University of Miami
Scholarly Repository

Open Access Dissertations | Electronic Theses and Dissertations

2014-07-02

Syndrome Specific and Non-Syndrome Specific Predictors of Developmental Change in Higher Functioning Children with Autism

Kim E. Ono
University of Miami, kim.e.ono@gmail.com

Follow this and additional works at: https://scholarlyrepository.miami.edu/oa_dissertations

Recommended Citation
Ono, Kim E., "Syndrome Specific and Non-Syndrome Specific Predictors of Developmental Change in Higher Functioning Children with Autism" (2014). *Open Access Dissertations*. 1241.
https://scholarlyrepository.miami.edu/oa_dissertations/1241

This Embargoed is brought to you for free and open access by the Electronic Theses and Dissertations at Scholarly Repository. It has been accepted for inclusion in Open Access Dissertations by an authorized administrator of Scholarly Repository. For more information, please contact repository.library@miami.edu.

UNIVERSITY OF MIAMI

SYNDROME SPECIFIC AND NON-SYNDROME SPECIFIC PREDICTORS OF DEVELOPMENTAL CHANGE IN HIGHER FUNCTIONING CHILDREN WITH AUTISM

By

Kim E. Ono

A DISSERTATION

Submitted to the Faculty
of the University of Miami
in partial fulfillment of the requirements for
the degree of Doctor of Philosophy

Coral Gables, Florida

August 2014

©2014
Kim E. Ono
All Rights Reserved

UNIVERSITY OF MIAMI

A dissertation submitted in partial fulfillment of
The requirements for the degree of
Doctor of Philosophy

SYNDROME SPECIFIC AND NON-SYNDROME SPECIFIC PREDICTORS OF DEVELOPMENTAL CHANGE IN HIGHER FUNCTIONING CHILDREN WITH AUTISM

Kim E. Ono

Approved:

_____ Heather A. Henderson, Ph.D. Associate Professor of Psychology	_____ Neena Malik, Ph.D. Psychology Training Director Department of Pediatrics
_____ Daniel S. Messinger, Ph.D. Professor of Psychology	_____ Michael Alessandri, Ph.D. Clinical Professor of Psychology
_____ Rebecca B. Shearer, Ph.D. Associate Professor of Psychology	_____ M. Brian Blake, Ph.D. Dean of the Graduate School

ONO, KIM E. (Ph.D., Psychology)
Syndrome Specific and Non-Syndrome Specific Predictors of Developmental Change in Higher Functioning Children with Autism (August 2014)

Abstract of a dissertation at the University of Miami.

Dissertation supervised by Professor Heather A. Henderson.
No. of pages in text. (100)

There is a wide range of variability in symptoms and comorbid behaviors among individuals with Autism Spectrum Disorder (ASD). Variability is seen both between individuals at a given point in time and in patterns of change within individuals over time. The goals of the current study were to examine (1) initial levels and rates of change in social reciprocity and internalizing and externalizing behavior problems, and (2) the effects of initial temperament, verbal IQ, and symptom severity on mean initial levels and rates of change in social reciprocity and comorbid internalizing and externalizing behaviors. The sample consisted of higher functioning children with autism (HFA) and an age- and gender- matched non-ASD comparison sample (non-ASD), 8-19 years old. One fifty four adolescents (80 HFA, 74 non-ASD) and their parents participated in a series of visits, in which temperament, autism symptoms, and comorbid internalizing and externalizing problems were assessed. Using multilevel modeling (MLM) in HLM6 results indicated that all children decreased in internalizing behaviors and social reciprocity over childhood and adolescence. Externalizing behaviors showed trend level improvements for both adolescents diagnosed with HFA and non-ASD adolescents. In general, higher levels of negative affect, lower levels of effortful control (EC), and lower levels of surgency were associated with concurrent maladjustment in internalizing,

externalizing, and ASD symptom related problems, such as social reciprocity. Lower levels of negative affect and higher verbal IQ were associated with greater reductions in emotional, behavioral, and social reciprocity problems across critical adolescent years. Importantly, these predictors and their relations to patterns of development were generally consistent for both adolescents diagnosed with HFA and non-ASD adolescence, emphasizing a generalized effect of temperament on behavior change, regardless of a diagnosis. Results suggest the importance of assessing and acknowledging temperament to inform targeted intervention and goodness-of-fit for both children with HFA and non-ASD children.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Chapter 1: Introduction

Autism is a neurodevelopmental disorder characterized by varying degrees of social and communicative impairment, as well as restricted interests and repetitive behavior (American Psychiatric Association, 2000; World Health Organization, 2007). The term Autism Spectrum Disorder (ASD) describes the widespread continuum of traits that these individuals exhibit. Particularly high levels of heterogeneity have been noted among higher functioning ASD individuals (HFA), or those with IQ's greater than 70 (Prior et al., 1998). Contributing to this heterogeneity in presentation are high rates of comorbidity with other psychological and medical conditions, such as Anxiety, Depression, Attention Deficit Hyperactivity Disorder (ADHD), aggression, and sleep disturbances (Dominick, Davis, Lainhart, Tager-Flusberg, & Folstein, 2007). These emotional and behavioral comorbidities often complicate diagnosis, interfere with treatment, and exacerbate impairments in adaptive functioning (Lane, Young, Baker, & Angley, 2010), parent-child relationships (Davis & Carter, 2008), and health related quality of life in their caregivers (Allik, Larsson, & Smedje, 2006). Importantly, these comorbidities can adversely impact adaptive functioning even more than the ASD symptoms themselves. Despite the impact of ASD and the associated burden of emotional and behavioral problems, little is known about developmental changes in symptoms and associated comorbidities that take place over the course of childhood and adolescence. The purpose of the current study is to examine developmental changes in ASD related symptoms, such as social reciprocity, as well as comorbid internalizing and externalizing problems, over the transition from childhood through adolescence in a

sample of individuals with HFA as well as an age- and gender-matched typically-developing sample. A variety of within-child predictors related to ASD presentation (i.e., IQ, initial symptom severity) and non-syndrome specific factors (i.e., temperamental effortful control, negative affect, and approach withdrawal) will be examined in relation to both of initial levels and rates of change in social reciprocity difficulties, internalizing and externalizing behavior problems. Findings will contribute to a better understanding of individual differences in the progression and prognosis for individuals with ASD, and will facilitate improved diagnostic, prevention, and intervention efforts for the ASD community.

Comorbidity:

There is extensive research on comorbid difficulties associated with ASD, including internalizing problems (Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; Tantam, 2000), and externalizing problems (Gadow, Devincent, & Pomeroy, 2006; Goldstein & Schwebach, 2004). Internalizing problems can take the form of anxiety, fearfulness, depression, and withdrawal. Externalizing problems can appear in the form of hyperactivity, defiance, destructive behavior, and aggression (Achenbach, 1991, 1992; Achenbach et al., 1991; Campbell, 1995). Several studies have shown higher rates of clinical and subclinical anxiety and depression in children diagnosed with ASD compared to non-ASD peers. Noteworthy, higher functioning individuals with ASD show even more pronounced internalizing difficulties than both lower functioning individuals diagnosed with ASD and non-ASD children (Gillott, Furniss, & Walter, 2001; Kanne, Abbacchi, & Constantino, 2009; Kim et al. 2000; Mazurek & Kanne, 2010; Sukhodolsky

et al., 2008). Leyfer et al. (2006) assessed comorbid internalizing symptoms in 109 children and adolescents, ages 5-17 years, diagnosed with autism (VIQ ranges: 46-142). Results indicated that 12% of the sample met criteria for separation anxiety, 7% for social phobia, 44% for specific phobia, 2% for generalized anxiety disorder, and 37% for obsessive compulsive disorder. Additionally, 10% of the sample had a history of major depressive disorder. Simonoff et al. (2008) found that anxiety related disorders were the most common comorbid diagnoses among children with ASD. Given the high rates of anxiety and mood related difficulties, many have questioned whether internalizing problems are a central feature of ASD, or whether they are a secondary effect to other symptoms. Consistent with this latter theory, Bellini (2004) and Chamberlain, Kasari, and Rotheram-Fuller (2007) posited that the high rates of anxiety among higher functioning individuals on the spectrum may stem from an awareness of their own social deficits. This perspective has been corroborated with evidence that children diagnosed with ASD with higher IQ generally have greater anxiety when compared to children with lower IQ (Mazurek & Kanne, 2010; Sukhodolsky et al., 2008).

This model of emerging anxiety based on awareness of atypical social interactions and relationships is consistent with the literature on anxiety development in non-ASD children. Within typical development, negative emotional outcome often transpires from early social difficulties. For example, peer rejection is not only concurrently associated with heightened rates of anxiety and depression, but also predictive of increased levels of internalizing problems later in life (Kupersmidt et al., 1990; Prinstein & Aikins, 2004; Strauss et al., 1988). Furthermore, temperamentally withdrawn children, who also

experience peer rejection, are at a heightened risk for developing internalizing problems across the 5 to 12 age period (Ladd, 2006). Results suggest that environment (peer rejection) and biological based factors (temperament) both contribute to an enhanced risk for development of internalizing problems. With repeated exposure to alienation and rejection, children develop more pronounced self-doubt, lower self-esteem, and heightened loneliness (Chorpita & Barlow, 1998; Troop-Gordon & Ladd, 2005). This bidirectional relationship between negative self-concept and social difficulties is associated with increased depression and anxiety over time (Caldwell et al., 2004; Ladd & Troop-Gordon 2003; Troop-Gordon & Ladd 2005). For higher functioning adolescents with ASD, who exhibit the capability to reflect and are self-aware, the social difficulties inherent to ASD make navigating peer relationships and friendships a particular challenge, resulting in negative emotional outcomes.

With respect to externalizing problems, ASD is highly comorbid with hyperactivity, inattention, aggression, irritability, and behavior problems (Barkley, 2006; Brereton, Tonge, & Einfeld, 2006; Connor, Steeber, McBurnett, 2010; Dickerson et al., 2011; Efron & Sciberras, 2010; Gadow, Devincent, & Pomeroy, 2006; Goldstein & Schwebach, 2004; Green et al., 2000; Mayes et al., 2012; Sturm, Ferbell, & Gillberg, 2004). These symptoms overlap closely with criteria for ADHD and present complications when making differential diagnoses. In fact, the autism description in the DSM-IV makes special reference to account for ADHD symptomatology, stating that "attention-deficit/hyperactivity disorder is not diagnosed if the symptoms of inattention and hyperactivity occur exclusively during the course of a pervasive developmental disorder" (American Psychiatric Association, 2000, p.91). Nonetheless, the overlap in

symptoms between ADHD and ASD often delays ASD diagnosis or results in initial misdiagnoses of ADHD (Hartley & Sikora, 2009). In addition, there are common neurocognitive deficits in ASD and ADHD, such as executive function deficits (Corbett et al., 2009; Happe et al., 2006), slow processing speed (Calhoun & Mayes, 2005), and deficits in attention, motor control, and planning (Sturm, Fernell, & Gillberg, 2004). The high rates of comorbidity between ADHD and ASD may be due in part to shared neurocognitive deficits, thus resulting in overlapping behavioral expression, and/or lack of specificity in assessment measures. Despite evidence that both internalizing and externalizing problems are highly comorbid with ASD (American Psychiatric Association, 2000), little is known about the development and progression of these comorbidities in this population (Lecavalier, 2006).

Developmental Trajectories of Emotional/Behavioral Problems:

With comorbidities already complicating the clinical profile of children with HFA, developmental changes over the course of childhood and adolescence adds a further layer of dynamic complexity. Within typical human development, adolescence is a transitional period fraught with emotional and behavioral changes and problems (Zahn-Waxler, Shirtcliff, & Marceau, 2008). Young teens struggle with behavioral changes, social and relationship challenges, family and education related difficulties, and parental conflict with independence (Ernst et al., 2005). Furthermore, evidence suggests that differential rates of development in the subcortical limbic and prefrontal cortical regions may exacerbate internalizing and externalizing symptoms in adolescents prone to emotional reactivity, and increase the likelihood of poor outcomes (Casey, Jones, & Hare,

2008). Symptoms of anxiety are common in childhood and adolescence, however specific forms of anxiety vary with age (Craske, 1997). Separation anxiety is deemed more of a childhood problem, whereas social phobia and generalized anxiety are more common in adolescence. On the other hand, symptoms of depression tend to be relatively infrequent in childhood, but tend to manifest mid puberty and increase into adulthood (Angold, Costello, & Worthman, 1998; Birmaher et al., 1996; Cohen et al., 1993; Fleming & Offord, 1990; Laitinen-Krispijn, Van der Ender, & Verhulst, 1999). Studies on developmental trajectories suggest that non-ASD children tend to show a gradual increase in internalizing behaviors from infancy through late childhood (Gilliom & Shaw, 2004), however they show considerable stability over the course of adolescence (Masten et al., 2005). Obradovic, Burt, and Masten (2010) corroborated and extended these findings by reporting continued stability of internalizing problems into young adulthood (20 years old). Furthermore, Bongers, Koot, van der Ender, and Verhulst (2003) used a multilevel model design to assess 2,076 children, aged 4 to 18 years, from the general population with the Child Behavior Checklist. Again, results suggested a gradual increase in internalizing behaviors until roughly age 10, then a level trajectory until 18 years of age.

Similar to internalizing behaviors, externalizing problems change in both form and prevalence over development (Bongers et al., 2004; Brame, Nagin, & Tremblay, 2001; Moffit, 1993; Stanger, Achenbach, & Verhulst, 1997). Based on a meta-analysis of 44 studies, Frick et al. (1993) derived four broad categories of externalizing behaviors, which included: property violations (e.g., lying, cruelty to animals), aggression (e.g., bullies, fights), status violations (e.g., running away, substance use), and oppositional

behavior (e.g., temper, stubbornness). Aggression often appears in toddlers, but with the development of cognitive abilities and emotion regulation, these externalizing behaviors tend to decrease over preschool and school years (Coie & Dodge, 1998). Using a multi-level growth analysis and semiparametric mixture model, Bongers et al. (2004) corroborated these findings. Results indicated a decline in aggression and oppositional behavior in non-ASD children and adolescents, from 4 to 18 years of age. However, property violations increased over the study period.

In summary, within typical human development, internalizing problems appear to increase over childhood and then stabilize over adolescence, while externalizing problems show a declining trajectory over childhood and adolescence. Interesting to consider, with the added layer of complexity and challenges associated with their ASD symptoms, how would an individual on the spectrum deal with inherent challenges associated with puberty and adolescence, and what would the influence be on the expression of internalizing and externalizing problems?

Development and ASD:

Although one would assume ASD symptoms may be exacerbated by challenges associated with transitioning through adolescence, current literature suggests the contrary. Reviews indicate that from childhood to adulthood, there is a general tendency for modest improvement in ASD symptoms (e.g., Billstedt, Gillberg, & Gillberg, 2007; Howlin, Goode, & Rutter, 2000; Mawhood, Howlin, & Rutter, 2000; Shattuck et al., 2007; Taylor & Seltzer, 2010). Over early childhood, higher functioning individuals show improvements in standard scores of language and cognitive functioning, while

lower functioning individuals diagnosed with ASD tend to decline in language and cognitive functioning with age (mean age of 4.7 years at baseline and retested at either 7 or 9 years of age) (Stevens et al., 2000). In a longitudinal study of 400 adolescents and adults with a community diagnosis of autism (initial ages 10-53 years), retrospective parent ratings of autism symptoms on the "current" Autism Diagnostic Interview – Revised (ADI-R) indicated that by the age of 21 years, 41.7% of the sample no longer met criteria for autism (Seltzer et al., 2003). Several other studies have replicated these findings of symptom improvements as individuals transition from adolescence into adulthood (Fecteau, Mottron, Berthiaume, & Burack, 2003; McGovern & Sigman, 2005; Piven, Harper, Palmer, & Arndt, 1996). Possible explanations for improvement in symptoms are intervention effects, changing diagnostic practices, and natural developmental progression (Dawson & Osterling, 1997; Lord & McGee, 2001; Rogers, 1996; Seltzer et al., 2003; Volkmar et al., 1992).

Because of the wide range of variability in patterns of developmental change, recent research has focused on identifying within-child predictors of relative rates of change in symptom severity. To date, the most consistent findings suggests that lower language and cognitive functioning are associated with greater ASD symptom severity, poorer overall outcome, and a decreased likelihood of improvement (Carter et al., 2007; Eaves, Ho, & Eaves, 1994; Lord & Bailey, 2002; Matson & Shoemaker, 2009; Mayes et al., 2009; Mayes & Calhoun, 2011; McGovern & Sigman, 2005; Miller & Ozonoff, 2000; Nordin & Gillberg, 1998; Perry, Condillac, Freeman, Dunn-Geier, & Belair, 2005; Pilowsky, Yirmiya, Shulman, & Dover, 1998; Prior et al., 1998; Seltzer et al., 2004; Shea & Mesibov, 2005). Consistent with these reports, higher language and cognitive abilities

have been shown to be associated with greater improvements and functioning over time (Howlin, Goode, Hutton, & Rutter, 2004; Howlin, Mawhood, & Rutter, 2000; Lord & Bailey, 2002; Nordin & Gillberg, 1998; Seltzer et al., 2004; Shattuck et al., 2007; Shea & Mesibov, 2005; Taylor & Seltzer, 2010). In contrast, changes in autism symptom severity were not influenced by more global demographic factors such as gender, social economic status, or race (Carter et al., 2007; Mayes & Calhoun, 2011; Murphy et al., 2009; Szatmari et al., 2006).

Given the significant developmental changes in core ASD symptoms, including social reciprocity, one might expect to see comparable changes in emotional and behavioral problems (Shattuck et al., 2007; Lounds et al., 2007; Taylor & Seltzer, 2010). Although limited research exists on the developmental change of emotional and behavioral maladjustment in ASD, two studies have reported a reduction in both internalizing and externalizing symptomatology as individuals with ASD age. Shattuck et al. (2007) conducted a prospective study examining both ASD symptoms and emotional/behavioral deficits during a 4.5-year period in 241 adolescents and adults on the spectrum (10-52 years old; mean age = 22.0). Results indicated a general ASD symptom reduction and improvements on internalizing (39.4% of sample showed improvements) and externalizing (30.7% of sample showed improvements) behaviors on the Scales of Independent Behavior-Revised (SIB-R). Additionally, the likelihood of improvement for emotional and behavioral problems was greater among individuals 31 years and older compared to the younger cohort (10-21 years old), although improvements were seen in both age groups. Once again, higher cognitive functioning predicted a better prognosis and greater reductions in emotional/behavioral problems.

However, other factors, such as gender and overall language level were not predictive of improvements in symptoms and emotional/behavioral maladjustment.

Taylor and Seltzer (2010) extended these findings, following 242 youth with ASD over a 10-year period as they transitioned out of high school (average age 16.3 years, SD = 3.1 at Time 1; range = 10.1 to 23.5). Utilizing a multi-level modeling approach incorporating 5 waves of data collection, results indicated an overall improvement in core ASD symptoms, as well as a reduction in internalizing symptoms over the study period. Higher cognitive functioning and higher family income were associated with more improvements in symptoms and emotional problems (Mayes & Calhoun, 2011; Taylor & Seltzer, 2010), but gender and maternal education were not. One limitation of both this study and the Shattuck et al. (2007) study is the use of only parent report measures of functioning. The use of a single informant limits the ability to control for functional or response bias (Huber & Power, 1985). For example, it is possible that repeated interviews alone would lead parents to report improvement. The current study addresses this limitation by using a multi-informant methodology, sampling from both parent- and self-reports at each time point, thus enabling analysis to control for response bias. Furthermore, both Shattuck et al. (2007) and Taylor and Seltzer (2010) do not utilize a matched control group and thus can not adequately determine if the observed changes in the participants diagnosed with ASD are significantly different than those that would be observed over the course of typical development. Therefore, in the current study, I will assess both individuals diagnosed with HFA and an age- and gender-matched non-ASD comparison sample to account for this limitation.

In summary, most existing studies of change in children with ASD use cross-sectional comparisons and retrospective data, often analyzing historical clinical reports, and thus may not represent an accurate account of the ontogenesis and dynamic nature of presentation over the course of development. Furthermore, most studies focus on developmental trajectories of core ASD symptoms, but neglect to assess the significant impact that emotional/behavioral comorbidities have on clinical presentation. The few studies that do assess developmental changes in emotional/behavioral problems in ASD have other limitations, such as lacking a control group and potential response bias. Finally, past literature has focused on a limited range of individual difference predictors that are focused on the ASD phenotype itself (e.g., cognitive functioning, gender, initial symptomatology), however have neglected to examine non-syndrome specific predictors, such as temperament, that have demonstrated both 1) longitudinal associations with emotional/behavioral maladjustment in typical development and 2) concurrent associations with symptoms and behavioral expression in ASD. In order to extend the existing literature, the current study employs a prospective, longitudinal, multi-informant approach to model developmental trajectories (8-19 years of age) of both symptoms and emotional/behavioral comorbidities, examining both ASD specific as well as temperamental predictors of initial functioning and developmental change.

Temperamental Predictors of Change:

Research pertaining to risk factors and prognosis has thus far focused on indicators more directly associated with core ASD symptoms, including social reciprocity (e.g., Shattuck et al., 2007; Taylor & Seltzer, 2010). However, several lines of research

suggest that much of the variability in functioning and development can be accounted for by non-syndrome specific, within-child predictors of individual differences. Non-syndrome specific constructs, such as temperament, may interact with autism symptoms to modify the behavioral expression of ASD at different points in development (Mundy et al., 2007). Temperament is generally regarded as a biologically based, stable construct, that influences one's behavioral and affective reactions to the environment (Caspi & Shiner, 2006; McCrae et al., 2000; Rothbart, Ahadi, & Evans, 2000). These differences have been hypothesized to exacerbate symptoms or comorbid behaviors by altering the way individuals perceive and interact with the world around them. Within typical development, a difficult temperament and deficits in cognitive functioning are the two most consistent within individual predictors of chronic externalizing problems (Lynam, Moffit, & Stouthamer-Loeber, 1993; Miner & Clarke-Stewart, 2008; Moffit, 1990, 1993) and internalizing problems (Booth-LaForce & Oxford, 2008; Bub, McCartney, & Willett, 2007; Keenan, Shaw, Delliquadri, Giovannelli, & Walsh, 1998). The current study extends the literature by assessing the developmental effects of temperament on symptoms and emotional/behavioral maladjustment, which to our knowledge has never been reported on in the ASD literature.

Temperament:

Rothbart (2005) identified three broad factors of temperament, based on parent report from infancy to adulthood: 1) surgency or Extraversion, which is related to positive affect and activity, 2) negative affectivity, which is related to negative emotions, and 3) EC, which is related to attentional, inhibitory, and activational control. The

current study explores three of these dimensions, surgency/Extraversion (i.e., approach/withdrawal), EC, and negative affect as predictors of initial levels and rates of change in development, due to their concurrent association with ASD related symptoms and problem behavior in autism and longitudinal associations with emotional/behavioral maladjustment in typical development.

Effortful Control and Self Regulation:

Within developmental research, the term "Effortful Control" (EC) refers to the "efficiency of executive attention including the ability to inhibit a dominant response and/or to activate a subdominant response, to plan, and to detect errors" (Rothbart & Bates, 2006, p. 129). Conventionally, measures of EC include attentional control (i.e., maintaining attention and focus or to shift one's attention as needed to deal with task demands) and inhibitory control (i.e., the ability to plan and effortfully suppress inappropriate or inaccurate responses) (Derryberry & Rothbart, 1988; Kochanska, Murray, & Harlan, 2000; Muris & Ollendick, 2005; Rothbart et al., 2001). The abilities to focus attention, switch between tasks, and inhibit prepotent responses underlie the emergence of self regulation, and are deemed a major milestone in child development (Rothbart, Ahadi, Hersey, & Fisher, 2001).

Across typical development, low levels of EC have been linked to emotional and behavioral maladjustment and this link is hypothesized to be mediated through differences in information processing. For instance, the ability to flexibly shift attention from negative thoughts to neutral or positive thoughts is important for modulating levels of anger, anxiety, and depression (Derryberry & Reed, 2002; Derryberry & Rothbart,

1988; Silk, Steinberg, & Morris, 2003), and reducing distress (Erber & Tesser, 1992; Harman, Rothbart, & Posner, 1997). The ability to focus attention aids in planning and organization, which may in turn affect one's ability to cope with stressful situations (Eronen, Nurmi, & Salmela-Aro, 1997; National Institute of Child Health and Human Development [NICHD] Early Child Care Research Network, 2005). Thus, evidence suggests that EC may be important for reducing internalizing problems (e.g., anxiety, depression). However, the most notable and well-documented association is between EC and externalizing difficulties/impulsivity (Eisenberg, Fabes, Guthrie, & Reiser, 2000).

Deficits in EC have been empirically related to children's externalizing behaviors, concurrently and over time (Kochanska & Knaack, 2003; Lengua, 2006; Lengua, West, & Sandler, 1998; Martel et al., 2007; Oldehinkel, Hartman, Ferdinand, Verhulst, & Ormel, 2007; Olson, Sameroff, Kerr, Lopez, & Wellman, 2005; Rydell, Berlin, & Bohlin, 2003; Spinrad et al., 2007). While the association between EC and externalizing difficulties is well documented (Eisenberg et al., 2004, 2009; Krueger, Caspi, Moffit, White, & Stouthamer-Loeber, 1996; Lemery, Essex, & Smider, 2002; Martel et al., 2007), the findings regarding the relation between EC and internalizing are somewhat more complex. Some studies have reported an inverse relation between EC and internalizing problems (Eisenberg et al., 2001, 2007, 2009; Lengua, 2006; Muris, 2006; Muris, de Jong, & Engelen, 2004; Oldehinkel et al., 2007; Zeman, Shipman, & Suveg, 2002), whereas others have not (Oosterlaan, Logan, & Sergeant, 1998; Rydell, Berlin, & Bohlin, 2003). Nonetheless, EC has demonstrated a true relation with both emotional and behavioral problems within typical development and clinical populations (i.e., ADHD,

Anxiety, and Depression), and may be seen as an important contributing factor in the phenotypic presentation of adolescents with autism.

Negative Affect:

Negative affect is a temperamental dimension of subjective distress that subsumes a variety of negative mood states, including anger, discomfort, frustration, fear, and nervousness. Low negative affect is described as a state of calmness and serenity. Positive affectivity has been regarded as a marker for self-regulatory capacity or may be involved with the development of self regulation (Komsi et al., 2006). In infancy, negative affect is expressed as general distress proneness. As a child develops, early forms of environmental reactivity are replaced with reactivity to novelty and limitations, such as frustration and fear (Eisenberg et al., 1993, 2000). It has been suggested that individuals with lower levels of negative affect, rely on global, category-based information as the basis of their judgments (Isbell, Burns, & Haar, 2005). This enhanced global perspective also diminishes bias, and broadens one's perspective on their environment (Johnson & Frederickson, 2005). Furthermore, individuals with lower levels of Negative Affect display greater cognitive flexibility and creativity (Frederickson, 2004; Isen, 2008, & Rowe, Hirsh, & Anderson, 2007), which may lead to more attention to detect and learn from others' social cues (Wellman, Lane, LaBounty, & Olsen, 2011). Negative affect may be associated with outcome, not only by how a child reacts to their environment, but also how the environment responds to the child. Research supports the view that negative affect is associated with increased vulnerability to life stressors. While on the other hand, positive affect and regulation skills are seen as

a protective factor against environmental stress (Eisenberg et al., 2000; Rothbart & Bates, 2006). Trait negative affectivity is associated with extraversion and neuroticism personality traits (Watson & Clark, 1984). Individuals described as having high negative emotionality tend to exhibit poor psychosocial functioning and experience more difficulty coping with negative life events (Ellenbogen & Hodgins, 2004). The ability to self regulate negative affect is generally regarded as a key developmental task in childhood and adolescence (Cole, Martin, & Dennis, 2004; Eisenberg & Morris, 2002). Prospective and longitudinal studies have reported a link between negative affect in early childhood and preschool years and development of internalizing symptoms during middle childhood and adolescence (Goodman, 2007; Goodman & Gotlib, 1999; Kovacs, Joorman, & Gotlib, 2008). In fact, Martin and Bridger (1999) suggested that high levels of negative affect may be the single most predictive temperamental factor for developing poorer social and emotional outcomes.

Approach Withdrawal:

Approach withdrawal also has implications in its relation to emotional and behavioral maladjustment. Approach behaviors are thought to reflect activity of the Behavioral Activation System (BAS), described as a motivational tendency toward approaching novel situations, and has been associated with extraversion, surgency, and sensitivity to reward seeking cues (Gray, 1982; Putnam & Stifter, 2005; Rothbart et al., 2001; Watson & Clark, 1997). From 2- to 4- months of age, infants seem to have their own distinct approaching tendencies, falling along a continuum characterized by lower or higher levels of extraversion and surgency (Rothbart, 1988; Rothbart, Derryberry, &

Hershey, 2000). As children grow, immediate approach movements are replaced with hesitation and reluctance towards unfamiliar situations and people (Schaffer, Greenwood, & Parry, 1972). Gray (1982) described this wariness and fear response as the Behavioral Inhibition System (BIS).

Conventionally, high inhibition/ high withdrawal is associated with introversion, while low inhibition/ high approach is associated with extraversion concurrently and over time (Kagan, Reznick, Snidman, 1987). The Behavioral Activation System (BAS) is hypothesized to control approach behavior in response to novel stimuli, whereas the Behavioral Inhibition System (BIS) is hypothesized to be sensitive to threat cues and related to avoidance (Depue & Collines, 1999; Gray & McNaughton, 2000; Zuckerman, 1991). Overreactivity and underreactivity of the BIS and BAS systems have been related to risk for various forms of psychopathology (Johnson, Turner, & Iwata, 2003). Specifically, depression has been associated with high BIS and low BAS (Kasch et al., 2002), anxiety disorders (Carver, 2004) and internalizing behaviors with high BIS sensitivity (Colder & O'Connor, 2004), and ADHD with low BIS (Matthys et al., 1998). Additionally, temperamental inhibition at 18 months has been identified as a risk factor for anxiety and depression at age 13 (Karevold, Røysamb, Ystrom, & Mathiesen, 2009). In general, higher BAS sensitivity is suggested to underlie externalizing problems (Newman et al., 1997; 2005), while higher BIS sensitivity is suggested to underlie internalizing problems (Colder & O'Connor, 2004). Although originally conceptualized to describe typical development, emerging literature suggests that temperamental factors, such as EC and approach withdrawal, may provide valuable information regarding heterogeneity in phenotypic presentation in individuals with ASD (Eaves, Ho, & Eaves,

1994; Garon et al. 2009; Hepburn & Stone 2006; Ozonoff et al. 2005; Schwartz et al. 2009; Wing 1997).

Temperament in ASD:

Temperament has been studied in two ways in children with ASD: 1) early temperamental predictors of an ASD diagnosis and 2) as a moderator of phenotypic expression of ASD (Adamek et al., 2011; Garon et al., 2009; Konstantareas & Stewart, 2006; Schwartz et al., 2009; Soderstrom, Rastam, & Gillberg, 2002; Sutton et al., 2005). Several studies have attempted to describe an early temperamental profile of children later diagnosed with autism. Garon et al. (2009) prospectively investigated the temperamental profiles of at-risk infants with an older sibling diagnosed with ASD (*N*=138) and infants with no family history of ASD (*N*=73). At-risk younger siblings who later received an ASD diagnosis at 36 months were distinguished from controls and siblings of children with ASD who did not receive a diagnosis, by a temperamental profile marked by reduced positive affect, difficulty regulating negative emotions, and low effortful control (EC). A similar study found that at-risk infants (6-36 months of age), who later displayed ASD symptoms, were characterized by a temperamental profile marked with irritability and negative affect (Bryson et al., 2007). Konstantareas and Stewart (2006) examined temperament in 3-10 year old children diagnosed with ASD and a control group (mean age = 6.16 years for ASD, 6.37 years for non-ASD comparison). Children with ASD were reported by parents to have lower levels of EC compared to control children. Furthermore, Schwartz et al. (2009) reported lower levels of self-reported temperamental surgency (i.e., low activity and impulsivity, high shyness, and

reduced enjoyment of high-intensity, sensation-seeking activities) and higher levels of temperamental negative affectivity in adolescents (8-16 years) diagnosed with HFA, compared to age- and gender- matched non-ASD adolescents. Therefore, there appears to be consistent predictive and concurrent associations between temperament and a diagnosis of ASD from infancy through adolescence. However, temperament not only distinguishes between diagnostic groups, but it is also predictive of variations in the expression of symptoms and emotional/behavioral maladjustment within samples of children with autism.

Garon et al. (2009) reported that low levels of behavioral approach were associated with more ASD symptoms, even after controlling for IQ and gender in a sample of at-risk 3-year-old toddlers. Schwartz et al. (2009) documented that within HFA adolescents, temperament predicted variations in concurrent internalizing and externalizing problems. Specifically, higher surgency was associated with lower levels of internalizing symptoms, in both individuals diagnosed with HFA and the non-ASD comparison sample. High EC was predictive of lower levels of internalizing symptoms in the non-ASD sample and lower levels of externalizing symptoms in both individuals diagnosed with HFA and the non-ASD samples. Results from Adamek et al. (2011) indicated that high negative affectivity, high surgency, and low EC was related to problem behavior (measured by the Aberrant Behavior Checklist), as reported by parents, in 111 children diagnosed with ASD (mean age = 4.2 years).

Collectively, these results highlight the importance of temperament as both a predictor of an ASD diagnosis and a concurrent modifier of phenotypic presentation. Within ASD, temperamental approach (i.e., surgency) shows mixed findings. However,

in general, low surgency demonstrates concurrent associations with greater emotional problems, such as social stress and anxiety and greater symptom severity. Low temperamental EC is related to problem behavior and externalizing problems. The current study adds to the existing literature on temperament in ASD, by assessing both the concurrent and developmental effects of EC and Approach/Withdrawal on symptoms and internalizing and externalizing problems.

Current Study:

Life course accounts of patients with ASD describe variable trajectories of development, with some individuals progressively losing skills with time, others reaching a plateau in adolescence, and still others acquiring or manifesting new difficulties in adulthood (Kranner, 1971; Sperry, 2001; Tantam, 2000; Wolf & Goldberg, 1986). Despite the important role of development in the expression of ASD, few prospective studies exist examining the trajectories of both symptoms and comorbid internalizing and externalizing difficulties across adolescence (Seltzer, Shattuck, Abbeduto, & Greenberg, 2004). More importantly, predictors of differential change in ASD have thus far focused on syndrome specific factors, neglecting to account for the significant longitudinal association that temperament may play on the behavioral expression of symptoms and comorbid behaviors. Therefore, the goals of the current study were to examine (1) rates of change in social reciprocity and comorbid behaviors in adolescents 8-19 years old, and (2) the effects of initial temperament, verbal IQ, and initial symptom severity on mean initial levels and rates of change in social reciprocity and emotional/behavioral

maladjustment among children diagnosed with HFA and an age- and gender-matched non-ASD comparison sample.

Specific Aims:

Specific Aim 1: To assess whether the children diagnosed with HFA and non-ASD children differ on mean initial levels and rates of change in social reciprocity, as well as internalizing and externalizing problems.

Hypothesis: I hypothesized higher levels of severe social reciprocity, internalizing, and externalizing problems in the children with HFA at baseline, compared to the comparison sample. Additionally, I hypothesized a linear decreasing trajectory of social reciprocity, internalizing, and externalizing problems for the children diagnosed with HFA. For the non-ASD sample, I hypothesized slight reductions in internalizing and externalizing problems over time.

Specific Aim 2: To test whether the associations between individual differences in Verbal IQ, initial symptom severity, and temperament (Approach Withdrawal, EC, and negative affect) and baseline levels and rates of change in social reciprocity, internalizing, and externalizing problems.

Hypothesis: I hypothesized that for all participants, higher initial IQ and lower levels of lifetime symptom severity would be associated with lower levels of current social reciprocity difficulties and lower levels of internalizing and externalizing problems at baseline, as well as greater improvements over time. Furthermore, across both diagnostic groups, I hypothesized that (1) higher levels of surgency would be associated with lower levels of internalizing problems and higher levels of externalizing problems at

baseline and over time, (2) higher levels of EC would be associated with lower levels of externalizing problems at baseline and over time, and (3) lower levels of negative affect would be associated with lower levels of internalizing and externalizing problems at baseline and over time in both individuals with HFA and the non-ASD comparison group.

Chapter 2: Method

Participants:

Participants were part of several ongoing studies of social and emotional functioning in children and adolescents diagnosed with HFA (8-19 years) and an age- and gender-matched sample of non-ASD comparison children. For the purpose of this study, the term higher functioning simply refers to a verbal IQ greater than 70, and the sample therefore includes participants with Asperger Disorder, Pervasive Developmental Disorder – Not Otherwise Specified, or high functioning autism. Two hundred thirty one adolescents were recruited for the study (123 HFA, 108 non-ASD). Of the original 231 participants in the sample, adolescents were excluded from analyses if (a) they had a VIQ lower than 70 (6 HFA) (b) there was insufficient diagnostic information to confirm diagnosis for inclusion criteria (5 HFA, 5 non-ASD), (c) they did not meet inclusion criteria (4 HFA, 3 non-ASD), (d) they did not speak English (1 non-ASD), (e) they had genetic disorders (3 HFA, 3 non-ASD), and (f) there was insufficient information to conduct longitudinal analysis, e.g., predictor or dependent measures were not collected (25 HFA, 22 non-ASD). Thus, 154 children/adolescents were included in the final analysis (80 HFA, 74 non-ASD). There were 121 males (65 HFA, 56 non-ASD) and 33 females in the sample (15 HFA, 18 non-ASD). Diagnostic groups were matched on age and gender distribution at first assessment (see Table 1). The ethnic distribution of the sample was 40.7% Caucasian, 41.1% Hispanic, 2.5% Asian, 6.1% African American, 1.5% mixed race, and 8.1% unknown or not given. Ethnicity did not differ between the diagnostic groups, $\chi^2(4, N = 149) = 3.18$, *ns*.

Participants in the HFA group had a community diagnosis of autism and were recruited from the University of Miami Center for Autism and Related Disabilities (CARD) center. To recruit participants for this study, families of children with autism were sent a letter announcing the project through the University of Miami Center for Autism and Related Disabilities (UM-CARD) database mailing system. The comparison sample was recruited through the local Miami-Dade school district through a similar mailing, targeted for children between the ages of 8 and 17 years of age. Families who responded to the recruitment letters and volunteered to participate were then scheduled for assessment sessions in the Department of Psychology F. H. Flipse Building on the Coral Gables Campus of the University of Miami. Upon consent, a thorough background history was conducted to ensure that all participants in the non-ASD group did not have a family history of autism. Furthermore, diagnostic status was confirmed during the first laboratory session, based on: (1) parent report on the Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003), (2) parent report on the High-Functioning Autism Spectrum Screening Questionnaire (ASSQ; Ehlers et al., 1999), and (3) direct observations using the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2001). Cutoff scores of 12 on the SCQ Total score, 13 on the ASSQ Total score and 7 on the ADOS Communication and Social Interaction domain were used to confirm ASD diagnosis. All children/adolescents included in the HFA sample met the established cutoffs on at least 2 of the 3 diagnostic measures. Likewise, all children/adolescents included in the non-ASD sample scored below the cutoffs on at least 2 of the 3 measures. Five children in the non-ASD group who scored above cutoff on the ADOS, but below on both the ASSQ and SCQ, were initially included in all analyses. HLM results were re-

run without the inclusion of these 5 children, which produced comparable results, thus these children were retained in the sample.

Procedures:

Within the parent project, adolescents participated in a series of visits in which genetic, electrophysiological, behavioral, and cognitive measures were completed. For the current study, participants and parents who completed one to three prior visits in the lab were asked to fill-out additional questionnaires on current social reciprocity, and internalizing and externalizing problems. The additional measures enabled us to conduct higher-order statistical analyses that were used to model developmental change (using multilevel modeling; HLM6; Raudenbush, Bryk, Cheong & Congdon, 2004). This provided valuable information on the progression of symptoms and comorbidities within an already well-characterized autism population.

Diagnostic Measures Used to Confirm Inclusion/Exclusion Criteria:

Autism Diagnostic Observation Schedule (ADOS; Lord et al, 2001) is a semi-structured observational assessment of Pervasive Developmental Disorders that measures social, communicative, cognitive, and self-regulatory behaviors. The ADOS consists of a series of standard play based activities designed to allow the examiner to observe social, communication, and repetitive behaviors. The ADOS provides multiple items rated on a qualitative scale of 0 (not abnormal) to 3 (most abnormal) to assess 5 main domains: Language and Communication; Reciprocal Social Interactions; Imagination; Restricted, Repetitive Behaviors and Interests Scale; and Other Abnormal Behavior. The ADOS has

sound inter-rater reliability (coefficients ranging from .82 to .93 for the subscales), excellent sensitivity (1.0), and specificity (.79) in a sample of 54 children ranging in age from 15 months to 10 years (Lord et al., 2001). A cut-off score of 7 on the ADOS Communication and Social Interaction domain was used in order to verify community diagnoses for inclusion/exclusion in the study.

High-Functioning Autism Spectrum Screening Questionnaire (ASSQ; Ehlers et al., 1999) is a brief 27-item, parent-reported screening instrument used to identify symptoms associated with ASD in children and adolescents. The ASSQ is rated on a 3-point scale (0 indicating normality, 1 some abnormality, and 2 definite abnormality). Normed on a sample of 1,407 children, the ASSQ has sound test-retest reliability (Pearson $r = .90$, $p = .001$), and inter-rater reliability ($r = .79$, $p = .001$). Ehlers et al. (1999) suggest a cutoff score of 13 to correctly identify a child in the HFA and non-ASD sample. A cut-off score of 13 on the ASSQ total score was used to verify community diagnoses for inclusion/exclusion in the study.

Social Reciprocity Questionnaire Used as Dependent Variables:

The Social Responsiveness Scale (SRS; Constantino & Gruber, 2005), is a brief parent-reported, quantitative measure of autistic behaviors in children and adolescents ages 4 to 18 years of age. The measure was standardized on a sample of 1,636 children drawn from the general population. A T-score of 60 or higher is considered an association with a clinical diagnosis of autism. SRS Total T-scores were utilized to index social reciprocity at each assessment point.

Intelligence Measures Used as Benchmark for IQ Cutoff and Predictor of Change:

Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV; Wechsler, 2003) measures intellectual functioning in four separate cognitive domains: Verbal Comprehension (VCI), Perceptual Reasoning (PRI), Working Memory (WMI), and Processing Speed (PSI). The WISC-IV was normed on a sample of 2,200 children divided in to eleven groups. The WISC-IV has strong internal consistency (coefficients ranging from .88 to .97 for composite scores), high test-retest stability (effect sizes ranging from .08 to .60), and strong validity (r = .89 with WISC-III). For the purposes of this study, an abbreviated version of the subtests: Similarities, Vocabulary, were used to calculate index scores for the VCI. These subtests were chosen for several reasons: they have the highest loadings on the VCI factors, strongest estimates of internal consistency, best test-retest reliabilities, and the narrowest standard errors of measurement among the WISC-IV scales (Williams et al., 2003). The WISC-IV was used to verify higher functioning cognitive abilities in both samples (IQ > 70) and as a predictor of change.

Emotional and Behavioral Questionnaires Used as Dependent Variable:

The Behavioral Assessment System for Children – Second Edition (BASC-2; Reynolds & Kamphaus, 2004) parent-report (PRS) and self-report (SRP) versions were used to evaluate the behaviors, thoughts, and emotions of children (ages 6 to 11) and adolescents (ages 12- 21). Children with ASD were included in the general and clinical normative samples in the BASC-2, and were included in the reliability and validity studies (Reynolds & Kamphaus, 2004). The BASC-2 Parent-Report of Personality has strong internal consistency, α, ranged from .73 to .95, and .76 to .95, respectively, for the

child, and adolescent forms. Furthermore, reported median test retest reliabilities for the child, and adolescent versions were found to be .84 and .81, respectively. Median inter-rater reliability for the child and adolescent forms were found to be .69 and .77 (Reynolds & Kamphaus, 2004). Of interest for the current study is the BASC-2 PRS externalizing behavior composite, composed of Hyperactivity, Aggression, and Conduct Problems subscales, and the internalizing composite, composed of Anxiety, Depression, and Somatization subscales. The BASC-2 Self-Report of Personality consists of 16 subscales. BASC-2 – SRP has a median reliability value for combined sex group of .78, with the Anxiety and Depression subscales exhibiting the highest reliabilities across all norm groups (from .80 to .83). Test-retest reliability for all subscales ranges from .56 to .79, with a median value of .70 (Reynolds & Kamphaus, 2004). Internal reliability of the PRS and SRP composite scales range from the low to mid .90's using coefficient alpha (De Los Reyes & Kazdin, 2004). Of interest for the current study is the BASC-2 SRP internalizing composite, which is composed of the Atypicality, Locus of Control, Social Stress, Anxiety, Depression, and Sense of Inadequacy subscales, as well as the Inattention/Hyperactivity composite, composed of Inattention and Hyperactivity subscales. Parent- and self-report data were assessed individually in separate models of change. Previous studies have used the BASC PRS and SRP composite T-scores to assess change in symptoms over time (see Evans, Axelrod, & Lamberg, 2004; Lehner-Dua, 2002; McClendon et al., 2011; Packman, 2002 for examples).

In order to determine accurate reporting among the lower IQ (i.e., VIQ = 70-89) and higher IQ adolescents (VIQ ≥ 90), Cronbach's alphas for each of the self-report measures of emotional and behavioral problems were analyzed. The internalizing

subscale consisted of 6 subtests. Cronbach's alpha for the lower IQ adolescents was (α = .91) and for the higher IQ adolescents (α = .91), respectively. The externalizing subscale consisted of two subscales. The Cronbach's alpha for the lower IQ adolescents was (α = .83) and for the higher IQ adolescents was (α = .76). Results suggest high internal consistency on self-report measures of emotional and behavior problems among both lower and higher IQ adolescents.

Diagnostic Measures Used to Confirm Inclusion/Exclusion Criteria and as a Predictor:

Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003) is a 40-item, parent-report screening device that measures lifetime communication skills and social functioning in children diagnosed with autism. In a sample of 200 children and adolescents, diagnostic differentiation of the SCQ was significant in all ranges of IQ (30-49, 50-69, > 70) but was strongest in the highest IQ cluster. The SCQ has documented sensitivity of 0.88 and specificity of 0.72 for the discrimination between ASD and non-ASD cases and a sensitivity of 0.90 and specificity of 0.86 for the discrimination between autism and non-autism cases (Bolte, Holtmann, & Poustka, 2008). Analyses of differentiation by domain score suggest that all three domain scores contribute to diagnostic differentiation, but that the total score followed by the social interaction domain score provide the strongest differential. A cut-off score of 12 on the SCQ total score was used to confirm diagnostic status for inclusion in the study and as a predictor of change (i.e., initial symptomatology).

Temperament Questionnaires Used as Predictor Variables:

The Early Adolescent Temperament Questionnaire- Revised (EATQ-R; Ellis & Rothbart, 2001), is a questionnaire that measures reactive and regulative temperament traits, in a large sample of children and adolescents. The EATQ-R has good internal consistency for most of the subscales (Cronbach's alphas ranging from .51 to .77). Test-retest reliability is strong, with intra-class correlation coefficients ranging from 0.55 (perceptual sensitivity) to 0.85 (frustration) (Muris & Meesters, 2009). The current study uses both the parent- and self-reported measures, utilizing three composite scores: EC, which is comprised of activation control, attention, and inhibitory control; surgency, comprised of high intensity pleasure, activity level, and low levels of shyness; and negative affect, comprised of frustration, depressive mood, and aggression. Parent and child data were used as temperamental predictors of variability in symptom and internalizing and externalizing maladjustment.

Study Design:

In order to test the study hypotheses, multilevel modeling (MLM) was employed to examine individual-level variables and their associations with adolescents' rates of change in (1) social reciprocity, (2) internalizing problems and (3) externalizing problems. MLM has the advantage of being able to include individuals with less than complete data and also allows for individuals to be sampled at unequal intervals across time. In addition, multiple assessments can be considered simultaneously, and one growth function can be determined within a single model. Thus, the overall form of change (e.g., linear) can be determined. Then, because of the way in which age of

assessment (Level 1) is nested within individuals (Level 2), a second step allows the initial mean level as well as the trajectory of change for each individual in the sample to be determined (Bryk & Raudenbush, 1987). For the current study, a series of two-level models were conducted using HLM6 (Raudenbush, Bryk, Cheong, & Congdon, 2004). Missing data were handled using full information maximum likelihood (FIML), which uses all available data when estimating parameters (Hancock & Mueller, 2006; Kline, 2005) and is recommended for use in developmental research (McCartney, Burchinal, & Bub, 2006).

Analyses were conducted in several steps. First, a fully unconditional model for each of the outcome variables (i.e., social reciprocity from the SRS total, externalizing and internalizing composite scores) were specified to ensure that there was a significant proportion of variance attributed to differences within adolescents, as well as between adolescents. Second, outcome variables over childhood and adolescence (8-19 years) were plotted and examined. Third, unconditional growth models was specified by adding age in months (e.g., *Age*) as a predictor to determine if individuals' symptoms and comorbid behaviors change significantly across childhood and adolescence. Fourth, after significant change (and significant variability in change) was established, individual difference predictors (i.e., diagnostic status, Verbal IQ, initial symptom severity, temperament, and interaction terms) were entered as predictors of adolescents' baseline levels and rates of change in social reciprocity and comorbid behavior problems. In all models, social reciprocity and behavior problems were the level-1 dependent variable.

Predictor variables were assessed in relation to both univariate and multivariate models. First, predictors were entered in the intercept equation alone. Next, the predictor

variables were entered in both the intercept equation as well as the slope equation simultaneously (Raudenbush & Bryk, 2002). If the model variables were significant in either of the equations, they were retained as predictors of both. If predictor variables were non-significant in both equations, they were removed from subsequent models. Thus, the most parsimonious model was retained. These procedures have been shown in the past to avoid anomalies (e.g., uninterpretable negative explained variance values) associated with complex growth models that have correlated intercepts and slopes (cf. Snijders & Bosker, 1999). These analyses have been successfully used to examine predictors of growth in children's learning behaviors (Dominguez et al., 2010). Results are described for each step of the analyses.

Level 1 (Change in Symptoms Across Childhood and Adolescence):

Age (the age at which the child was assessed, in months) was entered as a predictor of the within-individual variability in adolescents' (1) social reciprocity (measured with the SRS T-score), (2) internalizing problems (measured with the BASC-2 parent- and self- report internalizing composite score), and (3) externalizing problems (measured with the BASC-2 parent- and self-report externalizing composite score). Parent- and self-reported measures were examined as separate variables. Examining the effect of *Age* on adolescents' symptoms determines whether there was a significant amount of change (positive or negative) across childhood and adolescence (8-19.5 years). The outcome measures of social reciprocity, internalizing and externalizing problems, designated as Y, were written as a function of an intercept (π_{0i}), plus the multiplication of a slope parameter (π_{1i}), plus a residual (e_{ti}). For example, Y could be the externalizing

problems for an adolescent and Age_{ti}, the age at the time the externalizing problems were assessed. At Level 1, even if the slopes were not statistically significant, indicating no change in symptoms across time, a statistically significant variance associated with π_{1i} would indicate that there was significant individual variability in the amount of change over time.

Level 2 (Adolescent Characteristics):

At Level 2, the parameter estimates from the Level 1 model (intercept, π_{0i} and slope, π_{1i}) were considered random dependent variables that can be predicted by adolescent-level characteristics. Parent- and self-reported temperamental EC, negative affect, and surgency, Verbal IQ, initial symptomatology (measured with the SCQ Lifetime score), and diagnostic group were entered as predictors of variability in adolescents' scores. The models were run separately for parent- and self-reported measures of temperament. Diagnostic group (0 = non-ASD, 1 = HFA) was dummy-coded, and entered uncentered. Level 1 (Age) and level 2 independent variables (e.g., temperament, IQ, symptoms, diagnostic group) were entered as grand-mean-centered values. In addition, interactions were added, which indicate whether, in addition to directly affecting the dependent variables, diagnostic group and adolescent characteristics differentially predict Level 1 variables. In other words, it indicated whether the predictor variables were longitudinally correlated with the dependent variables differently by diagnostic group.

The final models were analyzed separately for social reciprocity, externalizing problems, and internalizing problems:

Level 1: $Y = \pi_{0i} + \pi_{1i}\,(Age_{ti}) + e_{ti}$

Level 2: $\pi_{0i} = \beta_{00} + \beta_{01}\,(EC) + \beta_{02}\,(Approach\ Withdrawal) + \beta_{03}\,(Negative\ Affect) + \beta_{04}\,(VIQ) + \beta_{05}\,(Initial\ Symptom) + \beta_{06}\,(Diagnostic\ Group) + \beta_{07}\,(Group\ x\ EC) + \beta_{08}\,(Group\ x\ Approach\ Withdrawal) + \beta_{09}\,(Group\ x\ Negative\ Affect) + \beta_{010}\,(Group\ x\ VIQ) + \beta_{011}\,(Group\ x\ Initial\ Symptoms) + r_{0i}$

$\pi_{1i} = \beta_{10} + \beta_{11}\,(EC) + \beta_{12}\,(Approach\ Withdrawal) + \beta_{13}\,(Negative\ Affect) + \beta_{14}\,(VIQ) + \beta_{15}\,(Initial\ Symptom) + \beta_{16}\,(Diagnostic\ Group) + \beta_{17}\,(Group\ x\ EC) + \beta_{18}\,(Group\ x\ Approach\ Withdrawal) + \beta_{19}\,(Group\ x\ Negative\ Affect) + \beta_{110}\,(Group\ x\ VIQ) + \beta_{111}\,(Group\ x\ Initial\ Symptoms) + r_{1i}$

Chapter 3: Results

Descriptive Statistics and Intercorrelation:

Table 2 provides the intercorrelations between the dependent variables, presented separately by diagnostic group. There were positive correlations between internalizing and externalizing problems both within and between parent- and self-reports. In addition, the SRS total was positively correlated with parent-reported internalizing and externalizing problems within the children diagnosed with HFA and with both parent- and self-reported internalizing and externalizing problems in the non-ASD group. Table 3 provides the intercorrelations between the predictor variables, presented separately by diagnostic group. For both adolescents diagnosed with HFA and non-ASD adolescents, EC and negative affect were negatively correlated within both the parent- and self-reported measures, indicating that higher levels of EC were associated with lower levels of negative affect. There was a strong positive correlation between parent- and self-reported surgency. In summary, preliminary correlations demonstrate strong inter- and intra-rater agreement on behavioral and temperamental measures for both adolescents with HFA and non-ASD participants. Despite evidence for inter-rater agreement, parent- and self-report measures were not collapsed into a single variable in order to whether informant source influenced growth trajectories.

Table 4 presents means and standard deviations for the predictor and dependent variables in the adolescents with HFA and non-ASD group at the first assessment. At study onset, the individuals with HFA had significantly higher parent- and self-reported internalizing and externalizing problems, as well as social reciprocity deficits compared

to the non-ASD group. Furthermore, the adolescents with HFA were rated as having higher levels of negative affect and lower levels of surgency by both parent- and self-reports, compared to the non-ASD group. In addition, the adolescents with HFA were rated by parents as having lower levels of EC compared to the non-ASD group, and also tended (p = .09) to be lower on self-reported EC. In summary, preliminary analyses revealed that adolescents diagnosed with HFA differed from non-ASD adolescents on predictor variables as well as dependent measures of emotional/behavioral problems on their first assessment.

In the next step the trajectories of change for internalizing and externalizing problems, and social reciprocity difficulties were analyzed.

Assessing Change in Behavior Problems and Social Reciprocity over Childhood and Adolescence:

Figure 1 depicts growth trajectories across childhood and adolescence (8-19.5 years) for internalizing problems, externalizing problems, and social reciprocity difficulties for all participants.

To analyze change over time, a series of Hierarchical Linear Models (HLM, Raudenbush et al., 2004) were used to predict variability in the individual linear growth trajectories of BASC-2 internalizing and externalizing problems and SRS total scores. A linear function was fit for each adolescent's scores, resulting in two random-effect parameters (*intercept* and *slope*), which describe the course of development in behavior problems and social reciprocity difficulties.

Level 1(Unconditional Growth Model):

The unconditional growth models for parent- and self-reported internalizing and externalizing problems, as well as SRS total scores were analyzed to determine whether behaviors changed significantly over childhood and adolescence. For these models, the *intercept* was interpreted as the mean of parent- or self-reported measures at 8 years (baseline score), and the *slope* was interpreted as the monthly rate of change in problem behaviors and social reciprocity over childhood and adolescence (8-19.5 years). For parent- and self-rated internalizing problems and SRS total score (social reciprocity difficulties), the intercepts were statistically significant, and the slopes were significant and negative. These models demonstrated that across the full sample, children experienced a reduction in internalizing problems and SRS over childhood and adolescence. Parent- and self-reported externalizing behaviors showed a significant intercept and a trend level reduction in behavioral problems over childhood and adolescence (Externalizing Slope; self-report: $p = .06$; parent-report: $p = .13$; see Table 5).

The level 1 residual variance condenses the average scatter of an individual's observed outcome values around the child's own true change trajectory. By comparing the residual variance of the unconditional growth model to the residual variance of the empty model (unconditional means model), the percent of within-person variance accounted for by *Age* can be determined. For the models, *Age* accounted for 21.98 - 45.73% of the variance at level 1 attributed to differences within children (see Table 5).

The random effects associated with the intercept and slope were statistically significant for all models, indicating the appropriateness of entering predictors at level 2.

Level 2 (Temperamental and Syndrome Specific Predictors):

To assess whether the predictor variables influenced baseline levels and rates of change on the dependent measures, diagnostic status (HFA or non-ASD), Verbal IQ, initial symptom severity (SCQ Total), parent- and self-reported temperament (surgency, negative affect, EC), and the interaction between group and each temperament dimension were then added at the intercept and slope. As described previously, variables were retained in the equation if they significantly predicted either the intercept or slope, thus resulting in the most parsimonious model. As such, each equation had different predictor variables included in the final model. Within the final model, all predictors were entered simultaneously, thus allowing for examination of each predictor, while controlling for all other predictors.

Importantly, for all models except self-reported internalizing problems, the intercept and slope were strongly negatively correlated, indicating that the children rated higher in problems at baseline improved more over time (self-reported internalizing: r=-.28; self-reported externalizing: r=-.81; parent-reported internalizing: r=-.81; parent-reported externalizing: r=-.89; SRS: r = -.81). To account for the significant correlation between intercept and slope, latent variable regression analyses were implemented. This strategy utilized initial levels (intercept) as a predictor of growth rates, thus controlling for the effects of baseline levels of problem behavior and social reciprocity difficulties.

The difference in deviance values between the level 1 (age) and level 2 (predictors added) models is a likelihood ratio test describing whether the level 2 model significantly better explains the data compared to the level 1 model. All level 2 models explained the data significantly better than the level 1 models (self-reported internalizing:

p=.04; self-reported externalizing: *p*=.01; parent-reported internalizing: *p*=.004; parent-reported externalizing: *p*=.01; SRS: *p*<.001).

To assess the magnitude of fit for the final models, the proportion of the outcome variation unexplained by a model's predictors (level 2) was compared to the unconditional growth model (level 1) (i.e., the proportional reduction in residual variance). The addition of the predictor variables accounted for 31.68-88.92% of the level 2 model variance in the outcome variables (see Table 6). This implied that a large proportion of the outcome variance for the final models was explained by the addition of the predictor variables.

Diagnostic Status as a Predictor:

To assess the influence of diagnostic status on baseline levels and rates of change across adolescence, diagnostic group (non-ASD = 0; HFA=1) was entered as a predictor for level 2 intercept and slope. As described earlier, intercept was controlled for all models. As hypothesized and consistent with preliminary analyses, parent- and self-reported internalizing and externalizing problems, as well as social reciprocity difficulties, were rated as higher in adolescents diagnosed with HFA compared to non-ASD adolescents at baseline (see Table 7). Unexpectedly, there were no group differences in slope between the children with HFA and non-ASD children, thus indicating generally similar trajectories of change for both groups. Due to the significant association between diagnostic group and intercept, all following predictor models retained diagnostic group as a predictor.

Parent-Reported Internalizing Problems:

The following predictors were retained in the final model for parent-reported internalizing problems: diagnostic group, parent-reported negative affect, and the interaction between group and parent-reported negative affect. Parent-reported negative affect and the interaction term were significant predictors of baseline internalizing scores (see Table 8). That is, after controlling for all other predictor variables in the model, parent-reported negative affect had a concurrent positive association with parent-reported internalizing problems. This indicated that across both groups, high levels of negative affect were associated with more internalizing problems at age 8. The significant interaction suggested that variations in negative affect were associated with greater variability in baseline internalizing problems for the children diagnosed with HFA relative to the non-ASD group (see Figure 2).

In addition, diagnostic status and the interaction between diagnostic group and parent-reported negative affect were predictors of change in parent-reported internalizing problems (see Table 8). When controlling for negative affect and the interaction term, children in the non-ASD group showed a reduction in internalizing problems, whereas children in the children diagnosed with HFA did not show similar improvements over the study period. Higher levels of negative affect were seen as a risk factor and lower levels of negative affect were seen as protective factors for the development of internalizing problems across adolescence. Furthermore, high and low levels of negative affect as risk and protective factors may have been particularly important in HFA children, as evidenced by the significant interaction. This implied that internalizing problems may

have been exacerbated more so for higher negative affect adolescents diagnosed with HFA, compared to non-ASD adolescents. (see Figure 2).

Parent-Reported Externalizing Problems:

The following variables were retained in the final model for parent-reported externalizing problems: diagnostic status, parent-reported EC, and parent-reported negative affect. After controlling for diagnostic status, children rated by their parents as having lower levels of EC and higher levels of negative affect were rated by their parents as having higher concurrent levels of externalizing problems at baseline.

When controlling for diagnostic group, EC, and the intercept, lower initial levels of parent-reported negative affect were associated with greater reductions in parent-reported externalizing problems. Lower levels of negative affect can be seen as a protective factor against concurrent and longitudinal development of externalizing problems across adolescence for both HFA and non-ASD children (see Table 8).

Self-Reported Internalizing Problems:

The following predictors were retained in the final model for self-reported internalizing problems: diagnostic group, Verbal IQ, and parent-reported EC. When controlling for diagnostic group, children who scored higher on the WISC-IV Verbal Comprehension Index and children with lower levels of parent-reported EC, self-reported more internalizing problems at baseline (see Table 9).

Slope was predicted by Verbal IQ, such that children with higher IQ showed a greater reduction over childhood and adolescence in self-reported internalizing behavior

problems, when holding the intercept and diagnostic group constant (see Table 9). While higher verbal IQ was associated with greater baseline levels of internalizing problems, children with higher verbal functioning, regardless of diagnostic group, improved the most across adolescence.

Self-Reported Externalizing Problems:

The following predictor variables were retained in the final model for self-reported externalizing problems: diagnostic group, Verbal IQ, parent-reported EC, and self-reported surgency. Controlling for diagnostic group, children with higher Verbal IQ, lower levels of EC, and lower levels of surgency were rated as having higher levels of self-reported externalizing problems at baseline (see Table 9).

In addition, children with higher Verbal IQ showed a greater reduction in self-reported externalizing problems over childhood and adolescence (see Table 9).

Parent-Reported Social Reciprocity on the SRS:

To assess the predictors of change in social reciprocity within the children with HFA, the original file was split by diagnostic group and predictors were analyzed in the same way as described above, only for children with HFA. The following predictors were retained in the final model for parent reported social reciprocity on the SRS: parent-reported SCQ total score (initial symptom scores) and negative affect. SCQ total score and parent-reported negative affect significantly predicted baseline levels of ASD symptoms. Children with higher SCQ total scores and children with higher levels of

negative affect were rated as having higher levels of social reciprocity difficulties on the SRS at 8 years of age, within adolescents diagnosed with HFA.

Furthermore, parent-reported negative affect predicted greater reductions in social reciprocity difficulties over childhood and adolescence, within adolescents diagnosed with HFA (see Table 10).

Summary of Level 2 Growth Models:

In summary of all the final growth models, baseline levels and patterns of growth across adolescence were at least partially accounted for by temperamental characteristics and verbal IQ. Interestingly, these patterns were generally similar for both HFA and non-ASD adolescence. High levels of negative affect and low levels of EC were associated with greater maladjustment for children at age 8. In addition, higher parent reported negative affect was associated with poorer outcome and diminished reduction in social reciprocity difficulties, as well as internalizing and externalizing problems across the study period.

Post Hoc Analyses of Clinical Significant Change:

While the analyses above described statistically significant amounts of improvement in symptoms and behavior problems, they did not directly address the clinical significance of these changes. Thus, post hoc analyses were conducted to assess clinically significant improvements and identify children who displayed "optimal outcome." Fein et al. (2013) recently reported on a sample of individuals described as achieving "optimal outcome." These are individuals who were previously diagnosed

with autism who years later failed to meet diagnostic criteria. With the idea of optimal outcome in mind, an exploratory analysis was conducted to estimate the clinically significant changes in social reciprocity (measured with the SRS), as well as clinically significant changes in internalizing and externalizing problems on the BASC-2. On the BASC-2, a *T*-score of 60 and above is regarded as clinically elevated ("At Risk") and scores below 60 are regarded as within normal range (Reynolds & Kamphaus, 2004). Likewise, the recommended cutoff score for an autism diagnosis on the SRS is a *T*-score of 60, and scores falling below 60 are considered subclinical (Constantino et al., 2007).

To explore the developmental change within each diagnostic group, the original data file was split between diagnostic groups and the unconditional growth models were analyzed separately for HFA and non-ASD adolescents. Additionally, the *Age* variable was reverse coded (i.e., age – oldest age = recoded age) so that estimates of the average outcome value at 19.5 years of age could be determined. Each dependent variable (i.e., parent and self-reported internalizing, externalizing and parent-rated social reciprocity on the SRS) was examined in this fashion. Within the HFA group, there were trend level reductions in social reciprocity difficulties on the SRS, however estimates of SRS total T-scores at 19.5 years were still elevated above clinically significant levels (At 19.5 years: $\beta = 73.28$, $p = .06$) (see Table 11). Significant reductions were seen in parent-reported internalizing problems within the HFA group ($p < .01$). Noteworthy, score estimates at 8 years were above clinical levels (*T*-score = 65.63), and they subsequently fell below clinical levels by 19.5 years (*T*-score = 51.82). Within the non-ASD group, self-reported internalizing problems significantly improved over childhood and adolescence ($p = .01$), however scores at 8 and 19.5 years never exceeded clinical levels (*T*-score age 8 = 48.30;

T-score age 19.5 = 40.84) (see Table 11). Externalizing problems did not show significant amounts of change for either the HFA of non-ASD samples.

As an additional way to quantify the amount of clinically significant change observed, the percentage of children who moved from clinically elevated scores at study entry to subclinical scores by their final assessment was examined. For each dependent measure, participants were classified into one of four groups: Below/Below (*T-score* < 60 at both first and final visit), Below/Above (*T*-score < 60 at first visit; *T-score* > 60 at final visit), Above/Below (or "Optimal Outcome"; *T-score* > 60 at first visit, *T-score* < 60 at final visit), or Above/Above (*T-score* > 60 at both first and final visit). On the SRS, 49 HFA children remained consistently elevated, however 9 HFA children (15.52%) who were rated by their parents as having clinically elevated levels of social reciprocity difficulties on first assessment were subsequently rated as having subclinical levels on their last assessment (see Table 12). For comorbid symptoms of internalizing and externalizing problems, an even more pronounced number of HFA children showed clinically significant improvement. Within the HFA sample, rates of clinically significant improvement ranged from 48 - 70% on parent- and self-reported internalizing and externalizing problems (see Table 12). In fact, within the HFA sample, it was more common for participants to show clinically significant improvement in externalizing problems than it was to remain clinically elevated (Self-report: 64.71% improvement; parent-report: 70% improved).

Since not all children were included in the optimal outcome assessment, estimate attrition analyses were conducted. Children that discontinued after first assessment did not differ from children who completed repeated assessments on the SRS, $F(1,79) = .18$,

p=.80, parent reported internalizing problems, $F(1,79)$= .11, p=.85, parent-reported externalizing problems, $F(1,79)$=.20, p=.77, self-reported internalizing problems, $F(1,79)$=.16, p=.83, or self-reported externalizing problems, $F(1,79)$=.18, p=.80. No systematic differences in internalizing, externalizing or social reciprocity related problems were observed from the children that completed multiple assessments and children that did not. Thus, it was concluded that optimal outcome estimates, although based on a smaller subsample, generalize to the larger sample.

Chapter 4: Discussion

The current study followed the development of 80 higher functioning children diagnosed with autism and 74 non-ASD children and examined IQ, initial ASD symptoms, and initial temperament as predictors of improvements in social reciprocity difficulties and behavior problems. Across the entire sample, there were significant reductions in internalizing problems and social reciprocity difficulties from childhood through adolescence. These findings were not only statistically significant reductions, but were also observed in the assessment of clinically significant change. Current results are consistent with past studies documenting improvement but significantly extend this literature by describing the role of individual differences in verbal IQ and temperament in the prediction of differential rates of improvement. Importantly, variability in both initial levels of problems and the amount of subsequent change can be at least partially accounted for by characteristics of the child at study entry. All three temperament factors - higher levels of surgency, higher levels of EC, and lower levels of negative affect - predicted higher baseline levels of social reciprocity and comorbid internalizing and externalizing problems, across both diagnostic groups. This finding emphasizes the important role of temperamental reactivity and self-regulation in accounting for variability in concurrent presentation of children on the spectrum, as well as non-ASD children. Furthermore, consistent with current theory (Martin & Bridger,1999), parent reported lower negative affect was a significant predictor of greater reductions in internalizing, externalizing, and ASD related symptom problems, such as social reciprocity, across adolescence. A strength of this study was the use of multiple

informants to assess developmental change reported by parents and children. Interestingly, although there was a strong consensus between parent- and self-raters, results indicated that both reporters contributed to explain outcome variability differences.

Patterns of Growth Across Adolescence:

Interestingly, the patterns of declining social reciprocity, internalizing, and externalizing problems were relatively consistent for both the adolescents diagnosed with HFA and non-ASD groups, indicating a generalized effect of behavior change, regardless of diagnostic status. As for the non-ASD group, findings from the current study were largely consistent with previous literature, which suggest improvements or maintenance of internalizing and externalizing problems to be present across adolescence for typically developing children (Bongers, Koot, van der Ender, & Verhulst, 2003; Bonger et al., 2004; Crocetti et al., 2009). However, an unexpected finding was the reduction in social reciprocity over the study period observed within the non-ASD children. It was hypothesized that non-ASD children would display so few symptoms related to autism at first assessment that reductions or growth across adolescence would not be observed. However, reviews of the Social Responsiveness Scale (SRS), the measure used to assess social reciprocity, may help explain these unexpected findings. Results indicate that the SRS not only be assessing variability in social reciprocity, but may also be capturing internalizing and externalizing problems (Hus et al., 2013). Furthering this claim, significant correlations between the SRS and parent- and self-reported internalizing and externalizing problems at the first assessment were observed for both adolescents with

HFA and non-ASD adolescents, within the current study. Together these findings suggest that the reduction in SRS scores in the non-ASD group may reflect reductions in global emotional/behavioral problems as opposed to social reciprocity specific difficulties**.**

As for the adolescents diagnosed with HFA, results were consistent with past literature which describe social reciprocity problem reduction and internalizing and externalizing problem improvements across adolescence (Shattuck et al., 2007; Taylor & Setlzer, 2010). The current study expands upon these studies by investigating the reductions in comparison to a matched control sample, thus detailing observed changes as significantly different from what one would expect from typical development. In addition, the current study expands upon previous studies by utilizing multi-informant methodology, thus enabling analysis to control for response bias. The following sections will describe the significant predictors of change within the current study.

Temperament and Developmental Trajectories:

According to current models of temperament, early individual differences in temperament may affect development by altering a child's pattern of attending and responding to others and their social and nonsocial environments (Henderson & Wachs, 2007). These differences, in turn, influence the types of social situations a child is exposed to, what they learn from their environments, and the responses the child elicits from others. As such, individual differences in temperament may be either protective or risk factors, which foster or interfere with a child's development into adolescence.

Current models of temperament may explain the concurrent and longitudinal associations between temperament and problem behaviors observed within the present study.

Within both autism literature and research on typically developing children, high levels of negative affect and low levels of EC appear to be the most consistent predictors of concurrent negative social and emotional functioning (Adamek et al., 2011; Garon et al., 2009; Martin & Bridger, 1999; Schwartz et al., 2009). Consistent with this, results from the current study indicate that individual differences in negative affect and EC were concurrently and predictively associated with behavior problems and social reciprocity difficulties, even after controlling for diagnostic status and other aspects of temperament.

Consistent with ASD studies (e.g., Adamek et al., 2011) and research on typically developing children (e.g., Goodman, 2007; Goodman & Gotlib, 1999; Kovacs, Joorman, & Gotlib, 2008; Martin & Bridger, 1999), high levels of negative affect were associated with higher levels of parent-reported internalizing and externalizing problems at baseline as well as less reduction over childhood and adolescence for both adolescents diagnosed with HFA and non-ASD adolescents. Furthermore, within children diagnosed with HFA, high levels of negative affect were associated with greater baseline levels of social reciprocity difficulties, and less reduction across adolescence. Emotions such as fear, anger, frustration, sadness, and negative loadings of soothability comprise the dimensions of Rothbart's temperamental model of negative affect (Ahadi et al., 1993; Rothbart et al., 2001). Research has indicated that different aspects of negative affect are associated with both internalizing and externalizing problems. In general, fear related negative affect is related to internalizing problems, while anger and irritability are particularly related to externalizing problems (Rothbart, Ahadi, et al., 1994). Thus, it was not surprising that

negative affect was associated with both internalizing and externalizing problems concurrently and longitudinally.

It was interesting to note that the association between negative affect and internalizing problems was particularly important for adolescents diagnosed with HFA both at baseline and over the study period. The significant interaction indicated that high levels of negative affect were risk factors and low levels of negative affect were protective factors for development of internalizing problems, and this association was enhanced within children diagnosed with autism (compared to non-ASD children). The combination of deficits associated with core characteristics of autism, including difficulties understanding and interacting in social situations, in combination with high levels of fear, frustration, and anger may prevent these children from showing the normative decrease in internalizing problems, and therefore serves to maintain elevated levels of emotional maladjustment across adolescence.

A possible explanation for the particularly strong association between negative affect and internalizing problems in the children diagnosed with HFA may have to do with their reactions to common adolescent environmental stressors, including peer pressure and bullying. Reports indicate that children and adolescents diagnosed with ASD are at the greatest risk for experiencing repeated victimization due to their social impairments that are a manifestation of their disability (Blake, Lund, Zhou, Kwok, & Benz, 2012). Children with high negative affect have a biologically based tendency towards low frustration tolerance, difficulty regulating their negative emotions, and heightened irritability. Thus, when faced with stressors such as bullying, these children may not have the skills needed to cope with the negative emotions they experience. In

turn, this places these high negative affect children at a heightened risk for developing anxiety and depression over critical adolescent years. As described earlier, children with autism may be at the greatest risk for repeated peer victimization and children with higher functioning autism may be particularly affected because they have an awareness of their difficulties and bullying may be more salient to children diagnosed with HFA. While negative affect describes the ability to regulate ones negative emotions, the ability to regulate behavior and attention refers to one's EC.

Within the current study, EC predicted baseline levels of internalizing and externalizing problems. These findings were consistent with both ASD literature (e.g., Adamek et al., 2011; Burack, 1994; Chan et al., 2011; Goldstein et al., 2001; Nyden et al., 1999; Schmitz et al., 2006; Schwartz et al., 2009) as well as studies with typically developing children (e.g., Eisenberg et al., 2001, 2004, 2007, 2009; Martel et al., 2007; Krueger, Caspi, Moffit, White, & Stouthamer-Loeber, 1996; Lemery, Essex, & Smider, 2002). Core components of EC, including the ability to focus attention and shift from negative to positive thoughts and emotions, form the basis for effective emotion regulation (Derryberry & Reed, 2002; Derryberry & Rothbart, 1988; Erber & Tesser, 1992; Harman, Rothbart, & Posner, 1997; Silk, Steinberg, & Morris, 2003). Importantly, high levels of EC may increase a child's opportunity for participating in social exchange, which leads to more opportunities to engage in positive interactions with others. Learning from their enriching environment, children with high levels of EC have been reported to have enhanced empathy and conscience compared to low EC children (Eisenberg, 2000; Kochanska, 1997; Kochanska et al., 2000). On the other hand, low levels of EC may compromise a child's opportunity for interaction with peers (Salley &

Dixon, 2007; Todd & Dixon, 2010; Eisenberg, Hofer, & Vaughan, 2007) and is often associated with behavioral adjustment issues, such as aggression (Giancola & Zeichner, 1994; Seguin, Pihl, Harden, Tremblay, & Boulerice, 1995). Taken together, these findings suggest that EC may have profound effects on one's emotions and behavior.

A growing body of literature suggests that individual differences in EC may be attributed to developmental changes to the anterior cingulate cortex region (ACC) (Posner & Rothbart, 2007). Importantly, the ACC has also been thought to underlie some of the core symptoms of ASD (Haznedar, Buchsbaum, Metzger, Solimando, Spiegel-Cohen, & Hollander, 1997; Haznedar, Buchsbaum, Wei, Hof, Cartwright, et al., 2000; Mundy, 2003; Thakkar, Polli, Joseph, Tuch, Hadjikhani, et al., 2008; Vlamings, Jonkman, Hoeksma, van Engeland, & Kemner, 2008). This implies that EC may have biologically based links to ASD symptom presentation, including deficits in social reciprocity. Within the current study, children with HFA were rated by parents and themselves as having lower levels of EC on their first visit compared to non-ASD children, however, after controlling for both diagnostic group and initial levels of ASD symptoms, EC was not associated concurrently or longitudinally to social reciprocity on the SRS. This lack of association between EC and SRS in the growth models may indicate that initial ASD symptom levels account for the majority of the variance. Thus, EC was not a better predictor than diagnostic status of concurrent and longitudinal social reciprocity within this study.

The third temperamental factor examined, surgency, was inversely associated with baseline levels of externalizing problems. The surgency construct is comprised of ratings of positive emotion, impulsivity, and engagement with one's environment.

Individuals who have difficulty enjoying social situations may shy away from group interaction. Thus, not only are these children finding social engagement less enjoyable, but they also have less exposure to enriching environments over development (Rothbart & Derryberry, 1988).

In the current study, lower levels of *self*-reported surgency were associated with higher levels of self-reported externalizing problems at baseline. These results seem to contradict previous studies (Adamek et al., 2011; Newman et al., 1997; 2005), which indicate that higher levels of surgency are related to higher levels of externalizing problems. However, surgency may also be thought of as a protective factor for general adaptive functioning within typically developing children (Velez, 2011). Thus, high levels of approach behavior, particularly in children with ASDs, may support formal and informal social learning experiences and opportunities to develop adaptive coping skills, thus reducing their behavior problems and social reciprocity difficulties at an early age (Henderson & Wachs, 2007).

Intelligence as a Predictor for Greater Reductions in Internalizing and Externalizing Problems:

Verbal IQ was the only non-temperamental predictor of self-reported behavior problems across the full sample. Specifically, higher IQ was associated with greater reductions in internalizing and externalizing problems across adolescence. Results from this study corroborate previous literature, which report increased likelihood of improvements, and better outcome for emotional/behavioral problems in higher functioning children diagnosed with ASD (Mayes & Calhoun, 2011; Shattuck et al.,

2007; Taylor & Seltzer, 2010) and higher functioning typically developing children (Koenen et al., 2009; Masten et al., 2006). The current study expands upon the Shattuck et al. (2007) and Taylor and Seltzer (2010) findings by replicating results in an exclusively higher functioning sample (VIQ>70), whereas 68.5% of the sample in Shattuck et al.'s (2007) study and 63.2% of the sample in Taylor and Seltzer's (2010) study had an IQ lower than 70. Thus, results from the current study suggest that the effects of IQ on behavior can generalize to higher functioning children diagnosed with ASD. Interestingly, although high IQ was associated with greater reductions in problem behavior over the study period, these higher verbally functioning children, regardless of diagnostic group, self-reported more behavior problems at baseline. The concurrent association between IQ and anxiety has been reported previously among children with ASDs (Gadow, DeVincent, Pomeroy, & Azizian, 2005; Niditch, Varela, Kamps, & Hill, 2012; Weisbrot, Gadow, DeVincent, & Pomeroy, 2005). It may be that in children with HFA, internalizing problems develop secondary to ASD symptoms in response to social and environmental demands (Niditch et al., 2012). The current study extends prior findings by further documenting the importance of verbal IQ for the prediction of rates of change in behavior problems above and beyond the child's initial levels of problem behavior and their diagnosis. The clinical implications of this finding will be discussed further below.

Clinical Significance and Optimal Outcome:

An important addition to the growth analyses was the assessment of clinical significant change. These analyses enhanced the conclusions of social reciprocity

difficulties and comorbid problem reduction by addressing these changes in clinically meaningful terms. Children in the HFA group displayed an estimated average reduction of 10 points (equivalent to one standard deviation change in *T*-scores) from 8-19.5 years of age on the parent-rated SRS questionnaire. Furthermore, 16% (9 out of 58) children with HFA who were rating above clinical levels on the SRS on first assessment were subsequently rated in the subclinical range by their last assessment. Noteworthy, although only 15.52% of the HFA children moved from clinically elevated to subclinical levels on measures of social reciprocity, parent- and self-reported measures of internalizing and externalizing problems on the BASC-2 improved substantially (roughly 50-70%). This indicates that although social reciprocity difficulties may still persist over childhood and adolescence, significant clinical improvements in comorbid symptomatology can be seen. There were no systematic differences on measures of social reciprocity and problem behavior from children who completed multiple assessments verses children that only were assessed at time 1, implying that these results would generalize to the full sample. Thus, the data raise the question of whether these children displayed "optimal outcome."

This concept was first pioneered by Rutter (1970), who documented that 1.5% of adults who had a previous diagnosis of autism were functioning normally. Since then, many have investigated whether children with autism can display optimal outcomes. Consistent with the current findings, Sigman and Ruskin (1999) reported that 17% of their sample lost their ASD diagnosis, and "optimal outcome" was seen more among the higher verbally and cognitively functioning adolescents. Helt et al. (2008) concluded that roughly 3 to 25% of individuals with ASD eventually lose their diagnosis from childhood

to adulthood. It should be noted that although children may not meet criteria on conventional autism assessments, they may develop other comorbid symptoms or psychopathology. Thus, it is important to assess not only ASD diagnosis, but also adaptive functioning and comorbidity. Fein et al. (2013) conducted the first thorough investigation of adaptive functioning in children they deemed to have optimal developmental outcomes. Fein et al. (2013) assessed the behavioral and symptom differences of 34 "optimal outcome" participants, 8- 21 years old, in comparison to 44 individuals diagnosed with HFA and 34 typically developing individuals matched on gender, age, and nonverbal IQ. The optimal outcome participants were classified within this group if they displayed a clear diagnosis of autism before the age of 5, and subsequently did not meet ASD criteria at the initiation of the study as per the ADOS and clinical judgment (roughly 7 years later). Results indicated that the optimal outcome children could not be differentiated from a typically developing group in adaptive functioning. Furthermore, milder social difficulties early in development differentiated the optimal outcome group from children diagnosed with HFA. Although participants in the current study did not receive repeated diagnostic assessments (i.e., repeated ADOS and ADI-R administrations), the longitudinal analyses support this promising story of ASD related symptom (social reciprocity) reduction and improvements in comorbid symptoms from childhood through adolescence.

Implications for Practice:

These findings broaden our understanding of the relation between temperament and social reciprocity and comorbid behavior problems, and demonstrate the influence of

temperament on the developmental trajectories of all children – those with and without autism. Although results from the current study in conjunction with past literature support the role of early temperament in later development, the mechanisms through which temperament influence development are not fully understood. One potential mechanism is the "goodness-of-fit" between a child's temperament and the demands of his/her environment (Chess & Thomas, 1991, 1996, 1999; Thomas & Chess, 1977). Awareness of individual differences in temperament may help parents, teachers, and clinicians understand that the same environment may be experienced, and responded to, differently by children based on their temperament (Rothbart, Ahadi, & Hershey, 1994). Thus, the environment should be adapted to accommodate a child's needs for goodness-of-fit. For example, a child who possesses high levels of negative affect can present difficulties for teachers, parents, and clinicians, as this child would be more likely to become easily frustrated on difficult tasks and have difficulty regulating their anger. This child may act out as a result of his frustration or may become anxious. Therefore, a sensitive teacher or practitioner may select an activity that takes a shorter time to complete and break down the components to make the task easier to understand. In this way, knowledge of the influence of temperament should be embedded in teacher education, parenting styles, and therapy. Another way clinicians and practitioners can address temperamental influences on behavior is by targeting skills directly associated with deficits related to negative affect, such as mindfulness-based therapy.

Although temperament is conceptualized as a constellation of relatively stable reaction tendencies, children's abilities to regulate these reactions may be amenable to direct training. Teaching a child skills to enhance self regulation of one's fears and

frustrations, in addition to managing their emotional reactivity may have positive effects on temperamental negative affect, and in turn reduce or alleviate problem behaviors (Hofmann, Sawyer, Witt, & Oh, 2010; Kabat-Zinn 1990; Teasdale, Segal, & Williams, 1995). This concept forms the basis of mindfulness-based therapy (MBT). MBT has proven efficacy in treating mood disorders and behavior problems in clinical populations (Hofmann, Sawyer, Witt, & Oh, 2010; Teasdale, Segal, & Williams, 1995), and more recently, in HFA adolescents and adults (Bogels, Hoogstad, van Dun, de Singh et al., 2011; Spek et al., 2013; Singh et al., 2011). Early research into the effects of MBT in individuals with ASD report improvements in social interaction, concentration, impulsive behaviors, aggression, anxiety, and depression. For children with high levels of negative affect, therapy such as MBT, may help mitigate the long-term negative effects that develop as a result of this type of temperamental profile. Given the particularly strong coupling of negative affect and internalizing problems in the children diagnosed with HFA, such therapies may be particularly effective for reducing emotional problems in children with HFA.

Lastly, practitioners and clinicians should be highly aware of the association between high IQ and emotional/behavioral problems in early childhood. This may be particularly important among children diagnosed with ASD due to the compounded effects of having social deficits. Research indicates that emotional difficulties, such as anxiety and depression, often go undiagnosed as a separate disorder in children with ASD, and therefore go untreated (Bryson, 1996; Bryson & Smith, 1998; Gillberg & Billstedt, 2000; McNeil, et al., 2008; Tantam, 2000). This often leads to poorer outcomes as children transition into adulthood (Kim et al., 2000). Thus, identifying and treating

comorbid symptomatology in children with ASD is critical for positive developmental outcome. Therapies, such as cognitive behavior therapy (CBT), have been empirically validated and efficacious for use in higher functioning children with autism (Cardaciotto & Hebert, 2004; Chalfant et al., 2007; Lehmkuhl, Storch, Bodfish, & Gefken, 2008), as well as non-ASD children (Taylor, Lindsay, & Willner, 2008). Chalfant et al., (2007) reported significant anxiety symptom reduction in 47 children diagnosed with HFA (8-13 years old) using a family based, cognitive behavioural treatment. Thus, for children with higher cognitive functioning, it may be integral to utilize validated treatments, such as CBT, to ameliorate elevations in emotional and behavioral problems in early childhood.

Limitations and Future Directions:

Results from this study isolated lower levels of negative affect, higher levels of EC and surgency as protective factors against concurrent childhood behavior problems. Lower levels of negative affect and higher IQ were further isolated as supporting more optimal change in behavior problems. However, a limitation of the current study was the exclusive focus on developmental predictors in a person-oriented perspective. Future studies should also investigate the effects of environmental factors including treatment and intervention, parenting styles, and SES, on developmental trajectories. An additional limitation of the current study was that it did not assess the exact nature of interventions given to children (e.g., types of interventions, length of intervention received, etc.) and thus improvements in symptoms may be due in part to treatment and the relations of temperament to differential responses to treatment as opposed to time itself. Furthermore, controlling for baseline levels of the outcome variable in the growth model

confounds the interpretation of the results. Due to the significant and strong correlations between initial starting point and amount of change, intercept was controlled for so that predictions could be drawn about the variables associated with growth. However, in actuality, the children who improved the most were those who started out with the greatest levels of impairment. Finally, new lines of developmental research have used cluster analysis to group individuals with similar temperamental profiles together. These clustered groups are then used as the basis for analyzing developmental change. These methods allow for addressing issues of development in terms of a holistic profile as opposed to observing single temperamental factors as units, such as used in this study. Furthermore, study methods using HLM6 lack the sophistication to track all growth trajectories in a single model. Research suggests that internalizing, externalizing, and ASD related problem behaviors are bidirectionally associated (Hallett, Ronald, Rijsdijk, & Happe, 2010). Further validating this claim, there were significant concurrent correlations between symptoms and behavior problems. Thus, the use of structural equation modeling (SEM) and cross-lagged analysis can be a powerful tool in understanding the continuous interplay between outcome variables and how temperamental predictors affect change.

One of the benefits of using the Social Responsiveness Scale (SRS) is that it has been clinically validated in both non-ASD and autism samples. Thus, it can provide valuable information on a full range of participants. However, it should be noted that Hus et al. (2013) suggested that scores on the SRS total score (which was used in the current study as an outcome variable and optimal outcome post hoc analyses) may reflect general levels of impairment, as opposed to severity of ASD specific symptoms or social

impairments, because it contains queries about emotional and behavioral problems non-specific to an ASD diagnosis. As a result, Hus et al. (2013) suggest that comorbid behavior problems may inflate SRS total scores. Thus, the improvements seen in the SRS over childhood and adolescence may not only be assessing pure social reciprocity deficits, but may also be capturing comorbid symptoms of internalizing and externalizing problems. As noted earlier, future directions may utilize SEM to account for the overlap in assessments measuring internalizing and externalizing problems and the SRS.

The investigation of optimal outcome and recovery has become a critical topic of recent in the autism community. While the current study provides preliminary results on clinically significant reductions in social reciprocity difficulties and emotional/behavioral problems in higher functioning children diagnosed with autism, these results were not the main focus of the project. Thus, conclusions on optimal outcome and recovery cannot be made with certainty. Future studies should conduct prospective, comprehensive ASD workups, including multiple assessments on the ADOS and ADI-R, to assess true optimal outcome. Within this study design, it would be interesting to assess temperament as a predictor for optimal outcome.

Summary:

In conclusion, the current study provides evidence for the influence of within-child factors including temperament and verbal IQ on baseline levels and development of social reciprocity, internalizing, and externalizing problems. Lower levels of negative affect, higher levels of EC, and higher levels of surgency served as protective factors, associated with lower levels of problem behavior at baseline. Furthermore, low levels of

negative affect and higher verbal IQ were associated with greater reductions in emotional/behavioral problems and social reciprocity difficulties over childhood and adolescence (8-19.5 years). Importantly, the patterns of associations were more similar than different for the children diagnosed with HFA and non-ASD children. Findings from this study were largely consistent with previous studies examining the concurrent and longitudinal relations between temperament and social adaptation in typically developing children and children on the spectrum and emphasize the need for temperament evaluations for treatment utility.

References

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4–18 and 1991 Profile.* Burlington, VT: Department of Psychiatry, University of Vermont.

Achenbach, T. M. (1992). *Manual for the Child Behavior Checklist/2–3 and 1992 Profile*. Burlington, VT: Department of Psychiatry, University of Vermont.

Achenbach, T. M., Howell, C. T., Quay, H. C., & Conners, C. K. (1991). National survey of problems and competencies among 4- to 16-year olds: Parents' reports for normative and clinical samples. *Monographs of the Society for Research in Child Development, 56,* 1–131.

Adamek, L., Nichols, S., Tetenbaum, S. P., Bregman, J., Ponzio, C. A., & Carr, E. G. (2011). Individual temperament and problem behavior in children with autism spectrum disorder. *Focus on Autism and Other Developmental Disabilities, 26*(3), 173-183.

Allik, H., Larsson, J. O., & Smedje, H. (2006). Health-related quality of life in parents of school age children with Asperger Syndrome or Higher-Functioning Autism. *Health and Quality of Life Outcomes, 4,* 1.

American Psychiatric Association (APA). 2000. *Diagnostic and Statistical Manual of Mental Disorders: DSM-IV-TR.* Washington, DC: American Psychiatric Association.

Angold, A., Costello, E. J., & Worthman, C. M. (1998). Puberty and depression: The roles of age, pubertal status and pubertal timing. *Psychological Medicine, 28,* 51-61.

Barkley, R. A. (2006). Comorbid disorders, social and family adjustment, and subtypes. R.A. Barkley (Ed.), *ADHD: A Handbook for Diagnosis and Treatment*, Guilford, NY, 184-218.

Bellini, S. (2004). Social skill deficits and anxiety in high-functioning adolescents with autism spectrum disorders. *Focus on Autism and Other Developmental Disabilities, 19*(2), 78–86.

Billstedt, E., Gillberg, I. C., & Gillberg, C. (2007). Autism in adults: Symptom patterns and early childhood predictors. Use of the DISCO in a community sample followed since childhood. *Journal of Child Psychology and Psychiatry, 48,* 1102-1110.

Birmaher, B., Ryan, N. D., Williamson, D. E., Brent, D. A., Kaufman, J., Dahl, R. E., et al. (1996). Childhood and adolescent depression: A review of the past 10 years, Part I. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 1427-1439.

Blake, J. J., Lund, E. M., Zhou, Q., Kwok, O-M, & Benz, M. R. (2012). National prevalence rates of bully victimization among students with disabilities in the United States. *School Psychology Quarterly, 27*(4), 210-222.

Bongers, I. L., Koot, H. M., van der Ende, J., & Verhulst, F. C. (2004). Developmental trajectories of externalizing behaviors in childhood and adolescence. *Child Development, 75*, 1523–1537.

Bongers, I. L., Koot, H. M., van der Ende, J., & Verhulst, F. C. (2003). The normative development of child and adolescent problem behavior. *Journal of Abnormal Psychology, 112*, 179–192.

Booth-LaForce, C., & Oxford, M. L. (2008). Trajectories of social withdrawal from Grades 1 to 6: Prediction from early parenting, attachment, and temperament. *Developmental Psychology, 44,* 1298–1313.

Brame, B., Nagin, D. S., & Tremblay, R. E. (2001). Developmental trajectories of physical aggression from school entry to late adolescence. *Journal of Child Psychology and Psychiatry 42*, 503–512.

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,* 863-870.

Bryk, A. S., & Raudenbush, S. W. (1987). Application of hierarchical linear modeling to assessing change. *Psychological Bulletin, 101,* 147– 158.

Bryson, S. (1996). Brief report: Epidemiology of autism. *Journal of Autism and Developmental Disorders, 26*(2), 165-167.

Bryson, S., & Smith, I. (1998). Epidemiology of Autism: Prevalence, associated characteristics, and implications for research and service delivery. *Mental Retardation and Developmental Disabilities Research Reviews, 4,* 97-103.

Bryson, S.E., Zwaigenbaum, L., Brian, J., Roberts, W., Szatmari, P., Rombough, V., et al. (2007). A prospective case series of high-risk infants who developed autism. *Journal of Autism and Developmental Disorders, 37*, 12-24.

Bub, K. L., McCartney, K., & Willett, J. B. (2007). Behavior problem trajectories and first-grade cognitive ability and achievement skills: A latent growth curve analysis. *Journal of Educational Psychology, 99,* 653–670.

Burack, J. A. (1994). Selective attention deficits in persons with autism: Preliminary evidence of an inefficient lens. *Journal of Abnormal Psychology, 103*, 535–543.

Caldwell, M. S., Rudolph, K. D., Troop-Gordon, W., & Kim, D.-Y. (2004). Reciprocal influences among relational self-views, social disengagement, and peer stress during early adolescence. *Child Development, 75*(4), 1140–1154.

Calhoun, S. L., & Mayes, S. D. (2005). Processing speed in children with clinical disorders. *Psychology in the Schools, 42,* 333-343.

Campbell, S. B. (1995). Behavior problems in preschool children: A review of recent research. *Journal of Child Psychology and Allied Disciplines, 36,* 113–149.

Cardaciotto, L., & Herbert, J. (2004). Cognitive behavior therapy for social anxiety disorder in the context of Asperger's syndrome: A single-subject report. *Cognitive Behavioral Practice, 11*, 75-81.

Carter, A. S, Black, D. O., Tewani, S., Connolly, C. E., Kadlec, M. B., & Tager-Flusberg, H. (2007). Sex differences in toddlers with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 37*, 86–97.

Carver, C. S. (2004). Negative affects deriving from the behavioral approach system. *Emotion, 4*, 3–22.

Casey, B. J., Jones, R. M., & Hare, T. A. (2008). The adolescent brain. *Annals of the New York Academy of Science, 1124,* 111-126.

Caspi, A., & Shiner, R. L. (2006). Personality development. In W. Damon, R. Lerner, & N. Eisenberg (Eds.), *Handbook of Child Psychology: Vol. 3.* (6th ed., pp. 300–364). New York: Wiley.

Chalfant, A., Rapee, R., & Carroll, L. (2007). Treating anxiety disorders in children with high functioning autism spectrum disorders: A controlled trial. *Journal of Autism and Developmental Disorders, 37*, 1842-1857.

Chamberlain, B., Kasari, C., & Rotheram-Fuller, E. (2007). Involvement or isolation? The social networks of children with autism in regular classrooms. *Journal of Autism and Developmental Disorders, 37*(2), 230–242.

Chan, A. S., Han, Y. M., Leung, W. W., Leung, C., Wong, V. C., & Cheung, M-C. (2011). Abnormalities in the anterior cingulate cortex associated with attentional and inhibitory control deficits: A neurophysiological study on children with autism spectrum disorder. *Research in Autism Spectrum Disorders, 5*(1), 254-266.

Chess, S., & Thomas, A. (1991). Temperament and the concept of goodness of fit. In J. Strelau & A. Angleitner (Eds.), *Explorations in temperament: International perspectives on theory and measurement. Perspectives on individual differences*. New York: Plenum, pp. 15–28.

Chess, S., & Thomas, A. (1996). *Temperament. Theory and practice*. New York: Brunner Mazel.

Chess, S., & Thomas, A. (1999). *Goodness of fit. Clinical Applications from infancy through adult life*. Philadelphia: Brunner Mazel.

Chorpita, B. F., & Barlow, D. H. (1998). The development of anxiety: The role of control in the early environment. *Psychological Bulletin, 124*(1), 3–21.

Cohen, P., Cohen, J., Kasen, S., Velez, C. N., Hartmark, C., Johnson, J., et al. (1993). An epidemiological study of disorders in late childhood and adolescence: I. Age- and gender-specific prevalence. *Journal of Child Psychology and Psychiatry, 34,* 851-867.

Coie, J. D., & Dodge, K. A. (1998). Aggression and antisocial behavior. In W. Damon & N. Eisenberg (Eds.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (pp. 779–862). New York, NY: Wiley.

Colder, C. R., & O'Connor, R. M. (2004). Gray's reinforcement sensitivity model and child psychopathology: laboratory and questionnaire assessment of the BAS and BIS. *Journal of Abnormal Child Psychology, 32*(4), 435-451.

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development, 75*, 317– 333.

Connor, D.F., Steeber, J., & McBurnett, K. (2010). A review of attention-deficit/hyperactivity disorder complicated by symptoms of oppositional defiant disorder or conduct disorder. *Journal of Developmental and Behavioral Pediatrics, 31,* 427-440.

Constantino, J. N., & Gruber, C. P. (2005). Social Responsiveness Scale. Los Angeles: Western Psychological Services.

Constantino, J. N., LaVesser, P. D., Zhang, Y., Abbacchi, A. M., Gray, T., & Todd, R. D. (2007). Rapid quantitative assessment of autistic social impairment by classroom teachers. *Journal of the American Academy of Child and Adolescent Psychiatry, 46*(12), 1668–1676.

Cook, C. R., Williams, K. R., Guerra, N. G., Kim, T. E., & Shadek, S. (2010). Predictors of bullying and victimization in childhood and adolescence: A meta-analytic investigation. *School Psychology Quarterly, 25*(2), 65–83.

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*, 210-222.

Craske, M. G. (1997). Fear and anxiety in children and adolescents. *Bulletin of the Menninger Clinic, 61*, A4-A36.

Crocetti, E., Klimstra, T., Keijsers, T., Hale, W. W., III, & Meeus, W. (2009). Anxiety trajectories and identity development in adoles- cence: a five-wave longitudinal study. *Journal of Youth and Adolescence, 38*, 839–849.

Davis, N. O., & Carter, A. S. (2008). Parenting stress in mothers and fathers of toddlers with Autism Spectrum Disorder: Associations with child characteristics. *Journal of Autism and Developmental Disorders, 38*(7), 1278-1291.

Dawson, G., & Osterling, J. (1997). Early intervention in autism. In M. J. Guralnick (Ed.) *The effectiveness of early intervention* (pp. 307–326). Baltimore, MD: Paul H. Brookes.

De Los Reyes, A., & Kazdin, A. E. (2004). Measuring informant discrepancies in clinical child research. *Psychological Assessment, 16*, 330–334.

Depue, R. A., & Collins, P. F. (1999). Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. *Behavioral and Brain Science, 22,* 491-517.

Derryberry, D., & Reed, M. (2002). Anxiety-related attentional biases and their regulation by attentional control. *Journal of Abnormal Psychology, 111*, 225-236.

Derryberry, D., & Rothbart, M. K. (1988). Arousal, affect, and attention as components of temperament. *Journal of Personality and Social Psychology, 55,* 958-966.

Dominick, K. C., Davis, N. O., Lainhart, J., Tager-Flusberg, H., & Folstein, S. (2007). Atypical behaviors in children with autism and children with a history of language impairment. *Research in Developmental Disabilities, 28*(2), 145-162.

Dominguez, X., Vitiello, V. E., Maier, M. F., & Greenfield, D. B. (2010). A longitudinal examination of young children's learning behavior: child-level and classroom-level predictors of change throughout the preschool year. *School Psychology Review, 39*(1), 29-47.

Eaves, L. C., Ho, H. H., & Eaves, D. M. (1994). Subtypes of autism by cluster-analysis. *Journal of Autism and Developmental Disorders, 24*, 3–22.

Efron, D., & Sciberras, E. (2010). The diagnostic outcomes of children with suspected attention deficit hyperactivity disorder following multidisciplinary assessment. *Journal of Pediatrics and Child Health, 46,* 392-397.

Ehlers, S., Gillberg, C., & Wing, L. (1999). A screening questionnaire for Asperger syndrome and other high-functioning autim spectrum disorders in school age children. *Journal of Autism and Developmental Disorders, 28*, 527-533.

Eisenberg, N., Cumberland, A., Spinrad, T. L., Fabes, R. A., Shepard, S. A., Reiser, M., et al. (2001). The relations of regulation and emotionality to children's externalizing and internalizing problem behavior. *Child Development, 72,* 1112-1134.

Eisenberg, N., Fabes, R. A., Guthrie, I. K., & Reiser, M. (2000). Dispositional emotionality and regulation: Their role in predicting quality of social functioning. *Journal of Personality and Social Psychology, 78,* 136-157.

Eisenberg, N., Hofer, C., & Vaughan, J. (2007). EC and its socioemotional consequences. In J. J. Gross (Ed.), Handbook of emotion regulation (pp. 287–306). New York: Guilford.

Eisenberg, N., Ma, Y., Chang, L., Chang, L., Zhou, Q., West, S. G., & Aiken, L. (2007). Relations of EC, reactive under control, and anger to Chinese children's adjustment. *Development and Psychopathology, 19,* 385-409.

Eisenberg, N., & Morris, A. (2002). Children's emotion-related regulation. In R. V.Kail (Ed.), *Advances in Child Development and Behavior* (Vol. 30, pp. 189– 229). San Diego, CA: Academic Press.

Eisenberg, N., Spinrad, T. L., Fabes, R. A., Reiser, M., Cumberland, A., Shepard, S. A., et al. (2004). The relations of EC and impulsivity to children's resiliency and adjustment. *Child Development, 75,* 25-46.

Eisenberg, N., Valiente, C., Spinrad, T. L., Cumberland, A., Liew, J., Reiser, M., Zhou, Q., & Losoya, S.H. (2009). Longitudinal relations of children's EC, impulsivity, and negative emotionality to their externalizing, internalizing, and co-occurring behavior problems. *Developmental Psychology, 45*(4), 988-1008.

Ellenbogen, M. A., & Hodgins, S. (2004). The impact of high neuroticism in parents on children's psychosocial functioning in a population at high risk for major affective disorder: a family-environmental pathway of intergenerational risk. *Developmental Psychopathology, 16,* 113-136.

Ellis, L. K., & Rothbart, M. K. (2001). Revision of the Early Adolescent Temperament Questionnaire. Poster presented at the Biennial Meeting of the Society for Research in Child Development. Minneapolis, Minnesota.

Erber, R., & Tesser, A. (1992). Task effort and the regulation of mood: The absorption hypothesis. *Journal of Experimental Social Psychology, 28,* 339-359.

Ernst, M., Nelson, E. E., Jazbec, S., McClure, E. B., Monk, C.S., Leibenluft, E., et al. (2005). Amygdala and nucleus accumbens in responses to receipt and omission of gains in adults and adolescents. *Neuroimage, 25*(4), 1279–1291.

Eronen, S., Nurmi, J.E., & Salmela-Aro, K. (1997). Planning-oriented, avoidant, and impulsive social reaction styles: A person-oriented approach. *Journal of Research in Personality, 31*, 34-57.

Fecteau, S., Mottron, L., Berthiaume, C., & Burack, J. A. (2003). Developmental changes of autistic symptoms. *Autism, 7*(3), 255-268.

Fein, D., Barton, M., Eigsti, I-M., Kelley, E., Naigles, L., Schultz, R.T. et al. (2013). Optimal outcome in individuals with a history of autism. *Journal of Child Psychiatry, 54*(2), 195-205.

Fleming, J. E., & Offord, D. R. (1990). Epidemiology of childhood depressive disorders: A critical review. *Journal of the American Academy of Child and Adolescent Psychiatry, 29*, 571-580.

Fox, N. A., Henderson, H. A., Rubin, K. H., Calkins, S. D., & Schmidt, L. A. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. *Child Development, 72*, 1– 21.

Frederickson, B. L. (2004). The broad-and-build theory of positive emotions. *Philosophical Transaction of the Royal Society of London B: Biological Science, 359,* 1367-1377.

Frick, P. J., Lahey, B. B., Loeber, R., Tannenbaum, L., Van Horn, Y., Christ, M. A. G., Hart, E. L., & Hanson, K. (1993). Oppositional defiant disorder and conduct disorder: a meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clinical Psychology Review, 13*, 319–340.

Gadow, K. D., DeVincent, C. J., & Pomeroy, J. C. (2006). ADHD symptom subtypes in children with pervasive developmental disorder. *Journal of Autism and Developmental Disorders, 36*, 271-284.

Gadow, K. D., DeVincent, C. J., Pomeroy, J., & Azizian, A. (2005). Psychiatric symptoms in preschool children with PDD and clinic and comparison samples. *Journal of Autism and Developmental Disorders, 34*, 379–393.

Garon, N., Bryson, S., Zwaigenbaum, L., Smith, I., Brian, J., Roberts, W., et al. (2009). Temperament and its relationship to autistic symptoms in a high-risk infant sib cohort. *Journal of Abnormal Child Psychology, 37*, 59–78.

Giancola, P. R., & Zeichner, A. (1994). Neuropsychological performance on tests of frontal-lobe functioning and aggressive behavior in men. *Journal of Abnormal Psychology, 103*, 832–835.

Gillberg, C., & Billstedt, E. (2000). Autism and Asperger syndrome: Coexistence with other clinical disorders. *Acta Psychiatrica Scandinavica, 102*, 321-330.

Gilliom, M., & Shaw, D. S. (2004). Co-development of externalizing and internalizing problems in early childhood. *Development and Psychopathology, 16,* 313–333.

Gillott, A., Furniss, F., & Walter, A. (2001). Anxiety in high functioning children with autism. *Autism, 5*(3), 277–286.

Goldstein, G., Johnson, C. R., & Minshew, N. J. (2001). Attentional processes in autism. *Journal of Autism and Developmental Disorders, 31*, 433–440.

Goldstein, S., & Schwebach, A. (2004). The comobidity of Pervasive Developmental Disorder and Attention Deficit Hyperactivity Disorder: Results of a retrospective chart review. *Journal of Autism and Developmental Disorders, 34,* 329-339.

Goodman, S. H. (2007). Depression in mothers. *Annual Review of Clinical Psychology, 3*, 107– 135.

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review, 106*, 458– 490.

Gray, J. A. (1982). *The neuropsychology of anxiety*: An inquiry into the functions of the septohippocampal system. New York: Oxford University Press.

Gray, J. A., & McNaughton, N. (2000). *The neurophysiology of anxiety: An inquiry into the function of the septo-hippocampal system* (2nd ed.). Oxford, England: Oxford University Press.

Green J., Gilchrist, A., Burton, D., & Cox, A. (2000). Social and psychiatric functioning in adolescents with Asperger syndrome compared with conduct disorder. *Journal of Autism and Developmental Disorders, 30,* 279-293.

Hallett, V., Ronald, A., Rijsdijk, F., & Happe, F. (2010). Association of autistic-like and internalizing traits during childhood: a longitudinal twin study. *American Journal of Psychiatry, 167*(7), 809-817.

Hancock, G. R., & Mueller, R. O. (Eds.). (2006). *Structural equation modeling: A second course*. Greenwich, CT: Information Age Publishing.

Happe, F., Booth, R., Charlton, R., & Hughes, C. (2006). Executive function deficits in autism spectrum disorders and attention-deficit/hyperactivity disorder: Examining profiles across domains and ages. *Brain and Cognition, 61,* 25-39.

Harman, C., Rothbart, M. K., & Posner, M. I. (1997). Distress and attention interactions in early infancy. *Motivation and Emotion, 21,* 27-43.

Hartley, S. L., & Sikora, D. M. (2009). Which DSM-IV-TR criteria best differentiate high-functioning autism spectrum disorder from ADHD and anxiety disorders in older children? *Autism, 13,* 485-509.

Haznedar, M. M., Buchsbaum, M. S., Metzger, M., Solimando, A., Spiegel-Cohen, J., & Hollander, E. (1997). Anterior cingulate gyrus volume and glucose metabolism in autistic disorder. *American Journal of Psychiatry, 154*, 1047–1050.

Haznedar, M., Buchsbaum, M., Wei, T., Hof, P., Cartwright, C., Bienstock, C., & Hollander, E. (2000). Limbic circuitry in patients with autism spectrum disorders studied with positron emission tomography and magnetic resonance imaging. *American Journal of Psychiatry, 157*, 1994–2001.

Helt, M., Kelley, E., Kinsbourne, M., Pandey, J., Boorstein, H., Herbert, M., & Fein, D. (2008). Can children with autism recover? If so, how? *Neuropsychology Review, 18,* 339-366.

Henderson, H. A., & Wachs, T. (2007). Temperamental theory and the study of cognition-emotion interactions across development. *Developmental Review, 27*, 396-427.

Hepburn, S. L., & Stone, W. L. (2006). Using Carey temperament scales to assess behavioral style in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 36*, 637–642.

Hofmann, S. G., Sawyer, A. T., Witt, A. A., & Oh, D. (2010). The effect of mindfulness-based therapy on anxiety and depression: A meta-analytic review. *Journal of Consulting and Clinical Psychology, 78*, 169–183.

Hoglund, W. L. (2007). School functioning in early adolescence: Gender-linked responses to peer victimization. *Journal of Educational Psychology, 99,* 683–699.

Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry, 45*(2), 212-229.

Howlin, P., Mawhood, L., & Rutter, M. (2000). Autism and developmental receptive language disorder. A follow-up comparison in early adult. II: Social, behavioural, and psychiatric outcomes. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 41,* 561-578.

Huber, G. P., & Power, D. J. (1985). Retrospective reports of strategic-level managers: Guidelines for increasing their accuracy. *Strategic Management Journal, 6*(2), 171-180.

Hus, V., Bishop, S., Gotham, K., Huerta, M., & Lord, C. (2013). Factors influencing scores on the social responsiveness scale. *Journal of Child Psychiatry and Psychology*, *54,* 216–224.

Isbell, L. M., Burns, K. C., & Haar, T. (2005). The role of affect on the search for global and specific target information. *Social Cognition, 23,* 529-552.

Isen, A. (2008). Some ways in which positive affect influences decision making and problem solving. In M. Lewis & J. Haviland-Jones (eds.), *Handbook of emotions* (3rd ed., pp. 548-573). New York, NY: Guilford Press.

Johnson, K. J., & Fredrickson, B. L. (2005). "We all look the same to me": Positive emotions eliminate the own-race bias in face recognition. *Psychological Science, 16*, 875-881.

Johnson, S. L., Turner, R. J., & Iwata, N. (2003). BIS/BAS levels and psychiatric disorder: An epidemiological study. *Journal of Psychopathology and Behavioral Assessment, 25,* 25–36.

Kabat-Zinn, J. (1990). Full catastrophe living: Using the wisdom of your body and mind to face stress, pain, and illness. New York: Delacourt.

Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. *Child Development, 58*(6), 1459-1473.

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.

Keenan, K., Shaw, D. S., Delliquadri, E., Giovannelli, J., & Walsh, B. (1998). Evidence for the continuity of early problem behaviors: Application of a developmental method. *Journal of Abnormal Child Psychology, 26,* 441–454.

Karevold, E., Røysamb, E., Ystrom, E., & Mathiesen, K. S. (2009). Predictors and pathways from infancy to anxiety and depression in early adolescence. *Developmental Psychology, 45*(4), 1051-1060.

Kasch, K. L., Rottenberg, J., Arnow, B. A. & Gotlib, I. H. (2002). Behavioural activation and inhibition systems and the severity and course of depression. *Journal of Abnormal Psychology, 111*, 589 – 597.

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*, 117- 132.

Kincade, S. R., & McBride, D. L. (2009). CBT and autism spectrum disorders: A comprehensive literature review. *Online Submission*, 105.

Kline, R. B. (Ed.). (2005). *Principles and practice of structural equation modeling (2nd ed.).* New York, NY: The Guilford Press.

Kochanska, G. (1997). Multiple pathways to conscience for children with different temperaments: *From Toddlerhood to age 5. Developmental Psychology, 33*, 228–240.

Kochanska, G., & Knaack, A. (2003). EC as a personality characteristic of young children: Antecedents, correlates, and consequences. *Journal of Personality, 71,* 1087-1112.

Kochanska, G., Murray, L. T., & Harlan, E. T. (2000). EC in early childhood: Continuity and change, antecedents, and implications of social development. *Developmental Psychology, 36,* 220-232.

Koenen, K. C., Moffitt, T. E., Roberts, A. L. Martin, L. T., Kubzansky, L., Harrington, H. et al. (2009). Childhood IQ and adult mental disorders: A test of the cognitive reverse hypothesis. *American Journal of Psychiatry, 166*(1), 50-57.

Komsi, N., Raikkonen, K., Pesonen, A., Heinonen, K., Keskivaara, P., Jarvenpaa, A. et al. (2006). Continuity of temperament from infancy to middle childhood. *Infant Behavior and Development, 29,* 494-508.

Konstantareas, M. M., & Stewart, K. (2006). Affect regulation and temperament in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 36*(2), 143–154.

Kovacs, M., Joormann, J., & Gotlib, I. H. (2008). Emotion (dys)regulation and links to depressive disorders. *Child Development Perspectives, 2*, 149– 155.

Kranner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Childhood Schizophrenia, 1,* 119-145.

Krueger, R.F., Caspi, A., Moffit, T.E., White, J., & Stouthamer-Loeber, M. (1996). Delay of gratification, psychopathology, and personality: Is low self-control specific to externalizing problems? *Journal of Personality, 64*, 107-129.

Kupersmidt, J. B., Coie, J. D., & Dodge, K. A. (1990). The role of poor peer relationships in the development of disorder. In S. R. Asher & J. D. Coie (Eds.), *Peer rejection in childhood* (pp. 274– 305). New York, NY: Cambridge University Press.

Ladd, G. W. (2006). Peer rejection, aggressive or withdrawn behavior, and psychological maladjustment from ages 5 to 12: An examination of four predictive models. *Child Development, 77*(4), 822–846.

Ladd, G. W., & Troop-Gordon, W. (2003). The role of chronic peer difficulties in the development of children's psychological adjustment problems. *Child Development, 74*(5), 1344–1367.

Laitinen-Krispijn, S., Van der Ender, J., & Verhulst, F.C. (1999). The role of the pubertal progress in the development of depression in early adolescence. *Journal of Affective Disorders, 54,* 211-215.

Lane, A. E., Young, R. L., Baker, A. E., & Angley, M. T. (2010). Sensory processing subtypes in autism: Association with adaptive behavior. *Journal of Autism and Developmental Disorders, 40*(1), 112-122.

Lavigne, J. V., Arend, R., Rosenbaum, D., Binns, H. J., Christoffel, K. K., & Gibbons, R. D. (1998). Psychiatric disorders with onset in the preschool years: II. Correlates and predictors of stable case status. *Journal of the American Academy of Child & Adolescent Psychiatry, 37*, 1255–1261.

Lecavalier, L. (2006). Behavioral and emotional problems in young people with pervasive developmental disorders: Relative prevalence, effects of subject characteristics, and empirical classification. *Journal of Autism and Developmental Disorders, 36,* 1101-1114.

Lehmkuhl, H., Storch, E., Bodfish, J., & Gefken, G. (2008). Brief report: Exposure and response prevention for obsessive compulsive disorder in a 12-year-old with autism. *Journal of Autism and Developmental Disorders, 38*, 977-981.

Lemery, K. S., Essex, M. J., & Smider, N. A. (2002). Revealing the relation between temperament and behavior problem symptoms by eliminating measurement confounding: Expert ratings and factor analyses. *Child Development, 73*(3), 867-882.

Lengua, L. J., West, S. G., & Sandler, I. N. (1998). Temperament as a predictor of symptomatology in children: Addressing contamination of measures. *Child Development, 69,* 164-181.

Leyfer, O. T., Folstein, S. E., Bacalman, S., Davis, N. O., Dinh, E., Morgan, J., et al. (2006). Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal of Autism and Developmental Disorders, 36*(7), 849–861.

Lord, C., & Bailey, A. (2002). Autism spectrum disorders. In M. Rutter, & E. Taylor (Eds.), *Child and adolescent psychiatry* (pp. 664-681). Oxford: Blackwell Scientific.

Lord, C., & McGee, W. (2001). *Educating children with autism.* Committee on E Educational Interventions for Children with Autism. Division of Behavioral and Social Sciences and Education. Washington, DC: National Academy Press.

Lord, C., Rutter, M., Dilavore, P., & Risi, S. (2001). *Manual: Autism diagnostic observation schedule*. Los Angeles, CA: Western Psychological Services.

Lounds, J., Seltzer, M. M., Greenberg, J. S., & Shattuck, P. T. (2007). Transition and change in adolescents and young adults with autism: Longitudinal effects on maternal well-being. *American Journal of Mental Retardation, 112,* 401-417.

Lynam, D., Moffitt, T., & Stouthamer-Loeber, M. (1993). Explaining the relation between IQ and delinquency: Class, race, test motivation, school failure, or self-control? *Journal of Abnormal Psychology, 102,* 187–196.

Martel, M. M., Nigg, J. T., Wong, M. M., Fitzgerald, H. E., Jester, J. M., Puttler, L. I., et al. (2007). Childhood and adolescent resiliency, regulation, and executive functioning in relation to adolescent problems and competence in a high-risk sample. *Development & Psychopathology, 19*, 541-563.

Martin, R. P., & Bridger, R. C. (1999). *The Temperament Assessment Battery for Children-Revised.* Athens: University of Georgia.

Masten, A. S., Burt, K. B., & Coatsworth, J. D. (2006). Competence and psychopathology in development. In D. Cicchetti & D. J. Cohen (Eds.), Developmental psychopathology (2nd ed., Vol. 3, pp. 696 – 738). New York: Wiley.

Masten, A. S., Roisman, G. I., Long, J. D., Burt, K. B., Obradovic´, J., Riley, J. R., & Tellegen, A. (2005). Developmental cascades: Linking academic achievement and externalizing and internalizing symptoms over 20 years. *Developmental Psychology, 41*, 733–746.

Matson, J. L., & Shoemaker, M. (2009). Intellectual disability and its relationship to autism spectrum disorders. *Research in Developmental Disabilities, 30*, 1107–1114.

Matthys, W., van Goozen, S. H., de Vries, H., Cohen-Kettenis, P. T., & van Engeland, H. (1998). The dominance of behavioural activation over behavioural inhibition in conduct disordered boys with or without attention deficit hyperactivity disorder. *Journal of Child Psychology and Psychiatry, 39*(5), 643-651.

Mawhood, L., Howlin, P., & Rutter, M. (2000). Autism and developmental receptive language disorder- a comparative follow-up in early adult life. I: Cognitive and language outcomes. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 41,* 547-559.

Mayes, S. D., & Calhoun, S. L. (2011). Impact of IQ, SES, gender, and race on autistic symptoms. *Research in Autism Spectrum Disorders, 5*, 749-757.

Mayes, S. D., Calhoun, S. L., Mayes, R. D., & Molitoris, S. (2012). Autism and ADHD: Overlapping and discriminating symptoms. *Research in Autism Spectrum Disorders, 6*, 277-285.

Mayes, S. D., Calhoun, S. L., Murray, M. J., Morrow, J. D., Yurich, K. K. L., Mahr, F., et al. (2009). Comparison of scores on the Checklist for Autism Spectrum Disorder, Childhood Autism Rating Scale (CARS), and Gilliam Asperger's disorder scale (GADS) for children with low functioning autism, high functioning autism or Asperger's disorder ADHD, and typical development. *Journal of Autism and Developmental Disorders, 39*, 1682–1693.

Mazurek, M. O., & Kanne, S. M. (2010). Friendship and internalizing symptoms among children and adolescents with ASD. *Journal of Autism and Developmental Disorders, 40*, 1512-1520.

McCartney, K., Burchinal, M. R., & Bub, K. L. (2006). Best practices in quantitative methods for developmentalists. *Monographs of the Society for Research in Child Development, 71*, 1-145.

McCrae, R. R., Costa, P. T., Ostendorf, F., Angleitner, A., Hrebickova, M., Avia, M. D., et al. (2000). Nature over nurture: Temperament, personality, and life span development. *Journal of Personality and Social Psychology, 78*, 173–186.

McGovern, C. W. & Sigman M. (2005). Continuity and changes from early childhood to adolescences in autism. *Journal of Child Psychology and Psychiatry, 46*(4), 401-408.

McNeil, B. Lopes, V., & Minnes, P. (2008). Anxiety in children and adolescents with autism spectrum disorders. *Research in Autism Spectrum Disorders,3*, 1-21.

Miller, J. N., & Ozonoff, S. (2000). The external validity of Asperger disorder: Lack of evidence from the domain of neuropsychology. *Journal of Abnormal Psychology, 109*, 227–238.

Miner, J. L., & Clarke-Stewart, K. A. (2008). Trajectories of externalizing behaviors from age 2 to age 9: Relations with gender, temperament, ethnicity, parenting, and rater. *Developmental Psychology, 44,* 771–786.

Moffitt, T. E. (1990). The neuropsychology of delinquency: A critical review of theory and research. In N. Morris & M. Tonry (Eds.), *Crime and justice* (Vol. 12, pp. 99–169). Chicago, IL: University of Chicago Press.

Moffitt, T. E. (1993). Adolescence-limited and life-course persistent antisocial behavior: A developmental taxonomy. *Psychological Review, 100,* 674–701.

Mundy, P. (2003). Annotation: The neural basis of social impairments in autism: The role of the dorsal medial-frontal cortex and anterior cingulate system. *Journal of Child Psychology and Psychiatry, 44*, 793–809.

Mundy, P. C., Henderson, H. A., Inge, A. P., & Coman, D. C. (2007). The modifier model of autism and social development in higher functioning children. *Research and Practice for Persons with Severe Disabilities, 32*(2), 124–139.

Muris, P. (2006). Unique and interactive effects of neuroticism and EC on psychopathological symptoms in non-clinical adolescents. *Personality and Individual Differences, 40*, 1409-1419.

Muris, P., de Jong, P. J., & Engelen, S. (2004). Relationships between neuroticism, attentional control, and anxiety disorders symptoms in non-clinical children. *Personality and Individual Differences, 37,* 789-797.

Muris, P., & Meester, C. (2009). Reactive and regulative temperament in youths: Psychometric evaluation of the early adolescent temperament questionnaire-revised. *Journal of Psychopathological Behavioral Assessment, 31*, 7–19.

Muris, P., & Ollendick, T. H. (2005). The role of temperament in the etiology of child psychopathology. *Clinical Child and Family Psychology Review, 8*, 271-289.

Murphy, O., Healy, O., & Leader, G. (2009). Risk factors for challenging behaviors among 157 children with autism spectrum disorder in Ireland. *Research in Autism Spectrum Disorders 3*, 474–482.

National Institute of Child Health and Human Development Early Child Care Research Network. (2005). Predicting individual differences in attention, memory, and planning in first graders from experiences at home, childcare, and school. *Developmental Psychology, 41,* 99-114.

Newman, J. P., MacCoon, D. G., Vaughn, L. J., & Sadeh, N. (2005). Validating a distinction between primary and secondary psychopathy with measures of Gray's BIS and BAS constructs. *Journal of Abnormal Psychology, 114*(2), 319-323.

Newman, J. P., Wallace, J. F., Schmitt, W. A., & Arnett, P. A. (1997). Behavioural inhibition system functioning in anxious, impulsive and psychopathic individuals. *Personality and Individual Differences, 23*, 583-592.

Niditch, L. A., Varela, R. E., Kamps, J. L., & Hill, T. (2012). Exploring the association between cognitive functioning and anxiety in children with autism spectrum disorders: The role of social understanding and aggression. *Journal of Clinical Child and Adolescent Psychology, 41*(2), 127-137.

Nordin, V., & Gillberg, C. (1998). The long-term course of autistic disorders: Update on follow-up studies. *Acta Psychiatrica Scandinavica, 97,* 99-108.

Nyden, A., Gillberg, C., Hjelmquist, E., & Heiman, M. (1999). Executive function/attention deficits in boys with Asperger syndrome, attention disorder and reading/writing disorder. *Autism, 3*, 213–228.

Obradovic´, J., Burt, K. B., & Masten, A. S. (2010). Testing a dual cascade model linking competence and symptoms over 20 years from childhood to adulthood. *Journal of Clinical Child & Adolescent Psychology, 39*, 90–102.

Oldehinkel, A. J., Hartman, C. A., Ferdinand, R. F., Verhulst, F. C., & Ormel, J. (2007). EC as modifier of the association between negative emotionality and adolescents' mental health problems. *Development and Psychopathology, 19,* 523-539.

Olson, S. L., Sameroff, A. J., Kerr, D. C. R., Lopez, N. L., & Wellman, H. M. (2005). Developmental foundations of externalizing problems in young children: The role of EC. *Development and Psychopathology, 17,* 25-45.

Oosterlaan, J., Logan, G. D., & Sergeant, J. A. (1998). Response inhibition in AD/HD, CD, and comorbid AD/HD+CD, anxious, and control children: A meta-analysis of studies with the stop task. *Journal of Child Psychology and Psychiatry, 39,* 411-425.

Ozonoff, S., Garcia, N., Clark, E., & Lainhart, J. E. (2005). MMPI-2 personality profiles of high-functioning adults with autism spectrum disorders. *Assessment, 12*, 86–95.

Park, S., Belsky, J., Putnam, S., & Crnic, K. (1997). Infant emotionality, parenting, and 3-year inhibition: Exploring stability and lawful discontinuity in a male sample. *Developmental Psychology, 33*, 218– 227.

Perry, A., Condillac, R. A., Freeman, N. L., Dunn-Geier, J., & Belair, J. (2005). Multi-site study of the Childhood Autism Rating Scale (CARS) in five clinical groups of young children. *Journal of Autism and Developmental Disorders, 35*, 625–634.

Pilowsky, T., Yirmiya, N., Shulman, C., & Dover, R. (1998). The Autism Diagnostic Interview-Revised and the Childhood Autism Rating Scale: Differences between diagnostic systems and comparison between genders. *Journal of Autism and Developmental Disorders, 28*, 143–151.

Piven, J., Harper, J., Palmer, P., & Arndt, S. (1996). Course of behavioral change in autism: A retrospective study of high-IQ adolescents and adults. *Journal of the Academy of Child and Adolescent Psychiatry, 35*(4), 523-529.

Posner, M. I., & Rothbart, M. K. (2007). *Temperament and learning*. Washington, DC: American Psychological Association.

Prinstein, M. J., & Aikins, J. W. (2004). Cognitive moderators of the longitudinal association between peer rejection and adolescent depressive symptoms. *Journal of Abnormal Child Psychology, 32*(2), 147–158.

Prior, M., Eisenmajer, R., Leekam, S., Wing, L., Gould, J., Ong, B., et al. (1998). Are there subgroups within the autistic spectrum? A cluster analysis of a group of children with autistic spectrum disorders. *Journal of Child Psychology and Psychiatry, 39*(6), 893-902.

Putman, S. P., & Stifter, C. A. (2005). Behavioral approach-inhibition in toddlers: Prediction from infancy, positive and negative affective components, and relations with behavior problems. *Child Development, 76,* 212-226.

Raudenbush, S. W., & Bryk, A. S. (2002). *Heirarchical linear models: Applications and data analsysis.* Thoughsand Oaks, CA: Sage.

Raudenbush, S. W., Bryk, A. S., Cheong, Y. F., & Congdon, R. (2004). *Hierarchical Linear and Nonlinear Modeling* (Version 6.01a) [Computer software]. Lincolnwood, IL: Scientific Software International.

Reynolds, C. R. & Kamphaus, R. W. (1992). Behavior Assessment System for Children: Manual. Circle Pines, MN: American Guidance.

Rogers, S. J. (1996). Early intervention in autism. *Journal of Autism and Developmental Disorders, 26,* 243–246.

Rothbart, M. K. (1988). Temperament and the development of inhibited approach. *Child Development, 59,* 1241-1250.

Rothbart, M. K. (2005). Early temperament and psychosocial development. In: Tremblay RE, Barr RG, Peters RDeV, eds. *Encyclopedia on Early Childhood Development.* Montreal, Quebec: Centre of Excellence for Early Childhood Development; 1-6.

Rothbart, M. K., Ahadi, S. A., & Evans, D. E. (2000). Temperament and personality: Origins and outcomes. *Journal of Personality and Social Psychology, 78*, 122–135.

Rothbart, M. K., Ahadi, S. A., & Hershey, K. L. (1994). Temperament and social behavior in childhood. *Merrill-Palmer Quarterly, 40,* 21–39.

Rothbart, M. K., Ahadi, S. A., Hersey, K. L., & Fisher, P. (2001). Investigations of temperament at three to seven years: The children's Behavior Questionnaire. *Child Development, 72,* 1394-1408.

Rothbart, M. K., & Bates, J. E. (2006). Temerament. In W. Damon (Series Ed.) & N. Eisenberg (Vol. Ed), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed.; pp. 105-176). New York: Wiley.

Rothbart, M. K., & Derryberry, D. (1981). Development of individual differences in temperament. In M. E. Lamb & A. L. Brown (Eds), Advances in developmental psychology (Vol. I, pp. 37-86). Hillsdale, N. J.: Erlbaum.

Rothbart, M. K., Derryberry, D., & Hershey, K. (2000). Stability of temperament in childhood: Laboratory infant assessment to parent report at seven years. In VJ Molfese, & DL Molfese (Eds.), *Temperament and personality development across the life span* (pp. 85-119). Hillsdale, NJ: Erlbaum.

Rowe, G., Hirsh, J. B., & Anderson, A. K. (2007). Positive affect increases the breadth of attentional selection. *PNAS: Proceedings of the National Academy of Science of the USA, 104,* 383-388.

Rutter, M. (1970). Autistic children: Infancy to adulthood. *Seminars in Psychiatry, 2,* 435-450.

Rutter, M., Bailey, A., & Lord, C. (2003). *The social communication questionnaire*. Los Angeles, CA: Western Psychological Services.

Rutter, M., LeCouteur, A., & Lord, C. (2003). *Autism diagnostic interview-revised*. Los Angeles, CA: Western Psychological Services.

Rydell, A-M., Berlin, L., & Bohlin, G. (2003). Emotionality, emotion regulation, and adaptation among 5- to 8-year-old children. *Emotion, 3,* 30-47.

Salley, B. J., & Dixon W. E. (2007). "Temperamental and Joint Attentional Predictors of Language Development," *Merrill-Palmer Quarterly*: Vol. 53: Iss. 1, Article 7.

Schaffer, H. R., Greenwood, A., & Parry, M. H. (1972). The onset of wariness. *Child Development, 43*, 165-175.

Schmitz, N., Rubia, K., Daly, E., Smith, A., Williams, S., & Murphy, D. G. M. (2006). Neural correlates of executive functions in autistic spectrum disorders. *Biological Psychiatry, 59*, 7–16.

Schwartz, C. B., Henderson, H. A., Inge, A. P., Zahka, N., Coman, D. C., Kojkowski, N. M., et al. (2009). Temperament as a predictor of symptomatology and adatpive functioning in adolescents with high-functioning autism. *Journal of Autism and Developmental Disorders, 39*, 842-855.

Seguin, J. R., Pihl, R. O., Harden, P. W., Tremblay, R. E., & Boulerice, B. (1995). Cognitive and neuropsychological characteristics of physically aggressive boys. *Journal of Abnormal Psychology, 104,* 614–624.

Seltzer, M. M., Shattuck, P., Abbeduto, L., & Greenberg, J. S. (2004). Trajectories of development in adolescents and adults awith autism. *Mental Retardation and Developmental Disabilities Research reviews, 10,* 234-247.

Shattuck, P. T., Seltzer, M. M., Greenberg, J. S., Orsmond, G. I., Kring, S., Bolt, D. et al. (2007). Changes in autism symptoms and maladaptive behaviors among adolescents and adults with an autism spectrum disorder. *Journal of Autism and Developmental Disorders, 37,* 1735-1747.

Shea, V., & Mesibov, G. (2005). Adolescents and adults with autism. In F.R. Volkmar, R. Paul, A. Klin, & D.J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders.* Hoboken, NJ: John Wiley & Sons.

Sigman, M., & Ruskin, E. (1999). Continuity and change in the social competence of children with autism, Down syndrome, and developmental delays. *Monographs of the Society for Research in Child Development, 64,* 1-142.

Singh, N. N., Lancioni, G. E., Manikam, R., Winton, A. S. W., Singh, A. N. A., Singh, J., et al. (2011). A mindfulness-based strategy for self-management of aggressive behavior in adolescents with autism. *Research in Autism Spectrum Disorders, 5*, 1153–1158.

Singh, N. N., Lancioni, G. E., Singh, A. D. A., Winton, A. S. W., Singh, A. N. A., & Singh, J. (2011). Adolescents with Asperger syndrome can use a mindfulness-based strategy to control their aggressive behavior. *Research in Autism Spectrum Disorders, 5*, 1103–1109.

Silk, J. S., Steinberg, L., & Morris, A. S. (2003). Adolescents' emotion regulation in daily life: links to depressive symptoms and problem behavior. *Child Development, 74,* 1869-1880.

Simonoff, E. M., Pickles, A. P., Charman, T. P., Chandler, S. P., Loucas, T. O. M. P., & Baird, G. F. (2008). Psychiatric disorders in children with autism spectrum disorders: prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child & Adolescent Psychiatry, 47*(8), 921–929.

Simonoff, E., Jones, C. R., Pickles, A., Happe, F., Baird, G., & Charman, T. (2012). Severe mood problems in adolescents with autism spectrum disorder. *Journal of Child Psychology and Psychiatry, 53*(11), 1157-1166.

Snijders, T., & Bosker, R. (1999). Multilevel analysis. *An introduction to basic and advanced multilevel modeling.* London, England: Sage Publications.

Soderstrom, H., Rastam, M., & Gillberg, C. (2002). Temperament and character in adults with Asperger syndrome. *Autism, 6*(3), 284–297.

Spek, A. A., van Ham, N. C., & Nyklicek, I. (2013). Mindfulness-based therapy in adults with an autism spectrum disorder: A randomized controlled trial. *Research in Developmental Disabilities, 34*, 246-253.

Sperry, V. W. (2001). *Fragile success: Ten autistic children, childhood to adulthood*. Baltimore, MD: Paul H. Brookes.

Spinrad, T. L., Eisenberg, N., Gaertner, B., Popp, T., Smith, C. L., Kupfer, A. et al. (2007). Relations of maternal socialization and toddlers' EC to children's adjustment and social competence. *Developmental Psychology, 43,* 1170-1186.

Stanger, C., Achenbach, T. M., & Verhulst, F. C. (1997) Accelerated longitudinal comparisons of aggressive versus delinquent syndromes. *Developmental Psychopathology, 9,* 43–58.

Stevens, M. C., Fein, D. A., Dunn, M., Allen, D., Waterhouse, L. H., Feinstein, C. et al. (2000). Subgroups of children with autism by cluster analysis: A longitudinal examination. *Journal of Autism and Developmental Disorders, 33,* 15-22.

Strauss, C. C., Lahey, B. B., Frick, P., Frame, C. L., et al. (1988). Peer social status of children with anxiety disorders. *Journal of Consulting and Clinical Psychology, 56*(1), 137–141.

Sturm, H., Fernell, E., & Gillberg, C. (2004). Autism spectrum disorders in children with normal intellectual levels: Associated impairments and subgroups. *Developmental Medicine and Child Neurology, 46*, 444-447.

Sukhodolsky, D., Scahill, L., Gadow, K., Arnold, L., Aman, M., McDougle, C., et al. (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.

Sutton, S. K., Burnette, C. P., Mundy, P. C., Meyer, J., Vaughan, A., Sanders, C., et al. (2005). Resting cortical brain activity and social behavior in higher functioning children with autism. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 46*(2), 211–222.

Szatmari, P., Georgiades, S., Bryson, S., Zwaigenbaum, L., Roberts, W., Mahoney, W., et al. (2006). Investigating the structure of the restricted, repetitive behaviours and interests domain in autism. *Journal of Child Psychology and Psychiatry, 47*, 582–590.

Tantam, D. (2000). Psychological disorder in adolescents and adults with Asperger syndrome. *Autism, 4,* 47-62.

Taylor, J. L., Lindsay, W. R., & Willner, P. (2008). CBT for people with intellectual disabilities: Emerging evidence, cognitive ability and IQ effects. *Behavioural and Cognitive Psychotherapy, 36*(6), 723-733.

Taylor, J. L., & Seltzer, M. M. (2010). Changes in the autism behavioral phenotype during the transition to adulthood. *Journal of Autism Developmental Disorders, 40,* 1431-1446.

Teasdale, J. D., Segal, Z. V., & Williams, M. G. (1995). How does cognitive therapy prevent depressive relapse and why should attentional control (mindfulness training) help? *Behavior Research and Therapy, 33*, 25–39.

Thakkar, K. N., Polli, F. E., Joseph, R. M., Tuch,D. S., Hadjikhani, N., Barton, J. J., & Manoach, D. S. (2008). Response monitoring, re- 250 D. L. Santesso et al. petitive behaviour and anterior cingulate abnormalities in autism spectrum disorders (ASD). *Brain, 131,* 2464–2478.

Thomas, A., & Chess, S. (1977). *Temperament and development.* New York: Brunner Mazel.

Todd, J. T., & Dixon, W. E. Jr. (2010). Temperament moderates responsiveness to joint attention in 11-month-old infants. *Infant Behavior and Development, 33*(3), 297-308.

Troop-Gordon, W., & Ladd, G. W. (2005). Trajectories of peer victimization and perceptions of the self and schoolmates: Precursors to internalizing and externalizing problems. *Child Development, 76*(5), 1072–1091.

Velez, C. E. (2011). Children's coping efforts and coping efficacy: Effects of parenting, surgency, and effortful control. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 72*(1-B), 1-560.

Vlamings, P. H., Jonkman, L. M.,Hoeksma, M. R., van Engeland, H.,& Kemner, C. (2008). Reduced error monitoring in children with autism spectrum disorder: An ERP study. *European Journal of Neuroscience, 28*, 399–406.

Volkmar, F. R., Cicchetti, D. V., Bregman, J., & Cohen, D. J. (1992). Three diagnostic systems for autism: DSM-III, DSM-III-R, and ICD-10. *Journal of Autism and Developmental Disorders, 22,* 483–492.

Watson, D., & Clark, L. A. (1997). Measurement and mismeasurement of mood: Recurrent and emergent issues. *Journal of Personality Assessment, 68,* 267-296.

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*, 477–496.

Wellman, H. M., Lane, J. D., LaBounty, J., & Hamilton, B. (2008). Infant attention to intentional action predicts preschool theory of mind. *Developmental Psychology, 44,* 618-623.

Weschler, D. (2003). *Wechsler intelligence scale for children (4th Edition)*. San Antonio, TX: The Psychological Corporation.

Wing, L. (1997). The autistic spectrum. *Lancet, 350*, 1761–1766.

Wolf, L., & Goldberg, B. (1986). Autistic children grow up: An eight to twenty-four year follow-up study. *Canadian Journal of Psychiatry, 31,* 550-556.

Wood, J.J., Drahota, A., Sze, K., Dyke, M. V., Decker, K., & Fujii, C. et al. (2009). Brief report: effects of cognitive behavioral therapy on parent reported autism symptoms in school-age children with high-functioning autism. *Journal of Autism and Developmental Disorders, 39*(11), 1608-1612.

World Health Organization 2007. International statistical classification of diseases and related health problems; ICD-10 (10th revision ed.). Geneva: WHO.

Zahn-Waxler, C., Shirtcliff, E.A., & Marceau, K. (2008). Disorders of childhood and adolescence: Gender and psychopathology. *Annual Review of Psychology, 4*, 275-303.

Zeman, J., Shipman, K., & Suveg, C. (2002). Anger and sadness regulation: Predictions to internalizing and externalizing symptoms in children. *Journal of Clinical Child and Adolescent Psychology, 31,*393-398.

Zentner, M. A., & Bates, J. E. (2008). Child temperament: An integrative review of concepts, research programs, and measures. *European Journal of Developmental Science, 2*, 7-37.

Zuckerman, M. (1991). *Psychobiology of personality*. Cambridge, England: Cambridge University Press.

Table 1.

Demographic and Diagnostic Information.

	HFA			*non-ASD*			*Analysis*	
	N	*Mean (SD)*	*Range*	*N*	*Mean (SD)*	*Range*	*F value*	*p value*
T1 Age (yrs)	*N = 80*	12.62(2.43)	8.2-16.7	*N = 74*	12.99(2.31)	8.9-16.7	0.98	0.32
Gender	*N = 80*	65 M, 15 F		*N = 74*	56 M, 18 F		χ^2=.71	0.4
VIQ	*N = 80*	102.35(14.82)	77-140	*N = 74*	110.20(13.62)	81-155	11.66***	0.001
SCQ	*N = 80*	19.40(6.20)	3-33	*N = 74*	4.74(3.46)	0-20	320.76***	<.001
ADOS	*N = 78*	11.28(4.18)	0-21	*N = 69*	2.68(4.11)	0-19	157.67***	<.001
ASSQ	*N = 80*	26.89(8.62)	10-47	*N = 73*	4.25(4.15)	0-23	415.22***	<.001
T2 Age (yrs)	*N = 34*	13.85(2.88)	8.9-18.3	*N = 34*	14.65(2.11)	9.6-17.8	1.72	0.2
T3 Age (yrs)	*N = 42*	15.36(2.70)	10.1-19.5	*N = 35*	15.91(2.44)	9.5-19.2	0.9	0.35
T4 Age (yrs)	*N = 39*	16.45 (3.51)	9.8-19.5	*N = 47*	16.48(3.87)	10.8-19.5	0.01	0.99

Note: VIQ, SCQ, ADOS, ASSQ measured at first assessment. SCQ and ASSQ are Total Scores. ADOS is the Communication and Social Interaction Domain

*** $p < .001$

Table 2.

Correlation Table for Dependent Variables Presented Independently by Diagnostic Group.

HFA ($N = 80$)	1	2	3	4
Self Report Measures				
1.BASC Internalizing				
2.BASC Externalizing	.68***			
Parent Report Measures				
3.BASC Internalizing	.23*	.28*		
4.BASC Externalizing	.08	.23	.40***	
Symptom Severity				
5.SRS Total	.17	.12	.42***	.42***
TD ($N = 74$)	1	2	3	4
Self Report Measures				
1.BASC Internalizing				
2.BASC Externalizing	.67***			
Parent Report Measures				
3.BASC Internalizing	-.07	.27*		
4.BASC Externalizing	.38**	.38**	.19	
Symptom Severity				
5.SRS Total	.36**	.30*	.32**	.55***

* < .05

** < .01

*** < .001

Table 3.

Correlation Table for Predictors Presented Independently by Diagnostic Group

HFA (*N* = 80)	1	2	3	4	5	6	7
Self Report Measures							
1.EATQ Surgency							
2.EATQ Effort. Control	.24*						
3. EATQ Neg. Affect	-.37**	-.57***					
4.WISC Verbal Comp.	-.03	.15	.04				
Parent Report Measures							
5. EATQ Surgency	.34**	.14	-.02	.13			
6.EATQ Effort. Control	.08	.45***	-.19	.02	.16		
7.EATQ Neg. Affect	-.18	-.30**	.22	-.06	-.36**	-.48***	
8.SCQ Total	-.06	-.08	-.10	-.15	.01	-.07	.06
non-ASD (*N* = 74)	1	2	3	4	5	6	7
Self Report Measures							
1.EATQ Surgency							
2.EATQ Effort. Control	.14						
3. EATQ Neg. Affect	-.31**	-.54***					
4.WISC Verbal Comp.	.08	.24*	-.10				
Parent Report Measures							
5. EATQ Surgency	.38**	.14	-.03	.08			
6.EATQ Effort. Control	.10	.40***	-.32**	.25*	.13		
7.EATQ Neg. Affect	-.08	-.53***	.47***	-.17	-.21	-.60***	
8.SCQ Total	.08	-.03	-.10	-.13	-.16	-.11	.11

* < .05

** < .01

*** < .001

Table 4.
Predictor and Dependent Variables by Diagnostic Group

	HFA			*non-ASD*			*Analysis*	
	N	*Mean (SD)*	*Range*	*N*	*Mean (SD)*	*Range*	*F value*	*p value*
Child Report								
Surgency	80	-.85(.57)	-2.3	74	-.45(.48)	-2.1	22.68***	<.001
Effort Control	80	3.27(.49)	2.1 - 4.6	74	3.40(.46)	2.4 - 4.5	2.89	0.09
Neg. Affect	80	2.81(.55)	1.6 - 4.5	74	2.39(.56)	1.0 - 3.9	22.01***	<.001
Parent Report								
Surgency	80	-1.13(.60)	-3	74	-.33(.55)	-2.7	72.82***	<.001
Effort Control	80	2.55(.58)	1.5 - 4.2	74	3.47(.66)	2.1 - 4.73	83.60***	<.001
Neg. Affect	80	2.98(.59)	1.8 - 4.5	74	2.37(.55)	1.2 - 3.6	42.51***	<.001
Child Report								
Internalizing	77	53.19(9.99)	29 - 77	70	44.71(6.11)	36 - 64	37.61***	<.001
Externalizing	77	53.77(9.82)	33 - 84	70	48.69(9.48)	34 - 78	10.14**	0.002
Parent Report								
Internalizing	75	59.84(13.10)	36 - 93	69	46.13(8.12)	30 - 65	55.81***	<.001
Externalizing	75	55.24(10.75)	37 - 82	69	49.09(8.15)	34 - 78	14.78***	<.001
SRS	79	80.52(10.86)	54 - 91	73	46.37(7.72)	34 - 64	492.10***	<.001

Note: Analysis from first assessment
*** $p < .001$

Table 5.

Unconditional HLM model of growth for Parent and Child-reported Behavior Measures and Symptom Severity

	Parent Internalizing			Parent Externalizing			Child Internalizing			Child Externalizing			SRS		
	coeff	SE	Variance	coeff	SE	Variance	coeff	SE	Variance	coeff	SE	Variance	coeff	SE	Variance
Intercept (8yrs)															
Constant	51.29**	1.46	44.74**	54.23***	1.99	251.93***	51.29***	1.46	44.74**	54.22***	1.87	155.42**	68.76***	2.91	738.85***
Slope (growth rate)															
Constant	-0.03*	0.02	0.01***	-0.02	0.02	0.02***	-0.03*	0.02	0.001*	-0.03	0.02	0.09*	-0.07*	0.03	0.22***

Note. * ≤ .05
** ≤ .01
*** ≤ .001

Table 6.
Level 1 and level 2 Residual Variance

	Level 1	Level 2
ASD Symptoms	39.21%	88.92%
Child-Rated		
Internalizing	45.73%	28.72%
Externalizing	22.78%	31.68%
Parent-Rated		
Internalizing	37.32%	78.20%
Externalizing	21.98%	72.34%

Table 7.
HLM Models of Growth with Diagnostic Status as Predictor

	Parent Internalizing		Parent Externalizing		Child Internalizing		Child Externalizing		SRS	
	coeff	SE	coeff	SE	coeff	SE	coeff	SE	coeff	SE
Fixed Effect										
Intercept (8yrs)										
Constant (γ00)	45.59***	2.20	49.59***	2.37	46.62***	2.18	50.07***	2.73	49.70***	1.96
Diagnostic Group (γ01)	18.02***	3.83	8.45*	3.72	8.16**	2.91	7.77*	3.79	33.30***	3.73
Slope (growth rate)										
Constant (γ10)	0.01	0.02	-0.01	0.02	-0.03	0.02	-0.02	0.03	-0.04*	0.02
Diagnostic Group (γ11)	-0.06	0.03	-0.03	0.04	0.01	0.03	0.02	0.04	0.01	0.04
Random Effect										
Level 2										
Intercept (τπ00)	198.52***		218.83***		49.93**		188.83***		228.71***	
Slope (τπ1i)	0.01***		0.01***		0.01***		0.01***		0.01***	

Note. * ≤ .05
** ≤ .01
*** ≤ .001

Table 8.

HLM Models of Growth for Parent-Reported Internalizing and Externalizing Behaviors as a Function of Predictor Variables

	Parent Internalizing		Parent Externalizing	
	Estimate Coeff	SE	Estimate Coeff	SE
Fixed Effect				
Intercept (8yrs)				
Constant (γ00)	68.58***	7.77	55.64***	3.26
Diagnostic Group (γ01)	-25.07	14.69	-6.08	3.54
Parent Neg Affect (γ02)	11.87**	3.93	-9.17**	2.81
Group x Parent Neg Affect (γ03)	11.54*	5.40	9.64***	2.87
	Adjust Coeff	SE	Adjust Coeff	SE
Slope (growth rate)				
Constant (γ10)	-0.12	0.23	-0.35**	0.12
Diagnostic Group (γ11)	0.27*	0.12	0.01	0.02
Parent Neg Affect (γ12)	0.01	0.05	-0.03	0.02
Group x Parent Neg Affect (γ13)	-0.08*	0.05	0.11	0.03
Random Effect				
Level 2				
Intercept (τπ00)	64.01***		59.75***	
Slope (τπ1i)	0.01***		0.01***	

Note. * ≤ .05
** ≤ .01
*** ≤ .001

Table 9.

HLM Models of Growth for Child-Reported Internalizing and Externalizing Behaviors as a Function of Predictor Variables

	Child Internalizing		Child Externalizing	
	Estimate Coeff	SE	Estimate Coeff	SE
Fixed Effect				
Intercept (8yrs)				
Constant (γ00)	49.04***	2.34	53.78***	2.84
Diagnostic Group (γ01)	2.93	3.51	-2.08	4.30
Verbal IQ (γ02)	0.19*	0.09	0.42***	0.11
Parent Effort Control (γ03)	-7.80**	2.41	-12.43***	2.83
Child Surgency (γ04)			07.67**	2.83
	Adjust Coeff	SE	Adjust Coeff	SE
Slope (growth rate)				
Constant (γ10)	-0.14	0.79	-0.36*	0.11
Diagnostic Group (γ11)	0.04	0.07	0.01	0.02
Verbal IQ (γ12)	-0.01*	0.01	-0.01*	0.02
Parent Effort Control (γ13)	0.08	0.13	-0.01*	0.02
Child Surgency (γ14)			-0.01	0.03
Random Effect				
Level 2				
Intercept (τπ00)	21.57**		90.82**	
Slope (τπ1i)	0.01***		0.01***	

Note. * ≤ .05
** ≤ .01
*** ≤ .001

Table 10.
HLM Models of Growth for Social Reciprocity as a Function of Predictor Variables in Individuals with HFA

	SRS	
	Estimate Coeff	SE
Fixed Effect		
Intercept (8yrs)		
Constant (γ00)	72.71***	4.91
Initial Symptomatology (γ01)	4.15**	2.88
Parent Neg Affect (γ02)	2.52*	1.31
	Adjust Coeff	SE
Slope (growth rate)		
Constant (γ10)	-0.42	0.34
Initial Symptomatology (γ11)	0.11	0.04
Parent Neg Affect (γ12)	0.05*	0.11
Random Effect		
Level 2		
Intercept (τπ00)	54.79***	
Slope (τπ1i)	0.01***	

Note. * ≤ .05
** ≤ .01
*** ≤ .001

Table 11.

Developmental Change in Outcome Variables Presented Independently by Diagnostic Group

	HFA						*non-ASD*				
First Assessment		Self-Report		Parent Report		First Assessment		Self-Report		Parent Report	
8 years of age	SRS	Internal	External	Internal	External	8 years of age	SRS	Internal	External	Internal	External
Coefficient	83.62	55.16	55.28	65.63	57.47		47.35	48.3	50.76	45.64	50.76
Last Assessment						Last Assessment					
19.5 years of age	SRS	Internal	External	Internal	External	19.5 years of age	SRS	Internal	External	Internal	External
Coefficient	73.28	49.56	50.07	51.82	51.72		44.98	40.84	46.4	46.64	46.92
Intercept	-0.06	-0.03	-0.03	-0.08**	-0.03		-0.01	-0.04*	-0.03	0.01	-0.02
p value	0.06	0.18	0.21	<0.01	0.17		0.53	0.01	0.33	0.79	0.30

Note. * ≤ .05

** ≤ .01

Table 12.

Frequencies of Clinical Significant Change at an Individual Level

	HFA					non-ASD				
	Self-Report		Parent-Report			Self-Report		Parent-Report		
	Internal	External	Internal	External	SRS	Internal	External	Internal	External	SRS
First Assess/Last Assess										
Below/Below	43	38	20	36	2	56	50	49	47	46
Below/Above	7	9	6	3	2	1	4	6	8	8
Above/Below	8	11	16	14	9	0	2	2	2	3
Above/Above	6	6	17	6	49	0	0	0	2	1
Percent Recovered	57.14%	64.71%	48.48%	70%	15.52%	n/a	100%	100%	50%	75%

Note: Below: T-score < 60; Above: T-score ≥ 60.

Percent Recovered = "Above/Below" / ("Above/Below" + "Above/Above")

Figure 1.
Graph of Individual Trajectories of all Participants in Sample

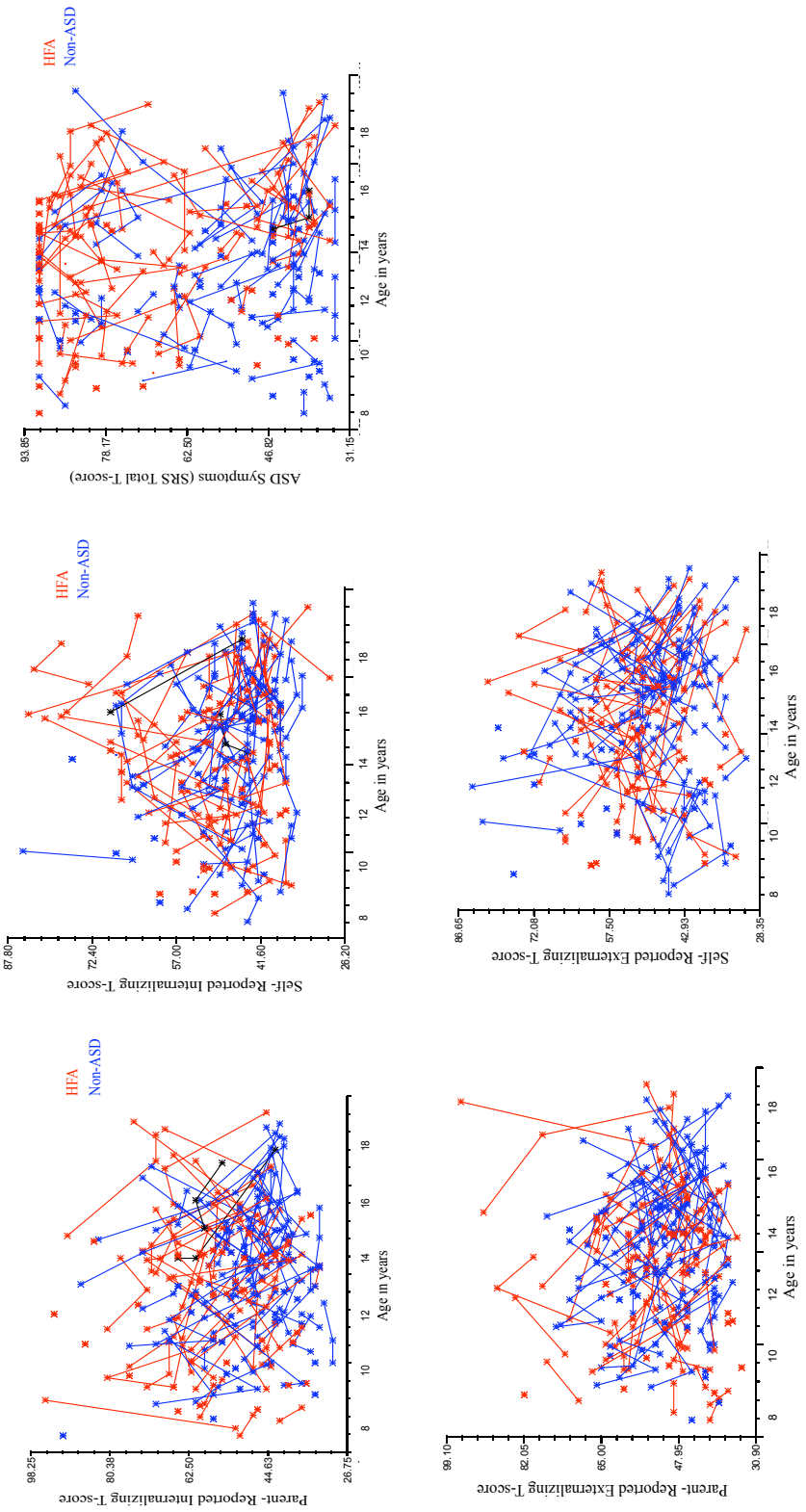

Figure 2.
Graph of the Final HLM model for Parent-Reported Internalizing Problems with all Predictors Included