THE HETEROGENEITY OF AUTISM SPECTRUM DISORDER: AN EXAMINATION OF CHILD OUTCOMES AND PARENTING BEHAVIORS

by

Deborah Eileen Rafferty

Bachelor of Science, 2013
University of Utah
Salt Lake City, Utah

Bachelor of Arts, 2013
University of Utah
Salt Lake City, Utah

Master of Science, 2016
University of Utah
Salt Lake City, Utah

Master of Science, 2020
Texas Christian University
Fort Worth, Texas

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Texas Christian University
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Autism spectrum disorder (ASD) is a heterogenous neurodevelopmental disorder with defining characteristics of social communication/interaction challenges (SC/I) and restrictive, repetitive behaviors (RRB; American Psychological Association, 2013; Maenner et al, 2023; Masi et al., 2017). The current prevalence rate of ASD in the United States is estimated to be one in 36 (Maenner, 2023). Beyond the core symptoms, autistic individuals display an array of strengths and challenges in cognitive abilities and daily living skills that present differently in each individual (Lord et al., 2020; Masi et al., 2017). With the heterogenous nature of ASD, it becomes important to explore how different presentations of ASD symptoms and cognitive abilities may affect child outcomes.

Many autistic children have difficulties with emotion regulation (e.g., Mazefsky et al., 2013), which may lead to negative physiological and behavioral outcomes (e.g., Bougeard et al, 2021; Doshi-Velez et al., 2014). Compared to non-autistic children, autistic children have a different physiological response to emotion stimuli, which may be indicative of emotion and behavior difficulties, such as depression, and anxiety (e.g., Barbier et al., 2022; Beauchaine, 2015a, 2015b). Cardiac vagal tone, a psychophysiological marker of emotion regulation, is measured through heart rate variability (HRV) – variation in heart rate and interbeat intervals – and respiratory sinus arrythmia (RSA), which examines how respiration affects HRV (Cheng et al., 2020; Kemp & Quintana, 2013; Quintana et al., 2016). Autistic children have lower baseline RSA compared to their non-autistic peers (Barbier et al., 2022; Neuhaus et al., 2014), suggesting autistic children may be hyperaware of their environment which activates their fight or flight response and reduces their ability to respond appropriately (Van Hecke et al., 2009). As a result of emotion regulation difficulties, autistic children and adolescents have higher rates of

internalizing (e.g., depression and anxiety), and externalizing behaviors (e.g., aggression, hyperactivity, and impulsivity; Bauminger et al., 2010; Rosen et al., 2018). As ASD symptoms present as a range of severity and cognitive ability, understanding these characteristics and determining how symptom profiles impact physiological and behavioral outcomes may lead to individualized interventions in the future.

Of particular interest is how the bidirectional relationship between parenting behaviors and child emotion and behavior difficulties are moderated by ASD symptom profiles. The tripartite model of the impact of the family on children's emotion regulation (Morris et al., 2007) proposes parenting practices, such as parental warmth, have an impact on child emotion regulation outcomes, which then influence parenting practices. This bidirectional relationship is then moderated by child characteristics (Morris et al., 2007). For example, as children display more problem behaviors (Totsika et al., 2013) and ASD symptoms (Greenberg et al., 2006), parents of autistic children display higher rates of stress and lower well-being that can negatively impact their child's behaviors. A bidirectional link between parental behaviors (e.g., warmth & supportiveness) and child behaviors (e.g., problem behaviors & ASD symptoms) in ASD populations has been found in a few studies (e.g., Dielman et al., 2017; Hickey et al., 2020).Therefore, understanding the nature of the bidirectional influence autistic children and their parents have on one another may allow greater insight into the relation between parenting behaviors and autistic children's emotion and behavior difficulties. Further, examining how ASD symptom profiles moderate the association between parenting behaviors and child outcomes may inform future interventions that are individualized for families based on symptom profiles.

As ASD presents with substantial differences in core symptoms and cognitive ability, examining the characteristics of autistic children may provide greater insight into the effects of emotion and behavior difficulties (e.g., physiological responses [RSA] and behavioral outcomes

[internalizing & externalizing behaviors]), and parenting behaviors on child emotion regulation. Therefore, the goals of the current study are to 1) analyze profiles of ASD symptoms and intelligence quotient (IQ) in order to examine profile differences in child emotion and behavior difficulties (i.e., RSA and internalizing & externalizing behaviors); 2.1) examine the bidirectional relations between parenting behaviors and autistic children's emotion and behavior difficulties, and 2.2) whether the symptom profiles moderate the association between parenting behaviors and child emotion and behavior difficulties.

The Heterogeneity of ASD

Autism spectrum disorder is typically diagnosed based on the child's behavioral presentation and impairment in two core areas: social communication/interaction (SC/I) and restrictive, repetitive behaviors (RRBs; APA, 2013). However, due to the heterogenous nature and complexity of ASD, these core symptoms can present differently and variation in symptomatology exists between and within individuals (Masi et al., 2017). Hill and colleagues (2015) found ASD symptom severity and IQ moderates the relationship between child age and adaptive functioning. Younger children with low IQ had worse adaptive functioning scores at high ASD symptom severity than low symptom severity. Conversely, older children with high IQ had worse adaptive functioning scores at high symptom severity than low symptom severity (Hill et al., 2015). Also, RRBs present differently based on cognitive functioning, as autistic children with high IQ have fewer repetitive sensory motor behaviors and more insistence on sameness behaviors (Jiujias et al., 2017). Jasim and Perry (2023) found autistic children with high cognitive functioning were more likely to engage in Ritualistic/Sameness behaviors, whereas those with lower cognitive functioning engaged in more self-injurious behaviors, compulsive behaviors, and restrictive interests.

Due to the heterogenous presentation of ASD symptoms, researchers should explore methods to address the issue of heterogeneity in ASD samples to better understand how symptom presentation impacts outcomes. Statistical methods exist to identify underlying patterns and differences within populations to create a more homogenous profile than can be compared to other profiles within a sample (e.g., Berlin et al., 2014; Weller et al., 2020). Latent profile analysis is used when examining the probability than an individual belongs to a subgroup (i.e., latent groups) existing within data. These person-oriented analyses allow researchers to identify more homogeneous groups to examine how these patterns affect variables of interest (Berlin et al., 2014; Weller et al., 2020).

Latent profile analysis has been used in numerous studies to examine latent profiles with ASD core symptoms and IQ as indicator variables to define profiles (e.g., Prefontaine et al., 2022; Reetzke et al., 2022; Sullivan et al., 2019). Zheng and colleagues (2019) used RRBs to develop subgroups of ASD and compare profiles on measures of cognitive ability, ASD symptom severity, and adaptive functioning. Three subgroups were identified (Low, Medium, & High Severity of RRBs) and significantly differed on severity for all measures. Prefontaine and colleagues (2022) used ASD symptoms, adaptive functioning, and IQ as indicator variables to create symptom profiles and found four profiles: mild impairment with average IQ, mild impairment with lower average IQ, moderate impairment, and severe impairment. Follow-up analyses found the profiles responded differently to early behavioral intervention as only some profiles maintained positive outcomes long-term. Reetzke and colleagues (2022) examined language and social communication profiles in autistic children and found the profiles were associated with different skill levels and clinical outcomes. Sullivan and colleagues (2019) used aggressive behaviors, ASD symptom severity, IQ, and adaptive behavior to develop a five-

profile model. While there was no difference in ASD symptoms, variation in aggressive behavior severity across profiles indicated differential relations with other child behaviors.

Overall, latent profile analysis can provide insight into the heterogenous presentation of ASD. More research using latent profile analysis is needed as ASD symptom severity and cognitive functioning can present differently in autistic children (Masi et al., 2017). Once profiles are discovered, profiles can be compared across various measures to examine whether profile membership affects outcomes. As autistic children have difficulties with emotion regulation (e.g., Mazefsky et al., 2013), they are at an increased risk for negative physiological and behavioral outcomes (e.g., Rosen et al., 2018). Through the use of latent profile membership, researchers may be able to better understand the heterogenous presentation of ASD symptoms and IQ and better examine outcomes of emotion regulation to create targeted therapies and interventions.

Emotion Regulation

Emotion regulation is an individual's attempt to adjust emotions within themselves (intrinsic) or others (extrinsic) to meet goals for any given situation (Gross, 2014; Gross, 2015a; Gross, 2015b; McRae & Gross, 2020). Through the process of emotion regulation, individuals can influence what emotions (positive or negative) they experience, as well as the duration and intensity of those emotions (Gross, 2014, Gross, 2015a). Emotion regulation can encompass not only down-regulation (decrease) of negative emotions (e.g., sadness & anger), but also up-regulation (increase) negative and positive (e.g., happiness & amusement) emotions (McRae & Gross, 2020).

Emotion dysregulation occurs when there is a disruption or interference with goal-directed activity and is a defining feature of most psychopathology (Thompson, 2019). Compared to their non-autistic peers, autistic children have poor emotion regulation abilities

which can lead to negative outcomes (e.g., Cai et al., 2018). Weiss and colleagues (2014) noted autistic children face many emotional and behavioral difficulties which are exacerbated by the impairments in social communication and restrictive and/or repetitive interests. In a study examining the association between ASD symptoms and emotion regulation, Fenning and colleagues (2018) found the strongest predictor of emotion dysregulation in autistic children was ASD symptom severity. While each of the core ASD symptoms (SC/I & RRB) are associated with poor emotion regulation (Cai et al., 2018; Masi et al., 2017), RRBs were the strongest predictor of emotion dysregulation as it is associated with difficulties with flexibility, perseveration, and inability to inhibit certain behaviors (Samson et al., 2014). Samson and colleagues (2014) suggested behaviors associated with RRBs (e.g., stereotyped and repetitive motor mannerisms, restrictive interests, resistance to change) could be the manifestation of emotion regulation deficits. Difficulties with social communication, language ability, and understanding social and emotional cues has been associated poor emotion regulation (Mazefsky & White, 2013), which can lead to self-injury (Martinez-Gonzalez et al., 2022), infrequent use of adaptive coping mechanisms such as reappraisal (Goldsmith & Kelley, 2018), and higher anxiety levels (Conner et al., 2020).

Although cognitive skills have been associated with emotion dysregulation in autistic children, the research examining the effects of IQ on emotion regulation is mixed. Zantinge and colleagues (2017) found IQ to be a significant predictor of use of constructive coping strategies, as higher IQ indicates use of more adaptive coping strategies and better emotion regulation. In addition, they found language ability was associated with avoidance coping strategies, with autistic children with lower language abilities more likely to engage in avoidance coping. Research has also found an association between IQ and internalizing and externalizing behaviors (Bolte et al., 1999, Mayes et al., 2022; Totsika et al., 2011). However, some researchers have

found no relation between IQ and emotion regulation (Fenning et al., 2018; Simonoff et al., 2012). The associations between internalizing and externalizing behaviors, and IQ will be discussed in greater detail in later sections. Yet, as some research has indicated that IQ and cognitive ability may influence emotion regulation, it is an important aspect to explore.

Emotion Regulation Outcomes and Respiratory Sinus Arrythmia

How an individual experiences and responds to an emotion is based on coordinated physiological response (Siegel et al., 2018). The autonomic nervous system (ANS) is comprised of two divisions that function in opposition: the sympathetic nervous system (SNS) that controls the body's fight or flight response and the parasympathetic nervous system (PNS) that is responsible for rest and recovery (Cheng et al., 2020). As most internal organ nerves are controlled by the ANS, it is possible to examine ANS activity through the vagus nerve and cardiac vagal tone, a psychophysiological marker of emotion regulation (Beauchaine, 2001). Excitatory cardiac activity (increased heart rate) is indicative of SNS activation, while inhibitory cardiac activity (decreased heart rate) suggests PNS activation (Beauchaine, 2001). Measuring physiological responses during emotional tasks or in response to emotion stimuli can provide insight into an individual's ability to regulate their emotions. Cardiac vagal tone is measured through heart rate variability (HRV), a physiological marker that describes variation in heart rate and interbeat intervals (Cheng et al., 2020; Quintana et al., 2016).

Emotion regulation researchers and theorists have contrasting ideas as to how the ANS is activated during an emotional response and its role in behavior response and psychopathology (Beauchaine, 2001; Porges 1995, 1997). Porges' (1995, 1997) Polyvagal theory and Beauchaine's (2001) Integrated Model of Autonomic Functioning are two of the prevailing theories that researchers use to help explain ANS activity during emotion regulation. Polyvagal theory (Porges, 1995, 1997) suggests a hierarchy within the ANS that is associated with social

communication, mobilization (e.g., fight or flight), or immobilization (e.g., freeze) behaviors. In response to emotional stimuli, initially, PNS activity withdrawal occurs. Then, based on the stimuli or the individual's response pattern, either the SNS will activate the body's fight or flight response or the PNS will reengage to return the body to a resting state.

Combining the Polyvagal theory (Porges, 1995, 1997) and Gray's (1987) motivational theory, Beauchaine's (2001) Integrated Model of Autonomic Functioning supposes the role of ANS in emotion regulation could be examined through clarifying how the SNS and PNS interact. Where Porges' (1995, 1997) theory links vagal tone and system regulation to both the SNS and the PNS, Beauchaine (2001) suggests regulation of body systems is controlled by the PNS alone. Instead of acting as a regulatory system of body functioning, the SNS is linked to motivation that dictates behavior responses based on the behavioral activation system and behavioral inhibition system described in Gray's (1987) motivation theory. In this theory, SNS activity controls brain responses, of which there are three: fight or flight, reward (behavior activation), and punishment (behavior inhibition). When the reward system is dominant, an individual either engages or actively avoids stimuli. During this time, SNS activity can be detected through increases in heart rate. When the punishment system is dominant, an individual will passively avoid stimuli, which can be seen through electrodermal activity (e.g., sweat on skin), a measure of SNS activity.

Therefore, Beauchaine's (2001) Model of Autonomic Functioning supposes that the PNS is responsible for regulation and is measured through the cardiac vagal tone, while the SNS controls brain systems responsible for behavior. Together, the SNS and PNS contribute to emotion and behavior predispositions and can be indexes of emotion regulation. Emotional dysregulation and maladaptive behavioral responses (e.g., externalizing or internalizing behaviors) occur when either the SNS behavior or PNS regulatory systems are out of sync. Under-responsivity of the SNS paired with PNS withdrawal is associated with externalizing

behaviors, while over-responsivity of the SNS and excessive PNS withdrawal is associated with internalizing behaviors (Beauchaine et al., 2001).

Respiratory sinus arrhythmia (RSA) is a phenomenon in which respiration can cause variation within HRV, as HRV increases with inhalation and decreases with exhalation (Kemp & Quintana, 2013). Higher RSA is an indicator of greater vagal cardiac control, suggesting calmer ANS (Patriquin et al., 2011). Conversely, lower RSA indicated poor vagal cardiac control which may suggest activated fight or flight defensive behaviors or the inability to regulate behavioral states. Neuhaus and colleagues (2014) suggested RSA may measure sensitivity to both positive and negative contextual factors. While low RSA may indicate vulnerability to stressful stimuli, high RSA may suggest the child is receptive to positive stimuli in the environment. As a result, RSA has been used in research as a biomarker of emotion regulation (e.g., Barbier et al., 2022; Patriquin et al., 2011) to examine associations between emotion regulation and cognitive ability (e.g., Patriquin et al., 2011; Staton et al., 2009), psychopathology (e.g., Cheng et al., 2020;) and internalizing and externalizing behaviors (e.g., Barbier et al., 2022; Hartman et al., 2019).

As low baseline RSA has been associated with psychopathology, RSA is also used to measure emotion dysregulation (Beauchaine, 2015a, 2015b). Reduced HRV and low resting RSA is associated with maladaptive emotional regulation and has been linked with clinical features of depression (e.g., Hartman et al., 2019), anxiety (e.g., Chalmers et al., 2014), and ASD (e.g., Cheng et al., 2020). Several studies have indicated that autistic children have lower baseline RSA compared to their non-autistic peers (e.g., Barbier et al., 2022; Neuhaus et al., 2014). For autistic children, research has suggested lower baseline RSA and HRV may be a result of hyperawareness and over-responsivity to changes in the environment (Cheng et al., 2020). Van Hecke and colleagues (2009) found autistic children had lower overall RSA when exposed to an unfamiliar person compared to non-autistic controls. They suggested autistic

children may view an unfamiliar person as threatening, which activates their fight or flight response and reduces their ability to respond with appropriate social behaviors. Barbier and colleagues (2022) suggested baseline RSA differences between autistic children and their non-autistic peers could be associated with presence of co-occurring behaviors, as anxiety, depression and ADHD have all be associated with decreases in baseline RSA (Bellato et al., 2020; Guy et al., 2014; Harman et al., 2019). Kushki and colleagues (2014) only found a marginal difference in baseline RSA when comparing autistic and non-autistic children, which they attributed to the medications the autistic children were taking.

Emotion and Behavior Difficulties

Emotion regulation failures (i.e., lack of regulation) and emotion misregulation (i.e., use of inappropriate regulation strategies for given situation) are present in various forms of psychopathology, which can create difficulties with emotion intensity, frequency, duration, and type (Beauchaine, 2015b; Gross & Jazaieri, 2014). Further, psychopathology can lead to emotion dysregulation when a) individuals have difficulties being aware of and understanding the context of specific emotions, b) knowledge of individuals desired goals and emotion states, and c) appropriate implementation of regulation strategies (Gross & Jazaieri, 2014). Disrupted emotion regulation processes and impaired capacity to appropriately regulate emotions lead to maladaptive behaviors which can be risk factors for psychopathology (Young et al., 2019).

Achenbach and Edelbrock (1978) broadly defined a spectrum of maladaptive behaviors as the internalizing-externalizing dichotomy. Where internalizing behaviors are directed internally and impact the child's psychological environment, externalizing behaviors are directed outward and affect the child's external environment (Achenbach & Edelbrock, 1978; Liu et al., 2004). While the internalizing-externalizing dichotomy is conceptualized as a continuum, it is possible for children to display many different behaviors, such as aggression and anxiousness, at

the same time (Achenbach & Edelbrock, 1978; Hinshaw, 1987; Huberty, 2017). Psychopathology occurs when persistent behavior patterns are severe and cause dysfunction or impairment (Bitsko et al., 2022). Internalizing and externalizing behavior disorders are associated with longer- term health outcomes, poor academic outcomes, poor social outcomes, and higher rates of suicide attempts (Bitsko et al., 2022; Huberty, 2017; Masi et al., 2017).

Internalizing Behaviors. Internalizing behaviors (e.g., withdrawal, apathy, excessive crying, need for sameness, irritability, & avoidance) stem from overcontrolled emotions through expending energy to inhibit emotions, behaviors, and responses (Hanson & Jordan, 2020; Huberty, 2017). These behaviors can be risk factors for psychopathology, the most common of which include major depressive disorder, dysthymia (persistent depression), and anxiety disorders (Hanson & Jordan, 2020; Huberty, 2017).

Compared to their non-autistic peers, autistic children experience anxiety and depression at a higher rate (e.g., Montazeri et al., 2020; Rai et al., 2018). Rai and colleagues (2018) examined depression trajectories of autistic and non-autistic children throughout childhood and adolescence. At age 10, autistic children reported higher levels of depressive symptoms than their non-autistic peers. The pattern of elevated depression rates for autistic children compared to non-autistic children continued through adolescence until age 18 years. Montazeri and colleagues (2020) found 28.8% of autistic children met clinic depression thresholds, more than double that of non-autistic children, and overall depression severity was higher for autistic children. However, Mayes and colleagues (2011) found that whereas depression and anxiety symptoms were more common in autistic children than non-autistic children, symptom severity was lower for autistic children than children with anxiety or depression. Prevalence rates of depression and anxiety in autistic children and adolescents can vary significantly (e.g., DeFilippis, 2018; Pezzimenti et al., 2019). It is estimated that depression rates in autistic children range from 0.9%

to 50%, with an overall lifetime prevalence rate of 10% to 53% (Chandrasekhar & Sikich, 2022; Hudson et al., 2018). Some studies have reported approximately 40% of autistic children have a co-occurring anxiety disorder but can range between 22 to 84%, with specific phobias (29-44%), separation anxiety (9-38%), obsessive compulsive disorder (17-37%), generalized anxiety disorder (15-35%), and social anxiety (16-30%) as the most common (Kent & Simonoff, 2017; Vasa & Mazurek, 2015; Zaboski & Storch, 2018).

Research has indicated cognitive ability is a factor in the prevalence and presentation of depression and anxiety in autistic children and adolescents (e.g., Chandrasekhar & Sikich, 2022; Mayes et al., 2022). Prior research agrees autistic children with average to above average IQs were at greater risk for high rates of depression and generalized anxiety (Mayes et al., 2022; Pezzimenti et al., 2019). Autistic children with higher IQs are more likely to have generalized anxiety disorder and obsessive-compulsive disorder than autistic children with lower IQs, who are more likely to have separation and social anxiety (Mayes et al., 2022). Generalized anxiety disorder occurred in 45% of autistic children with IQ $\geq$ 70, whereas 47% of autistic children with IQ $\leq$ 70 had separation anxiety (Mayes et al., 2022). Autistic children with higher IQs are more likely to report anhedonia, self-deprecating thoughts, and increased rigidity, whereas autistic children with lower IQs often present with behaviors such as crying, aggression, and self-injury (Pezzimenti et al., 2019).

Researchers have proposed that autistic children with higher IQs are more susceptible to internalizing behaviors because they have greater self-awareness and are cognizant of their impairments, which may lead to greater negative self-image and lower self-worth (Magnuson & Constanino, 2011; van Steensel & Heeman, 2017). They may have a greater desire for peer relationships but experience the stress of having to meet social norms or are bullied, increasing their risk for depression and anxiety (Chou et al., 2020; Rai et al., 2018). However, some

research has indicated no association between internalizing behaviors and cognitive ability (Brereton et al., 2006; Gotham et al., 2015; Strang et al., 2012). Gotham and colleagues (2015) found no association between IQ and depression symptoms in autistic adolescents and young adults with IQ ≥ 70. Strang and colleagues (2012) also found no relation between cognitive ability and depression or anxiety symptoms in autistic children with IQ ≥ 70. Brereton and colleagues (2006) also noted IQ was not associated with overall levels of anxiety or depression.

Externalizing behaviors. Broadly, externalizing behaviors are undercontrolled behaviors, as little energy is expended to control emotion and behavioral responses (Hinshaw, 1987; Huberty, 2017; Liu, 2004). Behaviors in this category include hyperactivity, attention problems, conduct problems, and aggression (Achenbach & Edelbrock, 1978). One in four autistic children exhibit disruptive behaviors, with aggression, antisocial behaviors (e.g., breaking the rules and theft), irritability, and non-compliance as the most common (Kaat & Lecavalier, 2013). Research has indicated 68% of autistic children and adolescents display aggression towards caregivers and 47% have been aggressive towards non-caregivers (Kanne & Mazurek, 2010). Van den Boogert and colleagues (2021) found that in autistic children sensory processing difficulties and hypersensitivity to the environment were a risk factor for elevated levels of aggression. While those with sensory processing difficulties were more likely to display reactive aggression, autistic children with sensory seeking behaviors had elevated levels of proactive and reactive aggression. Aggressive behaviors in autistic children are associated with poor social relationships, self-injury, victimization, increased hospitalization, increased family stress, and poor academic outcomes (Fitzpatrick et al., 2016).

Compared to internalizing behaviors, externalizing behaviors may lead to more mental health referrals as disruptive or aggressive behaviors are more obvious and easier to identify (Huberty, 2017). Historically, it has been difficult to distinguish between hyperactivity (i.e.,

motor overactivity) and attention deficits (i.e., inattention in structured settings) (Hinshaw, 1987; Liu, 2004). Many autistic children display inattentive behaviors and, while autistic children can maintain focused attention in certain contexts, the quality of attention and the ability to orient attention may be impaired (Hours et al., 2022) As a result, attention problems in autistic children may present as if they have difficulty shifting attention away from stimuli rather than being excessively distractable or having a short attention span (Hours et al., 2022). Additionally, while social interaction deficits are core symptoms of ASD, co-occurring ADHD symptoms may exacerbate these difficulties (Harkins et al., 2021). Impulsivity and hyperactivity associated with ADHD may further impact autistic children's social interactions by increasing behaviors such as interrupting others, impatience while waiting, and excessively talking (Antshel & Russo, 2019). Further, Harkins and colleagues (2021) found autistic children with ADHD exhibit greater social impairment than children with ADHD alone. Also, children with combined ASD+ADHD display greater adaptive functioning impairment than children with ASD only (Antshel et al., 2016).

The research examining the association between cognitive ability and externalizing behaviors is mixed (Mayes et al., 2022). IQ influences externalizing behaviors, as autistic children with IQ < 70 have had higher rates of hyperactivity, tantrums, lying and cheating, bullying, irritability, and conduct problems than those with IQ < 70 (Mayes et al., 2022; Rosen et al., 2018). Mayes and colleagues (2022) found 60.2% of autistic children with IQ > 70 had symptoms of oppositional defiance disorder, whereas oppositional defiance behaviors only occurred in 45.4% of autistic children with IQ < 70. They also reported the lowest rates of aggression, hyperactivity, self-injury behaviors, and inattention occurred in autistic children with IQ 120-149. Researchers have suggested the elevated rates of disruptive behaviors in autistic children with IQ > 70 may be related to increased social demands and higher verbal ability (Lerner et al., 2018; Mayes et al., 2022). Witwer and Lecavalier (2010) found no association

between oppositional defiance behaviors and IQ. Kanne and Mazurek (2010) also found no association between IQ, verbal ability, and disruptive behaviors. Therefore, it is important to consider IQ and cognitive ability when examining disruptive behaviors in autistic children.

Overall, emotion regulation is an important process that impacts physiological and behavioral functioning (e.g., Beauchaine, 2001; Gross, 2015a, 2015b, Gross & Jazaieri, 2014). ASD symptom severity and cognitive functioning may lead to emotion dysregulation in autistic children leading to poor outcomes (e.g., Cai et al., 2018; Cibralic et al., 2019). Therefore, examining physiological and behavioral outcomes of emotion regulation can provide insight into what might mitigate or exacerbate these outcomes for autistic children. Since research has shown heterogeneity in emotion regulation in autistic children based on ASD symptom severity and IQ (Mayes et al., 2022), latent profiles may allow for better understanding of symptom presentation and lead to more individualized treatment for autistic children.

Parenting and Child Emotion Regulation

Although emotion regulation can influence child outcomes, parents may influence their child's emotion and behavior outcomes through their parenting behaviors and styles (e.g., Belsky, 1984; Morris et al., 2007). As Bronfenbrenner's Bioecological Systems Theory (1979, 2005) suggests, a child's development is influenced by their environment. The microsystem is the most immediate environment that a child interacts with and includes family, school settings, and friends. Parents are one aspect of the microsystem as they can influence their child's emotion regulation through parenting behaviors and styles. The literature examining the association between parenting behaviors and child emotion regulation outcomes in non-autistic children is robust (e.g., Baker, 2018; Morris et al., 2017; van Lissa et al., 2019). Unfortunately, parenting research in ASD literature is limited as *refrigerator mothers* (i.e., mothers who were distant & cold toward their child; Kanner, 1943) were believed to cause ASD and created a hesitancy to

explore the parenting-child outcome association in the ASD field. However, examining parenting in families with autistic children may provide insight into emotion regulation outcomes (e.g., physiological & behavioral) for autistic children.

Morris and colleagues (2007) proposed the tripartite model of the impact of the family on children's emotion regulation and adjustment, which suggests child emotion regulation is influenced by three processes (i.e., observation, emotional climate of the family, and parenting practices) that then impact child outcomes. Observation refers to emotion modeling, referencing, and emotion cognition the child observes other family members engaging in. The emotional climate of the family encompasses family expressivity and marital relations. While observation and the emotional climate of the family are important contributors to children's emotion regulation outcomes, they will not be a focus of this review. Instead, the discussion will center on parenting practices, which Morris and colleagues define as parenting behaviors such as parental warmth and positive parenting. (Morris et al., 2017).

Parental warmth is the parental expressions of love, affection, comfort, and support toward their child (Khaleque, 2013). Positive parenting is defined as parenting behaviors that foster warm and supportive parent-child relationships through praise, listening, positive discipline, and mutual respect (Morris et al., 2017; Seay et al., 2014). Positive parenting helps to increase a child's sense of security and feelings of emotional safety as the parents recognize and respond supportively to their child's emotional cues (Morris et al., 2017). Parental warmth is associated with positive emotion regulation outcomes, such as lower rates of internalizing and externalizing behaviors (Pinquart, 2017), lower adolescent aggression (Klevens & Hall, 2014), and lower child anxiety (McLeod, et al., 2007). High parental warmth in mothers and fathers is associated with increased positive self-esteem, emotional stability, and emotional responsiveness, in addition to lower hostility and aggression (Khaleque, 2013). Peterson and

colleagues (2018) found better child emotion regulation was associated with elevated sensitive parenting, while poor emotion regulation was associated with low sensitive parenting. When parents exhibit low levels of positive parenting, child internalizing behaviors increased (Burlaka et al., 2017). Lee and colleagues (2018) found low parental warmth in fathers was associated with increased internalizing behaviors and marginally associated with higher externalizing behaviors.

Parental warmth may act as a protective factor and attenuate symptoms of depression and anxiety over time, as adolescents whose parents scored high in parental warmth were less likely to negatively respond to criticism and had lower rates of internalizing symptoms two years later (Butterfield et al., 2021). Lloyd and colleagues (2017) found that children with reactive temperament are at higher risk for increased depressive symptoms during adolescents. However, if those children have parents who display high parental warmth, they displayed lower rates of internalizing behaviors over time (Lloyd et al., 2017). Van der Voort and colleagues (2014) found mothers' parental sensitivity in infancy and middle childhood predicted lower rates of adolescent depressive and anxious behaviors. Rothenberg and colleagues (2020) examined how parental warmth influenced the internalizing and externalizing behavior trajectories of adolescents. Overall, their results support the hypothesis that parental warmth acts as a protective factor as it was associated with lower internalizing behaviors and lower externalizing behaviors in children and adolescents over time.

Emotionally supportive parenting practices are also a predictor of child baseline RSA and influences child physiological emotion regulation trajectories (Perry et al., 2013). When mothers engage in more emotionally supportive parenting practices, their children are more likely to have higher baseline RSA at an earlier age than those children with mothers who show low emotional support, suggesting children with more supportive parents are able to better regulate their

emotions physiologically. Low maternal emotional support also predicted poor child physiological emotion regulation and greater frustration (Perry et al., 2013). Additionally, child physiological emotion regulation moderates the relation between maternal emotional support and child behavior outcomes, suggesting that when children have low physiological emotion regulation, they have worse behavioral outcomes when their mothers engage in elevated non-supportive parenting practices (Perry et al., 2012). Adolescents who have low emotionally supportive parents and experienced high levels of stress over time were more likely to have low baseline RSA (Fox et al., 2019). Further, children had high resting RSA when their parents had better control of their own emotions (i.e., high resting RSA), used more adaptive coping strategies themselves, and engaged in adaptive emotion coaching with their child (Shih et al., 2017). Cui and colleagues (2015) also found parent positive affect during parent-adolescent interactions was positively associated with higher RSA in adolescents, while parent anger during those interactions was associated with decreased RSA in adolescents. The authors suggest their results indicate parenting practices may be an underlying mechanism as parent emotions and affect create an emotional climate for parent-child interactions which influences adolescent's physiological emotion regulation (Cui et al., 2015).

Overall, as theorized in the tripartite model of the impact of the family on parenting (Morris et al., 2007), parenting behaviors influence child emotion regulation outcomes, such as physiological responses and behavioral outcomes (e.g., Morris et al., 2017; Perry et al., 2013). Positive parenting behaviors, such as parental warmth, may serve as a protective factor against negative outcomes (Khaleque, 2013). However, while much of the parenting research examines the direct influence of parenting on child emotion regulation, developmental theory would suggest a bidirectional relation between parent and child behaviors. Therefore, research should

not only explore how parenting behaviors influence child outcomes, but also examine how child emotion regulation outcomes influence parenting behaviors.

Bidirectional Parenting Behaviors and Child Outcomes

The bidirectional relationship between parenting behaviors and child outcomes has been examined in many developmental theories (e.g., Belsky, 1984; Morris et al., 2007; Sameroff, 1975). The tripartite model of the impact of the family on children's emotion regulation (Morris et al., 2007) suggests each of the three processes (observation, parenting practices, & emotional climate of the family) have a bidirectional association with child emotion regulation and are moderated by child characteristics. Parents may engage in different parenting behaviors and respond differently to a child who is emotionally reactive than a child with a calmer temperament (Morris et al., 2007). For instance, Klein and colleagues (2016) found maternal warmth and child temperament are bidirectionally linked, as when children are calm and have few behaviors, mothers are more likely to engage in warm parenting behaviors which then increases positive child outcomes.

Belsky's (1984) determinants of parenting process model also supports the bidirectional relation between parenting and child outcomes. In Belsky's model (1984), factors associated with the parent (e.g., depression & stress), child (e.g., intellectual disability, maladaptive behaviors, & symptom severity), and environmental (e.g., family & marital conflict) can affect parenting quality (de Haan et al., 2013; Lisitsa et al., 2021). Therefore, when examining the effect of parenting on child outcomes, it may be necessary to explore the bidirectional relation as well. Child characteristics and behaviors can make it easier or more difficult for parents to interact with and meet their child's needs which then can affect the child's outcomes (Belsky, 1984). For instance, child internalizing and externalizing behaviors may impact parenting behaviors, as depression and aggression in adolescence may increase negative parenting

behaviors (e.g., de Haan et al., 2013; Roche et al., 2011). Indeed, higher levels of child internalizing and externalizing behaviors were associated with lower parental warmth over time, as parents are likely to react to negative behaviors by withdrawing warmth and support (Landsford et al., 2018).

Sameroff's (1975) transactional model of development proposed parent-child outcomes were a sequence of transactions in which parents of children with problem behaviors interacts with their child in such a way that creates a self-fulfilling prophecy of increased problem behaviors. Thus, the model suggests parents' perceptions of their child can affect how they interact with the child which may lead to increased behavioral difficulties (Sameroff, 1975). In support of Sameroff's (1975) transactional model of development, when children had increased externalizing behaviors, their parents were less likely to engage in positive parenting behaviors over time and predicted an increase in child externalizing behaviors, thus creating a positive feedback loop (Serbin et al., 2015). In contrast, child internalizing behaviors predicted an increase in positive parenting behaviors over time and was then associated with a decrease in child internalizing behaviors, creating a negative feedback loop. These feedback loops support the transactional model as the type of child behaviors influences whether parents use positive or negative parenting behaviors. Then, how parents respond to their child's behaviors can predict either an increase or decrease in child behaviors which completes the feedback loop. While externalizing behaviors and negative parenting behaviors exacerbate one another, the use of positive parenting behaviors decreases internalizing behaviors over time suggesting parents are adapting to the decrease in behaviors and engaging in different parenting behaviors (Serbin et al., 2015).

As supported by developmental theory, the research indicates a bidirectional relation between child emotion regulation and parenting behaviors (e.g., Hastings et al., 2019; Roche et

al., 2011; Serbin et al., 2015). However, most of the research exploring this association is focused on non-autistic children and their parents (e.g., Cui et al., 2014; de Haan et al., 2013). As previously discussed, autistic children have unique challenges related to emotion regulation and social communication (e.g., Veatch et al., 2021; Zantinge et al., 2017). Therefore, it is important to understand how these challenges may affect parenting behaviors and, in return, influence the emotion regulation outcomes of autistic children. Further, as the tripartite model of the impact of the family on children's emotion regulation (Morris et al., 2007) notes the bidirectional association is moderated by child characteristics, it is possible the heterogenous presentation of ASD symptoms and IQ could impact this association. Therefore, through the use of latent profile analysis using ASD symptoms and IQ as indicators, it may be possible to examine how profile membership influences parenting behaviors and child outcomes.

Parenting and ASD

While research examining the association between parenting and ASD is limited, a few studies have indicated parents interact with autistic children and non-autistic children differently (Gau et al., 2010; Maljaars et al., 2014). Maljaars and colleagues (2014) examined child behavior problems and parenting behaviors in both families with and without an autistic child. As supported by other research, autistic children displayed increased internalizing and externalizing behaviors. However, how parents responded to those behaviors differed for parents of autistic children compared to parents of non-autistic children. When responding to externalizing behaviors, both parents of autistic and non-autistic children had increased discipline and harsh parenting practices, while parents of autistic children engaged in more rule setting and parents of non-autistic children used less positive parenting behaviors. When children have high internalizing behavior, parents of autistic children are more likely to make attempts to control their child's environment. These parents were also more likely to adjust their communication

patterns by simplifying instructions and questions. As a result, it is possible that parents of autistic children attempt to prevent behaviors from occurring through rule setting and reducing environmental stimuli instead of focusing on consequences of negative behaviors. Parents of autistic adolescents were also more likely to encourage their child to make their own decisions, promote problem solving skills, and plan activities for their child. Parents of non-autistic children engage in similar behavior patterns but are more likely to engage in these behaviors during childhood rather than adolescence. Further, the study only found weak correlations between parenting practices and child behaviors, which the authors attributed to the measures used in the study (Maljaars et al., 2014).

Another study examined how parents interact with their autistic child or a child with Down syndrome compared to a non-autistic sibling (Gau et al., 2010). Overall, parents were less affectionate, more controlling, and more overprotective with autistic children than with children with Down syndrome and non-autistic siblings. Prior research also suggests parents interact less with their autistic children compared to non-autistic siblings, a pattern that was not found in parent of children with other developmental or intellectual disabilities (Kasari & Sigman, 1997; Konstantareas & Homatidis, 1992). While mothers of autistic children had lower affection with autistic children compared to non-autistic siblings and children with Down syndrome, fathers' parenting behaviors may also be more affected if they have an autistic child (Gau et al., 2010). Not only are fathers less affectionate with their autistic child, but studies have also indicated that having an autistic child was the strongest predictor of low father engagement (Gau et al., 2010; Konstantareas & Homatidis, 1992).

Overall, developmental theory and prior research would suggest a bidirectional association between parenting behaviors and child emotion regulation outcomes (e.g., Morris et al., 2007; Sameroff, 1975). However, the research examining this relation in autistic children and

their parents is limited. As autistic children and their parents face unique challenges related to both parenting and emotion regulation (e.g., Gau et al., 2010; Maljaars et al., 2014; Osborne & Reed, 2009), further examination may provide insights into what may exacerbate or ameliorate the relation. Additionally, by examining the bidirectional association and how it is mediated by ASD symptom severity and IQ profile membership, results may help future research understand the strength of the parent- and child-driven effects which may inform future interventions.

Current Study

Goal 1: ASD Symptom Profiles

Due to the heterogenous presentation of ASD, core ASD symptoms (SC/I and RRB) present differently in autistic children (Lord et al., 2018; Masi et al., 2017). Thus, the heterogeneity of ASD symptom severity and IQ may also influence child emotion and behavior difficulties. For Goal 1, I analyzed profiles of ASD symptoms and IQ in order to examine profile differences in child emotion and behavior difficulties (i.e., RSA and internalizing & externalizing behaviors). I used latent profile analysis to create profiles of autistic children (IQ ≥ 70) using IQ scores and the severity of ASD symptom domains (i.e., SC/I & RRB) as indicator variables to create the profiles. I used profile membership to compare resting RSA and internalizing and externalizing behaviors.

Due to the data-driven nature of latent profile analysis, it can be difficult to predict the total number of profiles. In latent profile analysis, underlying patterns in the data identified and subgroups are examined (Berlin et al., 2014; Weller et al., 2020). Prior research using latent profile analysis often discusses subgroups in terms of the degree to which they are associated with the indicator variables. For instance, Prefontaine and colleagues (2022) describes the profiles in their study as having mild, moderate, or severe impairment. Sullivan and colleagues (2019) characterized their subgroups by categorizing them by behavioral severity – low, mid,

and high. As the sample for the current study only included autistic children with IQ ≥ 70 (i.e., borderline to above average IQ), I expected to see profiles that have average IQ or above average IQ. As higher RSA was associated with higher cognitive ability (Staton et al., 2009), I predicted that Profiles with higher IQ would have higher RSA. Consistent with previous research suggesting that higher IQ is associated with greater awareness of their impairments (Magnuson & Constanino, 2011; van Steensel & Heeman, 2017), I expected to see higher levels of internalizing behaviors, such as anxiety or depression in profiles that have higher IQs compared to profiles with lower IQs. According to Mayes and colleagues (2022), autistic children with higher IQs have higher rates of disruptive behaviors as they have greater social demands but still experience challenges in social situations. Therefore, I expected profiles with higher IQs to have higher levels of externalizing behaviors compared to profiles with lower IQs. As autistic children have lower resting RSA (i.e., lower emotion regulation) compared to non-autistic children (Barbier et al., 2022), I expected profiles that have higher symptom severity to have lower RSA. Additionally, prior research suggests depression and anxiety have a higher prevalence rate in autistic children with lower symptom severity (e.g., DeFilippis, 2018; Pezzimenti et al., 2019). Thus, I expected profiles with lower ASD symptom severity to have higher levels of internalizing behaviors. As ASD symptoms and externalizing behaviors exacerbate one another (Harkins et al., 2021), I expected profiles with high ASD symptom severity to have higher levels of externalizing behaviors.

While I expected these patterns (e.g., high IQ is associated with high internalizing behaviors & high symptom severity is associated with high externalizing behaviors) to be the same across profiles, I expected differences between group based on the combination of ASD symptom severity and IQ (see Figure 1). For instance, a profile with high symptom severity and above average IQ would have high internalizing and externalizing behaviors, as well as low

resting RSA. A profile with low symptom severity and average IQ would have high internalizing behaviors, low externalizing behaviors, and low resting RSA. Therefore, I expected differences between profiles based on the combination of ASD symptom severity and IQ.

Goal 2: Bidirectional Association Between Parenting Behaviors and Child Outcomes

Developmental theory proposes a bidirectional relationship between parenting behaviors and child outcomes (e.g., Belsky, 1984; Morris et al., 2007; Sameroff, 1975). Parenting behaviors may influence autistic children's emotion and behavior difficulties, which then may impact parenting behaviors in the future. Examining these processes may provide better insight into interventions that may benefit autistic children and their parents. Further, these associations may be moderated by the heterogenous presentation of ASD symptoms and IQ.

For Goal 2.1 (see Figure 2), I examined the bidirectional association of parenting behaviors and child emotion and behavior difficulties (e.g., RSA and internalizing & externalizing behaviors). I examined Parent Effects models (i.e., parenting behaviors affecting child emotion and behavior difficulties) and Child Effects models (i.e., child emotion and behavior difficulties affecting parenting behaviors). Parental warmth is a protective factor for depression and anxiety (Butterfield et al., 2021), lower levels of aggression (Klevens & Hall, 2014), and higher resting RSA (Fox et al., 2019). Therefore, for Parent Effects models, I expected an increase in parental warmth to be associated with higher RSA (i.e., better emotion regulation), lower internalizing behaviors, and low externalizing behaviors. For Child Effects models, I expected an increase in internalizing behaviors would be associated with increased parental warmth while high externalizing behaviors is associated with lower parental warmth (Serbin et al., 2015). Also, high externalizing behaviors in autistic children was associated with harsher parenting practices (Maljaars et al., 2014). Additionally, high RSA (i.e., better emotion

Figure 1

Goal 1 Hypotheses for Profile Characteristics by Indicator Severity

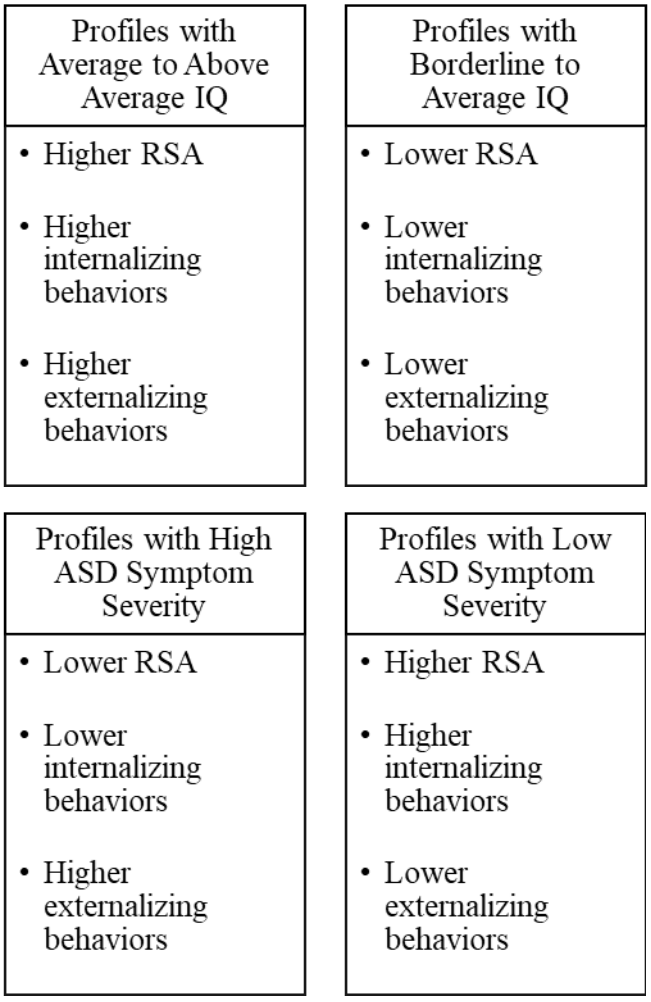
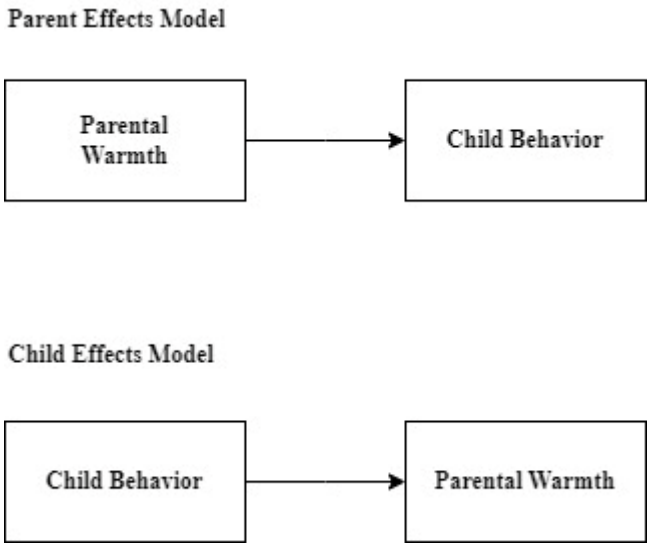


Figure 2

Goal 2.1: Proposed main effects for Parent Effects Model and Child Effects Model



regulation) in children is associated with better emotion regulation in their parents (Fox et al., 2019). Therefore, I expected an increase in child internalizing behaviors and child RSA to be associated with increased parental warmth. I also expected an increase in child externalizing behaviors to be associated with low parental warmth.

For Goal 2.2 (see Figure 3), I examined whether the ASD symptom profiles created in Goal 1 moderated the association between parenting behaviors and child emotion and behavior difficulties (e.g., RSA and internalizing & externalizing behaviors). As the total number of profiles was undetermined, I expected the profiles to moderate the association between parenting behaviors and child outcomes in a similar pattern to Goal 1. In Parent Effects models, I expected high levels of parenting warmth to be associated with higher RSA for profiles that had borderline to average IQ and high symptom severity (predicted lower RSA) compared to profiles with average to above average IQ and low symptom severity (predicted higher RSA). I also expected high levels of parental warmth to predict lower child internalizing and externalizing behaviors for profiles with average to above average IQ and ASD symptom severity (predicted higher internalizing and externalizing behaviors) compared to profiles with borderline to average IQ and high ASD symptom severity (predicted lower internalizing & externalizing behaviors). For Child Effects models, I expected high levels of RSA to predict higher parental warmth for profiles with borderline to average IQ and high symptom severity compared to profiles with average to above average IQ and low ASD symptom severity. I also expected high levels of internalizing and externalizing behaviors to predict lower parenting warmth for profiles with average to above average IQ and high ASD symptom severity.

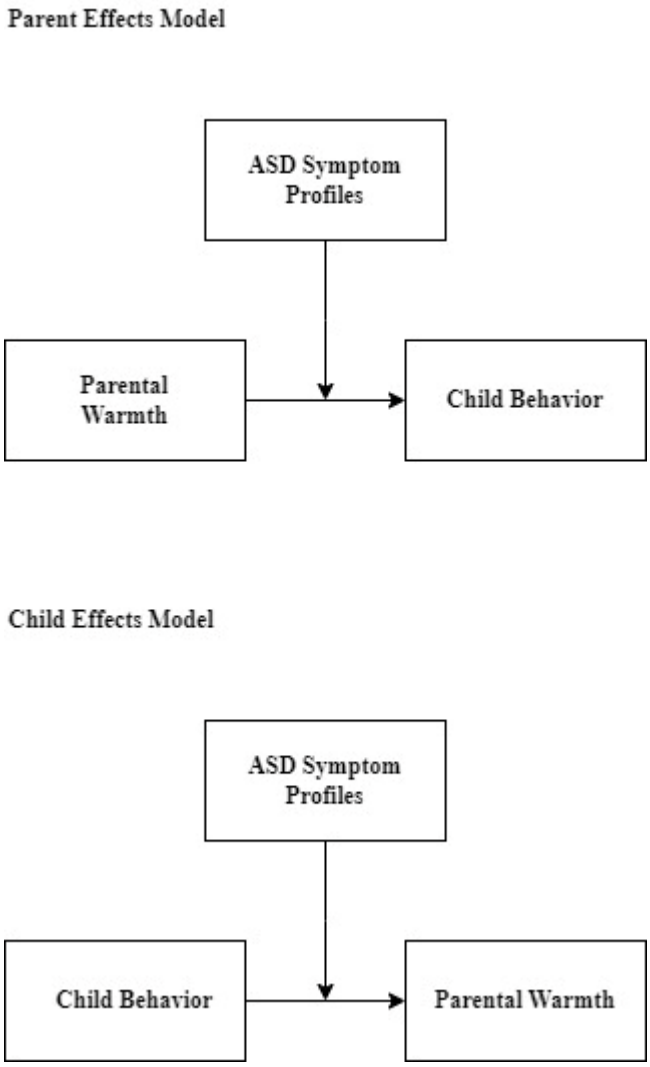
Method

Participants

The current study is part of a larger investigation of families of autistic children at Texas

Figure 3

Goal 2.2: Hypothesized Parent Effects Model and Child Effects Model Moderated by ASD Symptom Profiles



Christian University (TCU) and Southern Methodist University (SMU). Families with an autistic child between the ages of 10 to 17 years were recruited from the surrounding Dallas-Fort Worth metropolitan area and targeted ASD community events, adolescent psychologists, pediatricians, centers providing ASD services, schools, social media and SPARK, a foundation for increasing ASD research. Eligibility criteria for families required parents to be married or cohabitating for at least 1 year, to live with their child at least 50% of the time, and to be able to read and speak English.

The child also needed to meet diagnostic criteria to be eligible for the study, which included a community diagnosis of ASD (i.e., the child has received their diagnosis from a psychiatrist, diagnostician, school district, etc.). Children with co-occurring conditions of intellectual disability, bipolar, psychosis, schizophrenia, visual or hearing impairments, serious medical or neurological conditions, or genetic or metabolic disorders were excluded from the study. Families were screened for eligibility by trained graduate research assistants and children participated in an in-person assessment (described below) to confirm the family qualified for the study.

In the current study, 119 families were eligible for participation. Two families were excluded from analysis as they were not in heterosexual relationships. Therefore, the final sample included 117 mothers, fathers, and children. Most children were male (n = 94), non-Hispanic White (69.2%), and about 13 years old (M = 13.10; SD = 2.19). Parents were married (89.7%), non-Hispanic White (mothers: 73.5%; fathers: 76.1%), had an annual income greater than $50k (87.0%), and college educated (mothers: 87.2%; fathers: 86.3%). On average, mothers were 43-years-old (M = 43.23; SD = 6.40) and fathers were 45-years-old (M = 45.11; SD = 6.93).

Procedure

Participants were screened via phone by trained graduate research assistants at both SMU and TCU. If participants met the initial eligibility criteria for the study, at least one parent (both parents were able to attend if desired) and the child were scheduled for an in-person assessment visit to confirm they qualified for the study. Families were given the option to participate at either SMU or TCU and completed all visits at their chosen site. Parents gave informed consent and the child assented to participate in the study. Parents provided a copy of their child's diagnosis from a community provider (e.g., psychologist, school district, pediatrician, etc.).

Assessments: Time 1 Visit 1

At the first visit, the parent and child separately participated in a series of assessments. Parents completed the Autism Diagnostic Interview – Revised (ADI-R; Rutter et al., 2003) and the Vineland Adaptive Behavior Scales (Yang et al., 2016). After the interview portion, parents completed the Social Responsiveness Scales-2 (Bruni, 2014) to assess the child's current ASD symptom severity. In a separate room, the child completed assessments of their IQ (Differential Ability Scale-II [DAS-II]; Elliott, 2018), language ability (Peabody Picture Vocabulary Test [PPVT]; Dunn & Dunn, 2007), and ASD symptoms (Autism Diagnostic Observation Schedule – Module 3 [ADOS]; Lord et al., 2012).

Altogether, the session took about 3 hours to complete. For participating in the study, families were compensated with $75 in gift cards and the child received an additional $10 gift card. Assessments were scored after visit completion. If a child scored below a 75 on the DAS-II or below 70 on the PPVT, the family was deemed ineligible to continue in the study (n = 19). Families that were not eligible to continue in the study were notified within a week of their assessment visit and could request a copy of their child's assessment summary.

Family Visit: Time 1 Visit 2 and Time 2

Eligible families participated in the family visit about 2 weeks after their assessment visit. The T1 (T1) Visit 2 and T2 (T2) sessions were identical, with the T2 visit occurring about 6 months after T1 Visit 2. Some of the tasks that are not included in the current study are not described below. During this visit, the child completed their portion of the study separately from the parents. First, parents completed a set of questionnaires about their demographics, health, relationship, and child. At the same time, the child completed a set of questionnaires about their family, parents, and emotions.

Electrode sensors were placed on the child with the parents present after the family played a game together. The parents and child then completed another set of questionnaires in separate rooms, after which, the child's baseline physiological functioning was collected. At the end of the session, the parents and child were debriefed, and safety protocols were in place in case families were distressed after their visit. After the T1 Visit 2 session, families were given $75 gift cards as compensation and the child was given an additional $10 gift card. At the end of the T2 visit, the family received $100 in gift cards as compensation with the child receiving an extra $10 gift card.

COVID-19 and Remote Visits

Due to the COVID-19 pandemic, in-person research was halted on March 11, 2020. As a result, it was determined that families who had not completed at least the T1 visits 1 and 2 would not continue in the study and no new families would be added to the study. This decision was made as only a few families had not completed their assessment visit (n = 6) or their T1 family visit (n = 5). Therefore, all data from T1 was completed in-person prior to the March 2020 COVID-19 protocols.

The remaining families (n = 22) who had yet to complete their T2 family visit completed a modified remote visit via Zoom. Families were contacted to inform them of the changes and provide new informed consent to participate in the remote visits. Families were compensated up to $135 for participating in the remote visits. If families were still interested, parents were sent links to complete questionnaires prior to the start of their remote visits. The family visit was divided into two parts: a family remote visit and a child remote visit. In the family remote visit, the parents and child reviewed the consent and assent main points and gave permission to have the session recorded. The tasks associated with the family remote visit are not relevant to the current study.

The child remote visit occurred about one day after the family visit via Zoom. At the beginning of the visit, research assistants reviewed the main points of study and received verbal assent from the child for their participation and to record the session. Then, the child completed a set of questionnaires about their family, feelings, and parents with the help of a research assistant. To keep the visit to about 90 min, the child received a modified set of questionnaires from the in-person visit. The order in which the child completed the surveys changed to ensure the questionnaires that were most important to the research study were completed first in the event the child became tired or the visit ran too long. As the study was remote, no physiological data was collected. The session ended after the child completed the final set of questionnaires, after which the child was debriefed.

Measures

The following are the measures used for Aim 1 (Latent Profile Indicator Variables) and Aim 2 (Child Physiological Measurement, Emotion Regulation Difficulties and Behavioral Response surveys, and Parenting Behavior Surveys,). As there are several measures for each aim, time point, type, and reporter, Table 1 (T1) and Table 2 (2) provide a condensed version of

the measures used in the study. Therefore, Table 1 and Table 2 include relevant information, such as the name of the measure, the time point of collection, sample size for the measure, descriptive statistics, and Cronbach's alpha for each reporter (e.g., mother, father, and child). This information will not be reported below in text, but justifications for major changes were provided.

Latent Profile Indicator Variables

Autism Symptoms. The Autism Diagnostic Observation Schedule-II (ADOS-II; Lord et al., 2012) is a clinical instrument used to assess the presence of current ASD symptoms and to diagnose individuals of all ages with ASD. The current study employed Module 3, as children were required to have verbal language skills to meet eligibility criteria for the study. During administration of the ADOS-II, a trained research assistant conducted a semi-structured interview and play-based activities to assess the core characteristics of ASD (social communication/interaction and restrictive and repetitive behaviors). Administrators code the behaviors during the ADOS-II. Clinical cutoffs indicate the level of ASD the child has (e.g., autism, autism spectrum, or non-spectrum). ADOS-II is a valid measure in diagnosing ASD. Reliability across the two sites was established at 80%. Administrators from one site scored the video of the other to ensure reliability for the two domains (percent agreement overall = 78.76%; percent agreement with the algorithm = 72.85%) was maintained throughout the study (n = 24). In the event the reliability fell below 80%, coders discussed their differences and came to an agreement for a consensus code (consensus percent agreement overall = 86.53%; percent agreement with the algorithm = 83.05%). Social Affect and Restricted and Repetitive Behavior scores will be used in the current study. Social Affect is scored on a scale of 0 (least severe) to 20 (most severe). Restricted and Repetitive Behaviors is scored on a scale of 0 (least severe) to 8 (most severe).

Table 1.

Time 1 Descriptive Statistics and Cronbach's Alpha

	Child (*n* = 117)				Mother (*n* = 117)				Father (*n* = 117)			
	N	*M*	*SD*	α	*N*	*M*	*SD*	α	*N*	*M*	*SD*	α
Latent Profile Indicators												
General Conceptual Ability	117	102.72	18.41									
Social Affect	117	9.38	3.37									
Restricted and Repetitive Behaviors	117	2.56	1.84									
Child Physiological Measurement												
Baseline RSA	110	6.70	1.26									
Child Emotion & Behavior Difficulties												
Depressive Symptoms	116	10.89	7.42	.86	116	16.47	7.17	.83	117	15.82	5.96	.77
Anxiety Symptoms	115	22.68	12.81	.91	117	20.41	13.47	.93	117	16.88	10.09	.89
Internalizing Behaviors					116	15.41	9.07	.87	117	14.46	7.64	.84
Externalizing Behaviors					116	12.54	9.65	.92	117	12.63	8.88	.91
Parenting Behaviors												
Parent-report Parental Warmth					116	27.33	2.55	.77	117	25.52	3.26	.83
Child-report Maternal Warmth	114	26.02	4.04	.87								
Child-report Paternal Warmth	114	24.04	5.31	.92								

Note: Variables are arranged by outcome and reporter. Shaded boxes indicate no data was collected for that reporter. RSA = Respiratory Sinus Arrythmia

Table 2.

Time 2 Descriptive Statistics and Cronbach's Alpha

	Child (*n* = 102)				Mother (*n* = 101)				Father (*n* = 100)			
	N	*M*	*SD*	α	*N*	*M*	*SD*	α	*N*	*M*	*SD*	α
Child Physiological Measurement												
Baseline RSA	70	6.59	1.36									
Child Emotion & Behavior Difficulties												
Depressive Symptoms	102	10.44	7.47	.87	101	16.06	7.08	.83	99	15.25	6.58	.83
Anxiety Symptoms	102	20.30	14.33	.93	101	17.67	12.61	.93	99	14.88	10.46	.91
Internalizing Behaviors					100	14.39	8.76	.88	99	13.16	7.77	.86
Externalizing Behaviors					100	10.14	7.83	.89	99	10.53	7.92	.89
Parenting Behaviors												
Parent-report Parental Warmth					101	27.37	2.68	.81	99	25.25	3.48n	.84
Child-report Maternal Warmth	100	25.76	4.59	.91								
Child-report Paternal Warmth	98	23.11	5.61	.93								

Note: Variables are arranged by outcome and reporter. Shaded boxes indicate no data was collected for that reporter. RSA = Respiratory Sinus Arrythmia

IQ. The Differential Ability Scales-II (DAS-II; Elliott, 2017) is a clinical instrument used to assess the cognitive ability of children by identifying their strengths and weaknesses in the areas of Verbal Ability, Nonverbal Reasoning, and Spatial Ability. The instrument is administered individually to children by trained researchers and includes cognitive assessment such as defining words, design recall, and pattern construction. An individual's scores on the DAS are converted to standard scores, which are used for comparison to a normative sample. The DAS-II is approved for use in research for children in clinical populations and is a valid measure of a child's cognitive ability. In the current study, only the General Conceptual Ability (GCA), a composite score based on the calculated standard scores for each domain, will be used.

Child Physiological Measurement

Baseline Respiratory Sinus Arrythmia. After completion of the Jenga task, research assistants or the child's parents placed electrodes on the left and right ribcage, right collarbone, sternum, side of the throat, back of the neck, and mid-spine. Child physiological data was collected using Biolab 3.1.1 (MindWare Technologies LTD, Gahanna, OH, USA, 2011). Heart Rate Variability (HRV) 3.1 software (Mindware Technologies, Gahanna, OH, USA, 2014) was used to analyze and obtain R-spikes from the data automatically. Trained research assistants manually reviewed the data and corrected any misidentified R-spikes or R-R intervals. Segments used for the current study include two baselines: a quiet sitting baseline and a neutral video baseline.

Child Emotion Difficulties and Behavioral Response Surveys

Internalizing and Externalizing Behaviors.

Child Depressive Symptoms. The Children's Depression Inventory (CDI; Kovacs & MHS Staff, 2011) is an assessment of a child's depression symptoms (e.g., feelings of sadness and worth, loss of interest in activities, etc.) during the previous two weeks. The CDI has two

versions: child-report and parent-report. With the child-report version, the child is given 28 sets of three statements and then selects the one that best represents how they have felt in the last 2 weeks. Items are scored based on the severity of the symptom, with more severe symptoms receiving a higher score. For instance, statements may ask about their loss of interest (e.g., 0 = *I have fun in many things*; 1 = *I have fun in some things*; 2 = *Nothing is fun at all*). The child-report version is scored reverse coding positively worded statements and then summing all items together. For the current study, a question asking about suicide ideation was removed.

For the parent-report version, the parents rated 17 statements about their child's depression symptoms on a 4-point Likert scale from 0 (*Not at all*) to 3 (*Much or Most of the Time*). Example items include statements such as "My child cries or looks tearful" or "My child has to push himself or herself to do homework." After recoding positively worded items, all item scores are summed together to calculate a total score. For the current study, both mothers and fathers completed the survey.

Child Anxiety Symptoms. The Screen for Child Anxiety Related Disorders (SCARED; Birmaher et al., 1997) measures child anxiety-related symptoms with child- and parent-report versions. In the child-report version, the child answers 41 statements associated with anxiety symptoms, such as nervousness, nightmares, fear of attending school, and difficulty breathing. Each statement is rated on a three-point Likert scale from 0 (*Not True or Hardly Ever True*) to 2 (*Very True or Often True*) based on how well the statement described the child in the past 3 months. The SCARED includes a total score, calculated by summing all items together, in addition to five subscales: Generalized Anxiety Disorder, Panic, Separation Anxiety, Social Phobia, and School Phobia. The current study will only use the Total Score.

The parent-report version is similar as they rate 41 statements associated with their child's anxiety symptoms, such as headaches at school, worrying, and feeling of being alone.

These statements are rated on a three-point Likert scale from 0 (*Not True or Hardly Ever True*) to 2 (*Very True or Often True*) based on how well the statement described their child in the past 3 months. Statements include "When my child gets frightened, he/she feels like passing out" and "My child has nightmares about something bad happening to him/her." A total score is calculated by summing all items together. The parent-report version also includes General Anxiety Disorder, Panic, Separation Anxiety, Social Phobia, and School Phobia subscales. Only the Total Score will be used for the current study. For the current study, both mothers and fathers completed the survey.

Child Behavior Problems. The Child Behavior Checklist (CBCL; Achenback, 1999) is a parent-report measure of their child's internalizing and externalizing behaviors. Parents rate 63 behaviors on how often their child engaged in the behaviors in the last six months on a scale of 0 (*Not True [as far as you know]*) to 2 (*Very True or Often True*). The CBCL is comprised of eight syndrome scales and two higher order factors. The current study used the Internalizing factor and Externalizing factor scores. The Internalizing factor includes the Anxious/Depressed, Withdrawn/Depressed, and Somatic Complaints subscales. Example items include "Feels worthless or inferior" or "clings to adult or too dependent." The current study did not include Thought Problems subscale statements. The Externalizing factor includes the Rule-Breaking Behavior and Aggressive Behavior subscales. Example items include "temper tantrums or hot temper" or "vandalism." The Social Problems and Attention Problems subscales were not used in the current study. For the current study, both mothers and fathers completed the survey.

Parenting Behaviors Surveys

Parenting Warmth.

Child-Report of Parental Warmth. The Children's Report of Parental Behavior Inventory (CRPBI; Schaefer, 1965) measures parental behaviors on three subscales

Acceptance/Warmth, Psychological Control, and Firm Control. For the current study, only the Acceptance/Warmth subscale will be used. Children rate 30 statements about how similar the statement is to their parents' behaviors on a three-point Likert scale (0 = *Not Like*; 2 = *A Lot Like*). Example statements include "My mother/father is a person who makes me feel better after talking over my worries with her/him" and "My mother/father is a person who makes me feel like the most important person in their life." The child completed the measure twice, once for each parent. Due to COVID-19 protocol changes, the CRPBI for the father was moved to a later part of the study. To keep the remote sessions short to prevent attention and behavior problems, some children did not complete the CRPBI for the father at T2 (n = 5).

Parents also completed the CRPBI and rated their own parenting behaviors on a three-point Likert scale (0 = *Not Like*; 2 = *A Lot Like*) based on how true the statement was. Example statements include "I smile at my child often" and "I give my child a lot of care and attention." Items are summed together to receive an Acceptance and Warmth score. For the current study, both mothers and fathers completed the survey.

Analytical Plan

Missing Data and Attrition

For the current study, 117 families completed T1. Fifteen families were lost to follow-up, thus a total of 102 families (87.2%) completed T2. However, due to scheduling conflicts, three families only had one parent participate in the T2 visit. Therefore, the T2 sample contains 102 children, 101 mothers, and 100 fathers. Further, 22 T2 visits were completed over Zoom due to the COVID-19 pandemic and physiological data was not collected. Due to equipment malfunction, the child refusing to wear the sensors, or difficulties interpreting the data, some participants do not have physiological data. Additionally, as some participants completed their T2 visits over Zoom, collecting physiological data was not possible. For survey data, a multiple

imputation method was used when scales were missing less than 20% scale items (Enders, 2010). Table 1 (T1) and Table 2 (T2) provide the sample size for each study variable.

Descriptive and Covariate Analyses

All study variables were tested for required statistical assumptions (e.g., normality tests, linearity, & homoscedasticity). For each study variable, the means and standard deviations were calculated and were recorded by measure reporter (e.g., mother, father, or child). See Table 1 (T1) and Table 2 (T2) for descriptive statistics. Within each time point, paired samples *t*-test was performed for each measure with multiple reporters to determine if they are statistically different from one another. If there was no difference between reporters, a composite score for each variable was created and used in later analyses. If there was a significant difference between the variables, Pearson's correlation was used to determine which reporter will be used in the final model. Additionally, Pearson's correlations examined the strength and direction of the associations between study variables. Paired samples *t*-tests will examine if differences occur within each reporter between the two time points.

Covariate analyses determined the potential variables that will be controlled for in the final model. Study site, child age and gender, child puberty, household income, and parent education were tested for all study variables at each time point. For parenting measures at each time point, parent ethnicity and if the reporter is the biological parent was tested as covariates. Child ethnicity was tested as a covariate for physiological measures (i.e., RSA baselines) and child behavioral outcomes (e.g., internalizing and externalizing behaviors). Whether the family completed a remote visit (COVID-19) was tested as a covariate at T2. The length between family visits was tested as a covariate.

Latent Profile Analysis

Latent profile analysis (LPA) is a mixture modeling analysis that is used to discover unobserved groups within a set of continuous observed or indicator variables (Ferguson et al., 2020; Sinha et al., 2021). Each profile is distinct from one another while indicators within the profile are homogenous (Sinha et al., 2021). After achieving model fit, profile membership is determined by examining the probability that participants belong to that profile. These probabilities are then used to assign participants to a profile (Sinha et al., 2021). Bauer (2022) describes the four steps in latent profile analysis: 1) model specification, 2) profile enumeration, 3) substantive interpretation of the target models, and 4) examine distal outcomes of most likely latent class membership. In Step 1, indicator variables are selected based on prior research and theory. As discussed in the introduction, the current study used ASD symptom severity (ADOS-II) and IQ (DAS-II) as indicator variables. Scores from subscales for both the ADOS-II (Social Affect and Restricted and Repetitive Behavior) and the DAS-II (GCA) were used.

For Step 2, I used M*plus* (Muthen & Muthen, 1998-2017) to create the latent profile models. In this process, a series of models were estimated and then compared based on relative model fit (Bauer, 2022). The baseline model has one profile ($k = 1$), with subsequent models adding one additional profile each time (Sinha et al., 2021). Relative model fit indices were used to compare models and determine the number of profiles that best fit the data (Bauer, 2022). When comparing models, Ram and Grimm (2009) noted there is not a set of standard rules to determine model fit. Instead, prior theory and research, as well as statistical fit indices, should be used to determine model fit. The authors proposed guidelines to follow when evaluating model fit. First, they note the model should make mathematical sense, thus researchers should examine the estimation output for errors, parameter outliers, and estimators that do not fit with prior theory. Second, Bayesian Information Criteria (BIC) and Akaike Information Criteria (AIC) are

used to compare model fit. Lower BIC and AIC values indicate better model fit. If the BIC and the AIC are higher than the baseline model ($k = 1$), then the model should not be considered. Third, models are evaluated based on the confidence that observations have been accurately classified. Entropy is used as a summary indicator of group membership for individuals. High entropy (> .80) suggest high confidence in group membership and groups are distinct from one another. Once model fit is determined and the final model is selected, several random starts should be used to demonstrate replication. Profiles should also be inspected to ensure they are not scaled groupings of a single indicator (Sinha et al., 2021).

For Step 3, the profiles were assessed and compared to ensure they are distinguishable from the others and are interpretable (Bauer, 2022). For this step, profile size was assessed to ensure they are substantive and can be used in outcome analyses. Bauer argues that while small profiles can be of interest and indicate individuals did not fit within another profile, too many may be indicative that too many profiles were created. Therefore, if this occurs, examining another model solution may provide insight into whether the smaller profiles are substantive or one larger profile being split (Bauer, 2022). I used profile plots and compare the means of each indicator variable to determine if the profiles are distinctive from one another. To compare means, I exported group membership into SPSS and performed ANOVAs with each indicator variable. If the models are substantive and distinct, I will then move to Step 4. For this step. I exported profile membership into SPSS. Using ANOVAs, I compared the profiles on the following measures at T1: baseline RSA, child depression (CDI), child anxiety (SCARED), and child internalizing and externalizing behaviors (CBCL – internalizing and externalizing subscales).

Moderation

To examine how profile membership influences the association between parenting behaviors and child outcomes, I used moderated regression analysis. Moderation examines the effect of X on outcome Y that is influenced by variable W, which can be used to account for heterogeneity in the model (Hair et al., 2021; Hayes, 2017). I used SPSS and the PROCESS macro (Hayes, 2017) to run the moderated regression. I ran the model two ways, both of which will be moderated by the profiles created in Goal 1. First, I examined a Parenting Effects model which examined the effects of T1 parenting behaviors (warmth/acceptance) on T2 child outcomes (baseline RSA, child depression, child anxiety, and child internalizing and externalizing behaviors). Due to sample size limitations, each child outcome was run in separate models. Second, I examined a Child Effects model which will examine the effects of T1 child outcomes on T2 parenting behaviors. For both the Parenting Effects and Child Effects model, the data for mothers and fathers will be run separately. For each model, I centered each predictor variable prior to entering the variable into the PROCESS macro. I also used the multicategorical function in PROCESS to create dummy variables for the profiles. If the interactions are significant, I ran simple slope tests and use the re-centering strategy to examine differences at ±1 standard deviation (Aiken et al., 1991) as follow up analyses. Post hoc power analyses using G*Power (Faul et al., 2009) were conducted to determine the achieved power of the moderated regression.

Results

Descriptive Analyses

The means and standard deviations for RSA, child emotion and behavior difficulties, and parenting behavior variables are recorded by reporter in Table 1 (T1) and Table 2 (T2). All study variables were assessed for normality to determine if they met the assumptions for the planned

statistical analyses. Skewness, kurtosis, and Shapiro-Wilks test of normality for T1 are reported in Table 3 and for T2 reported in Table 4. The following variables had a non-significant Shapiro-Wilks test of normality result ($p \geq .054$): ADOS social affect, T1 and T2 RSA, and father-report T2 SCARED (child anxiety). All other variables had a significant Shapiro-Wilks result ($p \leq .05$), indicating non-normality. Further, some variables had a moderate positive and negative skew and were moderately leptokurtic. Additionally, most variables had at least one outlier. However, due to the clinical nature of the population, the choice was made to keep outliers in the data set as this would allow for better insight into the child and parenting outcomes. In examining ANOVA assumptions and the robustness of the *F*-test, Blanca and colleagues (2017) found that ANOVA results are valid with non-normal data when the homogeneity of variance assumption is met. Therefore, the decision was made not to transform the data in the current study. For all *t*-tests and ANOVAs, equivalent non-parametric tests (e.g., Kruskal-Wallis test and related-samples Wilcoxon signed ranked test) were also performed. Unless otherwise stated later in the results, all ANOVA and *t*-test results followed the same pattern of significance as their non-parametric counterparts. Therefore, only the results of the ANOVA and *t*-test results will be reported. There were three instances where the results of the non-parametric test did not match. In those instances, the non-parametric results were reported along with the ANOVA and *t*-test results.

A paired samples *t*-test indicated there was no significant difference between T1 RSA ($M = 66.66$, $SD = 1.37$) and T2 RSA ($M = 6.52$, $SD = 1.35$), $t(65) = 1.23$), $p = .224$, $d = 0.11$. Paired sample *t*-tests examined the difference between T1 and T2 for each variable for child- (Table 5), mother- (Table 6), and father-report (Table 7). For child-report child anxiety symptoms, T1 ($M = 22.39$, $SD = 12.61$) was significantly higher than T2 ($M = 19.65$, SD = 13.69), $p = .001$. For child-report depressive symptoms, there was no difference between time points, $p = .489$. There

Table 3

Time 1 Skewness, kurtosis, and Shapiro-Wilks Test of Normality

	Child-report (*n* = 117)			Mother-report (*n* = 117)			Father-report (*n* = 117)		
	Skewness	Kurtosis	Shapiro-Wilks	Skewness	Kurtosis	Shapiro-Wilks	Skewness	Kurtosis	Shapiro-Wilks
Latent Profile Indicators									
IQ	0.13	-0.72	.015						
Social Affect	0.30	0.08	.067						
RRB	0.46	-0.54	≤ .001						
Child Physiological Measurement									
Baseline RSA	0.11	-0.75	.322						
Child Emotion & Behavior Difficulties									
Depressive Symptoms	1.13	1.35	≤ .001	0.85	0.57	≤ .001	0.76	0.79	.001
Anxiety Symptoms	0.65	0.23	.005	0.88	0.36	≤ .001	0.80	0.87	.001
Internalizing Behaviors				1.05	0.75	.003	0.86	0.57	.020
Externalizing Behaviors				0.56	-0.29	≤ .001	0.62	0.47	≤ .001
Parenting Behaviors									
Parent-report Parental Warmth				-0.84	-0.30	≤ .001	-0.35	-0.70	≤ .001
Child-report Maternal Warmth	-1.19	0.98	≤ .001						
Child-report Paternal Warmth	-0.73	-0.31	≤ .001						

Note: Variables are arranged by outcome and reporter. Shaded boxes indicate no data was collected for that reporter. RSA = Respiratory Sinus Arrythmia.

Table 4

Time 2 Skewness, kurtosis, and Shapiro-Wilks Test of Normality

	Child-report (*n* = 102)			Mother-report (*n* = 101)			Father-report (*n* = 100)		
	Skewness	Kurtosis	Shapiro-Wilks	Skewness	Kurtosis	Shapiro-Wilks	Skewness	Kurtosis	Shapiro-Wilks
Child Physiological Measurement									
Baseline RSA	-0.17	-0.10	.531						
Child Emotion & Behavior Difficulties									
Depressive Symptoms	1.09	1.74	≤ .001	0.54	-0.13	.046	0.54	0.31	.054
Anxiety Symptoms	1.15	1.37	≤ .001	1.28	2.23	≤ .001	1.40	4.48	≤ .001
Internalizing Behaviors				1.13	0.89	≤ .001	0.96	1.05	≤ .001
Externalizing Behaviors				1.06	0.70	≤ .001	0.79	0.05	≤ .001
Parenting Behaviors									
Parent-report Parental Warmth				-1.04	0.35	≤ .001	-0.55	-0.26	.001
Child-report Maternal Warmth	-1.66	2.87	≤ .001						
Child-report Paternal Warmth	-0.56	-0.36	≤ .001						

Note: Variables are arranged by outcome and reporter. Shaded boxes indicate no data was collected for that reporter. RSA = Respiratory Sinus Arrythmia

was no difference between time points in maternal warmth or paternal warmth, *p*s ≥ .373.

For mother-report child anxiety symptoms, T1 (*M* = 19.59, *SD* = 13.26) was significantly higher than T2 (*M* = 17.67, *SD* = 12.61), *p* = .005. For mother-report externalizing behaviors, T1 (*M* = 11.97, *SD* = 9.13) was significantly higher than T2 (*M* = 10.19, *SD* = 7.85), *p* ≤ .001. For mother-report child depressive symptoms and internalizing behaviors, there was no difference between time points, ≥ .471. For mother-report maternal warmth, mothers rated themselves as having higher maternal warmth at T2 (*M* = 27.36, *SD* = 2.68) compared to T1 (*M* = 25.97, *SD* = 3.84), *p* = .002. However, a related samples Wilcoxon signed rank test indicated no significant difference in mother-reported maternal warmth between time points, *W* = 1005.50, *z* = .679 , *p* = .679.

For father-report child internalizing behaviors, results of the paired sample *t*-test indicated no significant difference between time points, *p* = .066. However, results of the related-samples Wilcoxon Signed Rank Tested indicated significantly higher father-report child internalizing behaviors at T1 (*M* = 14.18, *SD* = 7.50) compared to T2 (*M* = 13.16, *SD* = 7.77), *W* = 2767.5, *z* = 2.675, *p* = .007. For father-report child anxiety symptoms, fathers rated their child as having more anxiety at T1 (*M* = 16.93, *SD* = 9.78) compared to T2 (*M* = 14.88, *SD* = 10.46), *p* = .001. For father-report child externalizing behaviors, fathers rated their child as having more externalizing behaviors at T1 (*M* = 12.50, *SD* = 8.70) compared to T2 (*M* = 10.52, *SD* = 7.92), *p* ≤ .001. There was no difference in time points for father-report child depressive symptoms, *p* = .256

Reporter Comparisons

To determine if there were differences between child- and parent-report child depressive symptoms, child anxiety symptoms, and parental warmth, a series of paired sample *t*-tests were performed. For each reporter, scores for child depressive symptoms were converted to z-scores

Table 5

Comparison of Child-report Study Variables Across Time

	N	Time 1 (*SD*)	Time 2 (*SD*)	df	*t*	*p*	*d*
Child Depressive Symptoms	101	10.74 (7.46)	10.33 (7.42)	100	0.70	.489	0.06
Child Anxiety Symptoms	**100**	**22.39 (12.61)**	**19.65 (13.69)**	**99**	**3.30**	**.001**	**0.21**
Maternal Warmth	98	25.99 (3.85)	25.94 (4.34)	97	0.13	.897	0.01
Paternal Warmth	96	23.59 (5.26)	23.26 (5.50)	95	0.89	.373	0.06

Note: Variables with significant difference between timepoints are in bold.

Table 6

Comparison of Mother-report Study Variables Across Time

	N	Time 1 (*SD*)	Time 2 (*SD*)	df	*t*	*p*	*d*
Child Depressive Symptoms	100	16.08 (7.15)	16.14 (7.07)	99	0.12	.901	0.01
Child Anxiety Symptoms	**100**	**19.59 (13.26)**	**17.67 (12.61)**	**100**	**2.86**	**.005**	**0.15**
Child Internalizing Behaviors	99	14.65 (9.23)	14.27 (8.72)	98	0.72	.471	0.04
Child Externalizing Behaviors	**99**	**11.97 (9.13)**	**10.19 (7.85)**	**98**	**3.72**	**≤ .001**	**0.21**
Maternal Warmth	**98**	**25.97 (3.84)**	**27.36 (2.68)**	**97**	**3.20**	**.002[a]**	**0.42**

Note: Variables with significant difference between timepoints are in bold.

[a]A related samples Wilcoxon signed rank test indicated no significant difference in mother-reported maternal warmth between time points, W = 1005.50 , z = .679 , p = .679.

Table 7

Comparison of Father-report Study Variables Across Time

	N	Time 1 (*SD*)	Time 2 (*SD*)	df	*t*	*p*	*d*
Child Depressive Symptoms	99	15.85 (5.80)	15.26 (6.58)	98	1.14	.256	0.10
Child Anxiety Symptoms	**99**	**16.93 (9.78)**	**14.88 (10.46)**	**98**	**3.47**	**.001**	**0.20**
Child Internalizing Behaviors	**99**	**14.18 (7.50)**	**13.16 (7.77)**	**98**	**1.86**	**.066[a]**	**0.13**
Child Externalizing Behaviors	**99**	**12.50 (8.70)**	**10.52 (7.92)**	**98**	**3.97**	**≤ .001**	**0.24**
Paternal Warmth	99	25.52 (3.26)	25.25 (3.48)	98	0.64	.526	0.06

Note: Variables with significant difference between timepoints are in bold.

[a]A related-samples Wilcoxon signed rank test indicated a significant difference in father-reported CBCL internalizing between time points, W = 2767.5, z = 2.675, p = .007.

in order to compare child- and parent-reports. Paired sample *t*-test inferential statistics for child- and mother-report surveys are reported in Table 8. Results indicated there was no significant difference between child- and mother-report depressive symptoms z-scores at both T1 and T2, *p*s ≥ .883. The was no significant difference in anxiety symptoms at either time point, *p*s ≥ . 121. Therefore, for each time point, a mother-child child depressive symptoms composite z-score and a mother-child child anxiety symptoms composite score were created to use in later analyses. At both T1 (mothers: *M* = 27.36, *SD* =2.53; child: *M* = 26.05, *SD* = 2.53) and T2 (mothers: *M* = 27.34, *SD* = 2.69; child: *M* = 25.71, *SD* = 4.59), mothers reported significantly higher maternal warmth than their child, *p*s ≤ .002. Thus, the child-report parenting warmth and mother-report parenting warmth were used separately in later analyses.

Paired sample *t*-test inferential statistics for child- and father-report surveys are reported in Table 9. Results indicated there was no significant difference between child- and father-report depressive symptoms z-scores at both T1 and T2, *p*s ≥ .969. Therefore, a child-father composite depressive symptoms z-score was created to use in later analyses. At T1 (fathers: *M* = 16.97, *SD* = 10.12; child: *M* = 22.68, *SD* = 12.81) and T2 (fathers: *M* = 14.88, *SD* = 10.46; child: *M* = 20.46, *SD* = 14.40), fathers rated their child having lower anxiety symptoms than their child rating their own anxiety symptoms, *p*s ≤ .001. At T1 (fathers: *M* = 25.46, *SD* = 3.28; child: *M* = 24.04, *SD* = 5.31) and T2 (fathers: *M* = 25.16, *SD* = 3.51; child: *M* = 22.99, *SD* = 5.63), fathers rated paternal warmth higher than their child, *p*s ≤ .004.

Correlations

Within-timepoint correlations are reported in Table 10. Pearson's correlations were performed separately for Parent Effects Model (T1 parenting behaviors predicting T2 child behaviors). Results are reported in Table 11. T1 child-report maternal warmth was negatively correlated with T2 mother-child child depressive symptoms composite z-score, *r* = -.34, *p* = .001.

Table 8

Comparison of Child- and Mother-report Study Variables

		Child-Report		Mother-Report		Paired-Sample *t*-Test				Composite Score	
	N	Mean	*SD*	Mean	*SD*	df	*t*	*p*	*d*	Mean	*SD*
Time 1											
Child Depressive Symptoms[a]	**115**	**-0.01**	**1.00**	**0.01**	**1.00**	**114**	**0.15**	**.883**	**0.01**	**0.00**	**0.84**
Child Anxiety Symptoms	**115**	**22.67**	**12.81**	**20.63**	**13.47**	**114**	**1.36**	**.177**	**0.16**	**21.42**	**10.44**
Maternal Warmth	113	26.05	4.04	27.36	2.53	112	3.25	.002	0.39		
Time 2											
Child Depressive Symptoms[a]	**101**	**0.01**	**1.00**	**0.00**	**1.00**	**100**	**0.08**	**.940**	**0.01**	**0.00**	**0.83**
Child Anxiety Symptoms	**101**	**20.42**	**14.34**	**17.67**	**12.61**	**100**	**1.56**	**.121**	**0.20**	**18.94**	**10.21**
Maternal Warmth	99	25.71	4.59	27.34	2.69	98	3.66	≤ .001	0.43		

Note: Shaded box indicates no composite score was created for that measure. Variables with significant difference between reporters are in bold.

[a]Depressive Symptoms measure was converted to a z-score to compare mother- and child-report surveys.

T1 mother-report maternal warmth was also negatively correlated with T2 mother-child child depressive symptoms composite z-score, r = -.27, p = .006. The associations between T1 maternal warmth and T2 child RSA, T2 mother-child child anxiety symptoms composite, T2 child internalizing behaviors, and T2 child externalizing behaviors were non-significant, $p \geq$ .054. T1 child-report paternal warmth was positively correlated with T2 father-child child depressive symptoms composite z-score, r = .24, $p \leq .001$. T1 child-report paternal warmth was negatively correlated with T2 child externalizing behaviors, r = -.35, $p \leq .001$. T2 father-report child anxiety symptoms, child internalizing behaviors, and child RSA were non-significantly correlated with T1 child-report paternal warmth, ps $\geq .230$. T1 father-report paternal warmth was not significantly correlated with T2 father-child child depressive symptoms composite z-score, T2 child anxiety symptoms, T2 child internalizing behaviors, T2 child externalizing behaviors, and T2 RSA, $p \geq .052$. Overall, as maternal warmth increases, child depressive symptoms decrease. As paternal warmth increases, child depressive symptoms also increase. As child externalizing behavior increases, paternal warmth decreases.

Pearson's correlations were performed separately for Child Effects Model (T1 child RSA and child behaviors predicting T2 parenting behaviors). Results are reported in Table 12. T1 mother-child child depressive symptoms composite z-score was negatively correlated with T2 child-report maternal warmth, r = -.26, p = .010, and T2 mother-report maternal warmth, r = -.24, p = .014. T1 child-mother child anxiety symptoms composite, T2 child internalizing behaviors, T2 child externalizing behaviors, and child RSA were not significantly correlated with T2 child-report or T2 mother-report maternal warmth, $p \geq .079$. T1 father-child child depressive symptoms composite z-score was negatively correlated with T2 child-report paternal warmth, r = -.30, p = .002, and T2 father-report paternal warmth, r = -.21, p = .041. T1 father-report child internalizing behaviors was negatively correlated with T2 father-report paternal behaviors, r =

Table 9

Comparison of Child- and Father-report Study Variables

		Child-Report		Father-Report		Paired-Sample *t*-Test				Composite Score	
	N	Mean	*SD*	Mean	*SD*	df	*t*	*p*	*d*	Mean	*SD*
Time 1											
Child Depressive Symptoms[a]	**116**	**0.00**	**1.00**	**0.00**	**1.00**	**115**	**0.04**	**.969**	**.004**	**0.00**	**0.83**
Child Anxiety Symptoms	115	22.68	12.81	16.97	10.12	114	4.09	≤ .001	0.49		
Paternal Warmth	114	24.04	5.31	25.46	3.28	113	2.95	.004	0.32		
Time 2											
Child Depressive Symptoms[a]	**99**	**0.00**	**1.00**	**0.00**	**1.00**	**98**	**0.04**	**.971**	**.004**	**0.00**	**0.79**
Child Anxiety Symptoms	99	20.46	14.40	14.88	10.46	98	3.35	.001	0.44		
Paternal Warmth	95	22.99	5.63	25.16	3.51	94	3.67	≤ .001	0.46		

Note: Shaded box indicates no composite score was created for that measure. Variables with significant difference between reporters are in bold.

[a]Depressive Symptoms measure was converted to a z-score to compare father- and child-report surveys.

Table 10

Correlations Between Study Variables Within-Time

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1	1	.36**	.63**	.01	.08	-.42**	-.35**	-.17	-.13	.12	-.08	.12	-.19	-.08
2	.21*	1	.23*	.17	-.04	-.35**	-.30**	-.04	.01	.09	.04	.09	-.09	-.12
3	.54**	.06	1	.28**	.17	-.45**	-.40**	-.12	-.13	.02	-.18	-.04	-.32**	-.28**
4	.004	.12	.36**	1	.06	-.08	-.23*	.05	.13	-.05	.04	-.23*	-.04	-.31**
5	.07	-.06	.01	-.10	1	-.15	-.16	-.14	.00	.20	.14	.20	-.05	.04
6	-.48**	-.32**	-.36**	-.05	.03	1	.86**	.41**	.48**	.22*	.47**	.23*	.46**	.45**
7	-.45**	-.26**	-.36**	-.17	.06	.83**	1	.39**	.41**	.37**	.34**	.45**	.32**	.49**
8	-.06	.07	-.12	-.04	-.03	.34**	.36**	1	.79**	.14	.17	.08	.00	-.04
9	-.05	.18	-.08	.08	.02	.46**	.38**	.78**	1	.47**	.63**	.30**	.22*	.10
10	.11	.10	.02	-.05	-.01	.17	.31**	.16	.44**	1	.50**	.70**	.17	.30**
11	-.08	-.02	-.19*	.00	.00	.58**	.40**	.22*	.62**	.39**	1	.42**	.49**	.28**
12	-.05	-.03	-.11	-.18*	.00	.41**	.57**	.29**	.41**	.63**	.48**	1	.23*	.53**
13	-.24*	-.26**	-.30**	-.05	-.11	.55**	.37**	-.06	.11	.08	.54**	.27**	1	.71**
14	-.11	-.18	-.24**	-.21*	-.10	.37**	.42**	-.01	.04	.25**	.31**	.54**	.70**	1

Note: Time 1 correlations are reported below the divide and Time 2 correlations are reported above the divide. * $p \leq .05$, ** $p \leq .01$.

1. Child-Report Maternal Warmth, 2. Mother-Report Maternal Warmth, 3. Child-Report Paternal Warmth, 4. Father-Report Paternal Warmth, 5. Baseline RSA, 6. Child Depressive Symptoms Mother-Child Composite, 7. Child Depressive Symptoms Father-Child Composite, 8. Child-Report Anxiety Symptoms, 9. Child Anxiety Symptoms Mother-Child Composite, 10. Father-report Anxiety Symptoms, 11. Mother-report Internalizing Behaviors, 12. Father-report Internalizing Behaviors, 13. Mother-report Externalizing Behaviors, 14. Father-report Externalizing Behaviors

Table 11

Correlation Table for Parent Effects Model: Time 1 Parenting Behaviors with Time 2 Child Outcomes

	T1 Child-report Maternal Warmth	T1 Mother-report Maternal Warmth	T1 Child-report Paternal Warmth	T1 Father-report Paternal Warmth
T2 Child Depressive Symptoms Composite	-.34**	-.27**	.24*	-.05
T2 Child Anxiety Symptoms Mother-Child Composite	-.002	.08		
T2 Child-report Child Anxiety Symptoms			-.013	-.02
T2 Father-report Child Anxiety Symptoms			-.08	-.05
T2 Child Internalizing Behaviors	-.06	.08	-.14	-.19
T2 Child Externalizing Behaviors	-.20	-.18	-.35**	-.20
T2 Baseline RSA	.13	-.05	.15	-.21

Note: * $p \leq .05$, ** $p \leq .01$; Shaded boxes indicate there is no data for that measure and reporter. T1 = Time 1, T2 = Time 2, RSA = Respiratory Sinus Arrythmi

-.21, p = .042. T1 father-report child externalizing behaviors was negatively correlated with T2 child-report paternal warmth, r = -.22, p = .032, and T2 father-report paternal warmth, r = -.25, p = .015. T2 child-report and father-report paternal warmth was not significantly correlated with T1 child anxiety symptoms, T1 child internalizing behaviors, & child RSA, p ≥ .236. Overall, as child depressive symptoms increase, maternal and paternal warmth decreases. As child internalizing and externalizing behaviors increase, paternal warmth decreases.

Latent Profile Analysis

A latent profile analysis was performed using ASD symptom severity (SA & RRB) and IQ as indicator variables. Fit indices for the latent profile analysis are reported in Table 13. Model fit statistics were examined for two to four profiles. While the two-profile solution had a lower AIC, BIC, and SSABIC compared to the base model, entropy was .72 which is lower than the recommended .80. According to Ram and Grimm (2009), models with high entropy are preferred as the low entropy would be indicative of lower confidence in group membership and less distinction between groups. This suggests the two-profile solution is not a good fit and a model with more profiles should be examined.

When examining the three-profile solution, the model had a lower AIC and SSABIC compared to the base model and entropy was .81. Again, this suggests that the model is a good fit and has high confidence in group membership and distinction between groups. However, the BIC was slightly above the BIC of the base model. Ram and Grimm (2009) recommend the BIC in profile solutions with more than 1 profile should be higher than the base model. However, Chen and colleagues (2017) examined the efficacy of fit indices and found that SSABIC was more effective with a smaller sample size. While the three-model solution was potentially the best fitting model, the four profile-solution was examined to compare the two solutions to determine which best fit the data.

Similar to the three-profile solution, the four-profile solution also had a lower AIC and SSABIC compared to the base model and entropy was .81. Based on recommendations from Ram and Grimm (2009) and Chen and colleagues (2017), these results suggest the four-profile is a well-fitting model. Again, similar to the three-model solution, the BIC was higher than the base model although to a larger degree. The three-profile solution was 2.78 points higher than the base model, while the four-profile solution was 13.56. This suggests that the four-model solution would not be as good of a fit for the data as the three-model solution. Another method to determine which model has the best fit is to examine the number of individuals in each profile (Bauer, 2022). To determine which model to retain, the size of the profiles within the 3 and 4 profile model were examined. The three-profile solution had group membership sample sizes of 62, 43, and 12, while the four-profile solution had group membership sample size of 67, 26, 4, and 20. While profile solutions with small sample size profiles can be legitimate solutions, another model solution with more substantive or larger profiles may be the better solution (Bauer, 2022). As the four-profile solution had the highest BIC greater than the base model and a profile with a small sample size, it was not selected as the final solution. Therefore, the three-profile solution was selected as it had good model fit with the SSABIC, AIC, and entropy.

Table 14 includes the descriptive statistics for IQ and autism symptom severity for each of the three profiles. In the three-profile solution, Profile 1 (n = 62; Male: 79%) was characterized by average IQ, moderate SA severity, and low RRB severity. Profile 2 (n = 43; Male: 81.4%) was characterized by average IQ, moderate SA severity, and moderate RRB severity. Profile 3 (n = 12; Male: 83.3%) was characterized by borderline IQ, high SA severity, and high RRB severity. One-way ANOVAs were performed to examine the differences between profiles for each of the profile indicators (See Table 14 for inferential statistics and Tukey's HSD post-hoc tests). Results indicated that compared to Profile 1 (Average IQ-Moderate SA-Low

Table 12

Correlation Table for Child Effects Model: Time 1 Child Outcomes with Time 2 Parenting Behaviors

	T2 Child-report Maternal Warmth	T2 Mother-report Maternal Warmth	T2 Child-report Paternal Warmth	T2 Father-report Paternal Warmth
T1 Child Depressive Symptoms Composite	-.26**	-.24*	-.30**	-.21*
T1 Child Anxiety Symptoms Mother-Child Composite	.004	.08		
T1 Child-report Child Anxiety Symptoms			-.10	-.04
T1 Father-report Child Anxiety Symptoms			.10	-.08
T1 Child Internalizing Behaviors	-.05	-.01	.004	-.21*
T1 Child Externalizing Behaviors	-.18	-.10	-.22*	-.25*
T1 Baseline RSA	.05	-.04	.12	-.05

Note: * $p \leq .05$, ** $p \leq .01$; Shaded boxes indicate there is no data for that measure and reporter. T1 = Time 1, T2 = Time 2, RSA = Respiratory Sinus Arrythmia.

Table 13

Latent Profile Analysis Fit Indices

Number of Profiles	*N*	Fit Statistics			
		AIC	BIC	SSABIC	Entropy
1	1, *n* = 117	2113.98	2130.56	2111.59	-
2	1, *n* = 69 2, *n* = 48	2098.61	2126.23	2094.62	.72
3	1, *n* = 62 2, *n* = 43 3, *n* = 12	2094.68	2133.34	2089.09	.81
4	1, *n* = 67 2, *n* = 26 3, *n* = 4 4, *n* = 20	2094.40	2144.12	2087.22	.81

RRB) and Profile 2 (Average IQ-Moderate SA-Moderate RRB), Profile 3 (Borderline IQ-High SA-High RRB) had the lowest average IQ, *p*s ≤ .030, and the highest ASD symptom severity, *p*s ≤ .001. There was no difference in IQ between the Average IQ-Moderate SA-Low RRB Profile and Profile 2, $p = .994$. The Average IQ-Moderate SA-Low RRB Profile had the lowest symptom severity compared to the Average IQ-Moderate SA-Moderate RRB Profile and the Borderline IQ-High SA-High RRB Profile, *p*s ≤ .001. To further describe the characteristics of the profiles, I examined the differences between profiles in age, and verbal ability. On average, the participants in the Average IQ-Moderate SA-Low RRB Profile were older than those in Profiles 2 and 3, *p*s ≤ .030, with no difference in age between Profiles 2 and 3, $p = 1.00$. There was no difference in verbal ability between profiles, $p = .477$. See Table 15 for mean and inferential statistics.

Child Emotion Regulation Outcome Profile Comparisons

Profiles were compared using a series of one-way ANOVAs and examined profile differences in child RSA, child-, mother- and father-report child depressive symptoms, anxiety symptoms, internalizing behaviors, and externalizing behaviors and parental warmth at each time point. There was no significant difference in RSA between profiles at either time point, *p*s ≥ .219 (see Table 16 for inferential statistics).

For child-report internalizing and externalizing behaviors (see Table 17), there was no significant difference between profiles for depressive symptoms and anxiety symptoms at either time point. For child-report parental warmth, there was no significant difference between profiles for maternal or paternal warmth at either time point, *p*s ≥ .526. For mother-report (see Table 18), there was a significant difference in T1 anxiety symptoms between profiles, $p = 0.13$. Follow-up Tukey HSD tests indicated the Borderline IQ-High SA-High RRB Profile had significantly lower T1 anxiety symptoms compared to the Average IQ- Moderate SA-Low RRB Profile and the

Table 14

Profile Indicator Variables Descriptive Statistics and Comparisons Between Profiles

	Average IQ-Moderate SA-Low RRB (n = 62)		Average IQ-Moderate SA-Moderate RRB (n = 43)		Borderline IQ-High SA-High RRB (n = 12)		ANOVA			
	Mean	*SD*	Mean	*SD*	Mean	*SD*	df	*f*	*p*	η^2
General Conceptual Ability	104.10[a]	18.47	104.47[b]	17.93	89.33[ab]	15.43	2	3.70	.028	.06
Social Affect	8.37[cd]	3.17	10.07[ce]	2.95	12.17[de]	3.86	2	8.82	≤ .001	.13
Restricted and Repetitive Behavior	1.08[fg]	0.75	3.74[fh]	0.69	5.92[gh]	0.79	2	305.96	≤ .001	.84

Note: IQ = intelligence quotient, SA = social affect, RRB = Restricted and Repetitive Behavior. Post-hoc Tukey's HSD results indicated significant differences between profiles, [a] p = 0.28; [b] p = .030; [c] p = .021; [d] p = .001; [e] p = .001; [f] p = ≤ .001; [g] p = ≤ .001; [h] p = ≤ .001.

Average IQ-Moderate SA-Moderate RRB Profile, *p*s ≤ .036. T2 anxiety symptoms had no significant difference between profiles, *p* = .127. For both time points, the differences between profiles for child depressive symptoms, internalizing behaviors, and externalizing behaviors were not significant, *p*s ≥ .152. For mother-report maternal warmth, the differences between profiles at both time points were non-significant, *p*s ≥ .501.

For father-report (see Table 19 for inferential statistics), there was a significant difference between profiles in anxiety symptoms at both time points, *p*s ≤ .006. Follow-up Tukey's HSD tests indicated T1 and T2 anxiety symptoms were significantly lower for the Borderline IQ-High SA-High RRB Profile compared to the Average IQ-Moderate SA-Low RRB Profile and the Average IQ-Moderate SA-Moderate RRB Profile, ps ≤ .034. There was no significant difference between profiles in child depressive symptoms or externalizing behaviors at either time point, *p*s ≥ .220. Differences in T1 child internalizing behaviors were not significant between profiles, p = .070. The results of the one-way ANOVA for T2 child externalizing behaviors indicated no significant difference between profiles, p = .076. However, the Levene Test for T2 child externalizing behaviors was significant, indicating a violation of the homogeneity of variance. A Kruskal-Wallis test was performed and found a significant difference between profiles, $\chi^2(2) = 6.54$, p = .038. Post-hoc Dunn test pairwise comparisons indicated higher internalizing behaviors in the Average IQ-Moderate SA-Moderate RRB Profile compared to the Borderline IQ-High SA-High RRB Profile, *p* = .033. There was no significant difference in paternal warmth between profiles at either time point, *p*s ≥ .351Overall, RSA was not significantly different between profiles at either time point. For child-report child emotion and behavior difficulties, there was no significant difference in any of the measures at either time point. For both mother- and father-report, autistic children in the Borderline IQ-High SA-High RRB Profile had the lowest anxiety symptoms compared to the other profiles. For father-report child internalizing behaviors, the

Table 15

Profile Characteristics Descriptive Statistics and Comparisons Between Profiles

	Average IQ-Moderate SA-Low RRB (*n* = 62)		Average IQ-Moderate SA-Moderate RRB (*n* = 43)		Borderline IQ-High SA-High RRB (*n* = 12)		ANOVA			
	Mean	*SD*	Mean	*SD*	Mean	*SD*	df	*f*	*p*	η^2
Child Age	**13.87[ab]**	**2.19**	**12.23[a]**	**1.92**	**12.25[b]**	**1.68**	**2**	**9.26**	**≤ .001**	**.14**
Verbal Ability	102.77	14.04	105.30	18.52	108.92	20.29	2	0.81	.447	.01

Note: IQ = intelligence quotient, SA = social affect, RRB = Restricted and Repetitive Behavior. Post-hoc Tukey's HSD results indicated significant differences between profiles, [a] $p = \leq .001$; [b] $p = .036$. Variables with significant difference between profiles are in bold.

Table 16

Descriptive Statistics of Respiratory Sinus Arrythmia by Profile and Comparisons Between Profiles

	Average IQ-Moderate SA-Low RRB (*n* = 62)			Average IQ-Moderate SA-Moderate RRB (*n* = 43)			Borderline IQ-High SA-High RRB (*n* = 12)			ANOVA			
	N	Mean	*SD*	*N*	Mean	*SD*	*N*	Mean	*SD*	df	*f*	*p*	η^2
Time 1 RSA	59	6.76	1.23	39	6.68	1.34	12	6.48	1.17	2	0.25	.780	.004
Time 2 RSA	35	6.62	1.43	35	6.76	1.37	7	5.76	0.54	2	1.56	.219	.04

Note: IQ = intelligence quotient, SA = social affect, RRB = Restricted and Repetitive Behavior, RSA = Respiratory Sinus Arrythmia.

Table 17

Descriptive Statistics of Child-report Child Emotion and Behavior Difficulties by Profile and Comparisons Between Profiles

	Average IQ-Moderate SA-Low RRB (n = 62)			Average IQ-Moderate SA-Moderate RRB (n = 43)			Borderline IQ-High SA-High RRB (n = 12)			ANOVA			
	N	Mean	*SD*	*N*	Mean	*SD*	*N*	Mean	*SD*	df	*f*	*p*	η^2
Time 1													
Child Depressive Symptoms	62	10.06	6.11	43	12.02	8.09	11	11.09	11.00	2	0.89	.415	.02
Child Anxiety Symptoms	62	21.32	12.76	43	24.26	12.83	10	24.30	13.34	2	0.75	.475	.01
Maternal Warmth	62	26.39	3.99	43	25.53	4.17	9	25.78	3.96	2	0.58	.563	.01
Paternal Warmth	62	24.52	5.69	43	23.63	4.67	9	22.78	5.72	2	0.63	.535	.01
Time 2													
Child Depressive Symptoms	50	9.94	6.62	40	10.98	7.92	12	10.75	9.63	2	0.22	.802	.00
Child Anxiety Symptoms	50	18.00	13.38	40	22.23	13.24	12	23.50	20.41	2	1.31	.274	.03
Maternal Warmth	50	26.22	3.23	39	25.49	5.12	11	24.64	7.39	2	0.65	.526	.01
Paternal Warmth	49	23.08	5.59	39	23.49	5.31	10	21.80	7.13	2	0.36	.701	.01

Note: IQ = intelligence quotient, SA = social affect, RRB = Restricted and Repetitive Behavior.

Table 18

Descriptive Statistics of Mother-report Child Emotion and Behavior Difficulties by Profile and Comparisons Between Profiles

	Average IQ-Moderate SA-Low RRB (n = 62)			Average IQ-Moderate SA-Moderate RRB (n = 43)			Borderline IQ-High SA-High RRB (n = 12)			ANOVA			
	N	Mean	*SD*	*N*	Mean	*SD*	*N*	Mean	*SD*	df	*f*	*p*	η^2
Time 1													
Child Depressive Symptoms	62	16.31	6.96	42	17.33	8.09	12	14.25	4.00	2	0.89	.412	.02
Child Anxiety Symptoms	**62**	**22.29[a]**	**14.21**	**43**	**20.63[b]**	**12.77**	**12**	**9.92[ab]**	**5.73**	**2**	**4.51**	**.013**	**.07**
Child Internalizing Behaviors	62	16.02	9.20	42	15.57	9.32	12	11.67	6.95	2	1.17	.314	.02
Child Externalizing Behaviors	62	11.68	8.34	43	14.49	9.89	12	10.92	7.14	2	1.54	.220	.03
Maternal Warmth	62	27.23	2.77	42	27.48	2.32	12	27.33	2.35	2	0.12	.888	.002
Time 2													
Child Depressive Symptoms	49	15.96	6.98	40	16.55	7.82	12	14.83	4.82	2	0.28	.759	.01
Child Anxiety Symptoms	49	19.14	13.86	40	17.90	11.67	12	10.92	8.10	2	2.11	.127	.04
Child Internalizing Behaviors	48	15.13	9.60	40	14.58	8.57	12	10.83	4.45	2	1.17	.314	.02
Child Externalizing Behaviors	48	8.65	7.01	40	11.90	8.38	12	10.25	8.47	2	1.92	.152	.04
Maternal Warmth	49	27.04	3.04	40	27.68	2.32	12	27.67	2.23	2	0.70	.501	.01

Note: IQ = intelligence quotient, SA = social affect, RRB = Restricted and Repetitive Behavior. Post-hoc Tukey's HSD results indicated significant differences between profiles, [a] p = .009; [b] p = .036.

Average IQ-Moderate SA-Moderate RRB Profile had higher internalizing behaviors compared to the Borderline IQ-High SA-High RRB Profile. All other emotion difficulties and behavior measures were not significant at either time point.

Moderated Regression

Based on the Pearson's correlation results, only the correlation pairs (i.e., Parent Effects models with T1 parental warmth and T2 child outcome; Child Effects models with T1 child emotion & behavior difficulties and parental warmth) with significant results were run as moderated regression models. There are four Parent Effects moderated regression models: 1) T1 child-report mother parenting warmth to T2 child-mother depressive symptoms composite z-score, 2) T1 mother-report mother parenting warmth to T2 child-mother depressive symptoms composite z-score, 3) T1 child-report father parenting warmth to T2 child-father depressive symptoms composite z-score, and 4) T1 child-report father parenting warmth to T2 child externalizing behaviors. There are seven Child Effects moderated regression models: 1) T1 child-mother depressive symptoms composite z-score to T2 child-report mother parenting warmth, 2) T1 child-mother depressive symptoms composite z-score to T2 mother-report mother parenting warmth, 3) T1 child-father depressive symptoms composite z-score to T2 child-report father parenting warmth, 4) T1 child-father depressive symptoms composite z-score to T2 father-report mother parenting warmth, 5) T1 child internalizing behavior to T2 father-report father parenting warmth, 6) T1 child externalizing behavior to T2 child-report father parenting warmth, and 7) T1 child externalizing behavior to T2 father-report father parenting warmth.33

In each model, all T1 predictor variables were centered prior to being entered into PROCESS. As the ASD symptom profiles acted as the moderator, dummy variables were created using the multi-categorical function in PROCESS. In models where Profile 1 (Average IQ-Moderate SA-Low RRB) was the indicator variable, it was coded as 0 and Profiles 2 (Average

Table 19

Descriptive Statistics of Father-report Child Emotion and Behavior Difficulties by Profile and Comparisons Between Profiles

	Profile 1			Profile 2			Profile 3			ANOVA		
	N	Mean	*SD*	*N*	Mean	*SD*	*N*	Mean	*SD*	df	*f*	*p*	η^2
Time 1													
Child Depressive Symptoms	62	16.03	5.74	43	16.09	6.51	12	13.75	5.05	2	0.80	.450	.01
Child Anxiety Symptoms	**62**	**18.66[a]**	**10.82**	**43**	**16.63[b]**	**8.85**	**12**	**8.58[ab]**	**5.60**	**2**	**5.42**	**.006**	**.09**
Child Internalizing Behaviors	62	14.89	7.12	43	15.19	8.41	12	9.67	6.10	2	2.73	.070	.05
Child Externalizing Behaviors	62	11.68	8.34	43	14.49	9.89	12	10.92	7.14	2	1.54	.220	.03
Paternal Warmth	62	25.21	3.30	43	25.65	3.29	12	26.67	2.90	2	1.06	.351	.02
Time 2													
Child Depressive Symptoms	49	15.90	6.20	39	15.13	7.35	11	12.82	5.15	2	0.99	.374	.02
Child Anxiety Symptoms	**49**	**16.82[c]**	**11.58**	**39**	**15.05[d]**	**8.83**	**11**	**5.65[cd]**	**4.50**	**2**	**5.61**	**.005**	**.10**
Child Internalizing Behaviors	49	13.65	8.32	39	13.95[e]	7.60	11	8.18[e]	2.93	2	2.64	.076	.05
Child Externalizing Behaviors	49	9.88	7.12	39	11.95	9.27	11	8.36	5.45	2	1.21	.303	.02
Paternal Warmth	49	23.08	5.59	39	23.49	5.31	10	21.80	7.13	2	0.36	.701	.01

Note: IQ = intelligence quotient, SA = social affect, RRB = Restricted and Repetitive Behavior. Post-hoc Tukey's HSD results indicated significant differences between profiles, [a] $p = .004$; [b] $p = .034$; [c] $p = .003$; [d] $p = .019$; [e] The Levene Test for Time 2 CBCL Externalizing was significant, indicating a violation of the homogeneity of variance. A Kruskal-Wallis test was performed, $\chi^2(2) = 6.54$, $p = .038$ and found higher internalizing behaviors in Profile 2 compared to Profile 3, $p = .033$. Significant results are in bold.

IQ-Moderate SA-Moderate RRB) and Profile 3 (Borderline IQ-High SA-High RRB) were coded as 1 respectively. In models where Profile 3 was the indicator variable, it was coded as 0. and Profiles 2 and 3 were coded as 1 respectively. Simple slope tests and the re-centering strategy (Aiken et al., 1991) were used as follow-up tests when the interactions were significant. Post hoc power analyses using G*Power were conducted to determine achieved power.

Covariate Analyses

As detailed earlier, several covariates were examined to determine whether they would need to be controlled for in the moderated regression models. Pearson's correlation determined child age was negatively correlated with mother-report maternal warmth (T1: r = -.25, p = .007; T2, r = -.31, p = .003) and father-report paternal warmth (T1: r = -.26, p = .004; T2: r = -.291, p = .003). An independent t-test indicated children reported their mothers as having more T2 maternal warmth at Site 1 (M = 26.57, SD = 4.48) compared to Site 2 (M = 24.73, SD = 4.56), $t(98) = 2.03$, p = .045. A one-way ANOVA indicated a significant difference in father education for T2 father-reported paternal warmth, $f(5) = 3.84$, p = .003, as fathers who attended college (M = 24.00, SD = 3.87) rated themselves as having less T2 paternal warmth compared to fathers who had post-college professional degrees (M = 26.95, SD = 2.41), p = .011. A one-way ANOVA indicated a significant difference in child puberty for T2 father-reported externalizing behaviors, $F(3) = 220.93$, p = .013, with fathers rating their children who are in late puberty (M = 16.92, SD = 8.27) as having more T2 externalizing behaviors compared to fathers with children who are post-pubertal (M = 8.92, SD = 7.52), p = .006. All remaining potential covariates were non-significant.

Therefore, models with T2 child-report maternal warmth controlled for study site. Models with T2 father-report paternal warmth and T2 father-report child externalizing behaviors controlling

for father education. Models with T2 father-reported externalizing behaviors controlled for child puberty. Additionally, all models controlled for the T1 outcome.

Parenting Effects Models

Model 1: T1 Child-report Maternal Warmth → T2 Child Depressive Symptoms.

A 2-way moderated regression examined the interaction between T1 child-report maternal warmth (centered) and ASD profiles on T2 child depressive symptoms mother-child composite z-score while controlling for T1 child depressive symptoms mother-child composite z-score. See Table 20 for inferential statistics. When Profile 1 was coded as the reference group (dummy code = 0), the interactions with Profile 2, $p = .212$, and Profile 3, $p = .395$, were not significant. The main effect of T1 mother-report maternal warmth was also not significant, $p = .297$. When Profile 3 was coded as the reference group (dummy code = 0), the interaction with Profile 2 was not significant, $p = .115$. The main effect of T1 mother-report maternal warmth was non-significant, $p = .157$. A post-hoc power analysis using G*Power was performed to examine the achieved F-tests power. Given the effect size $F^2 = .016$, a sample size of 99, and 6 tested predictors, the power for the current model was .12.

Model 2: T1 Mother-report Maternal Warmth → T2 Child Depressive Symptoms.

A 2-way moderated regression examined the interaction between the T1 mother-report maternal warmth (centered) and ASD profiles on T2 child depressive symptoms mother-child composite z-score while controlling for T1 child depressive symptoms mother-child composite z-score and child age. See Table 21 for inferential statistics. When Profile 1 was coded as the reference group (dummy code = 0), there was a significant 2-way interaction between Profile 1 and Profile 2, $b = 0.13$, $p = .010$ (see Figure 4 for the interaction). The 2-way interaction between Profile 1 and Profile 3 was non-significant, $b = .03$, $p = .713$. When Profile 3 was coded as the reference group (dummy code =

0), the interaction with Profile 2 was not significant, p = .225, and no significant main effect, p = .792. Simple slope tests revealed that, for Profile 2, as maternal warmth increased, there was a marginal increase in child depressive symptoms, b = .08 (SE = .04), t = 1.95, p = .054, 95% CI [-.001, .162]. The simple slope for was not significant for Profile 1, p = .117, or Profile 3, p = .795. Examined differently, group differences were explored at 1 *SD* above and below the mean of T1 mother-report maternal warmth. At low levels of maternal warmth (-1 *SD*), Profile 1 (M = -0.27) had significantly lower depressive symptoms compared to Profile 2 (M = 0.15), b = .43 (SE = .18), t = 2.38, p = .019, 95% CI [0.07, 0.79]. At mean and high levels (+1 *SD*) of maternal warmth, the differences between Profile 1 and Profile 2 were non-significant, ps ≥ .235. The differences between Profile 1 and Profile 3 were non-significant at low, mean, and high levels of maternal warmth, ps ≥ .773. A post-hoc power analysis was performed to examine the achieved F-tests power. Given the effect size F^2 = .031, a sample size of 101, and 7 tested predictors, the power for the current model was .19. Overall, results indicate that at low levels of maternal warmth, those in Profile 1 (Average IQ-Moderate SA-Low RRB) have lower depressive symptoms compared to Profile 2 (Average IQ-Moderate SA-Moderate RRB).See Table 21 for inferential statistics. When Profile 1 was coded as the reference group (dummy code = 0), there was a significant 2-way interaction between Profile 1 and Profile 2, b = 0.13, p = .010 (see Figure 4 for the interaction). The 2-way interaction between Profile 1 and Profile 3 was non-significant, b = .03, p = .713. When Profile 3 was coded as the reference group (dummy code = 0), the interaction with Profile 2 was not significant, p = .225, and no significant main effect, p = .792. Simple slope tests revealed that, for Profile 2, as maternal warmth increased, there was a marginal increase in child depressive symptoms, b = .08 (SE = .04), t = 1.95, p = .054, 95% CI [-.001, .162]. The simple slope for was not significant for Profile 1, p = .117, or Profile 3, p = .795.

Table 20

Parent Effects Model 1: T1 Child-report Maternal Warmth → T2 Child Depressive Symptoms

	b	*SE*	*t*	*p*	LLCI	ULCI
Reference Group: Profile 1						
Constant	0.08	0.08	1.03	.304	-0.08	0.25
T1 Maternal Warmth	-0.03	0.02	-1.05	.297	-0.07	0.02
Profile 2	-0.13	0.12	-1.06	.291	-0.38	0.11
Profile 3	-0.16	0.21	-0.76	.447	-0.57	0.25
T1 Maternal Warmth X Dummy Code 1	0.04	0.03	1.26	.212	-0.02	0.10
T1 Maternal Warmth X Dummy Code 2	-0.05	0.06	-0.85	.395	-0.16	0.06
T1 Child Depressive Symptoms	**0.71**	**0.08**	**9.30**	**≤ .001**	**0.56**	**0.86**
Reference Group: Profile 3						
Constant	-0.07	0.19	-0.38	.703	-0.45	0.31
T1 Maternal Warmth	-0.07	0.05	-1.43	.157	-0.18	0.03
Profile 1	0.16	0.21	0.76	.447	-0.25	0.57
Profile 2	0.03	0.21	0.12	.902	-0.40	0.45
T1 Maternal Warmth X Dummy Code 3	0.05	0.06	0.85	.395	-0.06	0.16
T1 Maternal Warmth X Dummy Code 4	0.09	0.06	1.59	.115	-0.02	0.20
T1 Child Depressive Symptoms	**0.71**	**0.08**	**9.30**	**≤ .001**	**0.56**	**0.86**

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 = Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded.

had significantly lower depressive symptoms compared to Profile 2 (M = 0.15), b = .43 (SE = .18), t = 2.38, p = .019, 95% CI [0.07, 0.79]. At mean and high levels (+1 *SD*) of maternal warmth, the differences between Profile 1 and Profile 2 were non-significant, ps ≥ .235. The differences between Profile 1 and Profile 3 were non-significant at low, mean, and high levels of maternal warmth, ps ≥ .773. A post-hoc power analysis was performed to examine the achieved F-tests power. Given the effect size F^2 = .031, a sample size of 101, and 7 tested predictors, the power for the current model was .19. Overall, results indicate that at low levels of maternal warmth, those in Profile 1 (Average IQ-Moderate SA-Low RRB) have lower depressive symptoms compared to Profile 2 (Average IQ-Moderate SA-Moderate RRB).

Model 3: T1 Child-report Paternal Warmth → T2 Child Externalizing Behaviors.

A 2-way moderated regression examined the interaction between T1 child-report paternal warmth (centered) and ASD profiles on T2 father-report child externalizing behaviors while controlling for T1 father-report child externalizing behaviors and child puberty stage. See Table 22 for inferential statistics. When Profile 1 was coded as the reference group (dummy code = 0), the interactions with Profile 2, p = .460, and Profile 3, p = .300, were not significant. The main effect of T1 father-report paternal warmth was also not significant, p = .652. When Profile 3 was coded as the reference group (dummy code = 0), the interaction with Profile 2 was not significant, p = .581. The main effect of T1 mother-report maternal warmth was non-significant, p = .188. Given the effect size F^2 = .005, a sample size of 94, and 1 tested predictor, the power for the current model was .07.

Model 4: T1 Child-report Paternal Warmth → T2 Child Depressive Symptoms.

A 2-way moderated regression examined the interaction between T1 child-report paternal warmth (centered) and ASD profiles on T2 child depressive symptoms father-child composite z-score while controlling for T1 child depressive symptoms father-child composite z-score. See

Table 21

Parent Effects Model 2: T1 Mother-report Maternal Warmth → T2 Child Depressive Symptoms

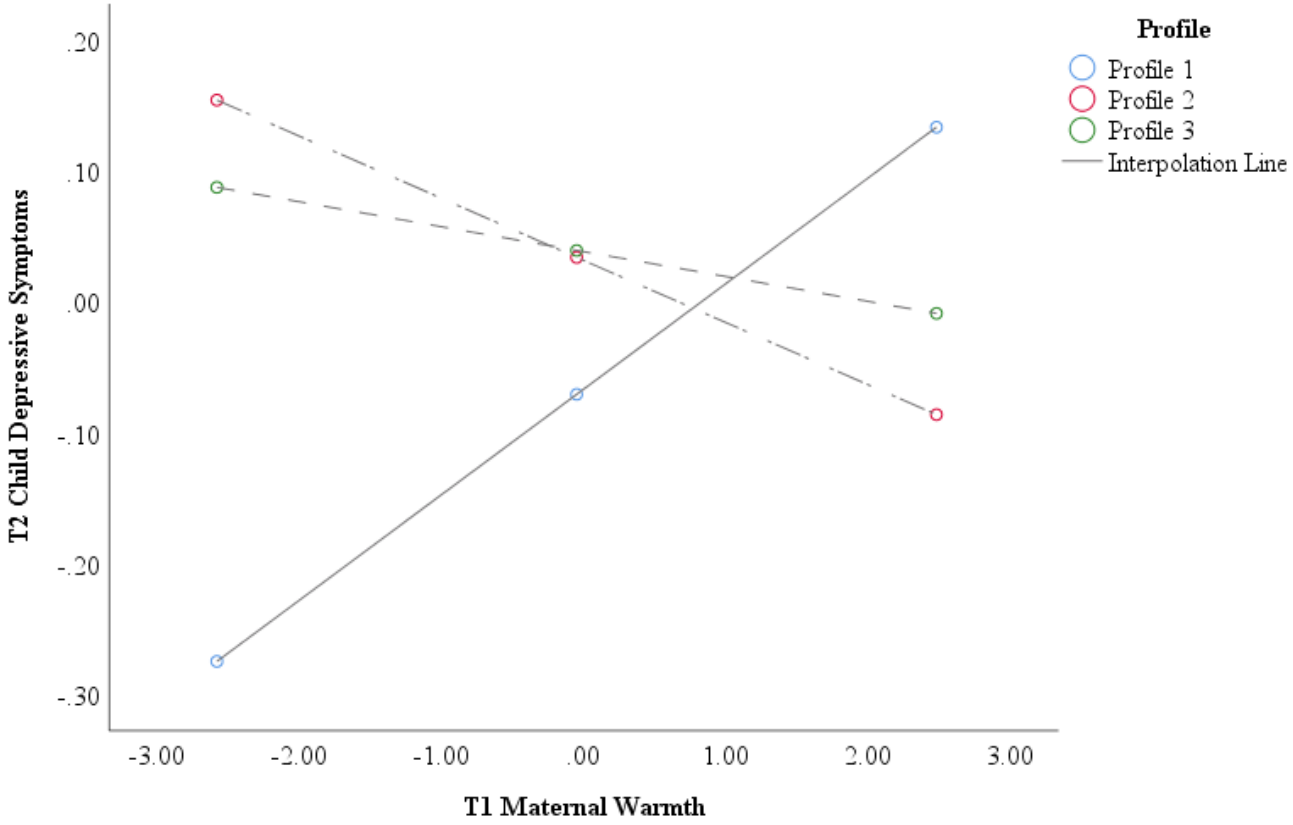
	b	*SE*	*t*	*p*	LLCI	ULCI
Reference Group: Profile 1						
Constant	-0.02	0.43	-0.05	.959	-0.87	0.82
T1 Maternal Warmth	-0.05	0.03	-1.58	.117	-0.11	0.01
Profile 2	-0.10	0.13	-0.74	.464	-0.36	0.17
Profile 3	0.01	0.19	0.04	.971	-0.36	0.38
T1 Maternal Warmth X Dummy Code 1	**0.13**	**0.05**	**2.62**	**.010**	**0.03**	**0.23**
T1 Maternal Warmth X Dummy Code 2	0.03	0.08	0.37	.713	-0.13	0.18
T1 Child Depressive Symptoms	**0.75**	**0.07**	**10.23**	**≤ .001**	**0.60**	**0.90**
Child Age	0.00	0.00	0.22	.829	≤ .001	0.01
Reference Group: Profile 3						
Constant	-0.02	0.40	-0.04	.970	-0.81	0.78
T1 Maternal Warmth	-0.02	0.07	-0.26	.792	-0.16	0.12
Profile 1	-0.01	0.19	-0.04	.971	-0.38	0.36
Profile 2	-0.11	0.19	-0.56	.574	-0.47	0.26
T1 Maternal Warmth X Dummy Code 3	-0.03	0.08	-0.37	.713	-0.18	0.13
T1 Maternal Warmth X Dummy Code 4	0.10	0.08	1.22	.225	-0.06	0.26
T1 Child Depressive Symptoms	**0.75**	**0.07**	**10.23**	**≤ .001**	**0.60**	**0.90**
Child Age	-0.02	0.40	-0.04	.970	≤ .001	0.01

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower

Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 = Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded.

Figure 4

Parent Effects Model 2: T1 Mother-report Maternal Warmth → T2 Child Depressive Symptoms



Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors

Table 22

Parent Effects Model 3: T1 Child-report Paternal Warmth → T2 Child Externalizing Behaviors

	b	*SE*	*t*	*p*	LLCI	ULCI
Reference Group: Profile 1						
Constant	4.32	2.33	1.85	.068	-0.32	8.96
T1 Paternal Warmth	-0.06	0.13	-0.45	.652	-0.31	0.20
Profile 2	-1.06	1.07	-0.99	.324	-3.19	1.07
Profile 3	-2.44	1.82	-1.34	.185	-6.06	1.19
T1 Paternal Warmth X Dummy Code 1	-0.15	0.20	-0.74	.460	-0.54	0.25
T1 Paternal Warmth X Dummy Code 2	-0.33	0.32	-1.04	.300	-0.96	0.30
T1 Child Externalizing Behavior	**0.74**	**0.06**	**12.22**	**≤ .001**	**0.62**	**0.86**
Child Puberty Stage	-0.58	0.49	-1.20	.233	-1.55	0.38
Reference Group: Profile 1						
Constant	1.88	2.52	0.75	.457	-3.12	6.88
T1 Paternal Warmth	-0.39	0.29	-1.33	.188	-0.97	0.19
Profile 1	2.44	1.82	1.34	.185	-1.19	6.06
Profile 2	1.37	1.84	0.75	.458	-2.29	5.03
T1 Paternal Warmth X Dummy Code 3	0.33	0.32	1.04	.300	-0.30	0.96
T1 Paternal Warmth X Dummy Code 4	0.18	0.33	0.55	.581	-0.47	0.83
T1 Child Externalizing Behavior	**0.74**	**0.06**	**12.22**	**≤ .001**	**0.62**	**0.86**
Child Puberty Stage	-0.58	0.49	-1.20	.233	-1.55	0.38

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 =

Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded.

Table 23

Parent Effects Model 4: T1 Child-report Paternal Warmth → T2 Child Depressive Symptoms

	b	*SE*	*t*	*p*	LLCI	ULCI
Reference Group: Profile 1						
Constant	0.07	0.08	0.86	.392	-0.10	0.24
T1 Paternal Warmth	-0.01	0.02	-0.90	.373	-0.04	0.02
Profile 2	-0.17	0.13	-1.30	.197	-0.42	0.09
Profile 3	-0.28	0.22	-1.25	.214	-0.72	0.16
T1 Paternal Warmth X Dummy Code 1	-0.03	0.03	-1.21	.230	-0.08	0.02
T1 Paternal Warmth X Dummy Code 2	-0.03	0.04	-0.86	.390	-0.12	0.05
T1 Child Depressive Symptoms	**0.55**	**0.08**	**6.69**	**≤ .001**	**0.38**	**0.71**
Reference Group: Profile 3						
Constant	-0.20	0.21	-0.99	.324	-0.61	0.21
T1 Paternal Warmth	-0.05	0.04	-1.27	.206	-0.12	0.03
Profile 1	0.28	0.22	1.25	.214	-0.16	0.72
Profile 2	0.11	0.23	0.49	.627	-0.34	0.57
T1 Paternal Warmth X Dummy Code 3	0.03	0.04	0.86	.390	-0.05	0.12
T1 Paternal Warmth X Dummy Code 4	0.00	0.04	0.11	.912	-0.08	0.09
T1 Child Depressive Symptoms	**0.55**	**0.08**	**6.69**	**≤ .001**	**0.38**	**0.71**

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower

Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 = Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded.

Table 23 for inferential statistics. When Profile 1 was coded as the reference group (dummy code = 0), the interactions with Profile 2, p = .230, and Profile 3, p = .390, were not significant. The main effect of T1 father-report paternal warmth was also not significant, p = .373. When Profile 3 was coded as the reference group (dummy code = 0), the interaction with Profile 2 was not significant, p = .627. The main effect of T1 mother-report maternal warmth was non-significant, p = .206. A post-hoc power analysis was performed to examine the achieved F-tests power. Given the effect size F^2 = .01, a sample size of 99, and 6 tested predictors, the power for the current model was .09.

Child Effects Models

Model 1: T1 Child Depressive Symptoms → T2 Child-report Maternal Warmth.

A 2-way moderated regression examined the interaction between T1 child depressive symptoms mother-child composite z-score (centered) and ASD profiles on T2 child-report maternal warmth while controlling for T1 mother-report maternal warmth and study site. When Profile 1 was coded as the reference group (dummy code), neither interaction with Profile 2, p = .141, or Profile 3, p = .328, were significant. The main effect of depressive symptoms was not significant, p = .346. See Table 24 for inferential statistics. When Profile 3 was coded as the reference group (dummy code = 0), the interaction between Profile 2 and Profile 3 was significant, b = 3.00, p = .048. See Figure 5 for the interaction. The simple slopes for Profile 1, b = -0.76, p = .346, Profile 2, b = 0.69, p = .261, and Profile 3, b = -2.31, p = .100, were not significant.

Group differences were explored at 1 *SD* above and below the mean of T1 child depressive symptoms. At 1 *SD* below mean T1 depressive symptoms, there was no significant difference in T2 maternal warmth between Profile 1 (M = 26.61) and Profile 2 (M = 25.03) or Profile 3 (M = 27.47) b = -0.86, p = .623. The difference between Profile 2 (M = 25.03) and Profile 3 was not

significant b = -2.44, p = .162. At mean T1 depressive symptoms, no significant difference in T2 maternal warmth between Profile 1 (M = 25.96) and Profile 3 (M = 25.51) was found, b = 0.45, p = .743. The difference in maternal warmth between Profile 2 (M = 25.62) and Profile 3 was not significant, b = 0.11, p =.934. At 1 *SD* above mean T1 depressive symptoms, there was no significant difference in T2 maternal warmth between Profile 1 (M = 25.31) and Profile 3 (M = 23.55) b = 1.77, p = .396. The difference between Profile 2 (M = 26.21) and Profile 3 was not significant b = 2.67, p = .184. A post-hoc power analysis was performed to examine the achieved F-tests power. Given the effect size F^2 = .036, a sample size of 98, and 7 tested predictors, the power for the current model was .21. Overall, these results indicate a significant interaction between child depression and ASD profile membership on maternal warmth. However, none of the follow up tests were significant.

Model 2: T1 Child Depressive Symptoms → T2 Mother-report Maternal Warmth. A 2-way moderated regression examined the effect of T1 child depressive symptoms mother-child composite z-score on T2 mother-report maternal warmth while controlling for T1 maternal warmth and child age. See Table 25 for inferential statistics. When Profile 1 was the reference group (dummy code = 0), there were no significant interactions with Profile 2, p = .820, or Profile 3, p = 874. The main effect of child depressive symptoms was not significant, p = .699. When Profile 3 was coded as the reference group (dummy code = 0), the interaction with Profile 2 was not significant, p = .749. The main effect of T1 child depressive symptoms was also not significant, p = .682. A post-hoc power analysis was performed to examine the achieved F-tests power. Given the effect size F^2 = .001, a sample size of 100, and 7 tested predictors, the power for the current model was .05.

Table 24

Child Effects Model 1: T1 Child Depressive Symptoms → T2 Child-report Maternal Warmth

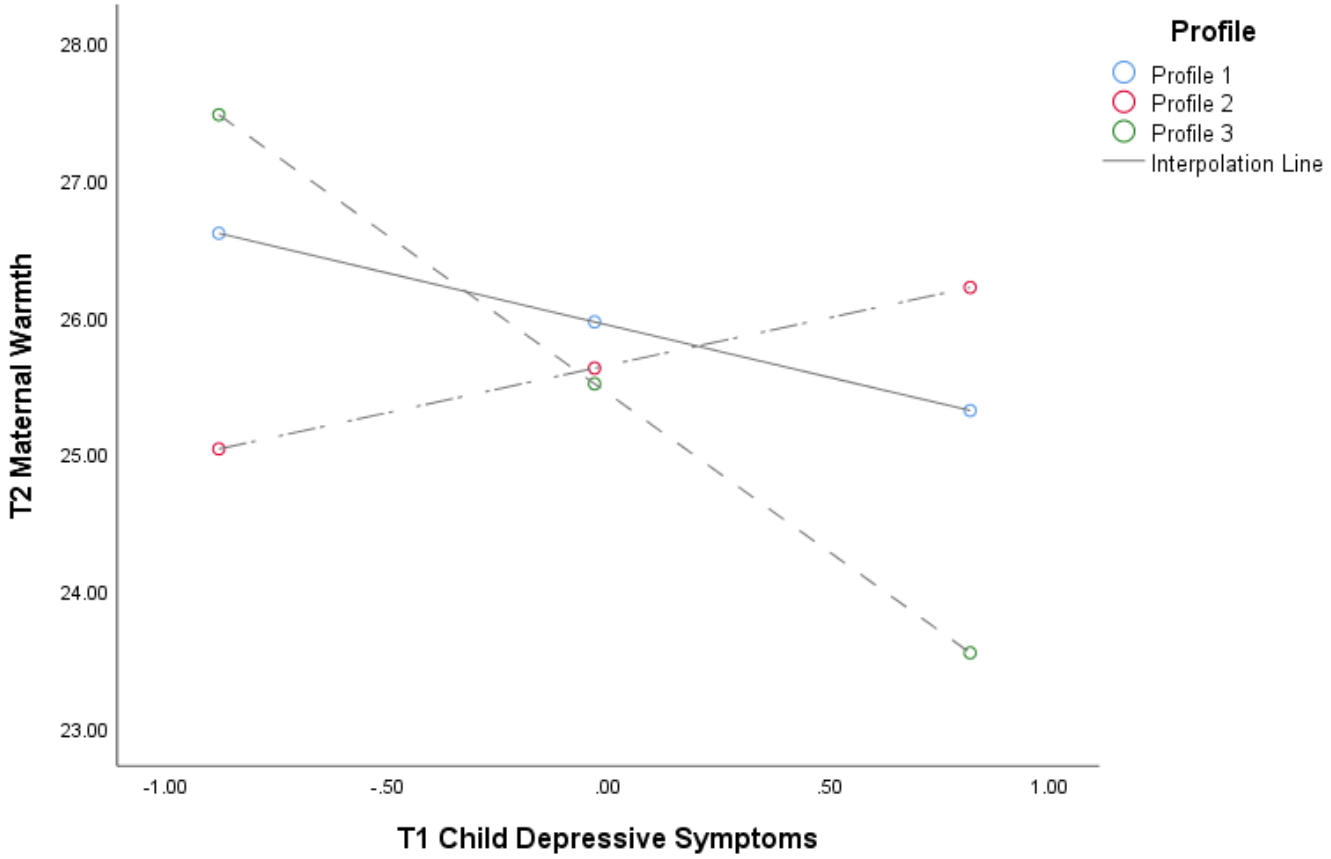
	b	*SE*	*t*	*p*	LLCI	ULCI
Reference Group: Profile 1						
Constant	**13.56**	**3.41**	**3.97**	**≤ .001**	**6.77**	**20.34**
T1 Child Depressive Symptoms	-0.76	0.80	-0.95	.346	-2.35	0.83
Profile 2	-0.29	0.81	-0.36	.718	-1.89	1.31
Profile 3	-0.50	1.38	-0.36	.719	-3.25	2.25
T1 Child Depressive Symptoms X Dummy Code 1	1.45	0.98	1.49	.141	-0.49	3.4
T1 Child Depressive Symptoms X Dummy Code 2	-1.55	1.57	-0.98	.328	-4.68	1.58
T1 Maternal Warmth	**0.56**	**0.11**	**5.17**	**≤ .001**	**0.35**	**0.78**
Site	-1.56	0.80	-1.94	.055	-3.15	0.04
Reference Group: Profile 3						
Constant	**13.06**	**3.34**	**3.91**	**≤ .001**	**6.41**	**19.7**
T1 Child Depressive Symptoms	-2.31	1.39	-1.66	.100	-5.07	0.45
Profile 1	0.50	1.38	0.36	.719	-2.25	3.25
Profile 2	0.21	1.37	0.15	.880	-2.52	2.93
T1 Child Depressive Symptoms X Dummy Code 3	1.55	1.57	0.98	.328	-1.58	4.68
T1 Child Depressive Symptoms X Dummy Code 4	**3.00**	**1.50**	**2.01**	**.048**	**0.03**	**5.97**
T1 Maternal Warmth	**0.56**	**0.11**	**5.17**	**≤ .001**	**0.35**	**0.78**
Site	-1.56	0.80	-1.94	.055	-3.15	0.04

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-

High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 = Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded

Figure 5

Child Effects Model 1: T1 Child Depressive Symptoms → T2 Child-report Maternal Warmth



Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors

Model 3: T1 Child Depressive Symptoms → T2 Child-report Paternal Warmth.

A 2-way moderated regression examined the effect of T1 child depressive symptoms mother-child composite z-score on T2 child-report paternal warmth while controlling for T1 child-report paternal warmth. See Table 26 for inferential statistics. When Profile 1 was coded as the reference group (dummy code = 0), there were no significant interactions with either Profile 2, $p = .752$, or Profile 3, $p = .809$. The main effect of T1 child depressive symptoms was not significant, $p = .843$. When Profile 3 was coded as the reference group (dummy code = 0), the interaction with Profile 2 was not significant, $p = 906$. The main effect of T1 child depressive symptoms was not significant, $p = .672$. A post-hoc power analysis was performed to examine the achieved F-tests power. Given the effect size $F^2 = .001$, a sample size of 96, and 6 tested predictors, the power for the current model was .05.

Model 4: T1 Child Depressive Symptoms → T2 Father-report Paternal Warmth.

A 2-way moderated regression examined the effect of the T1 child depressive symptoms father-child composite z-score on T2 father-report paternal warmth, while controlling for T1 father-report paternal warmth, child age, and father education. See Table 27 for inferential statistics. When Profile 1 was coded as the reference group (dummy code = 0), the interaction with Profile 2 was not significant, $p = 0.813$. The interaction with Profile 3 was significant, $b = -2.02$, $p = .015$. When Profile 3 was coded as the reference group (dummy code = 0), the interaction with Profile 2 was significant, $b = 2.16$, $p = .005$. See Figure 6 for the interaction. Simple slope analyses indicated Profile 3 had a significant decrease in T2 paternal warmth as T1 child depressive symptoms increased, $b = -2.03$, $p = .003$. The simple slopes for Profile 1, $p = .988$, and Profile 2, $p = .711$, were non-significant.

Table 25

Child Effects Model 2: T1 Child Depressive Symptoms → T2 Mother-report Maternal Warmth

	b	*SE*	*t*	*p*	LLCI	ULCI
Reference Group: Profile 1						
Constant	**11.03**	**3.12**	**3.53**	**.001**	**4.82**	**17.23**
T1 Child Depressive Symptoms	-0.17	0.44	-0.39	.699	-1.03	0.70
Profile 2	0.21	0.48	0.43	.666	-0.75	1.17
Profile 3	0.26	0.68	0.39	.700	-1.09	1.62
T1 Child Depressive Symptoms X Dummy Code 1	0.12	0.54	0.23	.820	-0.94	1.19
T1 Child Depressive Symptoms X Dummy Code 2	-0.13	0.85	-0.16	.874	-1.81	1.55
T1 Maternal Warmth	**0.67**	**0.09**	**7.56**	**≤ .001**	**0.50**	**0.85**
Child Age	-0.01	0.01	-1.53	.130	-0.03	≤ .001
Reference Group: Profile 3						
Constant	11.29	3.08	3.67	≤ .001	5.18	17.41
T1 Child Depressive Symptoms	-0.30	0.74	-0.41	.682	-1.77	1.16
Profile 1	-0.26	0.68	-0.39	.700	-1.62	1.09
Profile 2	-0.05	0.68	-0.08	.936	-1.40	1.29
T1 Child Depressive Symptoms X Dummy Code 3	0.13	0.85	0.16	.874	-1.55	1.81
T1 Child Depressive Symptoms X Dummy Code 4	0.26	0.80	0.32	.749	-1.33	1.85
T1 Maternal Warmth	**0.67**	**0.09**	**7.56**	**≤ .001**	**0.50**	**0.85**
Child Age	-0.01	0.01	-1.53	.130	-0.03	≤ .001

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 = Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded.

Table 26

Child Effects Model 3: T1 Child Depressive Symptoms → T2 Child-report Paternal Warmth

	b	*SE*	*t*	*p*	LLCI	ULCI
Reference Group: Profile 1						
Constant	3.66	1.89	1.94	.056	-0.10	7.42
T1 Child Depressive Symptoms	0.17	0.85	0.20	.843	-1.52	1.85
Profile 2	0.78	0.78	1.00	.319	-0.77	2.34
Profile 3	0.61	1.40	0.44	.661	-2.16	3.39
T1 Child Depressive Symptoms X Dummy Code 1	-0.32	1.02	-0.32	.752	-2.34	1.70
T1 Child Depressive Symptoms X Dummy Code 2	0.35	1.43	0.24	.809	-2.50	3.20
T1 Paternal Warmth	**0.82**	**0.08**	**10.56**	**≤ .001**	**0.66**	**0.97**
Reference Group: Profile 3						
Constant	4.27	2.17	1.97	.052	-0.04	8.59
T1 Child Depressive Symptoms	0.52	1.22	0.43	.672	-1.90	2.93
Profile 1	-0.61	1.40	-0.44	.661	-3.39	2.16
Profile 2	0.17	1.42	0.12	.906	-2.66	2.99
T1 Child Depressive Symptoms X Dummy Code 3	-0.35	1.43	-0.24	.809	-3.20	2.50
T1 Child Depressive Symptoms X Dummy Code 4	-0.67	1.33	-0.50	.616	-3.31	1.97
T1 Paternal Warmth	**0.82**	**0.08**	**10.56**	**≤ .001**	**0.66**	**0.97**

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 = Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded.

Table 27

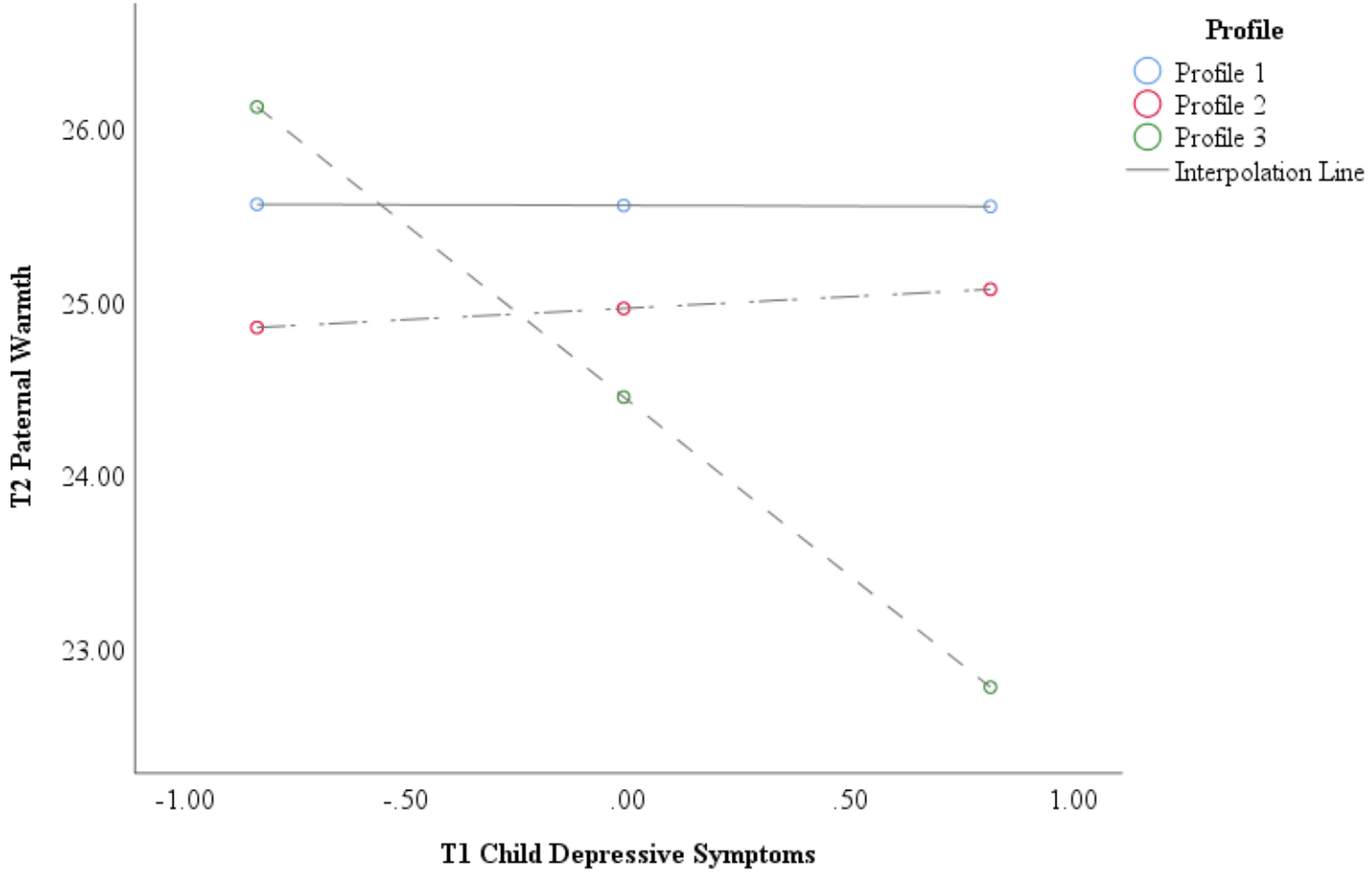
Child Effects Model 4: T1 Child Depressive Symptoms → T2 Father-report Paternal Warmth

	b	*SE*	*t*	*p*	LLCI	ULCI
Reference Group: Profile 1						
Constant	**5.83**	**2.63**	**2.22**	**.029**	**0.61**	**11.06**
T1 Child Depressive Symptoms	-0.01	0.48	-0.02	.988	-0.95	0.94
Profile 2	-0.59	0.50	-1.19	.238	-1.58	0.40
Profile 3	-1.13	0.74	-1.53	.130	-2.60	0.34
T1 Child Depressive Symptoms X Dummy Code 1	0.14	0.59	0.24	.813	-1.03	1.32
T1 Child Depressive Symptoms X Dummy Code 2	**-2.02**	**0.81**	**-2.49**	**.015**	**-3.64**	**-0.41**
T1 Paternal Warmth	**0.82**	**0.07**	**12.20**	**≤ .001**	**0.69**	**0.95**
Child Age	-0.01	0.01	-1.19	.236	-0.03	0.01
Father Education	0.15	0.19	0.79	.434	-0.23	0.52
Reference Group: Profile 3						
Constant	4.70	2.69	1.75	.084	-0.64	10.04
T1 Child Depressive Symptoms	**-2.03**	**0.66**	**-3.07**	**.003**	**-3.34**	**-0.71**
Profile 1	1.13	0.74	1.53	.130	-0.34	2.60
Profile 2	0.54	0.73	0.73	.464	-0.91	1.98
T1 Child Depressive Symptoms X Dummy Code	**2.02**	**0.81**	**2.49**	**.015**	**0.41**	**3.64**
T1 Child Depressive Symptoms X Dummy Code	**2.16**	**0.75**	**2.90**	**.005**	**0.68**	**3.65**
T1 Paternal Warmth	**0.82**	**0.07**	**12.20**	**≤ .001**	**0.69**	**0.95**
Child Age	-0.01	0.01	-1.19	.236	-0.03	0.01
Father Education	0.15	0.19	0.79	.434	-0.23	0.52

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 = Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded.

Figure 6

Child Effects Model 4: T1 Child Depressive Symptoms → T2 Father-report Paternal Warmth



Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors.

Examined differently, group differences were explored at 1 *SD* above and below the mean of T1 child depressive symptoms. There was no significant difference in T2 paternal warmth between Profile 1 and Profile 2 at low (Profile 1: $M = 25.56$; Profile 2: $M = 24.85$), $p = .316$, mean (Profile 1: $M = 25.56$; Profile 2: $M = 24.96$), $p = .237$, and high levels of T1 child depressive symptoms (Profile 1: $M = 25.55$; Profile 2: $M = 25.07$), $p = .491$. There was no difference in T2 paternal warmth between Profile 1 and Profile 3 at low (Profile 3: $M = 26.12$), $p = .543$, and mean levels of T1 child depressive symptoms (Profile 3: $M = 24.45$), $p = .0137$. There was no difference in T2 paternal warmth between Profile 2 and Profile 3 at low, $p = .159$, and mean levels, $p = .484$ of T1 child depressive symptoms. At high levels of T1 child depressive symptoms, Profile 1 had significantly higher T2 paternal warmth compared to Profile 3 ($M = 22.77$), $b = -2.77$, $p = .011$. Profile 2 also had significantly higher T2 paternal warmth at high levels of T1 child depressive symptoms compared to Profile 3, $b = 2.30$, $p = .025$. A post-hoc power analysis was performed to examine the achieved F-tests power. Given the effect size $F^2 = .031$, a sample size of 99, and 8 tested predictors, the power for the current model was .17. Overall, when autistic children in Profile 3 (Borderline IQ-High SA-High RRB) experience high depressive symptoms, their fathers display less paternal warmth compared to Profile 1 (Average IQ-Moderate SA-Low RRB) and Profile 2 (Average IQ-Moderate SA-Moderate RRB).

Model 5: T1 Child Externalizing Behaviors → T2 Father-report Paternal Warmth.

A 2-way moderated regression examined the effect of the T1 father-report child internalizing behaviors on T2 father-report paternal warmth while controlling for T1 father-report paternal warmth, child age, and father education. See Table 28 for inferential statistics. When Profile 1 was coded as the reference group (dummy code = 0), the interaction between Profile 1 and Profile 2 was not significant, $p = .540$. The interaction between Profile 1 and Profile 3 was also not significant, $p = .690$. The main effect of T1 child internalizing behaviors

was not significant, p = .194. When Profile 3 was coded as the reference group (dummy code = 0), the interaction between Profile 2 and Profile 3 was not significant, p = .880. The main effect of T1 child internalizing behaviors was not significant, p = 1.000. A post-hoc power analysis was performed to examine the achieved F-tests power. Given the effect size F^2 = .001, a sample size of 99, and 8 tested predictors, the power for the current model was .05.

Model 6: T1 Child Externalizing Behaviors → T2 Child-report Paternal Warmth. A 2-way moderated regression examined the effect of T1 father-report child externalizing behaviors on T2 child-report paternal warmth while controlling for T1 child-report paternal warmth. See Table 29 for inferential statistics. When Profile 1 was coded as the reference group (dummy code = 0), the interaction between Profile 1 and Profile 2 was not significant, p = .270. The interaction between Profile 1 and 3 was also not significant, p = .196. The main effect of T1child externalizing behaviors was non-significant, p = .415. When Profile 3 was coded as the reference group (dummy code = 0), the interaction between Profile 2 and Profile 3 was not significant, p = .387. The main effect of T1 child externalizing behaviors was not significant, p = .264. A post-hoc power analysis was performed to examine the achieved F-tests power. Given the effect size F^2 = .014, a sample size of 96, and 6 tested predictors, the power for the current model was .11.

Model 7: T1 Child Externalizing Behaviors → T2 Father-report Paternal Warmth.

A 2-way moderated regression examined the effect of T1 father-report child externalizing behaviors on T2 father-report paternal warmth while controlling for T1 father-report paternal warmth, child age, and father education. See Table 30 for inferential statistics. When Profile 1 was coded as the reference group (dummy code = 0), there were no significant interactions between Profile 1 and Profile 2, p = .641, or Profile 3, p = .828. The main effect of T1 child externalizing behaviors was not significant, p = .0113. When Profile 3 was coded as the

Table 28

Child Effects Model 5: T1 Child Internalizing Behaviors → T2 Father-report Paternal Warmth

	b	*SE*	*t*	*p*	LLCI	ULCI
Reference Group: Profile 1						
Constant	**7.84**	**2.77**	**2.83**	**.006**	**2.34**	**13.34**
T1 Child Internalizing Behaviors	-0.06	0.05	-1.31	.194	-0.16	0.03
Profile 2	-0.69	0.51	-1.36	.177	-1.70	0.32
Profile 3	-0.83	1.15	-0.72	.472	-3.12	1.46
T1 Child Internalizing Behaviors X Dummy Code 1	0.04	0.06	0.61	.540	-0.09	0.17
T1 Child Internalizing Behaviors X Dummy Code 2	0.06	0.16	0.40	.690	-0.25	0.38
T1 Paternal Warmth	**0.79**	**0.07**	**11.23**	**≤ .001**	**0.65**	**0.93**
Child Age	-0.02	0.01	-1.98	.051	-0.04	0.00
Father Education	0.16	0.20	0.81	.419	-0.23	0.55
Reference Group: Profile 3						
Constant	**7.01**	**2.85**	**2.46**	**.016**	**1.35**	**12.66**
T1 Child Internalizing Behaviors	0.001	0.15	0.00	1.000	-0.30	0.30
Profile 1	0.83	1.15	0.72	.472	-1.46	3.12
Profile 2	0.14	1.16	0.12	.905	-2.16	2.43
T1 Child Internalizing Behaviors X Dummy Code 3	-0.06	0.16	-0.40	.690	-0.38	0.25
T1 Child Internalizing Behaviors X Dummy Code 4	-0.02	0.15	-0.15	.880	-0.33	0.28
T1 Paternal Warmth	**0.79**	**0.07**	**11.23**	**≤ .001**	**0.65**	**0.93**
Child Age	-0.02	0.01	-1.98	.051	-0.04	0.00
Father Education	0.16	0.20	0.81	.419	-0.23	0.55

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 = Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded.

Table 29

Child Effects Model 6: T1 Child Externalizing Behaviors → T2 Child-report Paternal Warmth

	b	*SE*	*t*	*p*	LLCI	ULCI
Reference Group: Profile 1						
Constant	**3.83**	**1.82**	**2.10**	**.038**	**0.21**	**7.44**
T1 Child Externalizing Behaviors	0.06	0.07	0.82	.415	-0.08	0.19
Profile 2	0.87	0.78	1.11	.270	-0.69	2.42
Profile 3	-0.58	1.63	-0.35	.724	-3.81	2.66
T1 Child Externalizing Behaviors X Dummy Code 1	-0.12	0.09	-1.38	.170	-0.30	0.05
T1 Child Externalizing Behaviors X Dummy Code 2	-0.37	0.28	-1.30	.196	-0.93	0.19
T1 Paternal Warmth	**0.81**	**0.07**	**10.86**	**≤ .001**	**0.66**	**0.96**
Reference Group: Profile 3						
Constant	3.25	2.23	1.46	.148	-1.18	7.68
T1 Child Externalizing Behaviors	-0.31	0.28	-1.12	.264	-0.86	0.24
Profile 1	0.58	1.63	0.35	.724	-2.66	3.81
Profile 2	1.44	1.65	0.87	.385	-1.84	4.73
T1 Child Externalizing Behaviors X Dummy Code 3	0.37	0.28	1.30	.196	-0.19	0.93
T1 Child Externalizing Behaviors X Dummy Code 4	0.25	0.28	0.87	.387	-0.32	0.81
T1 Paternal Warmth	**0.81**	**0.07**	**10.86**	**≤ .001**	**0.66**	**0.96**

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 =

Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded.

Table 30

Child Effects Model 7: T1 Child Externalizing Behaviors → T2 Father-report Paternal Warmth

	b	*SE*	*t*	*p*	LLCI	LLCI
Reference Group: Profile 1						
Constant	**8.57**	**2.78**	**3.09**	**.003**	**3.05**	**14.08**
T1 Child Externalizing Behaviors	-0.07	0.04	-1.60	.113	-0.15	0.02
Profile 2	-0.57	0.50	-1.13	.262	-1.57	0.43
Profile 3	-0.84	0.76	-1.11	.272	-2.34	0.67
T1 Child Externalizing Behaviors X Dummy Code 1	0.03	0.06	0.47	.641	-0.08	0.14
T1 Child Externalizing Behaviors X Dummy Code 2	-0.02	0.10	-0.22	.828	-0.22	0.18
T1 Paternal Warmth	**0.77**	**0.07**	**11.16**	**≤ .001**	**0.64**	**0.91**
Child Age	**-0.02**	**0.01**	**-2.15**	**.034**	**-0.04**	**≤ .001**
Father Education	0.13	0.20	0.66	.513	-0.26	0.52
Reference Group: Profile 3						
Constant	**7.73**	**2.83**	**2.73**	**.008**	**2.10**	**13.35**
T1 Child Externalizing Behaviors	-0.09	0.09	-0.96	.341	-0.27	0.09
Profile 1	0.84	0.76	1.11	.272	-0.67	2.34
Profile 2	0.27	0.74	0.36	.716	-1.21	1.75
T1 Child Externalizing Behaviors X Dummy Code 3	0.02	0.10	0.22	.828	-0.18	0.22
T1 Child Externalizing Behaviors X Dummy Code 4	0.05	0.10	0.48	.631	-0.15	0.24
T1 Paternal Warmth	**0.77**	**0.07**	**11.16**	**≤ .001**	**0.64**	**0.91**
Child Age	**-0.02**	**0.01**	**-2.15**	**.034**	**-0.04**	**≤ .001**
Father Education	0.13	0.20	0.66	.513	-0.26	0.52

Note: Profile 1: Average IQ-Moderate SA-Low RRB; Profile 2: Average IQ-Moderate SA-Moderate RRB; Profile 3: Borderline IQ-High SA-High RRB. IQ = Intelligence Quotient, SA = Social Affect, RRB = Restricted and Repetitive Behaviors, LLCI = Lower Limit Confidence Interval, ULCI = Upper Limit Confidence Interval. Dummy Code 1 = Profile 1 vs. Profile 2. Dummy Code 2 = Profile 1 vs. Profile 3. Dummy Code 3 = Profile 3 vs. Profile 1. Dummy Code 4 = Profile 3 vs. Profile 2. Significant results are bolded

reference group (dummy code = 0), there was no significant interaction between Profile 2 and Profile 3, $p = .631$. The main effect of T1 child externalizing behaviors was also not significant, $p = .341$. A post-hoc power analysis was performed to examine the achieved F-tests power. Given the effect size $F^2 = .001$, a sample size of 96, and 8 tested predictor8, the power for the current model was .05.

Discussion

The goal of the current study was to 1) identify profiles of ASD symptoms and IQ in order to examine profile differences in child emotion and behavioral difficulties (i.e., RSA and internalizing & externalizing behaviors); 2.1) examine the bidirectional relations between parenting behaviors and autistic children's emotion regulation, and 2.2) determine whether the previously identified symptom profiles moderated the association between parenting behaviors and child behaviors. Latent profile analysis indicated three profiles: 1) average IQ, moderate SA, and moderate RRB, 2) average IQ, moderate SA, moderate RRB, and 3) borderline IQ, high SA, and high RRB. Profile 1 had the lowest SA and RRB severity of the three profiles. Profile 1 and 2 had no difference in IQ. Profile 3 had the lowest IQ and highest SA and RRB of the three profiles. Profile 3 had the lowest child anxiety symptoms compared to Profiles 1 and Profile 2. All other comparisons between profiles were not significant. One Parenting Effects model was significant, and two Child Effects models were significant. The Parenting Effect model found that high levels of maternal warmth predicted lower levels of child depression for autistic children with average IQ and low symptom severity compared to other symptom profiles. For the Child Effects models, high levels of child depressive symptoms predicted lower parental warmth for both mothers and fathers for autistic children with borderline IQ and higher symptom severity compared to other symptom profiles. Some hypotheses were confirmed while others were not.

Goal 1: ASD Symptom Profiles

Using latent profile analysis to determine if there were underlying subgroups in the population using IQ and ASD symptom severity as indicator variables, a three-profile solution was selected. Two of the profiles had average IQ with low to moderate symptom severity. The third profile had borderline average with higher ASD symptom severity. The profile pattern is consistent with Zheng and colleagues (2019) who examined ASD subgroups ages 4 to 18 using IQ, symptom severity, and adaptive functioning as indicator variables. In that study, they found four profiles that ranged from average IQ with mild impairment to low IQ with high impairment. Sullivan and colleagues (2019) examined autistic children ages 6 to 28 and found a five-profile solution using ASD symptom severity, behavior severity, IQ, and adaptive behavior. These profiles followed a similar pattern to the current study in that they found profiles with low IQ and adaptive behavior with high behavior severity to higher IQ and adaptive behavior with low behavior severity. However, they did not find that profiles had significantly different ASD symptom severity levels (Sullivan et al., 2019). Overall, it appears as if IQ and ASD symptom severity follow a pattern of higher IQ with low ASD symptom severity to lower IQ with higher ASD symptom severity. While Zheng and colleagues (2019) and Sullivan and colleagues (2019) found four- and five-profile solutions, respectively, they included additional indicator variables that would influence results. Additionally, both studies had much larger sample sizes, a wider age range, and participant inclusion was not restricted to IQ $\geq$ 70. When examining the four-profile solution for the current study, one of the profiles only had 4 members. These 4 members had the above average IQs with the lowest symptom severity. Therefore, with a larger sample, it is possible that a solution with more profiles is a better fit for the data.

Profiles were then compared to determine if there were differences in child emotion and behavioral difficulties. For child internalizing and externalizing behaviors, it was hypothesized

that profiles with higher IQs (i.e., Profiles 1 and 2 v. Profile 3) would have higher internalizing and externalizing behaviors. However, the profiles only differed on anxiety symptoms as Profile 1 and Profile 2 had significantly higher anxiety symptoms compared to Profile 3. This is consistent with prior research which suggests autistic children with higher IQs and lower symptom severity are more likely to have generalized anxiety disorders than children with lower IQs (Magnuson & Constanino, 2011; Mayes et al., 2022; van Steensel & Heeman, 2017). Autistic children with higher IQs have higher rates of anxiety because they have greater self-awareness of their impairments (Magnuson & Constanino, 2011; van Steensel & Heeman, 2017). They may have a greater desire for peer relationships but experience the stress of having to meet social norms or are bullied, increasing their risk for depression and anxiety (Chou et al., 2020; Rai et al., 2018). Indeed, Eussen and colleagues (2012) found anxiety was associated with poor social relationships in autistic children with lower symptom severity; however, this association was not moderated by IQ. Contrary to the current study and Eussen and colleagues (2012), Rieske and colleagues (2012) who found a moderate positive association between anxiety and ASD symptom severity. The authors also reported a positive correlation between anxiety and IQ, though the effect was smaller. Rodgers and colleagues (2012) also found autistic children who experienced higher levels of insistence on sameness and increased RRB had the highest levels of anxiety. In the current study, Profile 3 had the highest RRB severity in addition to the lowest anxiety scores.

It is possible the contrary results could be explained by the measures used to examine anxiety symptoms and ASD symptom severity. For the current study, ASD symptom severity was divided into SA and RRBs but did not specifically examine behaviors within the subdomains, such as insistence on sameness or sensory motor behaviors. Rieske and colleagues (2013) used a total ASD symptom severity score and total anxiety scores in their study. Perhaps

examining a total ASD symptom score would be positively associated, but the individual subdomains have different effects on anxiety. Rodgers and colleagues (2012) only reported on RRBs in their study. For the current study, the ADOS-II was used to measure RRBs. However, as the ADOS-II is reliant on observing behaviors that occur during testing sessions, if a behavior is not observed at that time, it may not be accurately captured (Freudenstein et al., 2020). Overall, Eussen and colleagues (2012) found each of the three ASD symptom domains (language and communication, reciprocal social impairments, & stereotyped behaviors and restricted interests) were negatively correlated with anxiety symptoms, which is consistent with the current study. Further, these studies included autistic children with IQ < 70. Thus, the inclusion of lower IQ autistic children may influence results. Rodgers and colleagues (2012) and Eussen and colleagues (2013) had slightly lower average participant age, though they still included school age and adolescents in their study. However, Rieske and colleagues (2013) only included preschool age children in their study. Therefore, it is possible that child age may also have a role in how anxiety symptoms present for different IQ and symptom severity subgroups.

For RSA, it was hypothesized that profiles with higher IQs and lower symptom severity would have higher RSA (i.e., better emotion regulation). However, the current study found no difference between profiles at either timepoint, which is consistent with Gao and colleagues (2015) who found no association between RSA and IQ. This finding is contrary to Staton and colleagues (2009) who found resting RSA was associated with higher cognitive functioning and better performance on IQ tests. Additionally, there was no difference between profiles for child depressive symptoms, internalizing and externalizing behaviors, and parental warmth. Much of current research is mixed in regard to the influence of IQ on internalizing and externalizing behaviors in autistic children. While several studies have shown associations between IQ and internalizing and externalizing behaviors (e.g., Chandrasekhar & Sikich, 2022; Mayes et al.,

2022), many studies have also found no association between IQ and these behaviors (Brereton et al., 2006; Gotham et al., 2015; Strang et al., 2012).

The lack of results may be attributed to discrepancies in IQ profiles and measures used in ASD research as the validity of the IQ measurement can vary based on which test is used (Wolff et al., 2022). Further, the design of the IQ test and full-scale IQ score calculations may not reflect actual intelligence but rather the ability of a specific skill. Additionally, IQ tests may not accurately account for ASD symptom presentation and the nature of the impairment (e.g., slower processing speed or social communication abilities impacting pre-requisite understanding of tasks) can lead to an underestimation of abilities (Wolff et al., 2022). As a result, epidemiological and clinical studies examining rates of ID in ASD populations often report cognitive ability in ASD populations is skewed more to the left (Baio, 2018; Wolff et al., 2022). In addition to the challenges associated with measuring IQ, recruitment strategies for many epidemiological and clinical studies rely heavily on tertiary-care centers that serve a population with higher core-ASD symptoms, leading to overrepresentation of participants who are more severely impaired and have the means to access therapeutic services (Wolff et al., 2022). Therefore, examining average (IQ $\leq$ 85) to above average (IQ $\leq$ 115) IQ in studies is inconsistent and typically only reported as a group mean and insufficient within group and subgroup details are provided (Wolff et al., 2022). While prior research has found an association between IQ and internalizing and externalizing behaviors, due to the challenges with IQ measurement and bias in study samples, it is possible no relation exists.

Further, it may be difficult to differentiate between ASD symptom severity and internalizing and externalizing behaviors, as many psychiatric symptoms present differently in ASD children (Chandrasekhar & Sikich, 2022; Rosen et al., 2018). ASD core symptoms can overlap and mask symptoms of depression and anxiety (Chandrasekhar & Sikich, 2022; Zaboski

& Storch, 2018). For instance, repetitive behaviors and resistance to change may be indicative of obsessive-compulsive disorder and impairments in social and communication skills may be related to social anxiety (Zaboski & Storch, 2018). Additionally, common symptoms of ADHD, such as interrupting others, impatience while waiting, and excessively talking, may be mistaken for social interaction difficulties (Antshel & Russo, 2019). As a result, it can inhibit correctly identifying whether an autistic child is exhibiting anxiety or hyperactivity as they may have trouble communicating their thoughts and feelings (Mannion et al., 2014). Avni and colleagues (2018) examined the association between ASD and co-occurring anxiety and ADHD. The ASD+ADHD+Anxiety group had the highest symptom severity across all three domains (i.e., social interaction, communication, and RRB), followed by the ASD+ADHD group, with the ASD-only group having the lowest symptom severity. They did not find a difference in cognitive ability between groups (Avni et al., 2019).

Overall, prior research is mixed as to the association between IQ, ASD symptom severity and child emotion and behavior difficulties. The contrary results found in the current study highlight the need to continue research examining the heterogenous presentation of ASD. Further research could provide insight into whether IQ and ASD symptom severity profiles are more consistent with the principles of equifinality or multifinality (Cicchetti & Rogosch, 1996). Equifinality (i.e., a diversity of developmental pathways that lead to the same outcome) would help to explain a lack why ASD symptom profiles, despite having different IQs and ASD symptom severity, would not have differences in emotion and behavior difficulty outcomes. Multifinality (i.e., pathology affects individuals differently based on a set of conditions) would support differences between profiles, as the variation in IQ and ASD symptom severity would then impact emotion and behavioral outcomes (Cicchetti & Rogosh, 1996). Since IQ is considered one of the major factors contributing to heterogeneity in ASD and is associated with

variability in symptom severity (e.g., Ankenman et al., 2014; Zheng et al., 2019), more research is needed examining subgroups using latent profile analysis is needed.

Goal 2: Bidirectional Association Between Parenting Behaviors and Child Outcomes

Parenting Effects Models

For Parenting Effects models, there was one significant model: T1 mother-report maternal warmth predicting T2 child depressive symptoms moderated by ASD symptom profiles. Higher levels of maternal warmth predicted lower depressive symptoms 6 months later; however, this association differed depending on the child's IQ and symptom severity profile. Specifically, for autistic children in the average IQ and moderate ASD symptom severity group, when maternal warmth was high, child depressive symptoms decreased and were lower than other symptom profiles. This supports the hypothesis that maternal warmth acts as a protective factor and ameliorates child depressive symptoms over time. Prior research has also found higher parental warmth acts as a protective factor, reducing depression symptoms over time in non-autistic children (Butterfield et al., 2021).

The lack of results in the other Parenting Effects models may be due to the type of parenting behaviors examined. Aside from parental warmth, positive parenting includes behaviors that include caring, teaching, leading, and communicating behaviors (Seay et al., 2014). Much of the existing literature on positive parenting behaviors and child outcomes examines parents of non-autistic children (e.g., Butterfield et al., 2021; Pinquart, 2017). Studies examining positive parenting with parents of autistic children are limited. However, some studies have indicated parents of autistic children engage with their child differently than non-autistic children (Maljaars et al., 2014; Ventola et al., 2017). Maljaars and colleagues (2014) found parenting of autistic children were more likely to adjust their parenting behaviors based on their child's needs and are less focused on punitive behaviors compared to parents of non-autistic

children. Ventola and colleagues (2017) found parents of autistic children are more likely to use psychological control and firm control with their children compared to parents with non-autistic children. Perhaps future research should examine other types of positive parenting behaviors to determine if they predict child emotion and behavior difficulties in autistic children. Overall, the Parenting Effects model was partially supported as increased parenting warmth was associated with lower depressive symptoms for children with average IQ and low symptom severity.

Child Effects Models

For Child Effects models, higher levels of child depressive symptoms predicted lower paternal warmth 6 months later depending on the child's IQ and symptom severity profile. Specifically, for autistic children in the profile with the lower IQ and higher ASD symptom severity (Profile 3), higher levels of child depression predicted lower paternal warmth than the other symptom profiles. Additionally, high levels of child depression predicted lower maternal warmth 6 months later, though no follow-up tests were significant. These results are contrary to the hypothesis that internalizing and externalizing behaviors have a greater negative impact on parenting warmth for profiles with average to above average IQ and high ASD symptom severity. While Profile 3 had the highest symptom severity of all profiles, it also had the lowest IQ. It is possible then that ASD symptom severity has a greater role in how child depressive symptoms affect parental warmth. In fact, ASD symptom severity is the strongest predictor of parenting stress, which negatively affects parental sense of competence and parent-child closeness (Dieleman et al., 2018; Lyons et al., 2010). Therefore, it may be that for child depressive symptoms, parenting stress mediates the relationship with parental warmth, which is then moderated by ASD symptom severity. Overall, the current study found that as child depressive symptoms increased, both maternal and paternal warmth decreased for the profile

with lower IQ and higher symptom severity. While prior research does support these findings, parenting stress may be a mediating factor that should be explored in future research.

Bidirectional Relation Between Parental Warmth & Child Emotion and Behavior Difficulties

As results indicate that in the Child Effects models child depressive symptoms influence parental warmth and in the Parenting Effects models maternal warmth, there is some support for the bidirectional association between parenting behaviors and child outcomes. Belsky's (1984) determinants of parenting and Sameroff's (1975) transactional model are both partially supported by the current study. Belsky (1984) proposed child behaviors can affect the way parents interact with their child, which then influences child outcomes. Roche and colleagues (2011) found that when early adolescents display externalizing behaviors, their parents experience anger and frustration. The parents then change how the parents respond to their child and may affect their motivation for using more punitive parenting behaviors which increases negative outcomes for their child. Sameroff (1975) suggests the transactions between parents and children are cyclical and influence each other. For instance, when children have an increase in internalizing behaviors, parents are more likely to engage in positive parenting behaviors that is then associated with a decrease in child internalizing behaviors (Serbin et al., 2015). While child effects and parenting effects were not tested in the same model in the current study, the association between child depressive symptoms and parental warmth was significant in both the Child Effects and Parenting Effects models. Therefore, future research should focus on studies that have both parenting and child effects in the models.

For the non-significant models in the current study, parenting stress may better explain the bidirectional association between parent effects and child effects. Parenting roles and demands may increase levels of distress and discomfort (i.e., parenting stress) which may negatively affect parenting behaviors and styles (Deater-Deckard, 1998; Mak et al., 2020).

Building from Belsky's (1984) determinants of parenting, Deater-Deckard (1998) hypothesized parenting stress negatively affects parenting behaviors, in that stressed parents are more likely to be reactive, more authoritarian, and less responsive. As a result of poor parenting behaviors, children of high stress parents will exhibit increased problem behaviors. Further, Deater-Deckard hypothesized parenting behavior mediates the association between parenting stress and child behaviors. According to Mak and colleagues (2020), increased parenting stress was positively associated with authoritarian and permissive parenting style and had a direct relation to elevated child internalizing and externalizing behaviors. Additionally, negative parenting styles mediated the association between parenting stress and child problem behaviors, with increased parenting stress resulting in more negative parenting styles and, therefore, leading to elevated child problem behaviors (Mak et al., 2020).

Parent-driven effects, such as parenting stress in parents of autistic children, may also be associated with negative child emotion regulation outcomes, including increased irritability, withdrawal, aggression, and impulsivity (Osborne & Reed, 2009; Rodriguez et al., 2019). While controlling for cognitive functioning and ASD symptom severity, Osborne and Reed (2009) found in a series of studies, overall, that while child behavior problems predicted parenting stress, the effect of parenting stress on child behaviors over time was stronger. However, child problem behaviors have a stronger effect on parentings stress for parents of young autistic children, while parenting stress has a greater influence on child behaviors for older autistic children. The authors suggest the discrepancy is a result of differences in developmental stages and the unique problems at each stage. Rodriguez and colleagues (2019) also found parenting stress for both mothers and fathers of autistic children was associated with increases in child internalizing behaviors, but the increase was not related to future parenting stress. For both mothers and fathers, high parenting stress also predicted an increase in child externalizing

behaviors and ASD symptom severity over time. However, increases in child externalizing behavior was only associated with future parenting stress for fathers, while high ASD symptom severity predicted parenting stress for both mothers and fathers over time (Rodriguez et al., 2019).

Overall, the results of the current study suggest there is an association between parental warmth and child depressive symptoms. While not directly measured or analyzed in the current study, there is some support for a bidirectional relation between these variables. This is in support of Belsky's (1984) determinants of parenting and Sameroff's (1975) transactional model, which suggests a bidirectional association between child behaviors and parenting behaviors. However, more research is needed to examine parent effects and child effects in the same model. Additionally, future research should examine whether parenting stress has a more significant association with child behaviors compared to parenting warmth.

Clinical Implications

The heterogeneity of ASD creates several challenges when trying to develop interventions and treatments for autistic children, especially when current treatments are already limited (Masi et al., 2017). Many of the current therapies available for autistic children have small sample sizes, are inconsistent in treatment measurements, or only have a small effect size. Current behavior interventions are expensive to implement and require intensive delivery (Masi et al., 2017). To address challenges associated with the heterogeneity of autism, Rizzo and Pavone (2016) recommend creating subgroups based on symptom presentation and considering the underlying cause of behaviors when developing treatments. As the current study found difference in anxiety symptoms between ASD symptom profiles and differences in the association between parenting behaviors and child depressive symptoms, interventions used to

treat depression and anxiety in autistic children would benefit from examining latent profiles in the data.

Additionally, there are some interventions for non-autistic children and their parents that are designed to address child depression and anxiety. Eckshtain and colleagues (2017) examined the effectiveness of behavioral parent training (BPT) for parents of children with conduct disorder and depression. BPT is a parent-oriented treatment that provides time for the parents to interact exclusively with their child while receiving training and feedback on their parenting behaviors. Despite the fact BPT did not specifically target child depression and receiving fewer treatment sessions, BPT was as effective as reducing child depressive symptoms as child-focused cognitive behavioral therapy. The authors suggest their results indicate that BPT is an effective treatment for child depression that could be used alone or in combination with cognitive behavioral therapy (Eckshtain et al., 2017).

Fernando and colleagues (2018) developed the *Parenting Resilient Kids* (PaRK) program focused on helping parents learn better parenting through customized online parenting modules and reports that assessed their preventive parenting behaviors (e.g., supporting increasing autonomy, minimizing conflict in the home, & helping their children problem solve). Sim and colleagues (2022) found parents who participated in the PaRK program were more to have higher parental acceptance, use less psychological control, and have an increased parent and child health-related quality of life at the 12-month follow-up. However, there was no effect on child depression or anxiety, which the authors suggest was due to a reduction in intervention effects over time. Yet, the study results suggest targeted programs can lead to improvements in parenting behaviors and child outcomes (Sim et al., 2022). While this study was performed in families of non-autistic children with depression and anxiety, it would be interesting to reproduce this in families with autistic children to determine if there is a similar effect. While these

parenting-focused treatments may be effect for non-autistic children with anxiety, future research should examine the efficacy of similar interventions in families with autistic children.

Heterogeneity in ASD presents several challenges when developing interventions for autistic children. As research continues and new interventions are developed, researchers should prioritize investigating subgroups within their sample. Masi and colleagues (2017) note treatments and interventions are moving toward a personalized medicine approach which would address the individual needs of autistic children and their families. Genovese and Butler (2020) note as researchers learn about the etiology of ASD and environmental factors that influence presentation, better treatment based on targeted symptoms and co-occurring conditions will be developed. As ASD is associated with increased negative child emotion and behavior outcomes (e.g., Barbier et al., 2022; Rosen et al., 2018), future research needs to focus on better understanding how subgroups within samples influence child outcomes.

Limitations

While the current study adds to the literature by examining the heterogeneity of ASD through the use of latent profile analysis and examining the bidirectional association between parental warmth and child emotion and behavior difficulties, there are several limitations that affect the results. First, the study was limited in statistical power due to a smaller overall sample size and the sample size differences between groups. As latent profile analysis is data driven, estimating the number of profiles and how many individuals will be in each profile is difficult to predict. As such, a priori power analysis is used to determine whether the sample size is sufficient to detect significant results for planned analyses is not possible. Unfortunately, the current study had many non-significant results that were contrary to previous research. Due to sample size limitations and lack of a prior power analysis, it is difficult to determine if these are true null results or if they are the result of an underpowered statistical analysis. However,

attempting to determine that through the use of post-hoc power analysis is problematic. Post-hoc power analyses assume that the observed effect size within the study is the true effect size in the population, and thus, cannot be used to determine whether the results are statistical noise or meaningful negative effects (Griffith & Feyman, 2021). Goodman and Berlin (1994) also note that post-hoc power analyses will always indicate low power, which makes them meaningless. Therefore, it is difficult to know for the current study whether the null results were a product of sample size limitations or were meaningful negative results through the use of post-hoc power analyses.

Additionally, several measures in the study had multiple reporters. Initially, it was planned to create composite scores if there were no significant differences between reporters. However, that was not the case for most of the measures in the study. Correlations were performed to determine whether one reporter should be used over another. However, in some instances, both reporters had significant correlations between variables. Thus, multiple models were run for each reporter. While some had the same pattern of results, others did not. Having multiple reporters can give different perspectives, as mothers and fathers may view and interact with their child differently and children may perceive emotions and behaviors differently from their parents. Yet, when examining the same effect, multiple reporters can add unnecessary complications to analyses.

Further, the results of the current study may not be generalizable. Latent profile analysis can provide useful information on subgroups within data. However, for the current sample, the children were mostly male and non-Hispanic white. Parents were typically married, college educated, with an annual income greater than $50k. Thus, these results may not be reflective of families of autistic children who do not fit within these demographics. Further, the distribution of IQ and symptom severity was not equal across the study inclusion. While the focus of the study

was on autistic children with IQs ≥ 70, two of the three profile groups had average IQs and moderate to low ASD symptom severity. The third profile had borderline IQ and high symptom severity, but only had 12 members. Generalizing their results to others with the same IQ and ASD symptom presentation should be done cautiously.

Conclusion

The goal of the current study was to examine the heterogeneity of ASD through the use of latent profile analyses and examined the bidirectional relationship between parenting warmth and child emotion and behavior difficulties. Latent profile analysis indicated a solution with three profiles: 1) average IQ, moderate SA, and low RRB, 2) average IQ, moderate SA, and moderate RRB, and 3) borderline IQ, high SA, and high RRB. Profile 3 had the lowest IQ and the highest SA and RRB severity. Profile 3 also had the lowest anxiety compared to Profile 1 and Profile 2. Moderated regression analyses indicated a Parent Effects model where an increase in maternal warmth was associated with lower child depressive symptoms for Profile 1. Child Effects models found an increase in child depressive symptoms was associated with lower maternal and paternal warmth for Profile 3. Overall, the study provides support for the need for future research to examine subgroups within ASD samples and support for a bidirectional relationship between parenting behaviors and child outcomes.

References

Achenbach, T. M. & Ruffle, T. M. (2000). The Child Behavior Checklist and Related Forms for Assessing Behavioral/Emotional Problems and Competencies. https://doi.org/10.1542/pir.21-8-265

Achenbach, T. M., & Edelbrock, C. S. (1978). The classification of child psychopathology: a review and analysis of empirical efforts. *Psychological Bulletin, 85*(6), 1275. https://doi.org/10.1037/0033-2909.85.6.1275

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions.* Sage Publications, Inc.

American Psychiatric Association, D., & American, P. A. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. American psychiatric association Washington, DC. https://doi.org/10.1176/appi.books.9780890425596

Ankenman, K., Elgin, J., Sullivan, K., Vincent, L., & Bernier, R. (2014). Nonverbal and verbal cognitive discrepancy profiles in autism spectrum disorders: Influence of age and gender. *American Journal on Intellectual and Developmental Disabilities*, *119*(1), 84-99. https://doi.org/10.1352/1944-7558-119.1.84

Antshel, K. M., & Russo, N. (2019). Autism spectrum disorders and ADHD: Overlapping phenomenology, diagnostic issues, and treatment considerations. *Current Psychiatry Reports, 21*, 1-11. https://doi.org/10.1007/s11920-019-1020-5

Avni, E., Ben-Itzchak, E., Saban-Bezalel, R., & Zachor, D. A. (2023). Parents' and Teachers' Perspectives of Autism and Co-Morbidity Symptom Severity in Young Children with ASD Over One School Year. *Journal of Autism and Developmental Disorders*, 1-13. https://doi.org/10.1007/s10803-023-06183-4

Baker, S. (2018). The effects of parenting on emotion and self-regulation. *Handbook of Parenting and Child Development Across the Lifespan,* , 217-240. https://doi.org/10.1111/cdep.12238

Baio, J. (2018). Prevalence of autism spectrum disorder among children aged 8 years—autism and developmental disabilities monitoring network, 11 sites, United States, 2014. *MMWR. Surveillance Summaries*, *67*. http://dx.doi.org/10.15585/mmwr.ss6706a1

Barbier, A., Chen, J., & Huizinga, J. D. (2022). Autism spectrum disorder in children is not associated with abnormal autonomic nervous system function: hypothesis and theory. *Frontiers in Psychiatry, 13*, 830234. https://doi.org/10.3389/fpsyt.2022.830234

Bauer, J. (2022). A primer to latent profile and latent class analysis. *Methods for researching professional learning and development: Challenges, applications and empirical illustrations* (pp. 243-268). Springer. https://doi.org/10.31234/osf.io/97uab

Bauminger, N., Solomon, M., & Rogers, S. J. (2010). Externalizing and internalizing behaviors in ASD. *Autism Research, 3*(3), 101-112. https://doi.org/10.1002/aur.131

Beauchaine, T. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology, 13*(2), 183-214. https://doi.org/10.1017/s0954579401002012

Beauchaine, T. P. (2015a). Future directions in emotion dysregulation and youth psychopathology. *Journal of Clinical Child & Adolescent Psychology, 44*(5), 875-896. https://doi.org/10.1080/15374416.2015.1038827

Beauchaine, T. P. (2015b). Respiratory sinus arrhythmia: A transdiagnostic biomarker of emotion dysregulation and psychopathology. *Current Opinion in Psychology, 3*, 43-47. https://doi.org/10.1016/j.copsyc.2015.01.017

Bellato, A., Arora, I., Hollis, C., & Groom, M. J. (2020). Is autonomic nervous system function atypical in attention deficit hyperactivity disorder (ADHD)? A systematic review of the evidence. *Neuroscience & Biobehavioral Reviews, 108*, 182-206. https://doi.org/10.1016/j.neubiorev.2019.11.001

Belsky, J. (1984). The determinants of parenting: A process model. *Child Development,* , 83-96. https://doi.org/10.1111/j.1467-8624.1984.tb00275.x

Berlin, K. S., Williams, N. A., & Parra, G. R. (2014). An introduction to latent variable mixture modeling (part 1): Overview and cross-sectional latent class and latent profile analyses. *Journal of Pediatric Psychology, 39*(2), 174-187. https://doi.org/10.1093/jpepsy/jst084

Birmaher, B., Khetarpal, S., Brent, D., Cully, M., Balach, L., Kaufman, J., & Neer, S. M. (1997). The screen for child anxiety related emotional disorders (SCARED): Scale construction and psychometric characteristics. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(4), 545-553. https://doi.org/10.1097/00004583-199704000-00018

Bitsko, R. H., Claussen, A. H., Lichstein, J., Black, L. I., Jones, S. E., Danielson, M. L., Hoenig, J. M., Jack, S. P. D., Brody, D. J., & Gyawali, S. (2022). Mental health surveillance among children—United States, 2013–2019. *MMWR Supplements, 71*(2), 1. http://dx.doi.org/10.15585/mmwr.su7102a1

Bölte, S., Dziobek, I., & Poustka, F. (2009). Brief report: The level and nature of autistic intelligence revisited. *Journal of Autism and Developmental Disorders, 39*, 678-682. https://doi.org/10.1007/s10803-008-0667-2

Bougeard, C., Picarel-Blanchot, F., Schmid, R., Campbell, R., & Buitelaar, J. (2021). Prevalence of autism spectrum disorder and co-morbidities in children and adolescents: a systematic

literature review. *Frontiers in Psychiatry, 12*, 744709. https://doi.org/10.3389/fpsyt.2021.744709

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. https://doi.org/10.1007/s10803-006-0125-y

Bronfenbrenner, U. (1979). *The ecology of human development: Experiments by nature and design*. Harvard university press. https://doi.org/10.2307/j.ctv26071r6

Bronfenbrenner, U. (2005). *Making human beings human: Bioecological perspectives on human development*. Sage Publications Ltd.

Bruni, T. P. (2014). Test review: Social responsiveness scale–Second edition (SRS-2). *Journal of Psychoeducational Assessment, 32*(4), 365-369.

Burlaka, V., Graham-Bermann, S., & Delva, J. (2017). Family factors and parenting in Ukraine. *Child Abuse & Neglect, 72*, 154-162. https://doi.org/10.1016/j.chiabu.2017.08.007

Butterfield, R. D., Silk, J. S., Lee, K. H., Siegle, G. S., Dahl, R. E., Forbes, E. E., Ryan, N. D., Hooley, J. M., & Ladouceur, C. D. (2021). Parents still matter! Parental warmth predicts adolescent brain function and anxiety and depressive symptoms 2 years later. *Development and Psychopathology, 33*(1), 226-239. https://doi.org/10.1017/S0954579419001718

Cai, R. Y., Richdale, A. L., Uljarević, M., Dissanayake, C., & Samson, A. C. (2018). Emotion regulation in autism spectrum disorder: Where we are and where we need to go. *Autism Research, 11*(7), 962-978. https://doi.org/10.1002/aur.1968

Chalmers, J. A., Quintana, D. S., Abbott, M. J. -., & Kemp, A. H. (2014). Anxiety disorders are associated with reduced heart rate variability: a meta-analysis. *Frontiers in Psychiatry, 5*, 80. https://doi.org/10.3389/fpsyt.2014.00080

Chandrasekhar, T., & Sikich, L. (2015). Challenges in the diagnosis and treatment of depression in autism spectrum disorders across the lifespan. *Dialogues in Clinical Neuroscience, 17*(2), 219-227. https://doi.org/10.31887/DCNS.2015.17.2/tchandrasekhar

Cheng, Y., Sun, F., D'Souza, A., Dhakal, B., Pisano, M., Chhabra, S., Stolley, M., Hari, P., & Janz, S. (2021). Autonomic nervous system control of multiple myeloma. *Blood Reviews, 46*, 100741. https://doi.org/10.1016/j.blre.2020.100741

Chou, W., Wang, P., Hsiao, R. C., Hu, H., & Yen, C. (2020). Role of school bullying involvement in depression, anxiety, suicidality, and low self-esteem among adolescents with high-functioning autism spectrum disorder. *Frontiers in Psychiatry, 11*, 9. https://doi.org/10.3389/fpsyt.2020.00009

Cibralic, S., Kohlhoff, J., Wallace, N., McMahon, C., & Eapen, V. (2019). A systematic review of emotion regulation in children with Autism Spectrum Disorder. *Research in Autism Spectrum Disorders, 68*, 101422. https://doi.org/10.1016/j.rasd.2019.101422

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology, 8*(4), 597–600. https://doi.org/10.1017/S0954579400007318

Conner, C. M., Golt, J., Shaffer, R., Righi, G., Siegel, M., & Mazefsky, C. A. (2021). Emotion dysregulation is substantially elevated in autism compared to the general population: Impact on psychiatric services. *Autism Research, 14*(1), 169-181. https://doi.org/10.1002/aur.2450

Cui, L., Morris, A. S., Harrist, A. W., Larzelere, R. E., & Criss, M. M. (2015). Dynamic changes in parent affect and adolescent cardiac vagal regulation: a real-time analysis. *Journal of Family Psychology, 29*(2), 180. https://doi.org/10.1037/fam0000067

Deater-Deckard, K. (1998). Parenting stress and child adjustment: Some old hypotheses and new questions. *Clinical psychology: Science and practice*, *5*(3), 314. https://doi.org/10.1111/j.1468-2850.1998.tb00152.x

De Haan, A.,D., Soenens, B., Deković, M., & Prinzie, P. (2013). Effects of childhood aggression on parenting during adolescence: The role of parental psychological need satisfaction. *Journal of Clinical Child & Adolescent Psychology, 42*(3), 393-404. https://doi.org/10.1080/15374416.2013.769171

DeFilippis, M. (2018). Depression in children and adolescents with autism spectrum disorder. *Children, 5*(9), 112. https://doi.org/10.3390/children5090112

Dieleman, L. M., De Pauw, S.,S.W., Soenens, B., Mabbe, E., Campbell, R., & Prinzie, P. (2018). Relations between problem behaviors, perceived symptom severity and parenting in adolescents and emerging adults with ASD: The mediating role of parental psychological need frustration. *Research in Developmental Disabilities, 73*, 21-30. https://doi.org/10.1016/j.ridd.2017.12.012

Dunn, L. M., & Dunn, D. M. (2007). *Peabody Picture Vocabulary Test--Fourth Edition (PPVT-4)* [Database record]. APA PsycTests. https://doi.org/10.1037/t15144-000

Eckshtain, Dikla, Rachel Horn, and John R. Weisz. "Family-based interventions for youth depression: Meta-analysis of randomized clinical trials." *Child Psychiatry & Human Development* 54.6 (2023): 1737-1748. https://doi.org/10.1007/s10578-022-01375-y

Elliott, C. D., Salerno, J. D., Dumont, R., & Willis, J. O. (2018). The Differential Ability Scales—Second Edition. In D. P. Flanagan & E. M. McDonough (Eds.), *Contemporary*

intellectual assessment: Theories, tests, and issues (4th ed., pp. 360–382). The Guilford Press.

Enders, C. K. (2022). *Applied missing data analysis*. Guilford Publications.

Erdfelder, E., Faul, F., & Buchner, A. (1996). GPOWER: A general power analysis program. Behavior Research Methods, *Instruments & Computers, 28*(1), 1–11. https://doi.org/10.3758/BF03203630

Eussen, M. L., Van Gool, A. R., Verheij, F., De Nijs, P. F., Verhulst, F. C., & Greaves-Lord, K. (2013). The association of quality of social relations, symptom severity and intelligence with anxiety in children with autism spectrum disorders. *Autism*, *17*(6), 723-735. https://doi.org/10.1177/1362361312453882

Fenning, R. M., Baker, J. K., & Moffitt, J. (2018). Intrinsic and extrinsic predictors of emotion regulation in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 48*, 3858-3870. https://doi.org/10.1007/s10803-018-3647-1

Ferguson, S. L., Moore, E. W.,G., & Hull, D. M. (2020). Finding latent groups in observed data: A primer on latent profile analysis in Mplus for applied researchers. *International Journal of Behavioral Development, 44*(5), 458-468. https://doi.org/10.1177/0165025419881721

Fernando, S. M., Tran, A., Taljaard, M., Cheng, W., Rochwerg, B., Seely, A. J., & Perry, J. J. (2018). Prognostic accuracy of the quick sequential organ failure assessment for mortality in patients with suspected infection: a systematic review and meta-analysis. Annals of internal medicine, 168(4), 266-275. https://doi.org/10.7326/M17-2820

Fitzpatrick, S. E., Srivorakiat, L., Wink, L. K., Pedapati, E. V., & Erickson, C. A. (2016). Aggression in autism spectrum disorder: presentation and treatment

options. *Neuropsychiatric Disease and Treatment,* , 1525-1538. https://doi.org/10.2147/NDT.S84585

Fox, A. R., Aldrich, J. T., Ahles, J. J., & Mezulis, A. H. (2019). Stress and parenting predict changes in adolescent respiratory sinus arrhythmia. *Developmental Psychobiology, 61*(8), 1214-1224. https://doi.org/10.1002/dev.21863

Freudenstein, O., Shimoni, H. N., Gindi, S., & Leitner, Y. (2020). Disagreement between assessment of ASD utilizing the ADOS-2 and DSM-5–A preliminary study. *Annales Universitatis Paedagogicae Cracoviensis. Studia Psychologica*, *13*, 17-26. https://doi.org/10.24917/20845596.13.1

Gao, Y., Borlam, D., & Zhang, W. (2015). The association between heart rate reactivity and fluid intelligence in children. *Biological psychology*, *107*, 69-75. https://doi.org/10.1007/s40489-014-0012-y

Gau, J. M. (2010). A longitudinal analysis of citizens' attitudes about police. *Policing: An International Journal of Police Strategies & Management, 33*(2), 236-252. https://doi.org/10.1108/13639511011044867

Genovese, A., & Butler, M. G. (2020). Clinical assessment, genetics, and treatment approaches in autism spectrum disorder (ASD). *International journal of molecular sciences*, *21*(13), 4726. https://doi.org/10.3390/ijms21134726

Goldsmith, S. F., & Kelley, E. (2018). Associations between emotion regulation and social impairment in children and adolescents with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 48*, 2164-2173. https://doi.org/10.1007/s10803-018-3483-3

Goodman, S. N., & Berlin, J. A. (1994). The use of predicted confidence intervals when planning experiments and the misuse of power when interpreting results. Annals of internal medicine, 121(3), 200-206. https://doi.org/ 10.7326/0003-4819-121-3-199408010-00008

Gotham, K., Brunwasser, S. M., & Lord, C. (2015). Depressive and anxiety symptom trajectories from school age through young adulthood in samples with autism spectrum disorder and developmental delay. *Journal of the American Academy of Child & Adolescent Psychiatry, 54*(5), 369-376. https://doi.org/10.1016/j.jaac.2015.02.005

Gotham, K., Pickles, A., & Lord, C. (2009). Standardizing ADOS scores for a measure of severity in autism spectrum disorders. *Journal of autism and developmental disorders*, *39*(5), 693–705. https://doi.org/10.1007/s10803-008-0674-3

Gray, J. A. (1987). Perspectives on anxiety and impulsivity: A commentary. https://doi.org/10.1016/0092-6566(87)90036-5

Greenberg, J. S., Seltzer, M. M., Hong, J., & Orsmond, G. I. (2006). Bidirectional effects of expressed emotion and behavior problems and symptoms in adolescents and adults with autism. *American Journal on Mental Retardation, 111*(4), 229-249. https://doi.org/10.1352/0895-8017(2006)111[229:BEOEEA]2.0.CO;2

Griffith, K. N., & Feyman, Y. (2021). Amplifying the noise: The dangers of post hoc power analyses. Journal of Surgical Research, 259, A9-A11. https://doi.org/10.1016/j.jss.2019.09.075

Gross, J. J. (2014). Emotion regulation: Conceptual and empirical foundations. *Handbook of Emotion Regulation, 2*, 3-20.

Gross, J. J. (2015a). Emotion regulation: Current status and future prospects. *Psychological Inquiry, 26*(1), 1-26. https://doi.org/10.1080/1047840X.2014.940781

Gross, J. J. (2015b). The extended process model of emotion regulation: Elaborations, applications, and future directions. *Psychological Inquiry, 26*(1), 130-137. https://doi.org/10.1080/1047840X.2015.989751

Gross, J. J., & Jazaieri, H. (2014). Emotion, emotion regulation, and psychopathology: An affective science perspective. *Clinical Psychological Science, 2*(4), 387-401. https://doi.org/10.1177/2167702614536164

Guy, L., Souders, M., Bradstreet, L., DeLussey, C., & Herrington, J. D. (2014). Brief report: Emotion regulation and respiratory sinus arrhythmia in autism spectrum disorder. *Journal of Autism and Developmental Disorders, 44*, 2614-2620. https://doi.org/10.1007/s10803-014-2124-8

Hair Jr, J.,F., Hult, G. T., Ringle, C. M., Sarstedt, M., Danks, N. P., Ray, S., Hair, J. F., Hult, G. T., Ringle, C. M., & Sarstedt, M. (2021). Moderation analysis. *Partial Least Squares Structural Equation Modeling (PLS-SEM) using R: A Workbook,* , 155-172. https://doi.org/10.1007/978-3-030-80519-7

Hansen, L. K., & Jordan, S. S. (2020). Internalizing behaviors. *Encyclopedia of personality and individual differences* (pp. 2343-2346). Springer. https://doi.org/10.1007/978-3-319-24612-3_907

Harkins, C., & Mazurek, M. O. (2023). The Impact of Co-occurring ADHD on Social Competence Intervention Outcomes in Youth with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders,* , 1-12. https://doi.org/10.1007/s10803-023-05987-8

Hartmann, R., Schmidt, F. M., Sander, C., & Hegerl, U. (2019). Heart rate variability as indicator of clinical state in depression. *Frontiers in Psychiatry, 9*, 735. https://doi.org/10.3389/fpsyt.2018.00735

Hastings, P. D., Kahle, S., Fleming, C., Lohr, M. J., Katz, L. F., & Oxford, M. L. (2019). An intervention that increases parental sensitivity in families referred to Child Protective Services also changes toddlers' parasympathetic regulation. *Developmental Science, 22*(1), e12725. https://doi.org/10.1111/desc.12725

Hayes, A. F. (2017). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. Guilford publications.

Helsel, W. J., & Matson, J. L. (1984). The assessment of depression in children: The internal structure of the Child Depression Inventory (CDI). *Behaviour Research and Therapy, 22*(3), 289-298. https://doi.org/10.1016/0005-7967(84)90009-3

Hickey, E. J., Bolt, D., Rodriguez, G., & Hartley, S. L. (2020). Bidirectional relations between parent warmth and criticism and the symptoms and behavior problems of children with autism. *Journal of Abnormal Child Psychology, 48*, 865-879. https://doi.org/10.1007/s10802-020-00628-5

Hill, T. L., Gray, S. A. O., Kamps, J. L., & Enrique Varela, R. (2015). Age and adaptive functioning in children and adolescents with ASD: The effects of intellectual functioning and ASD symptom severity. *Journal of Autism and Developmental Disorders, 45*, 4074-4083. https://doi.org/10.1007/s10803-015-2522-6

Hinshaw, S. P. (1987). On the distinction between attentional deficits/hyperactivity and conduct problems/aggression in child psychopathology. *Psychological Bulletin, 101*(3), 443. https://doi.org/10.1037/0033-2909.101.3.443

Hours, C., Recasens, C., & Baleyte, J. (2022). ASD and ADHD comorbidity: what are we talking about? *Frontiers in Psychiatry, 13*, 154. https://doi.org/10.3389/fpsyt.2022.837424

Huberty, T.J. (2017). Students With Emotional and Behavioral Problems. *Reference Module in Neuroscience and Biobehavioral Psychology.* https://doi.org/10.1016/B978-0-12-809324-5.05547-4

Hudson, C. C., Hall, L., & Harkness, K. L. (2019). Prevalence of depressive disorders in individuals with autism spectrum disorder: A meta-analysis. *Journal of Abnormal Child Psychology, 47*, 165-175. https://doi.org/10.1007/s10802-018-0402-1

Hus, V., Gotham, K., & Lord, C. (2014). Standardizing ADOS domain scores: separating severity of social affect and restricted and repetitive behaviors. *Journal of autism and developmental disorders*, *44*(10), 2400–2412. https://doi.org/10.1007/s10803-012-1719-1

Jasim, S., & Perry, A. (2023). Repetitive and restricted behaviors and interests in autism spectrum disorder: relation to individual characteristics and mental health problems. *BMC Psychiatry, 23*(1), 1-14. https://doi.org/10.1186/s12888-023-04766-0

Jiujias, M., Kelley, E., & Hall, L. (2017). Restricted, repetitive behaviors in autism spectrum disorder and obsessive–compulsive disorder: A comparative review. *Child Psychiatry & Human Development, 48*, 944-959. https://doi.org/10.1007/s10578-017-0717-0

Kaat, A. J., & Lecavalier, L. (2013). Disruptive behavior disorders in children and adolescents with autism spectrum disorders: A review of the prevalence, presentation, and treatment. *Research in Autism Spectrum Disorders, 7*(12), 1579-1594. https://doi.org/10.1016/j.rasd.2013.08.012

Kanne, S. M., & Mazurek, M. O. (2011). Aggression in children and adolescents with ASD: Prevalence and risk factors. *Journal of Autism and Developmental Disorders, 41*, 926-937. https://doi.org/10.1007/s10803-010-1118-4

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child, 2*(3), 217-250.

Kasari, C., & Sigman, M. (1997). Linking parental perceptions to interactions in young children with autism. *Journal of Autism and Developmental Disorders, 27*, 39-57. https://doi.org/10.1023/a:1025869105208

Kemp, A. H., & Quintana, D. S. (2013). The relationship between mental and physical health: insights from the study of heart rate variability. *International Journal of Psychophysiology, 89*(3), 288-296. https://doi.org/10.1016/j.ijpsycho.2013.06.018

Kent, R., & Simonoff, E. (2017). Prevalence of anxiety in autism spectrum disorders. *Anxiety in Children and Adolescents with Autism Spectrum Disorder,* , 5-32. https://doi.org/10.1016/B978-0-12-805122-1.00002-8

Khaleque, A. (2013). Perceived parental warmth, and children's psychological adjustment, and personality dispositions: A meta-analysis. *Journal of Child and Family Studies, 22*, 297-306. https://doi.org/10.1007/s10826-012-9579-z

Klein, M. R., Lengua, L. J., Thompson, S. F., Moran, L., Ruberry, E. J., Kiff, C., & Zalewski, M. (2018). Bidirectional relations between temperament and parenting predicting preschool-age children's adjustment. *Journal of Clinical Child & Adolescent Psychology, 47*, S113-S126. https://doi.org/10.1080/15374416.2016.1169537

Klevens, J., & Hall, J. (2014). The importance of parental warmth, support, and control in preventing adolescent misbehavior. *Journal of Child and Adolescent Behavior, 2*(1), 121-129. https://doi.org/10.4172/2375-4494.1000121

Konstantareas, M. M., & Homatidis, S. (1992). Mothers' and fathers' self-report of involvement with autistic, mentally delayed, and normal children. *Journal of Marriage and the Family*, 153-164. https://doi.org/10.2307/353283

Kushki, A., Brian, J., Dupuis, A., & Anagnostou, E. (2014). Functional autonomic nervous system profile in children with autism spectrum disorder. *Molecular Autism, 5*, 1-10. https://doi.org/10.1186/2040-2392-5-39

Lansford, J. E., Godwin, J., Al-Hassan, S., Bacchini, D., Bornstein, M. H., Chang, L., Chen, B., Deater-Deckard, K., Di Giunta, L., & Dodge, K. A. (2018). Longitudinal associations between parenting and youth adjustment in twelve cultural groups: Cultural normativeness of parenting as a moderator. *Developmental Psychology, 54*(2), 362. https://doi.org/10.1037/dev0000416

Lee, S. J., Pace, G. T., Lee, J. Y., & Knauer, H. (2018). The association of fathers' parental warmth and parenting stress to child behavior problems. *Children and Youth Services Review, 91*, 1-10. https://doi.org/10.1016/j.childyouth.2018.05.020

Lerner, M. D., Mazefsky, C. A., Weber, R. J., Transue, E., Siegel, M., & Gadow, K. D. (2018). Verbal ability and psychiatric symptoms in clinically referred inpatient and outpatient youth with ASD. *Journal of Autism and Developmental Disorders, 48*, 3689-3701. https://doi.org/10.1007/s10803-017-3344-5

Lisitsa, E., Bolden, C. R., Johnson, B. D., & Mezulis, A. H. (2021). Impact of stress and parenting on respiratory sinus arrythmia trajectories in early adolescence. *Developmental Psychobiology, 63*(6), e22165. https://doi.org/10.1002/dev.22165

Liu, J. (2004). Childhood externalizing behavior: Theory and implications. *Journal of Child and Adolescent Psychiatric Nursing, 17*(3), 93-103. https://doi.org/10.1111/j.1744-6171.2004.tb00003.x

Lloyd, B., Macdonald, J. A., Youssef, G. J., Knight, T., Letcher, P., Sanson, A., & Olsson, C. A. (2017). Negative reactivity and parental warmth in early adolescence and depressive

symptoms in emerging adulthood. *Australian Journal of Psychology, 69*(2), 121-129. https://doi.org/10.1111/ajpy.12129

Lord, C., Brugha, T. S., Charman, T., Cusack, J., Dumas, G., Frazier, T., Jones, E. J. H., Jones, R. M., Pickles, A., & State, M. W. (2020). Autism spectrum disorder. *Nature Reviews Disease Primers, 6*(1), 1-23. https://doi.org/10.1038/s41572-019-0138-4

Lord, C., Elsabbagh, M., Baird, G., & Veenstra-Vanderweele, J. (2018). Autism spectrum disorder. *The Lancet, 392*(10146), 508-520. https://doi.org/10.1016/S0140-6736(18)31129-2

Lord, C., Rutter, M., Goode, S., Heemsbergen, J., Jordan, H., Mawhood, L., & Schopler, E. (1989). Autism Diagnostic Observation Schedule (ADOS) [Database record].

Maenner, M. J., Warren, Z., Williams, A. R., Amoakohene, E., Bakian, A. V., Bilder, D. A., Durkin, M. S., Fitzgerald, R. T., Furnier, S. M., & Hughes, M. M. (2023). Prevalence and characteristics of autism spectrum disorder among children aged 8 years—Autism and Developmental Disabilities Monitoring Network, 11 sites, United States, 2020. *MMWR Surveillance Summaries, 72*(2), 1. http://dx.doi.org/10.15585/mmwr.ss7202a1

Magnuson, K. M., & Constantino, J. N. (2011). Characterization of depression in children with autism spectrum disorders. *Journal of Developmental and Behavioral Pediatrics: JDBP, 32*(4), 332. https://doi.org/10.1097/DBP.0b013e318213f56c

Mak, M. C. K., Yin, L., Li, M., Cheung, R. Y. H., & Oon, P. T. (2020). The relation between parenting stress and child behavior problems: Negative parenting styles as mediator. Journal of Child and Family Studies, 29, 2993-3003. https://doi.org/10.1007/s10826-020-01785-3

Maljaars, J., Boonen, H., Lambrechts, G., Van Leeuwen, K., & Noens, I. (2014). Maternal parenting behavior and child behavior problems in families of children and adolescents

with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 44*, 501-512. https://doi.org/10.1007/s10803-013-1894-8

Mannion, A., Brahm, M., & Leader, G. (2014). Comorbid psychopathology in autism spectrum disorder. *Review Journal of Autism and Developmental Disorders*, *1*, 124-134. https://doi.org/10.1007/s40489-014-0012-y

Martínez-González, A.,E., Cervin, M., & Piqueras, J. A. (2022). Relationships between emotion regulation, social communication and repetitive behaviors in Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders, 52*(10), 4519-4527. https://doi.org/10.1007/s10803-021-05340-x

Masi, A., DeMayo, M. M., Glozier, N., & Guastella, A. J. (2017). An overview of autism spectrum disorder, heterogeneity and treatment options. *Neuroscience Bulletin, 33*, 183-193. https://doi.org/10.1007/s12264-017-0100-y

Mayes, S. D., Baweja, R., Waschbusch, D. A., & Calhoun, S. L. (2022). Relationship between IQ and Internalizing and Externalizing Symptoms in Children with Autism and Children with ADHD. *Journal of Mental Health Research in Intellectual Disabilities, 15*(2), 95-110. https://doi.org/10.1080/19315864.2022.2029643

Mayes, S. D., Calhoun, S. L., Murray, M. J., Ahuja, M., & Smith, L. A. (2011). Anxiety, depression, and irritability in children with autism relative to other neuropsychiatric disorders and typical development. *Research in Autism Spectrum Disorders, 5*(1), 474-485. https://doi.org/10.1016/j.rasd.2010.06.012

Mazefsky, C. A., Herrington, J., Siegel, M., Scarpa, A., Maddox, B. B., Scahill, L., & White, S. W. (2013). The role of emotion regulation in autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 52*(7), 679-688. https://doi.org/10.1016/j.jaac.2013.05.006

Mazefsky, C. A., & White, S. W. (2014). Emotion regulation: Concepts & practice in autism spectrum disorder. *Child and Adolescent Psychiatric Clinics, 23*(1), 15-24. https://doi.org/10.1016/j.chc.2013.07.002

McLeod, B. D., Wood, J. J., & Weisz, J. R. (2007). Examining the association between parenting and childhood anxiety: A meta-analysis. *Clinical Psychology Review, 27*(2), 155-172. https://doi.org/10.1016/j.cpr.2006.09.002

McRae, K., & Gross, J. J. (2020). Emotion regulation. *Emotion, 20*(1), 1–9. https://doi.org/10.1037/emo0000703

MindWare Technologies LTD, Gahanna, OH, USA, 2011

Mindware Technologies, Gahanna, OH, USA, 2014

Montazeri, F., de Bildt, A., Dekker, V., & Anderson, G. M. (2020). Network analysis of behaviors in the depression and autism realms: Inter-relationships and clinical implications. *Journal of Autism and Developmental Disorders, 50*(5), 1580-1595. https://doi.org/10.1007/s10803-019-03914-4

Morris, A. S., Criss, M. M., Silk, J. S., & Houltberg, B. J. (2017). The impact of parenting on emotion regulation during childhood and adolescence. *Child Development Perspectives, 11*(4), 233-238. https://doi.org/10.1111/cdep.12238

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development, 16*(2), 361-388. https://doi.org/10.1111/j.1467-9507.2007.00389.x

Muthén, B., & Muthén, L. (2017). Mplus. *Handbook of item response theory* (pp. 507-518). Chapman and Hall/CRC.

Neuhaus, E., Bernier, R., & Beauchaine, T. P. (2014). Brief report: social skills, internalizing and externalizing symptoms, and respiratory sinus arrhythmia in autism. *Journal of Autism and Developmental Disorders, 44*, 730-737. https://doi.org/10.1007/s10803-013-1923-7

Osborne, L. A., & Reed, P. (2009). The relationship between parenting stress and behavior problems of children with autistic spectrum disorders. *Exceptional Children, 76*(1), 54-73. https://doi.org/10.1177/001440290907600103

Parker, G., Tupling, H., & Brown, L. B. (1979). A parental bonding instrument. *British Journal of Medical Psychology, 52*(1), 1–10. https://doi.org/10.1111/j.2044-8341.1979.tb02487.x

Patriquin, M. A., Lorenzi, J., Scarpa, A., & Bell, M. A. (2014). Developmental trajectories of respiratory sinus arrhythmia: Associations with social responsiveness. *Developmental Psychobiology, 56*(3), 317-326. https://doi.org/10.1002/dev.21100

Perry, N. B., Calkins, S. D., Nelson, J. A., Leerkes, E. M., & Marcovitch, S. (2012). Mothers' responses to children's negative emotions and child emotion regulation: The moderating role of vagal suppression. *Developmental Psychobiology, 54*(5), 503-513. https://doi.org/10.1002/dev.20608

Perry, N. B., Nelson, J. A., Swingler, M. M., Leerkes, E. M., Calkins, S. D., Marcovitch, S., & O'Brien, M. (2013). The relation between maternal emotional support and child physiological regulation across the preschool years. *Developmental Psychobiology, 55*(4), 382-394. https://doi.org/10.1002/dev.21042

Pezzimenti, F., Han, G. T., Vasa, R. A., & Gotham, K. (2019). Depression in youth with autism spectrum disorder. *Child and Adolescent Psychiatric Clinics, 28*(3), 397-409. https://doi.org/10.1016/j.chc.2019.02.009

Pinquart, M. (2017). Associations of parenting dimensions and styles with externalizing problems of children and adolescents: An updated meta-analysis. *Developmental Psychology, 53*(5), 873. https://doi.org/10.1037/dev0000295

Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory. *Psychophysiology, 32*(4), 301-318. https://doi.org/10.1111/j.1469-8986.1995.tb01213.x

Porges, S. W. (1997). Emotion: An evolutionary by-product of the neural regulation of the autonomic nervous system. *Annals of the New York Academy of Sciences-Paper Edition, 807*, 62-77. https://doi.org/10.1111/j.1749-6632.1997.tb51913.x

Préfontaine, I., Morizot, J., Lanovaz, M. J., & Rivard, M. (2022). A person-centered perspective on differential efficacy of early behavioral intervention in children with autism: A latent profile analysis. *Research in Autism Spectrum Disorders, 97*, 102017. https://doi.org/10.1016/j.rasd.2022.102017

Quintana, D. S., Elstad, M., Kaufmann, T., Brandt, C. L., Haatveit, B., Haram, M., Nerhus, M., Westlye, L. T., & Andreassen, O. A. (2016). Resting-state high-frequency heart rate variability is related to respiratory frequency in individuals with severe mental illness but not healthy controls. *Scientific Reports, 6*(1), 37212. https://doi.org/10.1038/srep37212

Rai, D., Heuvelman, H., Dalman, C., Culpin, I., Lundberg, M., Carpenter, P., & Magnusson, C. (2018). Association between autism spectrum disorders with or without intellectual disability and depression in young adulthood. *JAMA Network Open, 1*(4), e181465. https://doi.org/10.1001/jamanetworkopen.2018.1465

Ram, N., & Grimm, K. J. (2009). Methods and measures: Growth mixture modeling: A method for identifying differences in longitudinal change among unobserved

groups. *International Journal of Behavioral Development, 33*(6), 565-576. https://doi.org/10.1177/0165025409343765

Reetzke, R., Singh, V., Hong, J. S., Holingue, C. B., Kalb, L. G., Ludwig, N. N., Menon, D., Pfeiffer, D. L., & Landa, R. J. (2022). Profiles and correlates of language and social communication differences among young autistic children. *Frontiers in Psychology, 13*, 936392. https://doi.org/10.3389/fpsyg.2022.936392

Rieske, R. D., Matson, J. L., May, A. C., & Kozlowski, A. M. (2012). Anxiety in children with high-functioning autism spectrum disorders: Significant differences and the moderating effects of social impairments. *Journal of Developmental and Physical Disabilities*, *24*, 167-180. https://doi.org/10.1007/s10882-011-9264-y

Rizzo, R., & Pavone, P. (2016). Aripiprazole for the treatment of irritability and aggression in children and adolescents affected by autism spectrum disorders. *Expert Review of Neurotherapeutics*, *16*(8), 867-874. https://doi.org/10.1080/14737175.2016.1211007

Roche, K. M., Ghazarian, S. R., Little, T. D., & Leventhal, T. (2011). Understanding links between punitive parenting and adolescent adjustment: The relevance of context and reciprocal associations. *Journal of Research on Adolescence, 21*(2), 448-460. https://doi.org/10.1111/j.1532-7795.2010.00681.x

Rodgers, J., Glod, M., Connolly, B., & McConachie, H. (2012). The relationship between anxiety and repetitive behaviours in autism spectrum disorder. *Journal of autism and developmental disorders*, *42*, 2404-2409. https://doi.org/10.1007/s10803-012-1531-y

Rodriguez, Geovanna, Sigan L. Hartley, and Daniel Bolt. "Transactional relations between parenting stress and child autism symptoms and behavior problems." *Journal of autism and developmental disorders* 49 (2019): 1887-1898. https://doi.org/10.1007/s10803-018-3845-x

Rosen, T. E., Mazefsky, C. A., Vasa, R. A., & Lerner, M. D. (2018). Co-occurring psychiatric conditions in autism spectrum disorder. *International Review of Psychiatry, 30*(1), 40-61. https://doi.org/10.1080/09540261.2018.1450229

Rosen, T. E., Spaulding, C. J., Gates, J. A., & Lerner, M. D. (2019). Autism severity, co-occurring psychopathology, and intellectual functioning predict supportive school services for youth with autism spectrum disorder. *Autism : the international journal of research and practice*, *23*(7), 1805–1816. https://doi.org/10.1177/1362361318809690

Rothenberg, W. A., Lansford, J. E., Bornstein, M. H., Chang, L., Deater-Deckard, K., Di Giunta, L., Dodge, K. A., Malone, P. S., Oburu, P., & Pastorelli, C. (2020). Effects of parental warmth and behavioral control on adolescent externalizing and internalizing trajectories across cultures. *Journal of Research on Adolescence, 30*(4), 835-855. https://doi.org/10.1111/jora.12566

Rutter, M., Le Couteur, A., & Lord, C. (2003). Autism diagnostic interview-revised. *Los Angeles, CA: Western Psychological Services, 29*(2003), 30.

Sameroff, A. (1975). Transactional models in early social relations. *Human Development, 18*(1-2), 65-79. https://doi.org/10.1159/000271476

Samson, A. C., Phillips, J. M., Parker, K. J., Shah, S., Gross, J. J., & Hardan, A. Y. (2014). Emotion dysregulation and the core features of autism spectrum disorder. *Journal of Autism and Developmental Disorders, 44*, 1766-1772. https://doi.org/10.1007/s10803-013-2022-5

Schaefer, E. S. (1965). A configurational analysis of children's reports of parent behavior. *Journal of Consulting Psychology, 29*(6), 552. https://doi.org/10.1037/h0022702

Seay, A., Freysteinson, W. M., & McFarlane, J. (2014). Positive parenting. *Nursing forum*, *49*(3), 200–208. https://doi.org/10.1111/nuf.12093

Serbin, L. A., Kingdon, D., Ruttle, P. L., & Stack, D. M. (2015). The impact of children's internalizing and externalizing problems on parenting: Transactional processes and reciprocal change over time. *Development and Psychopathology, 27*(4), 969-986. https://doi.org/10.1017/S0954579415000632

Shih, E. W., Quiñones-Camacho, L.,E., & Davis, E. L. (2018). Parent emotion regulation socializes children's adaptive physiological regulation. *Developmental Psychobiology, 60*(5), 615-623. https://doi.org/10.1002/dev.21621

Siegel, E. H., Sands, M. K., Van den Noortgate, W., Condon, P., Chang, Y., Dy, J., Quigley, K. S., & Barrett, L. F. (2018). Emotion fingerprints or emotion populations? A meta-analytic investigation of autonomic features of emotion categories. *Psychological Bulletin, 144*(4), 343. https://doi.org/10.1037/bul0000128

Sim, W. H., Jorm, A. F., & Yap, M. B. (2022). The role of parent Engagement in a Web-Based Preventive Parenting Intervention for Child Mental Health in Predicting parenting, parent and child outcomes. *International Journal of Environmental Research and Public Health*, *19*(4), 2191. https://doi.org/10.3390/ijerph19042191

Simonoff, E., Jones, C. R. G., Pickles, A., Happé, 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. https://doi.org/10.1111/j.1469-7610.2012.02600.x

Sinha, P., Calfee, C. S., & Delucchi, K. L. (2021). Practitioner's guide to latent class analysis: methodological considerations and common pitfalls. *Critical Care Medicine, 49*(1), e63. https://doi.org/10.1097/CCM.0000000000004710

Staton, L., El-Sheikh, M., & Buckhalt, J. A. (2009). Respiratory sinus arrhythmia and cognitive functioning in children. *Developmental Psychobiology: The Journal of the International Society for Developmental Psychobiology, 51*(3), 249-258. https://doi.org/10.1002/dev.20361

Strang, J. F., Kenworthy, L., Daniolos, P., Case, L., Wills, M. C., Martin, A., & Wallace, G. L. (2012). Depression and anxiety symptoms in children and adolescents with autism spectrum disorders without intellectual disability. *Research in Autism Spectrum Disorders, 6*(1), 406-412. https://doi.org/10.1016/j.rasd.2011.06.015

Sullivan, M. O., Gallagher, L., & Heron, E. A. (2019). Gaining insights into aggressive behaviour in autism spectrum disorder using latent profile analysis. *Journal of Autism and Developmental Disorders, 49*, 4209-4218. https://doi.org/10.1007/s10803-019-04129-3

Thompson, R. A. (2019). Emotion dysregulation: A theme in search of definition. *Development and Psychopathology, 31*(3), 805-815. https://doi.org/10.1017/S0954579419000282

Totsika, V., Hastings, R. P., Emerson, E., Lancaster, G. A., Berridge, D. M., & Vagenas, D. (2013). Is there a bidirectional relationship between maternal well-being and child behavior problems in autism spectrum disorders? Longitudinal analysis of a population-defined sample of young children. *Autism Research, 6*(3), 201-211. https://doi.org/10.1002/aur.1279

van den Boogert, F., Sizoo, B., Spaan, P., Tolstra, S., Bouman, Y. H. A., Hoogendijk, W. J. G., & Roza, S. J. (2021). Sensory processing and aggressive behavior in adults with autism spectrum disorder. *Brain Sciences, 11*(1), 95. https://doi.org/10.3390/brainsci11010095

van der Voort, A., Linting, M., Juffer, F., Bakermans-Kranenburg, M., Schoenmaker, C., & van IJzendoorn, M.,H. (2014). The development of adolescents' internalizing behavior:

Longitudinal effects of maternal sensitivity and child inhibition. *Journal of Youth and Adolescence, 43*, 528-540. https://doi.org/10.1007/s10964-013-9976-7

Van Hecke, A. V., Lebow, J., Bal, E., Lamb, D., Harden, E., Kramer, A., Denver, J., Bazhenova, O., & Porges, S. W. (2009). Electroencephalogram and heart rate regulation to familiar and unfamiliar people in children with autism spectrum disorders. *Child Development, 80*(4), 1118-1133. https://doi.org/10.1111/j.1467-8624.2009.01320.x

Van Lissa, C.,J., Keizer, R., Van Lier, P.,A.C., Meeus, W. H. J., & Branje, S. (2019). The role of fathers' versus mothers' parenting in emotion-regulation development from mid–late adolescence: Disentangling between-family differences from within-family effects. *Developmental Psychology, 55*(2), 377. https://doi.org/10.1037/dev0000612

van Steensel, F.,J.A., & Heeman, E. J. (2017). Anxiety levels in children with autism spectrum disorder: A meta-analysis. *Journal of Child and Family Studies, 26*(7), 1753-1767. https://doi.org/10.1007/s10826-017-0687-7

Vasa, R. A., & Mazurek, M. O. (2015). An update on anxiety in youth with autism spectrum disorders. *Current Opinion in Psychiatry, 28*(2), 83. https://doi.org/10.1097/YCO.0000000000000133

Veatch, O. J., Malow, B. A., Lee, H., Knight, A., Barrish, J. O., Neul, J. L., Lane, J. B., Skinner, S. A., Kaufmann, W. E., & Miller, J. L. (2021). Evaluating sleep disturbances in children with rare genetic neurodevelopmental syndromes. *Pediatric Neurology, 123*, 30-37. https://doi.org/10.1016/j.pediatrneurol.2021.07.009

Ventola, P., Lei, J., Paisley, C., Lebowitz, E., & Silverman, W. (2017). Parenting a child with ASD: Comparison of parenting style between ASD, anxiety, and typical development. *Journal of autism and developmental disorders*, *47*, 2873-2884. https://doi.org/10.1007/s10803-017-3210-5

Weiss, J. A., Thomson, K., & Chan, L. (2014). A systematic literature review of emotion regulation measurement in individuals with autism spectrum disorder. *Autism Research, 7*(6), 629-648. https://doi.org/10.1002/aur.1426

Weller, B. E., Bowen, N. K., & Faubert, S. J. (2020). Latent class analysis: a guide to best practice. *Journal of Black Psychology, 46*(4), 287-311. https://doi.org/10.1177/0095798420930932

Witwer, A. N., & Lecavalier, L. (2010). Validity of comorbid psychiatric disorders in youngsters with autism spectrum disorders. *Journal of Developmental and Physical Disabilities, 22*, 367-380. https://doi.org/10.1007/s10882-010-9194-0

Wolff, N., Stroth, S., Kamp-Becker, I., Roepke, S., & Roessner, V. (2022). Autism spectrum disorder and IQ–A complex interplay. Frontiers in Psychiatry, 13, 856084. https://doi.org/10.3389/fpsyt.2022.856084

Yang, S., Paynter, J. M., & Gilmore, L. (2016). Vineland adaptive behavior scales: II profile of young children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 46*, 64-73. https://doi.org/10.1007/s10803-015-2543-1

Zaboski, B. A., & Storch, E. A. (2018). Comorbid autism spectrum disorder and anxiety disorders: a brief review. *Future Neurology, 13*(1), 31-37. https://doi.org/10.2217/fnl-2017-0030

Zantinge, G., van Rijn, S., Stockmann, L., & Swaab, H. (2017). Physiological arousal and emotion regulation strategies in young children with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 47*, 2648-2657. https://doi.org/10.1007/s10803-017-3181-6

Zheng, L., Grove, R., & Eapen, V. (2019). Spectrum or subtypes? A latent profile analysis of restricted and repetitive behaviours in autism. *Research in Autism Spectrum Disorders, 57*, 46-54. https://doi.org/10.1016/j.rasd.2018.10.003

VITA

Personal Background	Deborah Eileen Rafferty Chino Hills, California Daughter of Kelly Rafferty and Margaret Rafferty One fur child - Luna Rafferty
Education	Diploma, Grace M Davis High School, 2008 Bachelor of Science, University of Utah, 2013 Bachelor of Art, University of Utah, 2013 Master of Science, University of Utah, 2016 Master of Science, Texas Christian University, 2020
Experience	Research Assistant, University of Utah 2013-2016 Graduate Research Assistant, Texas Christian University, 2018-2022 Research Scientist, Cook Children's Health Care System, 2022-present
Certifications	Certified Child Life Specialist

THE HETEROGENEITY OF AUTISM SPECTRUM DISORDER: AN EXAMINATION OF CHILD OUTCOMES AND PARENTING BEHAVIORS

by Deborah Eileen Rafferty, PhD, 2024

Department of Psychology

Texas Christian University

Dissertation Advisor: Naomi Ekas, PhD, Professor

Cathy Cox, Professor of Psychology

Danica Knight, Professor of Psychology

Chrystyna Kouros, Associate Professor of Psychology, Southern Methodist University

Uma Tauber, Associate Professor of Psychology

Background: Autism spectrum disorder (ASD) is a heterogenous neurodevelopmental disorder with defining characteristics of social communication/interaction challenges and restrictive, repetitive behaviors Many autistic children have difficulties with emotion regulation, which may lead to negative physiological and behavioral outcomes. Parenting behaviors may influence autistic children's emotion and behavior difficulties, which then may impact parenting behaviors in the future. Examining these processes may provide better insight into interventions that may benefit autistic children and their parents. Further, these associations may be moderated by the heterogenous presentation of ASD symptoms and IQ.

Method: Families (mother, father, and child) with an autistic child between the ages of 10 to 17 years were recruited to participate in a larger study. Participants completed a set of questionnaires at two time points six months apart. Child respiratory sinus arrythmia was also collected.

Results: Latent profile analysis indicated a three-profile solution: 1) Average IQ-Moderate SA-Low RRB, 2) Average IQ-Moderate SA-Moderate RRB, and 3) Borderline IQ-High SA-High SA. Profile 3 had the lowest anxiety levels compared to Profile 1 and 3. Moderated regression analysis indicated at high levels maternal warmth, autistic children in Profile 1 had lower depressive symptoms. At high levels of depressive symptoms, maternal and paternal warmth was lower for autistic children in Profile 3.

Discussion: Using latent profile analysis to determine if there were underlying subgroups in the population using IQ and ASD symptom severity as indicator variables, a three-profile solution was selected. Two of the profiles had average IQ with low to moderate symptom severity. The third profile had borderline average with higher ASD symptom severity. Results are consistent with prior research indicating the need to examine subgroups within ASD research. Child Effects and Parenting Effects models were partially supportive of a bidirectional relationship between parenting behaviors and child effect. More research is needed examining additional positive parenting behaviors.