72	2.79	.30	12.38		

Note. N = 66; CI = 95% confidence interval for indirect effect; if CI does not include zero indirect effect is considered statistically significant. MHDep = maternal history of depression. Plan = Plan Shift subscale. Status = Developmental Status (ASD =1, TD = 0).

EF difficulties. Significant group differences were also found for EF variables with the

ASD group scoring on average significantly lower than the TD group. BRIEF Plan and

Shift were also significant predictors of parent and teacher reported of internalizing

symptoms and Plan was a significant predictor of teacher reported internalizing

symptoms. Shift demonstrated a trend towards significance as a predictor of teacher

report of internalizing symptoms. Status was not significantly associated with parent and

teacher reported internalizing symptoms via regression analyses but significant indirect

effects were identified.

Support was found for several significant indirect effect models examined during the current study. Specifically, an indirect effect was found between developmental status and parent report of internalizing symptoms through Shift and Plan with a substantial indirect effect size. With teacher report as the outcome, Plan demonstrated a significant indirect effect between developmental status and internalizing symptoms. Maternal history of depression was significantly related to parent report of internalizing symptoms through Shift and significantly related to teacher report of internalizing symptoms through planning skills.

Given the results of post hoc analyses demonstrating that components of EF (Shift & Plan) play an indirect role in the relation between maternal history of depression and parent and teacher report of internalizing symptoms, additional exploratory analyses investigated whether the magnitude of these specific indirect effects varied by developmental status. A significant interaction effect was found for developmental status on the relation between Shift and parent report of internalizing symptoms indicating that ratings of cognitive flexibility that are at the mean or below appear to have a greater buffering effect on internalizing symptoms for the ASD group versus the TD group. Graphical representation of the interaction effect indicated a positive relation for both groups whereby, increased difficulties in Shift were associated with higher levels of internalizing symptoms. The indirect effect at both levels of the moderator were significant suggesting that the overall indirect effect model remains significant with similar magnitudes regardless of group status.

Global performance on the ToH-R and descriptive measures (indexes of speed of responding and accuracy) did not evidence a significant direct or indirect effect with

105

internalizing symptoms. However, exploratory analyses evaluating specific components of the BASC-2 internalizing symptoms composite (i.e., anxiety, depression, and somatization subscales) did reveal a significant relation between developmental status and teacher report of somatization problems through ToH-R global performance although indirect effect size was small. Measures of EF including ToH-R, Shift, and Plan were significant correlated with each other.

CHAPTER IV

Discussion

The current study investigated the relations between developmental status (ASD vs. typically developing), two domains of executive functioning skills (cognitive flexibility and planning) and internalizing symptoms. These constructs were examined with preschool and early school aged children with autism spectrum disorder and average to high verbal skills and typically developing peers using both a performance-based measure of EF and rating scale assessing everyday manifestations of EF. The primary hypothesis was that an indirect link exists between developmental status and internalizing symptoms whereby ASD will be associated with higher rates of internalizing symptoms through deficits with executive functioning skills in the areas of planning and cognitive flexibility. In the following sections, I will review results of analyses involving primary study hypotheses, interpret and summarize findings, explore clinical implications of the results, discuss strengths and limitations associated with the current study, and new directions for future inquiries.

Interpretation of Results

Developmental status and internalizing symptoms. The hypothesis that children with ASD would exhibit significantly higher levels of internalizing symptoms than typically developing peers was not supported in the current study based on results of group differences and regression analyses although parent reported internalizing symptoms showed a trend towards significance (p = .068). This null finding is inconsistent with previous research demonstrating youth with ASD evidence increased levels of anxiety and depression symptoms and disorders compared to typically developing peers in clinical and community samples (Kim et al. 2000, MacNeil et al.,

2009; Russell & Sofronoff, 2005; Solomon, 201; Suhkodolsky et al., 2008). Although increased rates of internalizing symptoms have been reported in samples of preschool and young school aged children with ASD (Mayes et al., 2011), the majority of studies showing a discrepancy in prevalence rates have focused on older school aged children and adolescents. Recent studies showing increased rates of internalizing symptoms on the BASC-2 in youth with ASD have included samples of children between ages 8 and 18 years of age (Goldin, Matson, Konst, & Adams, 2014; Solomon et al., 2012). Given the well-established positive association between age and increased internalizing symptoms, the early age range of the current study sample (i.e., 3 years to 6 years, 11 months) may have obscured group differences due to delayed onset of more significant anxiety and depressive symptoms. In addition, many studies in this area have explored differences in clinical populations with elevated internalizing symptoms and diagnoses. The current study explored relations involving anxiety and depressive symptoms versus clinical levels of psychopathology. Specifically, only a few participants in each group received scores in the clinically significant range for internalizing symptoms per parent report (TD = 2, ASD = 5) and teacher report (TD = 2, ASD = 3) on the BASC-2. Posthoc power analyses with G*Power software (Paul et al., 2009) were conducted based on observed effect size for parent and teacher report of BASC-2. For parent report, one predictor variable (developmental status) was entered, with an alpha level set at .05 or less, a Cohen's $f^2 = 0.06$, and sample size of 22. Post-hoc computed power for parent report was 0.50. For teacher report, one predictor variable (developmental status) was entered, with an alpha level of .05 or less, a Cohen's $f^2 = .03$, and a sample size of 66. Post-doc computed power for teacher report of internalizing symptoms as the outcome

was 0.28. Therefore, the current study analyses were underpowered based on the sampling criteria for sufficient power with a small effect size.

Developmental status and executive functioning skills. The hypothesis that developmental status would predict executive functioning skills was supported. Children with ASD evidenced greater difficulties with EF skills in the areas of planning and set shifting after controlling for verbal mental age and maternal history of depression compared to TD peers. Group differences were evident for both the performance-based tasks of EF (ToH-R) and real world parent ratings of everyday EF skills (BRIEF). Developmental status accounted for a significant proportion of the variance in ratings on the BRIEF Shift scale (22%), BRIEF Plan scale (23%), and performance on the Tower of Hanoi-Revised (9%). These findings are congruent with a robust body of research reporting significant EF deficits in individuals with ASD of varying ages and levels of cognitive functioning (Gioia, Isquith, Kenworthy, & Barton, 2002; Hill, 2004; Kenworthy, Yerys, Anthony, & Wallace, 2008; Pennington & Ozonoff, 1996). Results also support previous research suggesting primary EF impairments in cognitive flexibility and planning for individuals with ASD (Hill, 2004; Yers, Wallace, Jankowski, Bollich, & Kenworthy, 2011) and neuroscience research demonstrating abnormal development of prefrontal areas involved in executive control including the dorsolateral prefrontal cortex in individuals with ASD (Courchesne, et al., 2011; McAlonan et al., 2009; Morgon et al., 2010; Zikopoulos & Barbas, 2010). Given history of mixed results in the area of EF dysfunction for preschool and early school aged children with ASD, the current study provides additional evidence that differences in EF abilities in children with ASD compared to typically developing peers are present and detectable during early childhood.

Executive functioning and internalizing symptoms. The hypothesis that executive functioning skills would significantly predict parent and teacher report of internalizing symptoms was partially supported. Specifically, measures of everyday EF functioning captured by parent report on the BRIEF suggested cognitive flexibility and planning skills were positively associated with internalizing symptoms such that as difficulties with EF increase, internalizing symptoms increased as well. BRIEF scores accounted for a significant proportion of the variance in internalizing symptoms including 33% for Shift and 22% for Plan based on parent report and 9% for Plan based on teacher report. The relation between cognitive flexibility and teacher reported internalizing symptoms showed a trend towards significance (p = .081). Performance scores on the Tower of Hanoi-Revised did not predict internalizing symptoms. This pattern of findings is contrary to previous studies displaying a connection between EF and internalizing symptoms using performance based tasks (Ciairano et al., 2007; Kusche, Cook, & Greenberg, 1993; Nigg et al., 1999; Riggs et al., 2003; Rinksy & Hinshaw, 2011) but consistent with research showing only a significant association between informant ratings of EF and internalizing symptoms (Jarratt et al, 2005). It is possible that ecologically valid assessments of EF such as the BRIEF are more significantly related to internalizing symptoms because everyday manifestations of EF deficits create significant challenges in navigating daily life and adapting to the environment which likely causes more distress and imparts greater vulnerability to feelings of demoralization, lack of success, and depression and anxiety symptoms (Boyd, McBee, Holtzclasw, Baranek, & Bodfish, 2009; Drayer, 2009). Deficits in EF are also likely to interfere with the application of effective coping skills to manage distress.

Further evaluation of the ToH-R pattern of scores indicated results may have been obscured by group effects. Specifically, ToH-R scores in the typically developing group evidenced greater range and variability (TD Range = 34.75, SD = 11.58, variance = 134.10) compared to the ASD group (ASD Range = 31.75, SD = 9.36, variance = 87.64). This observation is supported by Levene's test indicating unequal variance between groups (ASD vs. TD) on the ToH-R global scores. Visual inspection of the data showed typically developing children's ToH-R scores generally increased with age while children with ASD had scores that clustered on the lower end of the ToH-R scoring range regardless of age. This pattern of ToH-R scores for the TD group is congruent with typical developmental progression of lower order EFs (working memory, inhibition) followed by higher order EFs (planning, set shifting) across preschool to school age (De Luca & Leventer, 2008) and previous research showing greater difficulty on the ToH-R task measuring working memory, set shifting, and planning skills in preschool aged children followed by increased mastery in early school aged children (Bull, Espy, & Senn, 2004).

Developmental status, executive functioning, and internalizing symptoms. Support was found for a mediational model demonstrating a significant relation between developmental status and internalizing symptoms through executive functioning. Two separate multiple mediation analyses including three measures of EF (Shift, Plan, ToH-R) were conducted to examine this hypothesis including one with parent report of internalizing symptoms as the outcome and one with teacher report of internalizing symptoms as the outcome. For parent report as the outcome, bootstrapping results indicated the predictor variables jointly accounted for 48% of the variance in internalizing symptoms with significant specific indirect effects found for Shift and Plan but not for ToH-R performance scores. Exploration of significant findings indicated the presence of substantial indirect effects (*partially standardized total indirect effect* = 1.43) suggesting that the ASD group received on average, internalizing symptoms scores that were 1.43 standard deviations or approximately 19 points higher compared to the typically developing group as a result of the *total indirect effect* through executive functioning skills. With teacher report as the outcome, a significant specific indirect effect was found for BRIEF Plan. No significant indirect effects were observed for BRIEF Shift and ToH-R with teacher report as the outcome. Results indicated a moderate indirect effect size (specific indirect partially standardized effect = .46) with the ASD group on average receiving internalizing symptoms scores that were .46 standard deviations or approximately 6 points higher on internalizing symptoms scale compared to the typically developing group as a result of the indirect effect through planning skills. These results are congruent with research linking ASD and prefrontal pathology in the dIPFC with anxiety and depressive symptoms and disorders (Biver et al., 1994; Davidson, 2002; Koenigs & Grafman, 2009; Price & Drevets, 2010) and suggests that everyday manifestations of EF may be more meaningful in terms of impact on psychological functioning than performance based measures of EF which are collected in highly controlled settings. This research replicates Lawson and colleagues (2016) recent study demonstrating a mediational role of EF specifically cognitive flexibility between developmental status and internalizing symptoms in school aged children using the BRIEF as a measure of everyday EF. The current findings extend previous research by exploring this association in younger children. In addition, findings from the current

study demonstrate that planning skills as measured by the BRIEF also evidence an important role in explaining the higher rates of internalizing symptoms seen in children with ASD.

Lack of support for findings related to ToH-R, are curious given Hollocks and colleagues (2014) findings that performance-based EF measures evidence a significant association with anxiety symptoms in youth with ASD. However, this study was conducted with older youth with ASD specifically adolescents indicating that these effects may increase in significance with EF maturity and increasing internalizing symptoms with age. Additionally, previous research suggests performance on highly structured neuropsychological tasks of EF with individual with ASD may show decreased variability and deficits versus more open-ended EF tests in which no explicit instructions are given on how to accomplish the task (Van Eylen et al., 2015; White, Burgess, & Hill, 2009).

Executive functioning performance-based task and rating scale. Support was found for the hypothesis that performance on the ToH-R would be positively related to planning and set shifting abilities on the parental behavior rating scale of EF. BRIEF Shift evidenced a significant relation with ToH-R and accounted for 5% unique variance. Plan was also a significant predictor of ToH-R accounting for approximately 4% of unique variance in this measure. These results are consistent with previous work demonstrating significant moderate correlations with performance based measures of executive functioning in children (Bishop, 2011; Collins, 2012; Oberg & Lukomski, 2011).

Exploratory Analyses

Exploratory analyses were conducted to further assess discrepancies with previous research and investigate unanticipated findings. Further evaluation of the relations between developmental status, ToH-R including descriptive measures (index of accuracy and speed of responding), and internalizing symptoms was conducted. Examination of mediational models with ToH-R by specific internalizing symptom domains including anxiety, depression, and somatization largely did not support significant direct or indirect effects for either teacher or parent report with one exception. Support for an indirect effect between developmental status and somatization symptoms through executive functioning skills as measured by ToH-R was found but effect size was small. These results suggest that EF abilities as measured by a performance based assessment of planning and set shifting skills may be uniquely associated with physical manifestations of psychological distress suggesting that broader regulatory difficulties may underlie this association. This explanation is consistent with the young age of the sample who show greater variability in basic self-regulatory abilities than older children.

Maternal history of depression, executive functioning skills, and internalizing symptoms. Exploratory analyses examined the relations between maternal history of depression and other variables in the current study due to significantly higher frequency of reported maternal history of depression in ASD group and correlations with executive functioning variables. These exploratory analyses were also informed by a large body of research supporting the association between maternal depression and internalizing symptoms in TD youth (Goodman, 2007) and recent investigations demonstrating maternal depression predicted children's executive functioning skills (Hughes, Roman,

Hart, & Ensor, 201) and EF mediated the relation between maternal depression and internalizing symptoms in young children (Roman, Ensor, & Hughes, 2016). Support for various aspects of an indirect effect model were found for both parent and teacher report of internalizing symptoms. Specifically, results indicated an indirect for Shift, but not Plan or ToH-R, in the relation between maternal history of depression and parent report of internalizing symptoms and suggested a modest effect size. Findings also supported an indirect effect of Plan but not Shift or ToH-R in the relation between maternal history of depression and teacher report of internalizing symptoms. The effect size of Plan's indirect relation between maternal history of depression and internalizing symptoms also evidenced a modest effect size. The current study observed a pattern of results suggesting stronger associations between cognitive flexibility (Shift) and parent report of internalizing symptoms and planning skills (Plan) with teacher report of internalizing symptoms. These findings suggest that parents and teacher ratings of anxiety and depression symptoms are associated with difference EF skill deficits. Parent ratings of poor planning skills are associated with teacher ratings of higher internalizing symptoms. In the classroom, deficits in the ability to plan and organize behavior may relate more closely to what teachers observe and rate as internalizing symptoms or overall psychological distress. These findings support previous research with typically developing children demonstrating maternal depression predicts children's executive functioning skills (Hughes, Roman, Hart, & Ensor, 201) and an indirect effect of EF on the relation between maternal depression and internalizing symptoms in young children (Roman, Ensor, & Hughes, 2016) and extends this finding to include children with ASD.

Maternal history of depression, executive functioning skills, internalizing symptoms, and developmental status. Given the results of post hoc analyses demonstrating an indirect effect of maternal history of depression on internalizing symptoms through components of EF (Shift and Plan), additional exploratory analyses investigated whether the magnitude of these specific indirect effects varied by developmental status. Two separate conditional indirect effect models with parent and teacher report of internalizing symptoms as the outcome did not support a moderated mediation model. The conditional indirect effects at both levels of the moderater (ASD and TD) were significant suggesting that the overall indirect effects model remains significant with similar magnitude regardless of group. However, analyses involving the indirect effect of Shift on the relation between maternal history of depression and parent report of internalizing symptoms indicated a significant moderation effect of developmental stats on the b path between executive functioning skills and internalizing symptoms. Graphical representation of the interaction indicated both groups evidenced a positive relation whereby, higher levels of deficits in cognitive flexibility (Shift scores) were associated with increased internalizing symptoms. The simple slope value for the ASD group (1.07) was approximately twice as large as the value for the TD group (0.50)indicating that the positive relation between shift and internalizing symptoms changed more rapidly for children with ASD. Further evaluation of this relation through graphical representation with Shift as the moderator indicating that ratings of cognitive flexibility that are at the mean or below appear to have a greater buffering effect on internalizing symptoms for the ASD group versus the TD group. However, as mentioned above findings and interpretation are limited by unequal distribution of cases with the majority

of cases demonstrating Shift scores +1SD or above the mean coming from the ASD group and the majority of participants with very low Shift scores (-1SD or below) coming from the TD group.

There was no support for a conditional indirect effect model involving maternal history of depression symptoms, Plan, and teacher report of internalizing symptoms with developmental status as moderator of the b path. However, the current study with an *n* of 66 was underpowered for identifying these types of models (Preacher, Rucker & Hayes, 2007) which usually require hundreds of participants for adequate power. Therefore, continued research and accumulation of larger samples is needed to further explore moderators of the mediation models identified during this research (Preacher & Hayes, 2013).

Clinical Implications

These findings have a multitude of important clinical implications. Children with ASD exhibit elevated rates of anxiety and depression symptoms and disorders and increased prevalence of internalizing symptoms are associated with a range of maladaptive outcomes such as increased oppositional behavior, aggression (Kim et al., 2000), irritability, hypersensitivity (Sukhodolsky, 2008), exacerbation of core symptoms of ASD (Ghaziuddin, Ghaziuddin, & Greden, 2002; Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006), greater functional impairment (Chang, Quan, & Wood, 2012), lower life satisfaction, and greater social difficulties later life (Gotham, Brunwasser, & Lord, 2015). Internalizing symptoms increase with age and even early symptoms of subthreshold clinical severity may jeopardize learning and lead to poorer outcomes. Research exploring risk factors and possible mechanisms underlying increased risk of internalizing

symptoms in children with ASD is strikingly limited particularly in regards to neurocognitive factors. Early interventions aimed to improve emotional outcomes with a focus on reduction of internalizing symptoms are lacking (Bayer et al, 2009). The current study found support for a substantial indirect effect of executive functioning skills in the relation between ASD and increased anxiety and depression symptomology. Additionally, these results were identified in a sample of young children with ASD during a period of development when the prefrontal cortex and EF skills are rapidly developing and children may be more susceptible to intervention (Mezzacappa, 2004; Noble et al., 2005).

Support for an indirect effect of EF suggests targeting these skills may be important for addressing internalizing symptoms. These findings provide preliminary support for future investigations examining EF as a potential avenue for reducing internalizing symptoms and interventions targeting cognitive flexibility and planning skills in children with ASD to improve symptoms of anxiety and depression as well as other maladaptive outcomes associated with internalizing symptoms. The identified significant interaction effect of developmental status and Shift on parent reported internalizing symptoms suggests that mean or lower levels of Shift (cognitive flexibility) may have a greater buffering effect on internalizing symptoms for the ASD group versus the TD group. Research on interventions targeting EF in youth with ASD is limited particularly for preschool and early school aged children (Stichter et al., 2012). Cannon and colleagues (2011) developed a CBT based intervention targeting EF skills for school aged children titled, "Unstuck and on Target! (UOT) An executive function curriculum to improve flexibility for children with autism spectrum disorder". This intervention (UOT) is administered at both school and home and teaches children with ASD about flexibility. goal setting, and planning and how to use self-regulatory scripts and implement automatic cognitive and behavioral flexibility routines when faced with stressors or unexpected events. UOT utilizes "supported cognition" approaches in which new skills are taught with extensive scaffolding and practice with fading supports and consistency of supports across settings (e.g., school and home) with the goal of addressing difficulties with learning related to EF impairments in ASD. A recent randomized controlled effectiveness trial comparing UOT with social skills intervention in third to fifth grade children with ASD found that UOT was associated with significantly greater improvements in problem solving and EF (Kenworthy et al., 2014). Despite promising EF intervention findings in older children, research on treatments to promote EF in younger children is greatly needed. Researchers have explored school based programs for younger children designed to build self-regulatory skills, social-emotional development, and promote learning and achievement such as Tools of the Mind (Bodrova & Leong, 2007) and PATHS (Kusché & Greenberg, 1994) but these programs have not been applied to the ASD population or modified to support ASD specific learning styles. This study supports the need for future research exploring interventions targeting EF in younger children with ASD.

Improvement in EF skills may also help children better apply coping skills and strategies learned through traditional therapeutic interventions for anxiety and depression symptoms. The current findings suggest possible benefits of integrating nterventions specifically targeting cognitive flexibility and planning skills with evidence based approaches for the treatment of internalizing symptoms such as cognitive behavioral therapy in youth with ASD. Targeting EF and anxiety and depression symptoms in tandem may help children better acquire and access new coping skills in the moment given improved EF capabilities. Further assessment of EF skills by clinicians treating children with anxiety or depression may be warranted based on the results of this study with a focus on how identified deficits manifest in everyday life.

This research extends our understanding of the relation between maternal depression, EF, and internalizing symptoms in children with ASD and suggests that relations between these constructs are similar to those observed in children with TD. Given that maternal history of depression related to higher levels of EF difficulties and internalizing symptoms and previous literature demonstrating increased risk for psychological distress associated with parenting a child with ASD, it appears particularly important to identify and support mothers of children with ASD with a current or past history of depression or anxiety.

Strengths and Limitations

A range of strengths and limitations exist for the current study. A major asset of this study includes its extension of previous research to younger children. The current study adds to the limited literature exploring neurocognitive correlates of internalizing symptoms in youth with ASD (Hollocks et al., 2014; Lawson et al., 2015) by exploring these associations during early childhood. This is a particularly important given the opportunity for early intervention and prevention of downstream negative impacts of EF difficulties and internalizing symptoms. The current study also contributed to previous research by utilizing multiple measures specifically the utilization of both a performance based measure of EF (ToH-R) and ecologically valid measure of everyday EF

manifestations (BRIEF) and ratings of internalizing symptoms by multiple informants. Reports from both parents and teachers regarding internalizing symptoms allowed for examination of these symptoms within two difference contexts. The young age of the current sample provided an opportunity to study EF and internalizing symptoms during a critical period of development for these constructs. No study has examined the relation between developmental status, multiple domains of EF, internalizing symptoms as well as maternal history of depression in preschool and early school aged children with ASD. Therefore, the current study contributes to this field of research by providing preliminary findings and guiding future empirical endeavors.

Despite these strengths, several limitations are noted. One of the most significant limitations includes the cross sectional nature of this research which severely limits causal inferences regarding relations between variables and leaves the possibility that alternate models may be consistent with the results. Additionally the small sample size including only 26 participants with ASD (40 with TD) was a significant limitation of the current study and associated with insufficient power for statistical analyses. Specifically a post-hoc power analysis for the main effect of developmental status on internalizing problems indicated power of .50 for parent report as outcome and power of .28 for teacher report as outcome, whereas a sample of 133 (parent report) and 264 (teacher report) would have a power of .80. Therefore, my ability to detect anticipated relations between main study constructs was limited. The current investigation may be subject to Type II error in which significant effects are not identified due to low power. Additional limitations included restriction in range of internalizing symptoms with few participants exhibiting clinically significant levels of internalizing symptomology which limits

extensions of the current research to populations with diagnoses of anxiety and depressive disorders. In addition, the current study utilized a broad screener for internalizing symptoms (BASC-2) versus more comprehensive and specialized measures assessing only internalizing symptoms. Participants in the current study came from largely high socioeconomic status families which limits generalization of findings to other populations. Another major drawback of the current investigation relates to the measurement of maternal history of depression. Collection of data regarding maternal history of depression lacked information about time course, severity of maternal depression symptoms or disorders, and current depression symptoms. Therefore, I am unable to fully evaluate the nature of the association between maternal history of depression, child EF, and internalizing symptoms due to insufficient information regarding child exposure and severity and duration of maternal depression symptoms.

Conclusions and Future Directions

Elevated levels of anxiety and depression symptoms in youth with ASD is recognized as a significant co-occurring difficulty in children and adolescents with ASD yet research examining early predictors, risk factors, and correlates is very limited. While evidence for EF deficits in ASD are robust, neurocognitive factors associated with internalizing symptoms have not been widely explored. This was the first study to examine executive functioning specifically cognitive flexibility and planning skills as possible neurocognitive correlates underlying the association between ASD and internalizing symptoms in young children with both ASD and TD. Results encourage increased productivity and investigation in this area. This was also the first study to explore relations between history of maternal depression, EF, and internalizing symptoms in children with ASD. Important findings were identified for primary models including a substantial indirect effect of EF in the relations between developmental status and internalizing symptoms and maternal history of depression and internalizing symptoms which has significant clinical implications for this population. These findings support research suggesting that neuropathology of brain regions largely involved in the modulation of executive functions, including the dorsolateral prefrontal cortex (dIPFC), are associated with mood and anxiety disorders (Biver et al., 1994; Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Koenigs & Grafman, 2009; Price & Drevets, 2010, 2012) and extends this work to the ASD population. Overall, results suggest an indirect relation between cognitive flexibility and planning skills and internalizing symptoms and support future research exploring these relations to determine whether EF based interventions may reduce and/or prevent anxiety and depression symptomology in youth with ASD. Further longitudinal and experimental research including randomized controlled trials targeting EF deficits are needed to assess a possible causal mediation model of these relations (Shadish et al., 2002).

Many questions and areas of future investigation are warranted. As mentioned above, large sale and longitudinal and experimental research examining the associations among developmental status, EF, and internalizing symptoms is warranted to assist with interpretation of findings and understand the mechanisms of increased risk for anxiety and depression in children with ASD. Additionally, one can anticipate larger models involving the current variables including additional moderators of the indirect effect than evaluated in the current study. Larger scale studies involving substantial sample sizes are needed to further disentangle additional variables involved in the model with sufficient power. Additional research in the area of construct discrimination given overlapping ASD and internalizing symptoms and validation of traditional measures of internalizing symptoms for children with ASD is necessary to support future inquires examining internalizing symptoms in this population. Some research with traditional measures of anxiety applied to children with ASD show promise (Renno & Wood, 2014; White et al., 2013) but no exploration of measurement issues for co-occurring depression symptoms exist.

Additional research is needed to explore whether other traits and correlates of child internalizing symptoms found in typically developing populations apply to children with ASD. In particular, the current study did not explore how ADHD diagnoses or symptomology relate to the presentation or incidence of internalizing symptomology in children with ASD. Given high rates of comorbidity between ASD and ADHD and the possibility that ADHD symptoms may impact treatment response to interventions designed to reduce internalizing symptoms and/or build EF skills, this will be an important area for future research. Development of therapeutic interventions to improve EF skills and decrease the incidence of anxiety and depression in children with ASD is sorely needed. Lastly, more research is needed in the future to explore the environmental, genetic and neural underpinnings of internalizing symptoms in youth with ASD.

References

- Abrahams, B. S., & Geschwind, D. H. (2008). Advances in autism genetics: on the threshold of a new neurobiology. *Nature Reviews Genetics*, 9(5), 341-355. doi:10.1038/nrg2346
- Achenbach, T. M., & Edelbrock, C. (1991). Manual for the Child Behavior Checklist and 1991 Profile. Burlington, VT: University of Vermont Department of Psychiatry.
- Achenbach, T. M., & McConaughy, S. H. (1992). Taxonomy of internalizing disorders of childhood and adolescence. In W. M. Reynolds, W. M. Reynolds (Eds.), *Internalizing disorders in children and adolescents* (pp. 19-60). Oxford, England: John Wiley & Sons.
- Achenbach, T. M., & Rescorla, L. A. (2003). Manual for the ASEBA adult forms & profiles. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families.
- Allen, C. W., Silove, N. N., Williams, K. K., & Hutchins, P. P. (2007). Validity of the Social Communication Questionnaire in assessing risk of autism in preschool children with developmental problems. *Journal of Autism and Developmental Disorders*, 37(7), 1272-1278. doi:10.1007/s10803-006-0279-7
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders*: DSM-5. Washington, D.C: American Psychiatric Association.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR*. Washington, D.C.: American Psychiatric Publishing, Inc.
- Anderson, G. M. (2012). Twin studies in autism: What might they say about genetic and environmental influences. *Journal of Autism and Developmental Disorders*, 42(7), 1526-1527. doi:10.1007/s10803-012-1552-6
- Anderson, P. (2002). Assessment and development of executive function (EF) during childhood. *Child Neuropsychology*, 8(2), 71-82. doi:10.1076/chin.8.2.71.8724
- Anderson, V. (1998). Assessing executive functions in children: Biological, psychological, and developmental considerations. *Neuropsychological Rehabilitation*, 8(3), 319-349 doi:10.1080/713755568
- Anderson, V., Jacobs, R., & Anderson, P. (Eds.). (2008). *Executive functions and the frontal lobes: A lifespan perspective*. Philadelphia, PA US: Taylor & Francis.

- Anthony, J. L., Lonigan, C. J., Hooe, E. S., & Phillips, B. M. (2002). An affect-based, hierarchical model of temperament and its relations with internalizing symptomatology. *Journal of Clinical Child and Adolescent Psychology*, 31(4), 480-490. doi:10.1207/153744202320802151
- Alvarez, J. A., & Emory, E. (2006). Executive Function and the Frontal Lobes: A Meta-Analytic Review. *Neuropsychology Review*, 16(1), 17-42. doi:10.1007/s11065-006-9002-x.
- Bailey, A. A., Le Couteur, A. A., Gottesman, I. I., & Bolton, P. P. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. *Psychological Medicine*, 25(1), 63-77. doi:10.1017/S0033291700028099
- Baio, J. (2014). Prevalence of autism spectrum disorder among children aged 8 yearsautism and developmental disabilities monitoring network, 11 sites, United States, 2010. Morbidity and mortality weekly report. Surveillance summaries (Washington, DC: 2002), 63(2), 1.
- Barnard-Brak, L., Sulak, T., & Ivey Hatz, J. K. (2011). Macrocephaly in children with autism spectrum disorders. *Pediatric Neurology*, 44(2), 97-100. doi:10.1016/j.pediatrneurol.2010.09.011
- Barbaro, J., & Dissanayake, C. (2009). Autism spectrum disorders in infancy and toddlerhood: A review of the evidence on early signs, early identification tools, and early diagnosis. *Journal of Developmental and Behavioral Pediatrics*, 30(5), 447-459. doi:10.1097/DBP.0b013e3181ba0f9f
- Barlow, D. H., Farchione, T. J., Fairholme, C. P., Ellard, K. K., Boisseau, C. L., Allen, L. B., & Ehrenreich-May, J. (2011). Unified protocol for transdiagnostic treatment of emotional disorders: Therapist guide. New York, NY, US: Oxford University Press.
- Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, 51(6), 1173-1182. doi:10.1037/0022-3514.51.6.1173
- Baxter, L. R., Schwartz, J. M., Phelps, M. E., Mazziotta, J. C., Guze, B. H., Selin, C. E., & ...Sumida, R. M. (1989). Reduction of prefrontal cortex glucose metabolism common to three types of depression. *Archives of General Psychiatry*, 46(3), 243-250. doi:10.1001/archpsyc.1989.01810030049007
- Bayer, J. K., Hastings, P. D., Sanson, A. V., Ukoumunne, O. C., & Rubin, K. H. (2010) Predicting mid-childhood internalising symptoms: A longitudinal community study. *International Journal of Mental Health Promotion*, 12(1), 5-17. doi: 10.1080/14623730.2010.9721802

- Bayer, J. K., Rapee, R. M., Hiscock, H., Ukoumunne, O. C., Mihalopoulos, C., & Wake, M. (2011). Translational research to prevent internalizing symptoms early in childhood. *Depression and Anxiety*, 28(1), 50-57. doi:10.1002/da.20743
- Ben Bashat, D., Kronfeld-Duenias, V., Zachor, D. A., Ekstein, P. M., Hendler, T., Tarrasch, R.,... & Ben Sira, L. (2007). Accelerated maturation of white matter in young children with autism: a high b value DWI study. *Neuroimage*, 37(1), 40-47. doi: 10.1016/j.2007.04.060
- Berg, J. M., & Geschwind, D. H. (2012). Autism genetics: searching for specificity and convergence. *Genome Biology*, 13(7), 247. doi: 10.1186/gb-2012-13-7-247
- Berkel, S., Marshall, C. R., Weiss, B., Howe, J., Roeth, R., Moog, U., ... & Rappold, G. A. (2010). Mutations in the SHANK2 synaptic scaffolding gene in autism spectrum disorder and mental retardation. *Nature Genetics*, 42(6), 489-491. doi:10.1038/ng.589
- Berument, S. K., Rutter, M., Lord, C., Pickles, A., & Bailey, A. (1999). Autism screening questionnaire: Diagnostic validity. *British Journal of Psychiatry*, 175, 444-451. doi: 10.1192/bjp.175.5.444
- Bishop, T. L. (2011). Relationship between performance-based measures of executive function and the Behavior Rating Inventory of Executive Function (BRIEF), a parent rating measure. *Dissertation Abstracts International*, 72,
- Bishop, D., Aamodt-Leeper, G. G., Creswell, C. C., McGurk, R. R., & Skuse, D. H. (2001). Individual differences in cognitive planning on the Tower of Hanoi task: Neuropsychological maturity or measurement error? *Journal of Child Psychology* and Psychiatry, 42(4), 551-556. doi:10.1111/1469-7610.00749
- Bishop, D. M., Whitehouse, A. O., Watt, H. J., & Line, E. A. (2008). Autism and diagnostic substitution: Evidence from a study of adults with a history of developmental language disorder. *Developmental Medicine & Child Neurology*, 50(5), 341-345. doi:10.1111/j.1469-8749.2008.02057.x
- Biver, F., Goldman, S., Delvenne, V., Luxen, A., De Maertelaer, V., Hubain, P., ... & Lotstra, F. (1994). Frontal and parietal metabolic disturbances in unipolar depression. *Biological Psychiatry*, 36(6), 381-38. doi:10.1016/0006-3223(94)91213-0
- Bollen, K. A., & Stine, R. (1990). Direct and indirect effects: Classical and bootstrap estimates of variability. *Sociological Methodology*, 20(1), 15-140.
- Boyd, B. A., McBee, M., Holtzclaw, T., Baranek, G. T., & Bodfish, J. W. (2009). Relationships among repetitive behaviors, sensory features, and executive

functions in high functioning Autism. *Research in Autism Spectrum Disorders*, *3*(4), 959-966. doi:10.1016/j.rasd.2009.05.003

- Blumberg S., Bramlett, M., Kogan, M., Schieve, L., Jones, J., & Lu, M. (2013). Changes in prevalence of parent-reported autism spectrum disorder in school-aged U.S. children: 2007 to 2011-2012. *National Health Statistics Reports*, 65: 1-12. Hyattsville, MD: National Center for Health Statistics. Available online: http://www.cdc.gov/nchs/data/nhsr/nhsr065.pdf.
- Bradstreet, L. E., Juechter, J. I., Kamphaus, R. W., Kerns, C. M., & Robins, D. L. (2016). Using the BASC-2 parent rating scales to screen for autism spectrum disorder in toddlers and preschool-aged children. *Journal of Abnormal Child Psychology*, 1-12. doi:10.1007/s10802-016-0167-3.
- Braunschweig, D., Duncanson, P., Boyce, R., Hansen, R., Ashwood, P., Pessah, I. N., & Van de Water, J. (2012). Behavioral correlates of maternal antibody status among children with autism. *Journal of Autism and Developmental Disorders*, 42(7), 1435-1445. doi:10.1007/s10803-011-1378-7
- Brereton, A. V., Tonge, B. J., & Einfeld, S. L. (2006). Psychopathology in children and adolescents with autism compared to young people with intellectual disability. *Journal of Autism and Developmental Disorders*, 36(7), 863-870. doi:10.1007/s10803-006-0125-y
- Brody, A. L., Saxena, S., Mandelkern, M. A., Fairbanks, L. A., Ho, M. L., & Baxter, L. R. (2001). Brain metabolic changes associated with symptom factor improvement in major depressive disorder. *Biological Psychiatry*, 50(3), 171-178. doi:10.1016/S0006-3223(01)01117-9
- Bull, R., Espy, K., & Senn, T. E. (2004). A comparison of performance on the Towers of London and Hanoi in young children. *Journal of Child Psychology and Psychiatry*, 45(4), 743-754. doi:10.1111/j.1469-7610.2004.00268.x
- Bull, R., Espy, K., & Wiebe, S. A. (2009). Short-term memory, working memory, and executive functioning in preschoolers: Longitudinal predictors of mathematical achievement at age 7 years. *Developmental Neuropsychology*, 33(3), 205-228. doi:10.1080/87565640801982312
- Carlson, S. M. (2005). Developmentally sensitive measures of executive function in preschool children. *Developmental Neuropsychology*, 28(2), 595-616. doi:10.1207/s15326942dn2802_3
- Carter, A. S., Wagmiller, R. J., Gray, S. A., McCarthy, K. J., Horwitz, S. M., & Briggs-Gowan, M. J. (2010). Prevalence of DSM-IV disorder in a representative, healthy birth cohort at school entry: Sociodemographic risks and social adaptation. *Journal of the American Academy of Child & Adolescent Psychiatry*, 49(7), 686-

698. doi: 10.1097/00004583-201007000-00009

- Carter, A. S., Godoy, L., Wagmiller, R. L., Veliz, P., Marakovitz, S., & Briggs-Gowan, M. J. (2010). Internalizing trajectories in young boys and girls: The whole is not a simple sum of its parts. *Journal of Abnormal Child Psychology*, 38(1), 19-31. doi:10.1007/s10802-009-9342-0
- Chang, J., Gilman, S. R., Chiang, A. H., Sanders, S. J., & Vitkup, D. (2015). Genotype to phenotype relationships in autism spectrum disorders. *Nature Neuroscience*, 18(2), 191-198. doi: 10.1038/nn.3907
- Chang, Y., Quan, J., & Wood, J. J. (2012). Effects of anxiety disorder severity on social functioning in children with autism spectrum disorders. *Journal of Developmental and Physical Disabilities*, 24(3), 235-245. doi:10.1007/s10882-012-9268-2
- Ciairano, S., Visu-Petra, L., & Settanni, M. (2007). Executive inhibitory control and cooperative behavior during early school years: A follow-up study. *Journal of Abnormal Child Psychology*, *35*(3), 335-345. doi:10.1007/s10802-006-9094-z
- Clark, L. A., & Watson, D. (2006). Distress and fear disorders: An alternative empirically based taxonomy of the 'mood' and 'anxiety' disorders. *The British Journal of Psychiatry*, 189(6), 481-483. doi:10.1192/bjp.bp.106.03825
- Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences* (3rd ed). Mahwah, NJ: Lawrence Erlbaum Associates.
- Cohen, D., Pichard, N., Tordjman, S., Baumann, C., Burglen, L., Excoffier, E., ... & Heron, D. (2005). Specific genetic disorders and autism: Clinical contribution towards their identification. *Journal of Autism and Developmental Disorders*, 35(1), 103-116. doi. 10.1007/s10803-004-1038-2
- Collins, J., Mahone, E. M., DuCette, J. P., Rotheram-Fuller, E., & Thurman, S. K. (2011). Validity of teacher ratings on the Behavior Rating Inventory of Executive Function-Preschool Version. Temple University Libraries.
- Corbett, B. A., Constantine, L. J., Hendren, R., Rocke, D., & Ozonoff, S. (2009). Examining executive functioning in children with autism spectrum disorder, attention deficit hyperactivity disorder and typical development. *Psychiatry Research*, 166(2-3), 210-222. doi:10.1016/j.psychres.2008.02.005
- Courchesne, E., Campbell, K., & Solso, S. (2011). Brain growth across the life span in autism: Age-specific changes in anatomical pathology. *Brain Research*, *1380*, *138-145*. doi:10.1016/j.brainres.2010.09.101

- Courchesne, E., Mouton, P. R., Calhoun, M. E., Semendeferi, K., Ahrens-Barbeau, C., Hallet, M. J., ... & Pierce, K. (2011). Neuron number and size in prefrontal cortex of children with autism. *JAMA: The Journal of the American Medical Association*, 306(18), 2001-2010. doi:10.1001/jama.2011.1638.
- Courchesne, E., & Pierce, K. (2005). Why the frontal cortex in autism might be talking only to itself: Local over-connectivity but long-distance disconnection. *Current Opinion in Neurobiology*, *15*(2), 225-230. doi:10.1016/j.conb.2005.03.001
- Crawford, N. A., Schrock, M., & Woodruff-Borden, J. (2011). Child internalizing symptoms: Contributions of child temperament, maternal negative affect, and family functioning. *Child Psychiatry and Human Development*, 42(1), 53-64. doi:10.1007/s10578-010-0202-5
- Damasio, A. R., & Maurer, R. G. (1978). A neurological model for childhood autism. *Archives of Neurology*, 35(12), 777-786. doi:10.1001/archneur.1978.00500360001001
- Daniels, A. M., Rosenberg, R. E., Law, J., Lord, C., Kaufmann, W. E., & Law, P. A. (2011). Stability of initial autism spectrum disorder diagnoses in community settings. *Journal of Autism and Developmental Disorders*, 41(1), 110-121. doi:10.1007/s10803-010-1031-x
- Davidson, R. J., Pizzagalli, D., Nitschke, J. B., & Putnam, K. (2002). Depression: Perspectives from affective neuroscience. *Annual Review of Psychology*, 53(1), 545-574. doi:10.1146/annurev.psych.53.100901.135148
- Davidson, R. J. (2002). Anxiety and affective style: Role of prefrontal cortex and amygdala. *Biological Psychiatry*, *51*(1), 68-80. doi:10.1016/S0006-3223(01)01328-2
- Dawson, G., Meltzoff, A. N., Osterling, J., & Rinaldi, J. (1998). Neuropsychological correlates of early symptoms of autism. *Child Development*, 69(5), 1276-1285. doi:10.2307/1132265
- Dawson, G., Munson, J., Estes, A., Osterling, J., McPartland, J., Toth, K., & ... Abbott, R. (2002). Neurocognitive function and joint attention ability in young children with autism spectrum disorder versus developmental delay. *Child Development*, 73(2), 345-358. doi:10.1111/1467-8624.00411.
- Dawson, G., Munson, J., Webb, S., Nalty, T., Abbott, R., & Toth, K. (2007). Rate of head growth decelerates and symptoms worsen in the second year of life in autism. *Biological Psychiatry*, 61(4), 458-464. doi:10.1016/j.biopsych.2006.07.016

- Dawson, G. (2008). Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and Psychopathology*, 20(3), 775. doi:10.1017/S0954579408000370
- Diamond, A. (2002). Normal development of prefrontal cortex from birth to young adulthood: Cognitive functions, anatomy, and biochemistry. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function* (pp. 466–503). London, England: Oxford University Press.
- Diamond, A. (1988). Abilities and neural mechanisms underlying AB performance. *Child Development*, *59*(2), 523-527. doi:10.2307/1130330
- Dinstein, I., Pierce, K., Eyler, L., Solso, S., Malach, R., Behrmann, M., & Courchesne, E. (2011). Disrupted neural synchronization in toddlers with autism. *Neuron*, 70(6), 1218-1225. doi:10.1016/j.neuron.2011.04.018
- Derakshan, N., Smyth, S., & Eysenck, M. W. (2009). Effects of state anxiety on performance using a task-switching paradigm: An investigation of attentional control theory. *Psychonomic Bulletin & Review*, 16(6), 1112-1117. doi:10.3758/PBR.16.6.1112
- De Luca, C. R., & Leventer, R. J. (2008). Developmental trajectories of executive functions across the lifespan. In V. Anderson, R. Jacobs, P. J. Anderson (Eds.), *Executive functions and the frontal lobes: A lifespan perspective* (pp. 23-56). Philadelphia, PA US: Taylor & Francis.
- Doyle, A., Ostrander, R., Skare, S., Crosby, R. D., & August, G. J. (1997). Convergent and criterion-related validity of the Behavior Assessment System for Children– Parent Rating Scale. *Journal of Clinical Child Psychology*, 26(3), 276-284. doi:10.1207/s15374424jccp26036
- Drayer, J. D. (2008). *Profiles of executive functioning in preschoolers with autism* (Doctoral dissertation, NORTHEASTERN UNIVERSITY).
- Eaton, N. R., Krueger, R. F., Markon, K. E., Keyes, K. M., Skodol, A. E., Wall, M., & ... Grant, B. F. (2013). The structure and predictive validity of the internalizing disorders. *Journal of Abnormal Psychology*, 122(1), 86-92. doi:10.1037/a0029598
- Eikeseth, S. (2009). Outcome of comprehensive psycho-educational interventions for young children with autism. *Research in Developmental Disabilities*, *30*(1), 158-178. doi:10.1016/j.ridd.2008.02.003
- Eisenberg, N., Valiente, C., Spinrad, T. L., Cumberland, A., Liew, J., Reiser, M., & ... Losoya, S. (2009). Longitudinal relations of children's effortful control, impulsivity, and negative emotionality to their externalizing, internalizing, and

co-occurring behavior problems. *Developmental Psychology*, 45(4), 988-1008. doi:10.1037/a0016213

- Eisenberg, N., Cumberland, A., Spinrad, T. L., Fabes, R. A., Shepard, S. A., Reiser, M., .
 .. & Guthrie, I. K. (2001). The relations of regulation and emotionality to children's externalizing and internalizing problem behavior. *Child Development*, 72(4), 1112-1134. doi:10.1111/1467-8624.00337
- Elliott, C.D. (2007). *Differential Ability Scales–Second Edition* (DAS-II). San Antonio, TX: Psychological Corporation.
- Elsabbagh, M., Divan, G., Koh, Y., Kim, Y., Kauchali, S., Marcín, C., & ... Fombonne, E. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5(3), 160-179. doi:10.1002/aur.239
- Espy, K. (2004). Using developmental, cognitive, and neuroscience approaches to understand executive control in young children. *Developmental Neuropsychology*, *26*(1), 379-384. doi:10.1207/s15326942dn2601_1
- Espy, K. (1997). The Shape School: Assessing executive function in preschool children. *Developmental Neuropsychology*, *13*(4), 495-499. doi:10.1080/87565649709540690
- Estes, A., Dawson, G., Sterling, L., & Munson, J. (2007). Level of intellectual functioning predicts patterns of associated symptoms in school-age children with autism spectrum disorder. *American Journal on Mental Retardation*, 112(6), 439-449. doi:10.1352/0895-8017(2007)112[439:LOIFPP]2.0.CO;2
- Estes, A., Munson, J., Dawson, G., Koehler, E., Zhou, X., & Abbott, R. (2009). Parenting stress and psychological functioning among mothers of preschool children with autism and developmental delay. *Autism*, 13(4), 375-387. doi:10.1177/1362361309105658
- Faul, F., Erdfelder, E., Buchner, A., & Lang, A.G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods*, 41, 1149-1160.
- Field, A. (2009). Discovering Statistics Using SPSS (3rd ed.). London: Sage.
- Fisher, N., & Happé, F. (2005). A training study of theory of mind and executive function in children with autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, *35*(6), 757-771. doi:10.1007/s10803-005-0022-9
- Fombonne (2009). Epidemiology of pervasive developmental disorders. *Pediatric Research*, 65(6), 591-598. doi: 10.1203/PDR.0b013e31819e7203.

- Frazier, T. W., Youngstrom, E. A., Speer, L., Embacher, R., Law, P., Constantino, J., ... & Eng, C. (2012). Validation of proposed DSM-5 criteria for autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(1), 28-40. doi: 10.1016/j.jaac.2011.09.021
- Friedman, N. P., Miyake, A., Corley, R. P., Young, S. E., DeFries, J. C., & Hewitt, J. K. (2006). Not all executive functions are related to Intelligence. *Psychological Science*, 17(2), 172-179. doi:10.1111/j.1467-9280.2006.01681.x
- Gadow, K. D., DeVincent, C. J., Pomeroy, J., & Azizian, A. (2004). Psychiatric symptoms in preschool children with PDD and clinic and comparison samples. *Journal of Autism and Developmental Disorders*, 34(4), 379-393. doi: 10.1023/B:JADD.0000037415.21458.93
- Garon, N., Bryson, S. E., & Smith, I. M. (2008). Executive function in preschoolers: A review using an integrative framework. *Psychological Bulletin*, 134(1), 31-60. doi:10.1037/0033-2909.134.1.31
- Garth, J., Anderson, V., & Wrennell, J. (1997). Executive functions following moderate to severe frontal lobe injury. *Pediatric Rehabilitation*, *1*, 99-108.
- Gerstadt, C. L., Hong, Y., & Diamond, A. (1994). The relationship between cognition and action: Performance of children 3 1/2-7 years old on a Stroop-like day-night test. *Cognition*, 53(2), 129-153. doi:10.1016/0010-0277(94)90068-X
- Geschwind, D. H., & Levitt, P. (2007). Autism spectrum disorders: Developmental disconnection syndromes. *Current Opinion in Neurobiology*, *17*(1), 103-111. doi:10.1016/j.conb.2007.01.009
- Geurts, H. M., Sinzig, J., Booth, R., & Happé, F. (2014). Neuropsychological heterogeneity in executive functioning in autism spectrum disorders. *International Journal of Developmental Disabilities*, 60(3), 155-162. doi:10.1179/2047387714Y.0000000047
- Geurts, H. M., Verté, S., Oosterlaan, J., Roeyers, H., & Sergeant, J. A. (2004). How specific are executive functioning deficits in attention deficit hyperactivity disorder and autism? *Journal of Child Psychology and Psychiatry*, 45(4), 836-854. doi:10.1111/j.1469-7610.2004.00276.x
- Ghaziuddin, M., Ghaziuddin, N., & Greden, J. (2002). Depression in persons with autism: Implications for research and clinical care. *Journal of Autism and Developmental Disorders*, *32*(4), 299-306. doi:10.1023/A:1016330802348

- Ghaziuddin, M., Welch, K., Mohiuddin, S., Lagrou, R., & Ghaziuddin, N. (2010). Utility of the Social and Communication Questionnaire in the differentiation of autism from ADHD. *Journal of Developmental and Physical Disabilities*, 22(4), 359-366. doi:10.1007/s10882-010-9199-8
- Gilliom, M., & Shaw, D. S. (2004). Codevelopment of externalizing and internalizing problems in early childhood. *Development and Psychopathology*, 16(2), 313-333. doi:10.1017/S0954579404044530
- Gioia GA, Espy KA, Isquith PK. (2003). *Behavior Rating Inventory of Executive Function, Preschool Version* (BRIEF-P). Lutz, FL: Psychological Assessment Resources.
- Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). *Behavior Rating Inventory of Executive Function*. Lutz, FL: Psychological Assessment Resources.
- Gioia, G. A., Isquith, P. K., & Kenealy, L. E. (2008). Assessment of behavioral aspects of executive function. In V. Anderson, R. Jacobs, P. J. Anderson (Eds.), *Executive functions and the frontal lobes: A lifespan perspective* (pp. 179-202). Philadelphia, PA US: Taylor & Francis.
- Gioia, G. A., Isquith, P. K., Kenworthy, L., & Barton, R. M. (2002). Profiles of everyday executive function in acquired and developmental disorders. *Child Neuropsychology*, 8(2), 121-137. doi:10.1076/chin.8.2.121.8727
- Goldin, R. L., Matson, J. L., Konst, M. J., & Adams, H. L. (2014). A comparison of children and adolescents with ASD, atypical development, and typical development on the Behavioral Assessment System for Children, Second Edition (BASC-2). *Research in Autism Spectrum Disorders*, 8(8), 951-957. doi:10.1016/j.rasd.2014.04.005
- Goodman, R., Ford, T., Simmons, H., Gatward, R., & Meltzer, H. (2003). Using the strengths and difficulties questionnaire (SDQ) to screen for child psychiatric disorders in a community sample. *International Review of Psychiatry*, 15(1-2), 166-172. doi:10.1080/0954026021000046128
- Gotham, K., Brunwasser, S. M., & Lord, C. (2015). Depressive and anxiety symptom trajectories from school age through young adulthood in samples with autism spectrum disorder and developmental delay. *Journal of the American Academy of Child & Adolescent Psychiatry*, 54(5), 369-379. doi:10.1016/j.jaac.2015.02.005
- Granader, Y., Wallace, G. L., Hardy, K. K., Yerys, B. E., Lawson, R. A., Rosenthal, M., & ... Kenworthy, L. (2014). Characterizing the factor structure of parent reported executive function in autism spectrum disorders: The impact of cognitive inflexibility. *Journal of Autism and Developmental Disorders*, 44(12), 3056-3062. doi:10.1007/s10803-014-2169-8

- Greenberg, M. T., & Kusché, C. A. (1993). *Promoting social and emotional development in deaf children: The PATHS project*. Seattle, WA US: University of Washington Press.
- Grether J.K., Anderson M.C., Croen L.A., Smith D, & Windham G.C. (2009). Risk of autism and increasing maternal and paternal age in a large North American population. *American Journal of Epidemiology*, 170(9), 1118–1126. doi: 10.1093/aje/kwp247
- Griffith, E. M., Pennington, B. F., Wehner, E. A., & Rogers, S. J. (1999). Executive functions in young children with autism. *Child Development*, 70(4), 817-832. doi:10.1111/1467-8624.00059
- Gurney, J. G., McPheeters, M. L., & Davis, M. M. (2006). Parental report of health conditions and health care use among children with and without autism: National Survey of Children's Health. Archives of Pediatrics and Adolescent Medicine, 160(8), 825. doi:10.1001/archpedi.160.8.825
- Halari, R., Simic, M., Pariante, C. M., Papadopoulos, A., Cleare, A., Brammer, M., & ... Rubia, K. (2009). Reduced activation in lateral prefrontal cortex and anterior cingulate during attention and cognitive control functions in medication-naïve adolescents with depression compared to controls. *Journal of Child Psychology* and Psychiatry, 50(3), 307-316. doi:10.1111/j.1469-7610.2008.01972.x
- Hallett, V., Lecavalier, L., Sukhodolsky, D. G., Cipriano, N., Aman, M. G., McCracken, J. T., & ... Scahill, L. (2013). Exploring the manifestations of anxiety in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(10), 2341-2352. doi:10.1007/s10803-013-1775-1
- Hallett, V., Ronald, A., Rijsdijk, F., & Happé, F. (2010). Association of autistic-like and internalizing traits during childhood: A longitudinal twin study. *The American Journal of Psychiatry*, 167(7), 809-817. doi:10.1176/appi.ajp.2009.09070990
- Harrison, P. L., & Oakland, T. (2003). Adaptive behavior assessment—Second edition manual (ABAS-II). San Antonio, TX: Harcourt Assessment.
- Hartley, S. L., & Sikora, D. M. (2009). Sex differences in autism spectrum disorder: An examination of developmental functioning, autistic symptoms, and coexisting behavior problems in toddlers. *Journal of Autism and Developmental Disorders*, 39(12), 1715-1722. doi:10.1007/s10803-009-0810-8
- Hayes, A. F. (2009). Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Communication Monographs*, 76(4), 408-420. doi: 10.1080/03637750903310360

- Hayes, A. F. (2013). Introduction to mediation, moderation, and conditional process analysis: A regression-based approach. New York, NY, US: Guilford Press
- Hazlett, H. C., Poe, M., Gerig, G., Smith, R. G., Provenzale, J., Ross, A., ... & Piven, J. (2005). Magnetic resonance imaging and head circumference study of brain size in autism: Birth through age 2 years. *Archives of General Psychiatry*, 62(12), 1366. doi:10.1001/archpsyc.62.12.1366
- Hedley, D., & Young, R. (2006). Social comparison processes and depressive symptoms in children and adolescents with Asperger syndrome. *Autism*, 10(2), 139-153. doi:10.1177/1362361306062020
- Herbert, M. R. (2010). Contributions of the environment and environmentally vulnerable physiology to autism spectrum disorders. *Current Opinion in Neurology*, 23(2), 103-110.doi: 10.1097/WCO.0b013e328336a01f
- Hill, E. L. (2004). Evaluating the theory of executive dysfunction in autism. *Developmental Review*, 24(2), 189-233. doi:10.1016/j.dr.2004.01.001
- Hill, E. L. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8(1), 26-32. doi:10.1016/j.tics.2003.11.003
- Hollocks, M. J., Jones, C. G., Pickles, A., Baird, G., Happé, F., Charman, T., & Simonoff, E. (2014). The association between social cognition and executive functioning and symptoms of anxiety and depression in adolescents with autism spectrum disorders. *Autism Research*, 7(2), 216-228. doi:10.1002/aur.1361
- Huerta, M., Bishop, S. L., Duncan, A., Hus, V., & Lord, C. (2014). Application of DSM-5 criteria for autism spectrum disorder to three samples of children with DSM-IV diagnoses of pervasive developmental disorders. *American Journal of Psychiatry*. doi: 10.1176/appi.ajp.2012.12020276
- Hughes, C., & Ensor, R. (2008). Does executive function matter for preschoolers' problem behaviors? *Journal of Abnormal Child Psychology*, 36(1), 1-14. doi:10.1007/s10802-007-9107-6
- Hughes, C., Roman, G., Hart, M. J., & Ensor, R. (2013). Does maternal depression predict young children's executive function? A 4-year longitudinal study. *Journal* of Child Psychology and Psychiatry, 54(2), 169-177. doi:10.1111/jcpp.12014
- Hulvershorn, L. A., Cullen, K., & Anand, A. (2011). Toward dysfunctional connectivity: A review of neuroimaging findings in pediatric major depressive disorder. *Brain Imaging and Behavior*, 5(4), 307-328, doi:10.1007/s11682-011-9134-3

- Iossifov, I., Ronemus, M., Levy, D., Wang, Z., Hakker, I., Rosenbaum, J., ... & Kendall, J. (2012). De novo gene disruptions in children on the autistic spectrum. *Neuron*, 74(2), 285-299, doi: 10.1016/j.neuron.2012.04.009
- Isquith, P. K., Crawford, J. S., Espy, K., & Gioia, G. A. (2005). Assessment of executive function in preschool-aged children. *Mental Retardation and Developmental Disabilities Research Reviews*, 11(3), 209-215. doi:10.1002/mrdd.20075
- Jarratt, K., Riccio, C. A., & Siekierski, B. M. (2005). Assessment of Attention Deficit Hyperactivity Disorder (ADHD) using the BASC and BRIEF. *Applied Neuropsychology*, *12*(2), 83-93. doi:10.1207/s15324826an1202_4
- Jarusiewicz, B. (2002). Efficacy of neurofeedback for children in the autistic spectrum: A Pilot Study. *Journal of Neurotherapy*, 6(4), 39-49. doi:10.1300/J184v06n04_05
- Jonsdottir, S., Bouma, A., Sergeant, J. A., & Scherder, E. A. (2006). Relationships between neuropsychological measures of executive function and behavioral measures of ADHD symptoms and comorbid behavior. *Archives of Clinical Neuropsychology*, 21(5), 383-394. doi:10.1016/j.acn.2006.05.003
- Jurado, M., & Rosselli, M. (2007). The elusive nature of executive functions: A review of our current understanding. *Neuropsychology Review*, 17(3), 213-233. doi:10.1007/s11065-007-9040z
- Just, M., Keller, T. A., Malave, V. L., Kana, R. K., & Varma, S. (2012). Autism as a neural systems disorder: A theory of frontal-posterior underconnectivity. *Neuroscience and Biobehavioral Reviews*, 36(4), 1292-1313. doi:10.1016/j.neubiorev.2012.02.007
- Kanne, S. M., Abbacchi, A. M., & Constantino, J. N. (2009). Multi-informant ratings of psychiatric symptom severity in children with autism spectrum disorders: The importance of environmental context. *Journal of Autism and Developmental Disorders*, 39(6), 856-864. doi:10.1007/s10803-009-0694-7
- Karevold, E., Røysamb, E., Ystrom, E., & Mathiesen, K. S. (2009). Predictors and pathways from infancy to symptoms of anxiety and depression in early adolescence. *Developmental Psychology*, 45(4), 1051-1060. doi:10.1037/a0016123
- Kelly, N. C., Ammerman, R. T., Rausch, J. R., Ris, M., Yeates, K. O., Oppenheimer, S. G., & Enrile, B. G. (2012). Executive functioning and psychological adjustment in children and youth with spina bifida. *Child Neuropsychology*, 18(5), 417-431. doi:10.1080/09297049.2011.613814

- Kenworthy, L., Anthony, L. G., Naiman, D. Q., Cannon, L., Wills, M. C., Luong-Tran, C., ... Wallace, G. L. (2014). Randomized controlled effectiveness trial of executive function invention for children on the autism spectrum. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 55(4), 374–383. http://doi.org/10.1111/jcpp.12161
- Kenworthy, L., Black, D. O., Harrison, B., della Rosa, A., & Wallace, G. L. (2009). Are executive control functions related to autism symptoms in high-functioning children? *Child Neuropsychology*, 15(5), 425-440. dio:10.1080/09297040802646983
- Kenworthy, L. E., Black, D. O., Wallace, G. L., Ahluvalia, T., Wagner, A. E., & Sirian, L. M. (2005). Disorganization: The forgotten executive dysfunction in highfunctioning autism (HFA) spectrum disorders. *Developmental Neuropsychology*, 28(3), 809-827. doi:10.1207/s15326942dn2803_4
- Kennedy, D. P., & Courchesne, E. (2008). Functional abnormalities of the default network during self-and other-reflection in autism. *Social Cognitive and Affective Neuroscience*, 3(2), 177-190. doi: 10.1093/scan/nsn011
- Kenworthy, L., Yerys, B. E., Anthony, L. G., & Wallace, G. L. (2008). Understanding executive control in autism spectrum disorders in the lab and in the real world. *Neuropsychology Review*, 18(4), 320-338. doi:10.1007/s11065-008-9077-7
- Kim, J. A., Szatmari, P., Bryson, S. E., Streiner, D. L., & Wilson, F. J. (2000). The prevalence of anxiety and mood problems among children with autism and Asperger syndrome. *Autism*, 4(2), 117-132. doi:10.1177/1362361300004002002
- Kim, M., Gee, D. G., Loucks, R. A., Davis, F., & Whalen, P. J. (2011). Anxiety dissociates dorsal and ventral medial prefrontal cortex functional connectivity with the amygdala at rest. *Cerebral Cortex*, 21(7), 1667-1673. doi:10.1093/cercor/bhq237
- Koenigs, M., & Grafman, J. (2009). The functional neuroanatomy of depression: Distinct roles for ventromedial and dorsolateral prefrontal cortex. *Behavioural Brain Research*, 201(2), 239-243. doi:10.1016/j.bbr.2009.03.004
- Korkman, M., Kirk, U., & Kemp, S. (1998). *NEPSY: A developmental* neuropsychological assessment. San Antonio, TX: Psychological Corporation
- Kouijzer, M. J., de Moor, J. H., Gerrits, B. L., Buitelaar, J. K., & van Schie, H. T. (2009). Long-term effects of neurofeedback treatment in autism. *Research in Autism Spectrum Disorders*, 3(2), 496-501. doi:10.1016/j.rasd.2008.10.003
- Kovacs, M. (2004). *Manual for the Children's Depression Inventory Second Edition*. North Tonawanda, NJ: Multi-Health Systems.

- Kovacs, M., & Devlin, B. (1998). Internalizing disorders in childhood. *Journal of Child Psychology and Psychiatry*, 39(1), 47-63. doi:10.1017/S0021963097001765
- Kovacs, M., Obrosky, D., & Sherrill, J. (2003). Developmental changes in the phenomenology of depression in girls compared to boys from childhood onward. *Journal of Affective Disorders*, 74(1), 33-48. doi:10.1016/S0165-0327(02)00429-9
- Kumar, A., Sundaram, S. K., Sivaswamy, L., Behen, M. E., Makki, M. I., Ager, J., & ... Chugani, D. C. (2010). Alterations in frontal lobe tracts and corpus callosum in young children with autism spectrum disorder. *Cerebral Cortex*, 20(9), 2103-2113. doi:10.1093/cercor/bhp278
- Kusché, C. A., Cook, E. T., & Greenberg, M. T. (1993). Neuropsychological and cognitive functioning in children with anxiety, externalizing, and comorbid psychopathology. *Journal of Clinical Child Psychology*, 22(2), 172-195. doi:10.1207/s15374424jccp2202_5
- Lampi, K. M., Lehtonen, L., Tran, P., Cand, M., Suominen, A., Lehti, V., & ... Sourander, A. (2012). Risk of autism spectrum disorders in low birth weight and small for gestational age infants. *The Journal of Pediatrics*, 161(5), 830-836. doi:10.1016/j.jpeds.2012.04.058
- Lawson, R. A., Papadakis, A. A., Higginson, C. I., Barnett, J. E., Wills, M. C., Strang, J. F., & ... Kenworthy, L. (2015). Everyday executive function impairments predict comorbid psychopathology in autism spectrum and attention deficit hyperactivity disorders. *Neuropsychology*, 29(3), 445-453. doi:10.1037/neu0000145
- Lengua, L. J. (2006). Growth in temperament and parenting as predictors of adjustment during children's transition to adolescence. *Developmental Psychology*, 42(5), 819-832. doi:10.1037/0012-1649.42.5.819
- Lenroot, R. K., Schmitt, J. E., Ordaz, S. J., Wallace, G. L., Neale, M. C., Lerch, J. P., & Giedd, J. N. (2009). Differences in genetic and environmental influences on the human cerebral cortex associated with development during childhood and adolescence. *Human Brain Mapping*, 30(1), 163-174. doi:10.1002/hbm.20494
- Levy, D., Ronemus, M., Yamrom, B., Lee, Y. H., Leotta, A., Kendall, J. & Buja, A. (2011). Rare de novo and transmitted copy-number variation in autistic spectrum disorders. *Neuron*, 70(5), 886-897, doi:10.1016/j.neuron.2011.05.015
- Lewinsohn, P. M., Petit, J. W., Joiner, T. J., & Seeley, J. R. (2003). The symptomatic expression of major depressive disorder in adolescents and young adults. *Journal of Abnormal Psychology*, *112*(2), 244-252. doi:10.1037/0021-843X.112.2.244

- Lezak, M. D. (1982). The problem of assessing executive functions. *International Journal of Psychology*, *17*(2-3), 281-297. doi:10.1080/00207598208247445
- Lonigan, C. J., Vasey, M. W., Phillips, B. M., & Hazen, R. A. (2004). Temperament, anxiety, and the processing of threat-relevant stimuli. *Journal of Clinical Child and Adolescent Psychology*, *33*(1), 8-20. doi:10.1207/S15374424JCCP3301_2
- Lopez, B. R., Lincoln, A. J., Ozonoff, S., & Lai, Z. (2005). Examining the relationship between executive functions and restricted, repetitive symptoms of autistic disorder. *Journal of Autism and Developmental Disorders*, 35(4), 445-460. doi:10.1007/s10803-005-5035-x
- Lord, C., Luyster, R., Guthrie, W., & Pickles, A. (2012). Patterns of developmental trajectories in toddlers with autism spectrum disorder. *Journal of Consulting and Clinical Psychology*, 80(3), 477-489. doi:10.1037/a0027214
- Lord, C., Petkova, E., Hus, V., Gan, W., Lu, F., Martin, D. M., ... & Algermissen, M. (2012). A multisite study of the clinical diagnosis of different autism spectrum disorders. *Archives of General Psychiatry*, 69(3), 306-313. doi: 10.1001/archgenpsychiatry.2011.148
- Luby, J. L., Belden, A. C., Pautsch, J., Si, X., & Spitznagel, E. (2009). The clinical significance of preschool depression: Impairment in functioning and clinical markers of the disorder. *Journal of Affective Disorders*, 112(1-3), 111-119. doi:10.1016/j.jad.2008.03.026
- Luby, J. L., Mrakotsky, C., Heffelfinger, A., Brown, K., Hessler, M., & Spitznagel, E. (2003). Modification of DSM-IV criteria for depressed preschool children. *The American Journal of Psychiatry*, 160(6), 1169-1172. doi:10.1176/appi.ajp.160.6.1169
- Luciana, M., & Nelson, C. A. (2002). Assessment of neuropsychological function through use of the Cambridge Neuropsychological Testing Automated Battery: Performance in 4- to 12-year-old children. *Developmental Neuropsychology*, 22(3), 595-624. doi:10.1207/S15326942DN2203_3
- Luna, B., Minshew, N. J., Garver, K. E., Lazar, N. A., Thulborn, K. R., Eddy, W. F., & Sweeney, J. A. (2002). Neocortical system abnormalities in autism: An fMRI study of spatial working memory. *Neurology*, 59(6), 834-840. doi:10.1212/WNL.59.6.834
- MacKinnon, D. P., Krull, J. L., & Lockwood, C. M. (2000). Equivalence of the mediation, confounding and suppression effect. *Prevention Science*, 1(4), 173-181. doi:10.1023/A:1026595011371

- MacKinnon, D. P., Lockwood, C. M., Hoffman, J. M., West, S. G., & Sheets, V. (2002). A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods*, 7(1), 83-104. doi:10.1037/1082-989X.7.1.83
- MacNeil, B. M., Lopes, V. A., & Minnes, P. M. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Research in Autism Spectrum Disorders*, 3(1), 1-21. doi:10.1016/j.rasd.2008.06.001
- March, J. S., & Parker, J. A. (2004). The Multidimensional Anxiety Scale for Children (MASC). In M. E. Maruish (Ed.), *The use of psychological testing for treatment planning and outcomes assessment: Volume 2: Instruments for children and adolescents (3rd ed)*(pp. 39-62). Mahwah, NJ US: Lawrence Erlbaum Associates Publishers.
- Marangell, L. B., Martinez, M. M., Jurdi, R. A., & Zboyan, H. H. (2007). Neurostimulation therapies in depression: A review of new modalities. *Acta Psychiatrica Scandinavica*, *116*(3), 174-181. doi:10.1111/j.1600-0447.2007.01033.x
- Martel, M. M., Nigg, J. T., Wong, M. M., Fitzgerald, H. E., Jester, J. M., Puttler, L. I., & ... Zucker, R. A. (2007). Childhood and adolescent resiliency, regulation, and executive functioning in relation to adolescent problems and competence in a high-risk sample. *Development and Psychopathology*, 19(2), 541-563. doi:10.1017/S095457940707026
- Mayes, S., Calhoun, S. L., Murray, M. J., Ahuja, M., & Smith, L. A. (2011). Anxiety, depression, and irritability in children with autism relative to other neuropsychiatric disorders and typical development. *Research in Autism Spectrum Disorders*, 5(1), 474-485. doi:10.1016/j.rasd.2010.06.012
- Mayes, S., Calhoun, S. L., Murray, M. J., & Zahid, J. (2011). Variables associated with anxiety and depression in children with autism. *Journal of Developmental and Physical Disabilities*, 23(4), 325-337. doi:10.1007/s10882-011-9231-7
- Mazurek, M. O., & Kanne, S. M. (2010). Friendship and internalizing symptoms among children and adolescents with ASD. *Journal of Autism and Developmental Disorders*, 40(12), 1512-1520. doi:10.1007/s10803-010-1014-y
- McAlonan, G. M., Cheung, C. C., Cheung, V. V., Wong, N. N., Suckling, J. J., & Chua, S. E. (2009). Differential effects on white-matter systems in high-functioning autism and Asperger's syndrome. *Psychological Medicine*, 39(11), 1885-1893. doi:10.1017/S0033291709005728

- McCanlies, E. C., Fekedulegn, D., Mnatsakanova, A., Burchfiel, C. M., Sanderson, W. T., Charles, L. E., & Hertz-Picciotto, I. (2012). Parental occupational exposures and autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 42(11), 2323-2334.doi: 10.1007/s10803-012-1468-1
- McEvoy, R. E., Rogers, S. J., & Pennington, B. F. (1993). Executive function and social communication deficits in young autistic children. *Child Psychology & Psychiatry* & Allied Disciplines, 34(4), 563-578. doi:10.1111/j.1469-7610.1993.tb01036.x
- Miller, M. R., Giesbrecht, G. F., Müller, U., McInerney, R. J., & Kerns, K. A. (2012). A latent variable approach to determining the structure of executive function in preschool children. *Journal of Cognition and Development*, 13(3), 395-423. doi:10.1080/15248372.2011.585478
- Miyake, A., & Friedman, N. P. (2012). The nature and organization of individual differences in executive functions: Four general conclusions. *Current Directions in Psychological Science*, *21*(1), 8-14. doi:10.1177/0963721411429458
- Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., & ... Caspi, A. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, 108(7), 2693-2698. doi:10.1073/pnas.1010076108
- Morgan, J. T., Chana, G., Pardo, C. A., Achim, C., Semendeferi, K., Buckwalter, J., & ... Everall, I. P. (2010). Microglial activation and increased microglial density observed in the dorsolateral prefrontal cortex in autism. *Biological Psychiatry*, 68(4), 368-376. doi:10.1016/j.biopsych.2010.05.024
- Munson, J., Faja, S., Meltzoff, A., Abbott, R., & Dawson, G. (2008). Neurocognitive predictors of social and communicative developmental trajectories in preschoolers with autism spectrum disorders. *Journal of the International Neuropsychological Society*, 14(6), 956-966. doi:10.1017/S1355617708081393
- Nigg, J. T., Quamma, J. P., Greenberg, M. T., & Kusche, C. A. (1999). A two-year longitudinal study of neuropsychological and cognitive performance in relation to behavioral problems and competencies in elementary school children. *Journal of Abnormal Child Psychology*, 27(1), 51-63. doi:10.1023/A:1022614407893
- Nolen-Hoeksema, S., & Girgus, J. S. (1994). The emergence of gender differences in depression during adolescence. *Psychological Bulletin*, 115(3), 424-443. doi:10.1037/0033-2909.115.3.424
- Noordhof, A., Krueger, R. F., Ormel, J., Oldehinkel, A. J., & Hartman, C. A. (2015). Integrating autism-related symptoms into the dimensional internalizing and externalizing model of psychopathology. The TRAILS study. *Journal of Abnormal Child Psychology*, 43(3), 577-587. doi:10.1007/s10802-014-9923-4

- Oberg, E., & Lukomski, J. (2011). Executive functioning and the impact of a hearing loss: Performance-based measures and the Behavior Rating Inventory of Executive Function (BRIEF). *Child Neuropsychology*, 17(6), 521-545. doi:10.1080/09297049.2011.555760
- Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D., & Gross, J. J. (2004). For better or for worse: neural systems supporting the cognitive down-and up-regulation of negative emotion. *Neuroimage*, 23(2), 483-499. doi:10.1016/j.neuroimage.2004.06.030
- O'Roak, B. J., Vives, L., Girirajan, S., Karakoc, E., Krumm, N., Coe, B. P., & ... Eichler, E. E. (2012). Sporadic autism exomes reveal a highly interconnected protein network of de novo mutations. Nature, 485(7397), 246-250. doi:10.1038/nature10989
- Ozonoff, S., & Jensen, J. (1999). Brief report: Specific executive function profiles in three neurodevelopmental disorders. *Journal of Autism and Developmental Disorders*, 29(2), 171-177.doi: 10.1023/A:1023052913110
- Ozonoff, S., Pennington, B. F., & Rogers, S. J. (1991). Executive function deficits in high-functioning autistic individuals: Relationship to theory of mind. *Child Psychology & Psychiatry & Allied Disciplines*, *32*(7), 1081-1105. doi:10.1111/j.1469-7610.1991.tb00351.x
- Ozonoff, S., & Strayer, D. L. (2001). Further evidence of intact working memory in autism. *Journal of Autism and Developmental Disorders*, *31*(3), 257-263. doi:10.1023/A:1010794902139
- Pizzagalli, D. A. (2011). Frontocingulate dysfunction in depression: Toward biomarkers of treatment response. *Neuropsychopharmacology*, 36(1), 183-206. doi:10.1038/npp.2010.166
- Pennington, B. F., & Bennetto, L. (1993). Main effects of transactions in the neuropsychology of conduct disorder? Commentary on the neuropsychology of conduct disorder. *Development and Psychopathology*, 5(1-2), 153-164. doi:10.1017/S0954579400004314
- Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 37(1), 51-87. doi:10.1111/j.1469-7610.1996.tb01380.x
- Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments & Computers*, *36*(4), 717-731. doi:10.3758/BF03206553

- Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, 40(3), 879-891. doi:10.3758/BRM.40.3.879
- Price, J. L., & Drevets, W. C. (2010). Neurocircuitry of mood disorders. *Neuropsychopharmacology*, 35(1), 192-216. doi:10.1038/npp.2009.104
- Price, J. L., & Drevets, W. C. (2012). Neural circuits underlying the pathophysiology of mood disorders. *Trends In Cognitive Sciences*, 16(1), 61-71. doi:10.1016/j.tics.2011.12.011
- Puig-Antich, J. & Chambers, W. (1978). *The Schedule for Affective Disorders and Schizophrenia for School-Age Children*. New York: New York State Psychiatric Institute.
- Rai, D., Lee, B. K., Dalman, C., Golding, J., Lewis, G., & Magnusson, C. (2013). Parental depression, maternal antidepressant use during pregnancy, and risk of autism spectrum disorders: population based case-control study. *BMJ*, 1-15, doi: http://dx.doi.org/10.1136/bmj.f2059
- Renno, P., & Wood, J. J. (2013). Discriminant and convergent validity of the anxiety construct in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 43(9), 2135-2146. doi:10.1007/s10803-013-1767-1
- Reynolds, C. R., & Kamphaus, R. W. (2004). *Behavior Assessment System for Children*, Second Edition (BASC-2). Bloomington, MN: Pearson Assessments.
- Rezvan, P. H., Lee, K. J., & Simpson, J. A. (2015). The rise of multiple imputation: A review of the reporting and implementation of the method in medical research. BMC Medical Research Methodology, 15(1), 30. doi: 10.1186/s12874-015-00221
- Riggs, N. R., Blair, C. B., & Greenberg, M. T. (2003). Concurrent and 2-year longitudinal relations between executive function and the behavior of 1st and 2nd grade children. *Child Neuropsychology*, 9(4), 267-276. doi:10.1076/chin.9.4.267.23513
- Riggs, N. R., Greenberg, M. T., Kusché, C. A., & Pentz, M. (2006). The mediational role of neurocognition in the behavioral outcomes of a social-emotional prevention program in elementary school students: Effects of the PATHS curriculum. *Prevention Science*, 7(1), 91-102. doi:10.1007/s11121-005-0022-1
- Rinsky, J. R., & Hinshaw, S. P. (2011). Linkages between childhood executive functioning and adolescent social functioning and psychopathology in girls with ADHD. *Child Neuropsychology*, 17(4), 368-390. doi:10.1080/09297049.2010.544649

- Roman, G. D., Ensor, R., & Hughes, C. (2016). Does executive function mediate the path from mothers' depressive symptoms to young children's problem behaviors? *Journal of Experimental Child Psychology*, 142, 158-170. doi:10.1016/j.jecp.2015.09.022
- Romine, C. B., & Reynolds, C. R. (2005). A model of the development of frontal lobe functioning: Findings from a meta-analysis. *Applied Neuropsychology*, 12(4), 190. doi:10.1207/s15324826an1204_2
- Ronald, A., & Hoekstra, R. A. (2011). Autism spectrum disorders and autistic traits: a decade of new twin studies. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 156(3), 255-274. doi: 10.1002/ajmg.b.31159
- Rosenthal, M., Wallace, G. L., Lawson, R., Wills, M. C., Dixon, E., Yerys, B. E., & Kenworthy, L. (2013). Impairments in real-world executive function increase from childhood to adolescence in autism spectrum disorders. *Neuropsychology*, 27(1), 13-18. doi:10.1037/a0031299
- Rossignol, D. A., & Frye, R. E. (2011). A review of research trends in physiological abnormalities in autism spectrum disorders: Immune dysregulation, inflammation, oxidative stress, mitochondrial dysfunction and environmental toxicant exposures. *Molecular Psychiatry*, 17(4), 389-401. doi:10.1038/mp.2011.165
- Roth, R. M., Isquith, P. K., & Gioia, G. A. (2005). Behavior rating inventory of executive function—adult version (BRIEF-A). Lutz, FL: Psychological Assessment Resources.
- Rubin, D.B. (1987). *Multiple Imputation for Nonresponse in Surveys*, New York: John Wiley & Sons, Inc.
- Rucker, D. D., Preacher, K. J., Tormala, Z. L., & Petty, R. E. (2011). Mediation analysis in social psychology: Current practices and new recommendations. *Social and Personality Psychology Compass*, 5(6), 359-371. doi: 10.1111/j.1751-9004.2011.00355.x
- Rumsey, J. M., & Hamburger, S. D. (1988). Neuropsychological findings in highfunctioning men with infantile autism, residual state. *Journal of Clinical and Experimental Neuropsychology*, 10(2), 201-221. doi:10.1080/01688638808408236
- Russell, E., & Sofronoff, K. (2005). Anxiety and social worries in children with Asperger syndrome. *Australian and New Zealand Journal of Psychiatry*, 39(7), 633-638. doi:10.1111/j.1440-1614.2005.01637.x

- Russell, G., Kelly, S. & Golding, J. (2010). A qualitative analysis of lay beliefs about the aetiology and prevalence of autistic spectrum disorders. *Child: Care, Health and Development, 36*, 431-436. doi: 10.1111/j.1365-2214.2009.00994.x
- Rutter, M., Bailey, A., & Lord, C. (2003). SCQ: The social communication questionnaire-Manual. Los Angeles, CA: Western Psychological Services.
- Sanders, S. J., Murtha, M. T., Gupta, A. R., Murdoch, J. D., Raubeson, M. J., Willsey, A. J., & Walker, M. F. (2012). De novo mutations revealed by whole-exome sequencing are strongly associated with autism. *Nature*, 485(7), 237-241, doi: 10.1038/nature10945
- Santosh, P. J., Baird, G., Pityaratstian, N., Tavare, E., & Gringras, P. (2006). Impact of comorbid autism spectrum disorders on stimulant response in children with attention deficit hyperactivity disorder: A retrospective and prospective effectiveness study. *Child: Care, Health and Development*, 32(5), 575-583. doi:10.1111/j.1365-2214.2006.00631.x
- Schumann, C. M., Bauman, M. D., & Amaral, D. G. (2011). Abnormal structure or function of the amygdala is a common component of neurodevelopmental disorders. *Neuropsychologia*, 49(4), 745-759. doi: 10.1016/j.neuropsychologia.2010.09.028
- Scolnick, B. (2005). Effects of electroencephalogram biofeedback with Asperger's syndrome. *International Journal of Rehabilitation Research*, 28(2), 159-163.
- Sergeant, J. A., Geurts, H., & Oosterlaan, J. (2002). How specific is a deficit of executive functioning for attention-deficit/hyperactivity disorder? *Behavioural Brain Research*, 130(1-2), 3-28. doi:10.1016/S0166-4328(01)00430-2
- Senn, T. E., Espy, K., & Kaufmann, P. M. (2004). Using path analysis to understand executive function organization in preschool children. *Developmental Neuropsychology*, 26(1), 445-464. doi:10.1207/s15326942dn2601_5
- Shaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwab-Stone, M. E. (2000). NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child & Adolescent Psychiatry*, 39(1), 28-38. doi:10.1097/00004583-200001000-00014
- Shafritz, K. M., Dichter, G. S., Baranek, G. T., & Belger, A. (2008). The neural circuitry mediating shifts in behavioral response and cognitive set in autism. *Biological Psychiatry*, 63(10), 974-980. doi:10.1016/j.biopsych.2007.06.028

- Shallice, T. (2002). Fractionation of the supervisory system. In D. T. Stuss, R. T. Knight (Eds.), *Principles of frontal lobe function* (pp. 261-277). New York, NY US: Oxford University Press. doi:10.1093/acprof:oso/9780195134971.003.0017
- Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods*, 7(4), 422-445. doi:10.1037/1082-989X.7.4.422
- Sinzig, J., Morsch, D., Bruning, N., Schmidt, M. H., & Lehmkuhl, G. (2008). Inhibition, flexibility, working memory and planning in autism spectrum disorders with and without comorbid ADHD-symptoms. *Child and Adolescent Psychiatry and Mental Health*, 2. doi:10.1186/1753-2000-2-4
- Slemming, K., Sørensen, M. J., Thomsen, P. H., Obel, C., Henriksen, T. B., & Linnet, K. M. (2010). The association between preschool behavioural problems and internalizing difficulties at age 10–12 years. *European Child & Adolescent Psychiatry*, 19(10), 787-795. doi:10.1007/s00787-010-0128-2
- Smithson, P. E., Kenworthy, L., Wills, M. C., Jarrett, M., Atmore, K., & Yerys, B. E. (2013). Real world executive control impairments in preschoolers with autism spectrum disorders. *Journal of autism and developmental disorders*, 43(8), 1967-1975. doi:10.1007/s10803-012-1747-x
- Sobel, M. E. (1982). Asymptotic confidence intervals for indirect effects in structural equation models. *Sociological methodology*, *13*(1982), 290-312.
- Solomon, M., Miller, M., Taylor, S. L., Hinshaw, S. P., & Carter, C. S. (2012). Autism symptoms and internalizing psychopathology in girls and boys with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 42(1), 48-59. doi:10.1007/s10803-011-1215-z
- Sommerville, J. A., Woodward, A. L., & Needham, A. (2005). Action experience alters 3-month-old infants' perception of others' actions. *Cognition*, 96(1), B1-B11. doi:10.1016/j.cognition.2004.07.004
- Smithson, P., Kenworthy, L., Wills, M. C., Jarrett, M., Atmore, K., & Yerys, B. E. (2013). Real world executive control impairments in preschoolers with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, doi:10.1007/s10803-012-1747-x
- Stewart, M. E., Barnard, L., Pearson, J., Hasan, R., & O'Brien, G. (2006). Presentation of depression in autism and Asperger syndrome: A review. *Autism*, 10(1), 103-116. doi:10.1177/1362361306062013

- Sterba, S., Egger, H. L., & Angold, A. (2007). Diagnostic specificity and nonspecificity in the dimensions of preschool psychopathology. *Journal of Child Psychology* and Psychiatry, 48(10), 1005-1013. doi:10.1111/j.1469-7610.2007.01770.x
- Stewart, M. E., Barnard, L., Pearson, J., Hasan, R., & O'Brien, G. (2006). Presentation of depression in autism and Asperger syndrome: A review. *Autism*, 10(1), 103-116. doi:10.1177/1362361306062013
- Stuss, D. T., & Alexander, M. P. (2000). Executive functions and the frontal lobes: A conceptual Review. *Psychological Research*, 63(3-4), 289-298. doi:10.1007/s004269900007
- Sukhodolsky, D. G., Scahill, L., Gadow, K. D., Arnold, L., Aman, M. G., McDougle, C. J., & ... Vitiello, B. (2008). Parent-rated anxiety symptoms in children with pervasive developmental disorders: Frequency and association with core autism symptoms and cognitive functioning. *Journal of Abnormal Child Psychology*, 36(1), 117-128. doi:10.1007/s10802-007-9165-9
- Sullivan, P. F., Magnusson, C., Reichenberg, A., Boman, M., Dalman, C., Davidson, M., ... & Weiser, M. (2012). Family history of schizophrenia and bipolar disorder as risk factors for autism. *Archives of General Psychiatry*, 69(11), 1099-1103.
- Sundaram, S. K., Kumar, A., Makki, M. I., Behen, M. E., Chugani, H. T., & Chugani, D. C. (2008). Diffusion tensor imaging of frontal lobe in autism spectrum disorder. *Cerebral Cortex*, 18(11), 2659-2665. doi: 10.1093/cercor/bhn031
- Tandon, M., Cardeli, E., & Luby, J. (2009). Internalizing disorders in early childhood: A review of depressive and anxiety disorders. *Child and Adolescent Psychiatric Clinics of North America*, 18(3), 593-610. doi:10.1016/j.chc.2009.03.004
- Tordjman, S., Somogyi, E., Coulon, N., Kermarrec, S., Cohen, D., Bronsard, G., ... & Ginchat, V. (2014). Gene× Environment interactions in autism spectrum disorders: role of epigenetic mechanisms. *Frontiers in psychiatry*, 5(1), 1-17. doi: 10.3389/fpsyt.2014.00053
- Treiman, D. J. (2009). *Quantitative data analysis: Doing social research to test ideas*. San Francisco: Jossey-Bass.
- Valiente, C., Eisenberg, N., Fabes, R. A., Shepard, S. A., Cumberland, A., & Losoya, S. H. (2004). Prediction of children's empathy-related responding from their effortful control and parents' expressivity. *Developmental Psychology*, 40(6), 911-926. doi:10.1037/0012-1649.40.6.91

- Van Eylen, L., Boets, B., Steyaert, J., Wagemans, J., & Noens, I. (2015). Executive functioning in autism spectrum disorders: Influence of task and sample characteristics and relation to symptom severity. *European Child & Adolescent Psychiatry*, 24(11), 1399-1417. doi:10.1007/s00787-015-0689-1
- van Steensel, F. A., Bögels, S. M., & Perrin, S. (2011). Anxiety disorders in children and adolescents with Autistic spectrum disorders: A meta-analysis. *Clinical Child and Family Psychology Review*, *14*(3), 302-317. doi:10.1007/s10567-011-0097-0
- Vickerstaff, S., Heriot, S., Wong, M., Lopes, A., & Dossetor, D. (2007). Intellectual ability, self-perceived social competence, and depressive symptomatology in children with high-functioning autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, *37*(9), 1647-1664. doi:10.1007/s10803-006-0292-x
- Volker, M. A., Lopata, C., Smerbeck, A. M., Knoll, V. A., Thomeer, M. L., Toomey, J. A., & Rodgers, J. D. (2010). BASC-2 PRS profiles for students with high-functioning autism spectrum disorders. *Journal of Autism and Developmental Disorders*, 40(2), 188-199. doi: 10.1007/s10803-009-0849-6
- Volpe, J.J. (1995). Neurology of the newborn (3rd ed.). Philadelphia: W.B. Saunders.
- Wallace, G. L., Kenworthy, L., Pugliese, C. E., Popal, H. S., White, E. I., Brodsky, E., & Martin, A. (2016). Real-world executive functions in adults with autism spectrum disorder: Profiles of impairment and associations with adaptive functioning and co-morbid anxiety and depression. *Journal of Autism and Developmental Disorders*, 46(3), 1071-1083. doi:10.1007/s10803-015-2655-7
- Weisbrot, D. M., Gadow, K. D., DeVincent, C. J., & Pomeroy, J. (2005). The presentation of anxiety in children with pervasive developmental disorders. *Journal of Child and Adolescent Psychopharmacology*, 15(3), 477-496. doi:10.1089/cap.2005.15.477
- Welsh, J. A., Nix, R. L., Blair, C., Bierman, K. L., & Nelson, K. E. (2010). The development of cognitive skills and gains in academic school readiness for children from low-income families. *Journal of Educational Psychology*, 102(1), 43-53. doi:10.1037/a0016738
- Welsh, M. C., Pennington, B. F., & Groisser, D. B. (1991). A normative-developmental study of executive function: A window on prefrontal function in children. *Developmental Neuropsychology*, 7(2), 131-149. doi:10.1080/87565649109540483
- Werner, E., Dawson, G., Osterling, J., & Dinno, N. (2000). Brief report: Recognition of autism spectrum disorder before one year of age: A retrospective study based on home videotapes. *Journal of Autism and Developmental Disorders*, 30(2), 157-162. doi:10.1023/A:1005463707029

- White, S. J., Burgess, P. W., & Hill, E. L. (2009). Impairments on 'open-ended' executive function tests in autism. *Autism Research*, 2(3), 138-147. doi:10.1002/aur.78
- White, S. W., Lerner, M. D., McLeod, B. D., Wood, J. J., Ginsburg, G. S., Kerns, C., & ... Compton, S. (2015). Anxiety in youth with and without autism spectrum disorder: Examination of factorial equivalence. *Behavior Therapy*, 46(1), 40-53. doi:10.1016/j.beth.2014.05.005
- Wiebe, S. A., Espy, K., & Charak, D. (2008). Using confirmatory factor analysis to understand executive control in preschool children: I. Latent structure. *Developmental Psychology*, 44(2), 575-587. doi:10.1037/0012-1649.44.2.575
- Williams, J., & MacKinnon, D. P. (2008). Resampling and distribution of the product methods for testing indirect effects in complex models. *Structural Equation Modeling*, 15(1), 23-51. doi:10.1080/10705510701758166
- Wing, L., & Potter, D. (2002). The epidemiology of autistic spectrum disorders: Is prevalence rising? *Mental Retardation and Developmental Disabilities Research Reviews*, 8(3), 151-161. doi:10.1002/mrdd.10029
- Yerys, B. E., Wallace, G. L., Jankowski, K. F., Bollich, A., & Kenworthy, L. (2011). Impaired Consonant Trigrams Test (CTT) performance relates to everyday working memory difficulties in children with autism spectrum disorders. *Child Neuropsychology*, 17(4), 391-399. doi:10.1080/09297049.2010.547462
- Zablotsky, B., Black, L. I., Maenner, M. J., Schieve, L. A., & Blumberg, S. J. (2015). Estimated prevalence of autism and other developmental disabilities following questionnaire changes in the 2014 National Health Interview Survey. *National health statistics reports*, (87), 1-21.
- Zahn-Waxler, C., Klimes-Dougan, B., & Slattery, M. J. (2000). Internalizing symptoms of childhood and adolescence: Prospects, pitfalls, and progress in understanding the development of anxiety and depression. *Development and Psychopathology*, 12(3), 443-466. doi:10.1017/S0954579400003102
- Zelazo, P. D., & Carlson, S. M. (2012). Hot and cool executive function in childhood and adolescence: Development and plasticity. *Child Development Perspectives*, 6(4), 354-360. doi: 10.1111/j.1750-8606.2012.00246.x
- Zerbo, O., Iosif, A. M., Walker, C., Ozonoff, S., Hansen, R. L., & Hertz-Picciotto, I. (2013). Is maternal influenza or fever during pregnancy associated with autism or developmental delays? Results from the CHARGE (CHildhood Autism Risks from Genetics and Environment) Study. *Journal of Autism and Developmental Disorders*, 43(1), 25-33. doi: 10.1007/s10803-012-1540-x

- Zikopoulos, B., & Barbas, H. (2010). Changes in prefrontal axons may disrupt the network in autism. *The Journal of Neuroscience*, *30*(44), 14595-14609. doi:10.1523/JNEUROSCI.2257-10.2010
- Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal* of Developmental Neuroscience, 23(2-3), 143-152. doi:10.1016/j.ijdevneu.2004.05.001