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Family Factors as a Modifier of Individual Differences in Children with Higher Functioning Autism and Their Families

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FAMILY FACTORS AS A MODIFIER OF INDIVIDUAL DIFFERENCES IN
CHILDREN WITH HIGHER FUNCTIONING AUTISM AND THEIR FAMILIES

By

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FAMILY FACTORS AS A MODIFIER OF INDIVIDUAL DIFFERENCES IN
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<u>Family Factors as a Modifier of Individual</u>
<u>Differences in Children with Higher Functioning</u>
<u>Autism and Their Families.</u>

This study investigated the impact of family factors on individual differences in the social

and emotional development of children with autism and their families. Based on the

modifier model hypothesis suggested by Mundy, Henderson, Inge, and Coman (2007),

family factors may serve as a modifier that contributes to the variability in the phenotypic

presentation of children with higher functioning autism. Results indicated that Expressed

Emotion (EE) was associated with parent-reported hyperactivity and anxiety in children

and adolescents. Family cohesion was associated with parent-reported aggression and

depression. These results differed for typically developing and HFA children; higher EE

or lower cohesion was associated with greater impairment in the HFA group and less

impairment in the typically developing children. Family factors were not associated with

social symptoms, indicating these effects may be more related to the development of

comorbidity than to the core symptoms of autism. Expressed emotion was related

meaningfully to neutral attributions on the FMSS and provided validity for the measure.

Family factors were not associated with parental stress, which was not expected.

Implications for clinical interventions and future directions are discussed.

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Chapter 1: Introduction

Autism has risen to the forefront of the field of child development and its prevalence is higher than that of diabetes, cancer, or Down syndrome in children (Filipek et al., 1999; Centers for Disease Control and Prevention, 2009). Many areas are currently being investigated, including genetic and neurobiological risk factors, as well as assessment of the effectiveness of interventions and therapies. However, focus on one particular area is in its nascent stages; the study of family factors and how the bidirectional relations between the child and family may impact individual differences in the social and emotional functioning of children with autism and their families. Based on the modifier model hypothesis suggested by Mundy, Henderson, Inge, and Coman (2007), family factors may serve as a modifier that contributes to the variability in the phenotypic presentation of children with higher functioning autism. This is particularly thought to be true for individual differences in social and emotional development, more so than for the core symptoms of autism.

The investigation of family factors has received relatively little attention in research on autism possibly because autism is largely viewed as a biologically based disorder; therefore, research on the impact of environmental influences, including family factors has been minimal. However, family factors have been shown to have significant impact on the development and treatment of other biologically based forms of psychopathology, such as schizophrenia, and this may also be the case with autism. Although the role of family factors as primary in the etiology of autism has not been supported, it is possible that family factors moderate the expression of autism and relate to variations in both child and parent adjustment and well-being. It is additionally

important to explore this area because treatment strategies often call upon parents to act as interventionists. Therefore, a better understanding of family factors may be essential to accounting for the wide range of individual differences observed among higher functioning children with autism that currently complicate diagnosis and intervention. From the standpoint of better understanding the variation in individual differences in the social and emotional development of children with higher functioning autism, it is necessary to begin to investigate family factors more closely.

To begin to fill this gap in our understanding of family factors and individual differences in the development of children with autism, the goals of the current investigation were to examine the relations between family factors (i.e., family cohesion and expressed emotion) and: (1) comorbid internalizing and externalizing symptoms, (2) parental attributions about child behavior controllability, and (3) parental stress in a sample of higher functioning children with autism (HFA) and a matched comparison sample of children without autism. The following brief review is provided to better situate the study of family factors within the current state of knowledge of the broad phenotype of autism and its frequent comorbidity with other dimensions of psychopathology such as ADHD, Anxiety Disorders and Affective Disorders.

Autism

Autism is a complex neurodevelopmental disorder identified by impairments in three areas; social interaction, communicative language, and repetitive or stereotyped behaviors. According to the Diagnostic and Statistical Manual-Fourth Edition-Text Revision (DSM-IV-TR; American Psychiatric Association, 2002), a child meeting these criteria can be diagnosed with Autistic Disorder, Pervasive Developmental Disorder-Not

Otherwise Specified (PDD-NOS), or Asperger Disorder (AD). Variations in core symptom presentation and severity are present, as well as variations in intellectual and cognitive functioning. As much as there is variation in the behavioral presentation of individuals with autism, there is also significant variation in the cognitive profile. Approximately 41% of people with an autism diagnosis are characterized in the Impaired range of functioning (Centers for Disease Control and Prevention, 2009), and 59% of individuals are characterized as having "Higher Functioning Autism," which includes those with intellectual functioning measured to be equal to or above 70 (Centers for Disease Control and Prevention, 2009). Asperger Disorder and PDD-NOS can also fall under the HFA umbrella.

There is some debate between the diagnostic and symptomatic differences between HFA and Asperger Disorder, although the only clear-cut diagnostic difference is that to receive a diagnosis of AD there must be no history of language delay. Numerous researchers have attempted to clarify the cognitive profile of these two disorders, with limited success (Miller & Ozonoff, 2000). In essence, children who meet criteria for Asperger Disorder also often meet criteria for autism or high functioning autism (Macintosh & Dissanayake, 2004; Mayes, Calhoun, & Crites, 2001). Current prevalence of autism spectrum disorders, including diagnoses of Autistic Disorder, Asperger Disorder, and Pervasive Developmental Disorder- Not Otherwise Specified, are approximately 1 per 110 (Centers for Disease Control and Prevention, 2009), with some variation found across ethnicities. Five out of eleven monitoring sites identified higher prevalence rates for non-Hispanic White children than for non-Hispanic Black children. The prevalence of ASD is higher for boys than for girls, with 14.5 boys per 1000 and 3.2

per 1000 girls identified (Centers for Disease Control and Prevention, 2009). Fombonne (2003) reported the male/female ratio is 4:1 in higher functioning autism.

Current conceptualizations of autism suggest that it is a manifestation of atypical brain development, which is likely affected by both genetic and environmental factors (Bailey, Phillips, & Rutter, 1996). Twin studies provide support for genetic factors in the development of autism, with higher concordance in monozygotic than dizygotic twins as well as recurrence rates higher within families than the population (Santangelo & Tsatsanis, 2005). It is unlikely that the expression of autism symptoms is linked to a single gene. Instead, research suggests it may be polygenetic (Dawson et al., 2002). Dawson and colleagues suggested research on susceptibility genes for six potential traits involved in autism; face processing, social affiliation, motor imitation, memory, executive functioning, and language ability. With further work in these areas, it may be possible to elucidate gene-environment interactions such that increased early intervention targeting at-risk traits may change the developmental trajectory of the symptom presentation. With regard to the role of environmental factors, discordant MZ twin pairs indicate that some environmental factors may play a role as well (Dawson et al., 2002). While genes contribute to an individual's risk for autism, environmental factors may influence the expression of the particular genes (Newschaffer, 2006). Herbert et al. (2006) found support for the idea of autism being a genetic disorder whose presentation may be affected by how environmental factors alter brain development; these effects may be seen pre-, peri-, or possibly even post-natally.

Increasing attention has focused on the gene-environment interaction in autism to provide some explanation for the wide variation in core symptom presentation. It may be

that while gene-environment interactions have some responsibility for the primary symptoms of autism, a more specific person-environment interaction may affect the presentation of secondary symptoms (i.e., comorbidity) and overall levels of family stress. This may also be a reasonable explanation for the significant heterogeneity in symptom presentation and adaptation across children who carry the same diagnosis.

Comorbidity

A complicating factor in the diagnosis and treatment of children with HFA is the relatively higher rates of comorbid symptoms, including, but not limited to, anxiety, depression, ADHD, and conduct problems. Although the DSM-IV-TR (American Psychiatric Association, 2002) does not allow certain comorbid diagnoses with an autism or Asperger Disorder diagnosis, recent research has begun to investigate the presence of significant diagnostic features of externalizing and internalizing disorders in many children diagnosed with autism spectrum disorders. Symptoms consistent with a diagnosis of ADHD have been identified in 33-80% of children with PDD (Ehlers & Gillberg, 1993; Frazier et al., 2001; Goldstein & Schwebach, 2004; Leyfer et al., 2006). Children with comorbid PDD and clinical levels of attention problems experience higher rates of daily difficulty than do children with PDD alone (Goldstein & Schwebach, 2004; Holtmann, Bolte, & Poustka, 2007). Clark and colleagues found that even as young as age three to five years, children with PDD experience the most severe symptoms of comorbidity in comparison to ADHD, mood disorders, and ODD (Clark, Feehan, Tinline, & Vostanis, 1999). Yerys et al. (2009) explored the phenotypic characteristics of children with ASD and children with ASD and ADHD, and found the presence of ADHD exacerbated externalizing problems present in the ASD profile. This was consistent with

the modifier hypothesis presented by Mundy, Henderson, Inge, and Coman (2007) that certain within-individual processes (i.e., attention and executive functions) and environmental processes significantly modify the expression of ASD.

Exploring the shared characteristics of ADHD and ASD from an alternate perspective is equally informative. Characteristics of PDD, specifically difficulty in social interaction, are common in children with ADHD (Clark, Feehan, Tinline, & Vostanis, 1999). Reiersen, Constantino, Volk, and Todd (2007) found evidence of a relation between ADHD and autistic symptoms such that one-third of boys and three-fourths of girls with severe combined subtype ADHD met clinical cutoffs for autistic symptoms. Greene et al. (1996) found that 22% of a sample of children with ADHD were "socially disabled," while Clark et al. (1999) found that "a lack of awareness of feelings of others," was the most commonly reported autism symptom reported in their record review of children with ADHD.

With regard to anxiety and mood disorders, adolescents with Asperger Disorder have higher levels of anxiety than the general population and equivalent to those of typically developing adolescents diagnosed with anxiety disorders (Farrugia & Hudson, 2006). In comparing adolescent boys with Asperger Disorder to those with conduct disorder, high levels of anxiety and obsessions were present in Asperger Disorder, while depression and temper tantrums were common to both groups (Green, Gilchrist, Burton, & Cox, 2000). As this research indicates, children and adolescents with autism and Asperger Disorder are at risk for comorbidity and more serious psychopathology. In summary, similar to the findings on symptom overlap in ADHD and ASD, there are also higher levels of ASD symptoms in youths with mood and anxiety disorders. This finding

was particularly true above and beyond other confounding factors in children with mood disorders alone (Pine, Guyer, Goldwin, Towbin, & Leibenluft, 2008).

There are a few possibilities to explain the development of comorbidity in children with HFA. The first possibility is direct genetic transmission, without regard for environmental factors. In this pure genetic transmission model, we would assume that children develop comorbid psychiatric symptoms solely because of a genetic risk and not because of any gene-environment interaction. Significant research has occurred which investigates the psychiatric presentation of first-degree relatives of individuals with HFA. This construct, labeled as the "Broad Autism Phenotype (BAP)," represents the presence of sub-clinical levels of autism and related symptoms in the first-degree relatives of individuals with autism.

The second possibility for explaining the development of comorbidity in children with HFA is some combination of a genetic vulnerability and features in the environment that activate symptom presentation. It is possible then, that the family environment may play a role in this interaction. It may be that family factors provide a protective buffer against negative social experiences, or that family factors play some role that serves to exacerbate negative social experiences. That is, individual differences in susceptibility to adjustment problems and comorbid symptom expression are then potentially modified by the family environment and family factors.

Broad Autism Phenotype

Many studies have explored the presence of sub-clinical core or secondary symptoms consistent with a diagnosis of autism (e.g., social anxiety or language-based learning disabilities) in the first-degree relatives of individuals with autism. Essentially,

there may be an underlying genetic cause that may reveal itself to varying degrees in social, language, and behavioral functioning. The presence of the BAP concept has been confirmed through twin studies, which indicated concordance rates for autism in monozygotic twins to be 36-91% in comparison to 10% in dizygotic twins in the studies (Bailey et al., 1995; Folstein & Rutter, 1977; Steffenburg et al., 1989). Bailey et al. (1995) suggested these levels of concordance may indicate that autism has a heritability of > 90%. In fact, the rates of BAP found in multiple incidence autism families is higher than that of single incidence families, who in turn had greater rates than families with a child with Down Syndrome (Piven, 1999). Parents of a child with autism were found to be more aloof, rigid, anxious, and hypersensitive to criticism, and had fewer emotionally supportive friendships than parents of children with Down syndrome (Piven et al., 1997). With regard to specific psychiatric symptoms, elevated rates of affective disorders and social phobia were reported in relatives (Bailey, Palferman, Heavey, & Le Couteur, 1998; Bolton, Pickles, Murphy, & Rutter, 1998; Ghaziuddin & Greden, 1998; Micali, Chakrabarti, & Fombonne, 2004; J. Piven & Palmer, 1999). Further, Piven and Palmer (1999) found that, in multiple incidence families, depressive symptoms were present before the child's birth, indicating to some degree that the child's difficulties were not causing depression in the parent. In addition to affective disturbance, high rates of sub-clinical deficits in communication and executive functions in siblings and parents were reported (Hughes, Leboyer, & Bouvard, 1997; Ozonoff, Rogers, Farnham, & Pennington, 1993). These findings indicate that comorbid affective, attentional or behavioral disturbance could be explained by the presence of sub-clinical symptoms in the parents, who transmit the genetic risk directly to their child.

However, if a purely genetic transmission were responsible for the differences in HFA children's social and emotional functioning, one would expect the BAP theory to explain the individual differences in comorbidity. However, it is not the case that individual differences in social and emotional development are explained by the genetic risk, as not all children develop comorbidity, nor do all parents necessarily exhibit sub-clinical symptoms. Therefore, it is possible that other environmental factors, such as family factors, serve to explain the heightened, but variable, rates of comorbidity and child and parent adjustment problems.

Family Factors

Expressed emotion. A model involving environmental modifiers of the autism phenotype allows for a fuller explanation of the variable rates of comorbidity seen in HFA children (Mundy et al., 2007). One potentially important but infrequently examined modifier is the role family factors may play in the development of comorbid symptoms in autism. Very little research exists on the family system in autism (Sanders & Morgan, 1997). The research that does exist suggests that families of children with autism are not vastly different from families with a typically developing child. Rather the former families are stressed, they are also often characterized by positive and close relationships between a parent and their child with autism (Orsmond, Seltzer, Greenberg, & Krauss, 2006).

Hence, perturbations of family factors may not be so extreme to be viewed a singular "causes" of comorbid emotional or behavioral problems in autism. Nevertheless, it remains plausible, if not probable that the types of family factors that have been observed to modify symptom presentation and treatment responsiveness in other

neurodevelopmental disorders such as schizophrenia may also influence differences in autism. Prominent among these types of family factors is expressed emotion (e.g., Leff & Vaughn, 1985). Expressed emotion (EE), a construct developed to measure the family environment, may be a useful tool to begin to examine the variability in the behavioral adaptation of children, as well as parental adjustment. Expressed emotion was initially measured using the Camberwell Family Interview (Vaughn & Leff, 1976) and then by the Five Minute Speech Sample (FMSS; Magano-Amato, 1993). The FMSS asks the parent to speak about their child and their relationship with their child for five minutes, uninterrupted. The speech sample is coded for the presence of statements indicative of either criticism or emotional over-involvement toward a child. Using a coding matrix, frequency counts of positively and negatively-valenced statements, as well as more global attitudes about the child are summarized; the end result is a categorical rating of low or high expressed emotion. Notably, high expressed emotion is indicated by a high frequency of either positive, negative, or both positive and negative attitudes toward the child.

High expressed emotion, which is defined as either significant emotional overinvolvement, criticism, or both in a family system has been implicated in rates of relapse in neurobiological disorders, such as adult schizophrenia (Leff & Vaughn, 1985) and recovery in child depression (Asarnow, Goldstein, Tompson, & Guthrie, 1993). Although expressed emotion is a relatively new measurement tool in the field of child research, it is proving to be informative in conceptualizing the nature of family factors in relation to the parent-child relationship and child internalizing and externalizing problems.

Numerous studies have investigated the validity of the FMSS in non-clinically referred community samples. The FMSS has been found to relate to patterns of mother-child attachment security with High EE being associated with higher rates of disorganized attachment at age 6 (Jacobsen, Hibbs, & Ziegenhain, 2000), as well as suboptimal parenting behaviors including low levels of affection and high levels of direction during a play session with their preschool age child (Daley, Sonuga-Barke, & Thompson, 2003). More specific investigations of the criticism versus emotional overinvolvement domains have also been completed. The criticism dimension is more consistently related to child behavior than the emotional overinvolvement dimension (Peris and Baker, 2000). The presence of EOI is relatively more associated with internalizing disorders despite its limited consistency, whereas the presence of criticism is most consistently associated with externalizing disorders (Stubbe, Zahner, Goldstein, & Leckman, 1993; Vostanis, Nicholls, & Harrington, 1994). It may be that this is due to the more nebulous boundary of "good" versus "bad" emotional support in younger children and adolescents compared to adults (Wamboldt, O'Connor, Wamboldt, Gavin, & Klinnert, 2000).

Given the overall consistency of EE in relation to various maladaptive or disruptive problems, but the variation within these results, some researchers have attempted to clarify the nature of EE in the child and pediatric literature. In a study of children with chronic asthma, Wamboldt et al. (2000) found that the validity for the criticism scale used with children was well supported with regard to relations with observed parent-child interactions, parent report of family functioning, and increased behavior problems, although the emotional overinvolvement scale was not. Factor

analysis of the FMSS in a group of clinically referred children indicated a possible three-factor model of expressed emotion that included criticism, emotional overinvolvement and positivity (McCarty & Weisz, 2002). Their analyses indicated the criticism scale was most internally consistent and consistently related to externalizing behaviors, whereas the EOI scale was not internally consistent or consistently related to behaviors. Not surprisingly then the presence of criticism is most consistently associated with child outcome compared to emotional overinvolvement (McCarty, Lau, Valeri, & Weisz, 2004). Alternatively, the positivity scale, represented by the number of positive remarks made about a child, was associated with lower levels of externalizing and internalizing problems in children (Wamboldt et al, 2000). Hence, positivity during the FMSS may be a marker of family processes that buffer against the development of emotional and behavior problems. In summary, it appears that overall, criticism during the FMSS is a risk, and "positivity" may be relatively protective. The EOI scale overall appears to be relatively poorly understood at this point.

Further investigation on the subscales of the FMSS and their relation to child externalizing problems in clinically referred samples was completed. Criticism appears to be related specifically to externalizing behaviors. For example, Baker, Heller, and Henker (2000) reported that critical statements, but not emotional overinvolvement, was related to Externalizing Problems scale (broadband) on the Child Behavior Checklist in a sample of preschool aged children. Further support for the association between externalizing behaviors and EE was found in a study of family dynamics and girls with ADHD; high parental EE was associated with both ADHD and aggression, and the criticism scale showed stronger associations than did the EOI scale (Peris & Hinshaw, 2003). Parents

who were categorized as high on the criticism scale were more antagonistic in their interactions with their child, although the EOI scale was not related to observable behaviors (McCarty et al., 2004). Higher levels of maternal EE were related to a significant increase in risk for the child having a diagnosis of depressive disorder, substance abuse, or conduct disorder (Schwartz, Dorer, Beardslee, Lavori, & Keller, 1990).

Similar to findings of the relation between externalizing/ADHD symptoms, high EE was also associated with internalizing problems. Children with depressive disorders were more likely to have parents exhibiting high EE than both children with no symptoms and children with schizophrenia spectrum disorders (Asarnow, Tompson, Hamilton, Goldstein, & Guthrie, 1994).

To date, two studies explored expressed emotion specifically in families of individuals with autism. Orsmond et al., (2006) observed that mothers of an adult child with autism self-reported positive relationships with their child and 90% reported high levels of affection for the child. Higher levels of mother-reported positive affect were associated with lower levels of criticism and higher levels of warmth, both rated via the FMSS, whereas maternal over-involvement was associated with poorer language and more health limitations in the child. Although the core symptoms of autism (e.g., impaired communication or social reciprocity) were not related to positive affect, criticism, or warmth, the child's maladaptive behaviors were related to these dimensions; higher maladaptive behaviors were related to less positive affect, higher criticism and less warmth (Orsmond et al., 2006). This paper nicely relates to the modifier model presented

in this study and provides some support for variations in behavior as a result of family factors.

In a longitudinal study of individuals with autism, higher levels of expressed emotion were predictably related to increased severity and intensity of maladaptive, internalizing, and externalizing behaviors. Weaker effects were observed for symptom presentation (Greenberg, Seltzer, Hong, & Orsmond, 2006). Given the relatively low number of families exhibiting high expressed emotion, the researchers explored the possible explanations for this finding. One mechanism explored was the role of parental attributions about their child's level of control over maladaptive, internalizing, and externalizing behaviors and posited that if the mothers believed their child's behaviors were due to factors beyond their control, this minimized the criticism the child may receive (Greenberg, Seltzer, Hong, & Orsmond, 2006).

Our own preliminary research on family factors also provided a number of interesting findings with respect to children with autism. We examined family functioning in a sample of 37 families of children and adolescents with HFA. Overall, parents of HFA children did not express significantly different attitudes about their children, as measured via the component scales of the FMSS, than did parents of comparison children, with the exception of making fewer positive remarks on the FMSS. Consistent with other work in the field, high EE was relatively rare and found in only 11% of families (4 out of 37 families). Statements of attitude and the presence of negative initial statements on the FMSS were negatively correlated with the self-report of interpersonal peer relations (Zahka, 2005).

In considering the results of this study it is important to recall that higher levels of EE may be more likely to be expressed in families of children who display externalizing behaviors and symptoms of ADHD. Given that research suggests that some higher functioning children may also express ADHD symptoms and associated externalizing behaviors (Goldstein & Schweback, 2004; Leyfer et al., 2006), it is likely to be important to examine the possibility that the influence of EE related family factors in families of children with autism is associated with, or conditional on the presence of ADHD comorbidity.

Cohesion. In addition to measuring family factors via expressed emotion, or attitudes about a child, it is also worthwhile to examine the family environment in general, and not specific to an identified child. It may be that family closeness has either a protective or a detrimental effect on the presentation of comorbidity in children with autism. In a study of families of children with Fragile X, family cohesion was protective for the child in terms of lower levels of problem behaviors, and mothers felt less stressed when they were able to rely on family for social support (Johnson, Cohen, Kasen, Smailes, & Brook, 2001). Etiology of the child's disorder seems to play a role; families of a child with a disorder of known etiology (e.g., Down syndrome) reported higher family harmony, whereas families of a child with a disorder of unknown etiology (e.g., autism) reported lower levels of family harmony, defined as a combination of family cohesion and expressiveness. However, it should be clearly noted that levels of family harmony reported in families of a child with a developmental disability are close to the norm of non-distressed families (Perry, Harris, & Minnes, 2005). It may be that lack of clarity in the etiology of autism plays a role; parents may have more difficulty with, or

perceive child control of disruptive behaviors, for example, when they do not see a clear reason (e.g., genetic cause) for the behavior to occur. This is especially true for parents of a child with higher functioning autism, as there is significant variation not only in core symptom presentation, but also in secondary comorbidity. Research in our own laboratory revealed that higher cohesion was positively related to parents' observation of social skills, internalizing tendencies and social anxiety in children with HFA (Zahka, 2005). It may be that high cohesion in this group was protective against more disruptive behavior, or that this was a group who tended toward internalizing symptoms, and this created a better fit within the family system. Although more or less positive family environments may be associated with differences in social-emotional and behavioral outcomes for all children, the process behind this association is not yet clear. It may be that family environments are more positive when there is a relative fit between parents and children. In thinking about the BAP literature, a parent may have sub-clinical social anxiety, for example, and may have a more positive relationship, and by extension more positive family environment, when their child is more socially anxious as well. An alternative explanation is that the way parents think about their child's behavior may be impacted by the family environment; that is, parents may attribute more or less control to the child depending on the family environment. It may be that attribution theory can provide some explanation.

Attribution

When a causal attribution is made, concepts to be considered include perceived locus of event (internal or external), stability of the cause (stable or unstable), and controllability of the event (controllable or uncontrollable; Weiner, 1986). On a more

global level relating to beliefs about the cause of a child's autism diagnosis, mothers who reported higher levels of personal control after their child's diagnosis with an autism spectrum disorder also reported higher levels of depressed affect (Dale, Jahoda, & Knott, 2006). The authors' qualitative analyses indicated these mothers seemed to be dealing with a high level of personal responsibility for assisting their child. Given the broad scope of attribution theory, we are limiting our focus to the controllability of behavior for the purposes of this study. Fong (1991) found that parents of a child with autism vary in their attribution of a child's intentionality with regard to problem behaviors depending on the level of stress in the family. Parents classified as high stress were more likely to make negative appraisals about behavior, and exhibited more negative emotional reactions. This contributes to the idea that family factors may have some relation to parent stress. Looking specifically at parents' attributions about their own child's behavior revealed parents rated inattention as the least controllable in comparison to pro-social behaviors and oppositional behaviors that were rated as the most controllable. The co-occurrence of pro-social behaviors and disruptive behaviors affected maternal attributions; pro-social behaviors following negative oppositional behaviors were rated less positively (Freeman, Johnston, & Barth, 1997).

The role of perceptions of behavioral controllability has been investigated within the expressed emotion construct specifically. In a study investigating adults with schizophrenia and depression, Hooley and Campbell (2002) found high EE relatives attributed more control to the identified family member than did low EE relatives. The family members identified as high EE also behaved in a more controlling way. In contrast, Hooley and Licht (1997) found no associations between attribution style and

clinical outcome at nine months in a study of adults with schizophrenia. These studies either used or modified Weisman's attribution coding system for the FMSS, in which negative, positive, or neutral codes were assigned to incidental statements of behavioral attribution in the speech sample (Weisman, Lopez, Karno, & Jenkins, 1993; Weisman, Nuechterlein, Goldstein, & Snyder, 1998).

Extending on a line of research in our lab, Coman (2006) sought to provide some explanation for the patterns of relation between family factors and child social and emotional development. After developing a modified attribution coding system for the FMSS based on Weisman et al. (1998), preliminary findings indicated that parents made a greater number of negative attributions when their children with HFA displayed externalizing problems (i.e., report of hyperactivity, conduct problems, and attention problems; Coman, 2006).

These data provide an interesting pattern of results and it may be that, as posited by Greenberg et al. (2006), the level of control the parent believes their child has over behavior is related to negative or positive reactions to a child, and on a more global level, the family climate. It may be that children with HFA whose parents perceive them to have a higher level of behavioral control are at greater risk for family distress.

Minimal work has been completed in the area of perceptions of behavioral controllability and expressed emotion in the area of autism. In addition to the attributional coding derived from the FMSS, it may additionally be useful to have an independent measure of perceived behavioral control of core symptoms and comorbidities. This would allow for attributions related to expressed emotion, but also a more objective measure of the parents' beliefs about the controllability of their child's behavior.

Stress

Another possible factor to be considered in understanding the role of family factors in variability in symptom expression in autism is stress within the family may affect or be affected by expressed emotion or cohesion. Stress in families of children with autism may be brought on by many variables including variability in child symptom intensity, parental emotional and mental health status, and negative life events independent of the diagnosis of autism in a child in the family. Stress, child, parent, and life event variables may be expected to interact in their associations with family factors. For example, family cohesion may mitigate the effects of life event stress or in some cases life event stress may increase the potential for higher EE or lower cohesion. It may also be that parents who are more vulnerable because of external negative life events or internal psychopathology, may have less tolerance for problematic of disruptive behaviors in their children, leading to higher EE and lower cohesion. Just as it is difficult to examine effects associated with family factors without considering stress, it is also difficult to disentangle causal relations between family factors and sources of stress.

Mothers report higher levels of stress, especially in the areas of dependency, cognitive impairment, family opportunity, and physical illness, when their child has a chronic mental disorder (i.e., autism) than do mothers of a child with a chronic medical disorder (i.e., cystic fibrosis; Bouma & Schweitzer, 1990). Maternal stress also tends to be higher overall for mothers with a child in the PDD spectrum especially when their child displays maladaptive or disruptive behaviors (i.e., irritability or oppositionality), difficulty communicating, and social withdrawal (Tobing & Glenwick, 2002; Tomanik, Harris, & Hawkins, 2004). In fact, Baker et al. (2000) found that stress had a moderating

effect on expressed emotion in a family. Researchers found that the relationship between EE and child behavior problems was moderated by maternal stress and adjustment; maternal stress was better at predicting child behavior problems than EE status was.

With regard to more internally-based stressors, such as parental psychopathology, research has indicated a mediating role of negative parental attitudes on the relation between parent psychopathology and child psychopathology (Johnson et al., 2001). Further complicating the interaction are the child's coping strategies and their own attributions about parent behavior (Langrock, Compas, Keller, Merchant, & Copeland, 2002; Scherer, Melloh, Buyck, Anderson, & Foster, 1996). A bidirectional relation exists between parent and child pathology such that a parent's mood can affect child behavior (Dawson et al., 2003) as well as the effect of child behavior problems specifically associated with autism that affect parental mood (Lainhart, 1999). With regard to children diagnosed with ADHD, maternal depression predicted more conduct problems longitudinally, while positive parenting during a structured interaction predicted fewer problems (Chronis et al., 2007). A relatively large-scale epidemiological study with a community-based sample indicated a relation between parental psychopathology and punitive parenting but cautioned that the relation between parental psychopathology and child psychopathology is actually quite complex and non-linear (Vostanis et al., 2006).

Not all children with HFA are equally susceptible to the family environment modifying their social and emotional development or their behavior. There may be many factors related to susceptibility. Given the recent research on the presence of comorbid ADHD symptoms being related to less optimal functioning in multiple areas for children with both HFA and ADHD, one possible factor that requires further exploration is the

presence or absence of ADHD symptoms and its relation to family factors. One possibility is that family distress puts children with autism at greater risk for exhibiting disruptive or externalizing behaviors. In addition, this risk may be due to differences in parental beliefs about the degree to which children can exert control over the problematic behaviors. Finally, it may also be the case that family factors, such as higher levels of expressed emotion, are not only linked to externalizing problems in children with ASD but also how much life stress parents experience.

Current Proof of Principle Study

To definitively examine such a model, a large-scale longitudinal investigation would be needed to clarify the nature and direction of these relations. However, there is currently too little empirical information on family factors in autism to warrant the inception of a project with such high resource demands. What is needed is an intermediate "proof of principle" step to document that specific, expected associations exist between family factors (EE, family cohesion), child characteristics (ADHD symptoms), parental perceptions of child behavior control, and parental life stress in research with children with higher functioning autism. The study undertaken in this dissertation was designed to provide this type of intermediate step using a cross-sectional design and moderate sample sizes to test a-priori hypotheses about *some* of the more important relations between family factors and the development of children with HFA.

Three hypotheses were explored in this study. Hypothesis 1 stated that HFA and matched comparison children are expected to differ on ADHD and associated externalizing behavior problems, with the HFA group displaying greater symptoms of ADHD and externalizing problems. This relation is also predicted to be associated with

family factors (i.e., expressed emotion and family cohesion), such that diagnostic group differences in externalizing behaviors will be particularly pronounced (i.e., conditional) on the presence of higher EE or lower cohesion in the family. Based on the literature on EE and the modifier model of autism, the relation of family factors is predicted to be unique to externalizing behaviors, and not evident with internalizing symptoms or core social symptoms.

Second, it was hypothesized that groups will differ on parents' perceptions of their child's behavior, including perceptions of the child's level of control over his/her behavior and types of attributions about their child's behavior. Parents of children in the HFA group are predicted to report lower behavioral controllability. The relation between diagnostic group and perceptions of the child's behavior is expected to be moderated by family factors (i.e., expressed emotion and family cohesion). It is predicted particularly for children in the HFA sample, in the presence of high EE or low cohesion, parents will report a perception of greater behavioral control and a greater number of negative attributions.

Third, it was hypothesized that diagnostic groups will differ on measures of parents' experiences of exogenous and endogenous stress, with the parents of children in the HFA group reporting higher stress. The relation between diagnostic group and report of stress is predicted to be moderated by family factors (i.e., expressed emotion and family cohesion). It is predicted that higher EE or lower cohesion will be associated with higher parent stress. It is also predicted that diagnostic group differences in parent stress will be particularly pronounced in the presence of higher EE or lower cohesion.

Chapter 2: Method

Participants

Children and adolescents between the ages of 9 and 16 years of age who had a diagnosis of HFA, AD, or PDD-NOS were recruited from the University of Miami Center for Autism and Related Disabilities database. Letters were sent to parents describing the research project, asking for the participation of one child and one parent, as well as indicating the compensation to be received. Interested parents were encouraged to contact the research coordinator by phone or e-mail. Children who had complicating medical diagnoses (i.e., neurological impairments, genetic disorders) were excluded.

A comparison sample was recruited using a variety of methods. Children and adolescents between the ages of 9 and 16 years of age who are typically developing (i.e., no history of diagnosed learning, attention, or developmental disorders) were recruited from local public schools. Letters were sent to parents of students in the Miami-Dade school district who were in grades two through eleven describing the research project, asking for the participation of one child and one parent, as well as indicating the compensation to be received. Interested parents were encouraged to contact the research coordinator by phone or e-mail. Children who had complicating medical diagnoses (i.e., neurological impairments, genetic disorders) were excluded.

In order to attempt to match fro variability in comorbid symptom presentation within the comparison sample relative to the HFA sample, children and adolescents between the ages of 9 and 16 years of age who have a diagnosis of ADHD, LD, and/or an Anxiety Disorder were recruited into the comparison sample from the University of Miami Psychological Services Center. Letters were sent to parents of children and

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adolescents who have been evaluated at the center since 2003 describing the research project, asking for the participation of one child and one parent, as well as indicating the compensation to be received. In addition, local clinical psychologists specializing in working with children were asked to place pamphlets in their waiting rooms. Interested parents were asked to contact the research coordinator by phone or e-mail. Children with complicating medical diagnoses (i.e., neurological impairments, genetic disorders) were excluded.

Participants are all part of a larger study on individual differences in social-emotional development of children with higher functioning autism. The initially recruited HFA group ($n = 64$) was comprised of children diagnosed with Autism Spectrum Disorders without mental retardation (i.e., High Functioning Autism, Asperger Disorder, or Pervasive Developmental Disorder-Not Otherwise Specified). Five males with HFA who were initially recruited for this study were not included in the analyses because their WISC-IV VCI scores did not meet the criterion of 70. Two males with HFA who were diagnosed with a genetic disorder (22q11 deletion syndrome) were excluded from the analyses. One girl with HFA reportedly had a comorbid psychotic disorder and was excluded. Thus, the remaining HFA group included 56 participants (6 girls). In the HFA group, parent participation was divided as follows: mothers ($n = 53$) and fathers ($n = 3$). The comparison group ($n = 49$) was comprised of children with typical development and children with community-diagnosed Attention Deficit Hyperactivity Disorder (ADHD) or anxiety disorders. One female dropped out of the study due to language difficulties. Thus, the final comparison group included 48 children (37 male, 11 female; 40 TD, 83%, 6 ADHD, 12.5%, 2 anxiety, 4.5%).

Measures

Autistic symptoms. The Autism Spectrum Screening Questionnaire (ASSQ; Ehlers, Gillberg, & Wing, 1999) and Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003) were used to verify community diagnoses of autism spectrum disorders. To be retained in the HFA sample, participants were required to have a score greater than or equal to 13 on the ASSQ and the SCQ. For children in the comparison sample, their scores on the ASSQ and SCQ were required to be 12 or lower. The Autism Diagnostic Observation Schedule (ADOS; Lord, Rutter, DiLavore, & Risi, 1999) was used as a group differences measure only. The total scale from the Social Responsiveness Scale (SRS; Constantino, 2004) was utilized as a continuous individual difference measure of social symptoms and not as a diagnostic/symptom verification measure.

Autism Spectrum Screening Questionnaire (ASSQ; Ehlers, Gillberg, & Wing, 1999)

The ASSQ is a brief 27-item screening questionnaire used to identify symptoms associated with either Asperger Disorder (AD) or other high-functioning autism spectrum disorders in children and adolescents of normal intelligence or mild mental retardation. A cutoff score of 13 was used to verify diagnosis, as this is recommended for identifying those individuals who meet criteria for the disorder (Ehlers et al., 1999).

Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003)

The SCQ is a 40-item parent-report screening instrument that assesses autistic symptoms in three domains: communication, reciprocal social interaction, and restricted, repetitive and stereotyped patterns of behavior. The SCQ items are derived from the 40 (of 93) ADI-R items with the highest discriminant diagnostic validity. The questionnaire is presented in a yes/no format and assesses lifetime prevalence of symptoms (i.e.,

whether the behavior currently occurs or occurred during ages 4-5). A cutoff score of 13 has appropriate sensitivity to identify children who demonstrate symptoms consistent with a diagnosis of autism (Rutter et al., 2003).

Autism Diagnostic Observation Schedule- Generic (ADOS; Lord, Rutter, DiLavore, & Risi, 1999)

The ADOS is a semi-structured instrument that assesses social interaction, communication, and imaginative play. It is used in the assessment of individuals who may have an autism spectrum disorder. The goal of the ADOS is to provide a structured, standardized environment while also eliciting social communication, play and behaviors in a natural setting. One of four modules is used, depending on age and level of communicative language. This study utilized Modules 3 and 4, which require fluent speech and contain activities suitable for a child, adolescent, or young adult. The ADOS administration was coded by four trained graduate students via a standard scoring algorithm (Lord et al., 1999). Graduate students were trained in scoring by a primary coder who was reliable based on the requirements of the training program at the University of Michigan Autism and Communication Disorders Centers. The ADOS also is coded for repetitive behaviors, although this is not used in the diagnostic classification score. In the original normative sample, intraclass correlations ranged from .88 to .97 in the social domain, .74 to .90 for the communication domain, .84 to .98 for the Total social-communication score, and .75 to .90 for the repetitive behaviors domain. Inter-rater agreement was highest for autism vs. non-autism diagnoses (90-91% for Modules 3 and 4), but lower when PDD-NOS individuals were included (81-84%; Lord et al., 2000).

Social Responsiveness Scale (SRS; Constantino, J.N., 2004)

The SRS is a 65-item rating scale that assesses the severity of symptoms associated with Autism Spectrum Disorders in social settings. It is completed by the parent about the child using a five-point Likert-style scale, and provides continuous cores on the following dimensions: social impairments, social awareness, social information processing, reciprocal social communication, social anxiety, and autistic preoccupations. The Total Score was utilized as an index of impairment related to social communication difficulties in this investigation. Standardization data is based on national sample of 1600 children ages 4 through 18 from the general population. Internal consistency ranged from .93 to .97. Validity of the SRS when compared to the ADI-R algorithm scores was 0.7 (Constantino et al., 2003).

Intelligence.

Wechsler Intelligence Scale for Children-Fourth Edition (WISC-IV; Wechsler, 2003)

Four subtests from the WISC-IV were administered including Vocabulary, Block Design, Matrix Reasoning, and Similarities. These four subtests were chosen for their low standard error of the measure as well as their high loading on the factor scales (Wechsler, 2003). An estimated Verbal Comprehension Index and Perceptual Reasoning Index were calculated.

Parent report of child emotional and behavioral functioning.

Behavior Assessment System for Children-Second Edition-Parent Rating Scales (BASC2 PRS; Reynolds & Kamphaus, 2004)

The BASC2 PRS is a broadband parent report questionnaire used to assess a parent's perception and observation of their child's behaviors, emotions, and adaptive

skills. The questionnaire asks the parent to indicate how often a particular behavior occurs (never, sometimes, often, almost always). Two forms were used, depending on the age of the child (age 6-11) or adolescent (age 12-21). Normative data is based on a large, representative sample according to age, gender, and clinical status. The BASC2 includes validity scales. Dimensions of interest in the current study were: Attention Problems, Aggression, and Hyperactivity, which addressed externalizing problems, and Anxiety, Depression, and Withdrawal, which addressed internalizing problems. Test-retest reliability (over what period of time) as listed in the manual, yielded correlations in the .80s for composite scores and .70 to .80 for individual scales. Validity in comparison to other broadband measures (e.g., Achenbach Child Behavior Checklist) ranged from .70 to .80 and in comparison to the previous version of the Behavior Assessment System for Children was approximately .90. Norm-referenced T-scores (based on age and gender) were obtained for each scale.

Parent self-report measures of stress and psychological functioning.

Endler Multidimensional Anxiety Scales (EMAS; Endler, Edwards, & Vitelli, 1991)

The EMAS was designed to assess three different types of anxiety: State, trait, and perception of threat. For the purposes of this study, the EMAS-T was used to investigate trait anxiety in parents. The EMAS-T is a self-report measure that provides scales assessing levels of anxiety in evaluative, physically dangerous, and new situations, and daily routines. Level of distress is rated on a 5-point Likert scale from 1 (not at all) to 5 (very much). Four trait anxiety scores are provided: Social evaluation, physical danger, ambiguous, and daily routines. The Social Evaluation variable was used in this investigation to index social anxiety. Detailed information on the internal consistency and

test-retest reliability, in addition to construct and criterion validity, are reported in the manual. Norm-referenced T-scores based on age, gender, and education were obtained for the scale.

Symptom Checklist 90-Revised (SCL-90-R; Derogatis, 1994)

The SCL-90-R is a 90 item self-report inventory that measures the level of distress experienced from psychological symptoms in the past seven days. Level of distress for each symptom is rated on a Likert scale ranging from 0 (no distress) to 4 (extremely distressed). Nine primary symptom dimensions representing internalizing and externalizing symptoms are assessed as well as three broad indices of distress. The nine dimensions are: Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism. Three composite scores are calculated: Global Severity Index (GSI), which measures overall psychological distress, Positive Symptom Distress Index (PSDI), which measures the intensity of symptoms, and Positive Symptom Total (PST), which measures the number of reported symptoms. For the purposes of this study, the Global Severity Index was utilized as a summary measure of psychological distress in parents. Reliability and validity are adequate and are discussed in detail in the manual. Norm-referenced T-scores based on age and psychiatric status (i.e., non-clinical status) were obtained for each scale.

Life Stressors and Social Resources Inventory-Adult Form (LISRES-A; Moos & Moos, 1994b)

The LISRES-A is a comprehensive questionnaire measuring current level of stressors, change over time, and level of social resources. An abbreviated form of this inventory was used in this study, which included only the items that loaded on the Negative Life Events scale in the areas of Physical Health, Spouse/Partner, Finances,

Work, Home/Neighborhood, Children, Friends and Social Activities, and Extended Family). Participants were asked to respond yes or no to the presence or recent presence (within the last twelve months) of negative events in these domains. Positive (i.e., "yes") answers were summed to get a total score, which was then transformed to a standard score based on normative data in the manual. Standardized normative data (i.e., T-scores) were based on a national group of 1, 884 adult men and women.

Leyton Obsessional Inventory (LOI; Cooper, 1970)

The Leyton Obsessional Inventory, originally written in 1970, was an inventory of 69 questions investigating obsessional traits and symptoms. A card sorting method was used, where cards printed with each question were placed in yes or no boxes. Questions that were answered yes were then rated on the level of resistance from 1 (sensible) to 5 (try very hard to stop) as well as how much the trait or symptom interfered from 1 (no interference) to 4 (interferes a great deal) by pointing to cards laid out on the table. Due to time constraints, an updated self-report version based on a comparison study of the original version and a new written version was devised (Snowdon, 1980). The self-report version described by Snowdon will be used in this study due to its ease of administration. Scores obtained were not significantly different based on version administered. The LOI provides four scales for interpretation; total number of obsessional symptoms, obsessional traits, level of resistance, and level of interference. Cronbach's alpha indicated adequate internal consistency for the four LOI scales (Symptom = .88, Trait = .75, Resistance = .88, Interference = .90). Convergent validity is good, with the LOI Symptom, Resistance, and Interference scales correlating positively with the SCL-90-R Obsessive-Compulsive Scale. Further, Symptom, Resistance and

Interference scores were higher in the OCD group than the non-OCD group. No difference was noted in trait scores (Stanley et al., 1993).

Family factors.

Family Environment Scale (FES; Moos & Moos, 1994a)

The FES is true or false questionnaire measuring varying aspects of the family environment, including commitment, support and help provided for family members. For the purposes of this study, the Cohesion scale, consisting of ten items, was used to assess global aspects of the entire family system. A total score is obtained (range 1-9) and transformed to standardized scores presented in the normative manual (Moos & Moos, 1994a). High scores represent higher family cohesion. Internal consistency for the Cohesion subscale is .78 based on the normative data provided in the manual.

Five Minute Speech Sample (FMSS; Magano-Amato, 1993)

The FMSS is a brief method of measuring family process through the construct of expressed emotion. The participating parent is read the following directions verbatim: "*I'd like to hear your thoughts and feelings about (child's name), in your own words and without my interrupting you with any questions or comments. When I ask you to begin I'd like you to speak for five minutes, telling me what kind of person (child's name) is and how the two of you get along. After you begin I'd prefer not to answer any questions until after the five minutes.*" The instructions and speech sample were recorded using a portable audio recorder. If the parent continued speaking after the five minutes, only the first five minutes were coded.

The speech sample was rated as high or low in expressed emotion based on the presence of critical or emotionally over-involved statements. Further, subgroup ratings

were assigned based on more specific ratings of the initial statement or relationship, frequency count of critical statements, positive remarks, or statements of attitude, and the presence of dissatisfaction, an emotional display, self-sacrificing or overprotective statements, or excessive detail. In order to establish reliability for the coding of the Five Minute Speech Sample (FMSS), previously collected speech samples (not part of this study) were coded by a graduate student in the lab and by independent coders, one of whom had previously established inter-rater reliability with a primary researcher experienced in the use of FMSS. Speech samples were randomly selected from two groups (HFA; n=22 and typically-developing; n=12). Reliability was well established and single measures intraclass coefficients for the continuous measures were as follows: Critical statements $\alpha = 1.00$, Statements of Attitude $\alpha = .87$, and Positive remarks $\alpha = .85$. The subgroup score, which essentially acts as a continuous variable, had an intraclass coefficient of $\alpha = .77$. The reliability of the scoring of the categorical measures was evaluated using crosstabs and the results are as follows: Initial Statement kappa=.76, Relationship kappa=.82, Dissatisfaction kappa=.68, Emotional Display kappa =1.00, and EE group kappa = .87. Reliability analysis for the dimension of excess detail was unavailable because none of reliability samples contained instances of this. Based on use of the FMSS in the literature, the measures exhibited test-retest reliability, as well as concurrent and predictive validity in numerous studies with children (McCarty & Weisz, 2002).

Subgroup ratings were determined in the following manner: "Critical" was assigned if a negative initial statement, negative relationship, or ≥1 critical statements were present. "EOI" was assigned if self-sacrificing/over protective behavior or an

emotional display was present during the speech sample. "EOI" was also coded if two of the following were present: excess detail, ≥1 statement of attitude, or ≥5 positive remarks. "Borderline critical" was assigned if dissatisfaction was present in the speech sample. "Borderline EOI" was assigned if ≥1 statement of attitude, borderline self-sacrificing/over protective behavior, excess detail, or ≥5 positive remarks were present in the speech sample. "Low" was assigned when none of the former criteria were reached. Speech samples were then classified as High if their subgroup rating was Critical or EOI, and Low if their subgroup rating was Low, Borderline Critical or Borderline EOI. The classification of high or low expressed emotion was used as main dependent variable in this investigation.

Perceptions of behavioral controllability and attribution.

Controllability of Behavior Questionnaire (CBQ)

Designed for this study to measure parents' perceptions of their child's behavioral control, the questionnaire included twelve behaviors, six theoretically associated with autism and six theoretically associated with common comorbid symptoms. Parents rated the frequency with which each behavior occurred; the degree to which the parent felt (a) the child could control their own behavior and (b) they could control their child's behavior. Ratings were made on a five-point Likert scale, with 0 indicating never to 4 indicating all of the time. A principle component analysis of the Child Symptom Control (CSC) scores yielded one factor score based on Kaiser's criterion of retaining factor scores with eigenvalues greater than 1. The factor score explained 55.15% of the variance. Similarly, the Parent Symptom Control (PSC) scores yielded one factor score based on the same criteria and explained 50.26% of the variance. The Kaiser-Meyer-

Olkin measure of sampling adequacy had a value of .91 and Bartlett's Test of Sphericity was significant, χ^2 (66) = 554.58, p <.001 for the CSC variable. The Kaiser-Meyer-Olkin measure of sampling adequacy had a value of .90 and Bartlett's Test of Sphericity was significant, χ^2 (66) = 479.34, p <.001 for the PSC variable. The KMO statistics indicated correlations are not diffuse, and factor analysis theoretically yields distinct and reliable factors. Values greater than .8 are generally accepted as excellent (Hutcheson & Sofroniou, 1999). The CSC and PSC variables were negatively correlated with BASC SRP Locus of Control, r (79) = -.43, p < .001; r (80)= -.31, p < .01, respectively), with higher CSC and PSC scores being associated with a less external, or more internal, locus of control, supporting the validity of the measure as an index of controllability. The BASC2 SRP Locus of Control scale was used in earlier investigations in this lab and provided valid child self-report (Zahka, 2005).

FMSS Attribution (Coman, 2006)

In addition to the traditional coding of the FMSS, attributional statements were coded from the FMSS based on a modified attribution coding system (Coman, 2006). Statements were defined as attributions when "the parent attributes their child with a particular characteristic, behavior, belief, emotion, or motivation as being the cause of a particular event/outcome that affects the child himself/herself or his/her kin." (e.g., He has no friends because he acts strangely; She is good in school because she tries so hard). Scores yielded were the raw number of positive, negative, and neutral attributions. Reliability was established with two coders and single measures intraclass coefficients for the measures were as follows: positive attributions α = .72, negative attributions α = .94, neutral attributions α = .85.

Procedure

Families were scheduled for three two-hour sessions in the Social Development Lab in the Department of Psychology at the University of Miami. During the first session, the accompanying parent was asked to sign an IRB-approved informed consent form, while the child was asked to sign an IRB-approved informed assent. In addition, the Health Insurance Portability and Accountability Act (HIPAA) form was presented to parents and explained. Families were given $40 for each completed session. A trained graduate student gave participants an abbreviated form of the Wechsler Intelligence Scale for Children- Fourth Edition. Following, the child was administered the Autism Diagnostic Observation Schedule (ADOS). The child also completed a number of self-report measures of social and emotional development. Other cognitive and social-emotional measures, as well as two electroencephalograms, were administered during these three sessions as part of a larger investigation.

The accompanying parent completed several questionnaires assessing the child's behavior, symptom expression and social communication. Of interest in the current study were: the Behavior Assessment System for Children- 2nd Edition- Parent Report (BASC2-PRS; Reynolds & Kamphaus, 2004), Social Responsiveness Scale (SRS; Constantino, J.N., 2004), Autism Spectrum Symptom Questionnaire (ASSQ; Ehlers, Gillberg, & Wing, 1999), and Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003). In addition, the parent completed measures assessing family cohesion (Family Environment Scale (FES)-Cohesion; Moos & Moos, 1994a), attributions about the child's behavior (Controllability of Behavior Questionnaire), and expressed emotion via the Five Minute Speech Sample (FMSS; Magano-Amato, 1993).

Finally the parent completed questionnaires assessing their own psychological functioning including the Symptom Checklist 90-Revised (SCL90; Derogatis, 1994), an abbreviated form of Life Stressors and Social Resources Inventory measuring negative life events (LISRES-NLE; Moos & Moos, 1994b*)*, and the Endler Multidimensional Anxiety Scale (EMAS; Endler, Edwards, & Vitelli, 1991).

Chapter 3: Results

Descriptive Analyses

The alpha level for all statistical tests in the following analyses was .05 unless otherwise noted. The HFA and comparison groups were not well matched on WISC-IV VCI or gender. Specifically, the comparison sample contained more girls and had a higher mean VCI compared to the HFA sample. To address this issue a subsample of females with VCI greater than or equal to 110 were randomly removed from the comparison group ($n = 5$). Therefore, the final HFA sample included 56 children (50 male, 6 female) and the comparison sample included 43 children (37 male, 6 female); ADHD ($n = 6$; 14%), anxiety ($n = 2$; 5% See Table 1). After randomly removing comparison participants, diagnostic groups were statistically equivalent in terms of gender distribution, $\chi^2 (1) = .24$, $p = .63$, and age, $F (1, 97) = 1.60$, $p = .21$. In the HFA group, parent participation was divided as follows: mothers ($n = 53$) and fathers ($n = 3$). In the comparison group, parent participation was divided as follows: Mothers ($n = 32$), fathers ($n = 5$), grandmother ($n = 5$), and uncle ($n = 1$). The mean VCI for the HFA sample remained lower than for the comparison sample, $F (1, 97) = 5.65$, $p = .02$, See Table 2. Therefore, VCI was entered as a covariate in subsequent analyses.

The parents in the HFA group self identified ethnicity as follows: 17 White, non-Hispanic (30%), 27 Hispanic (48%), 3 Asian (5%), 3 Black or African-American (5%), 2 "other" (4%), and 5 parents (9%) did not identify ethnicity. The parents in the comparison group self identified ethnicity as follows: 16 White, non-Hispanic (37%), 20 Hispanic (47%), 1 Black or African-American (2%), 4 "other" (9%), and 2 parents did not identify ethnicity (5%). The groups were not significantly different on ethnicity, χ^2

(4) = 4.50, p = .34. Regarding marital status in the HFA group, 4 were single (7%), 38 were married (68%), 11 were divorced (20%), 1 was separated (2%), and 2 parents did not report (4%). In the comparison group, 7 were single (16%), 24 were married (56%), and 12 were divorced (28%). The groups did not differ on parent marital status, χ^2 (3) = 3.15, p = .37. Regarding maternal education, in the HFA group, 3 mothers completed high school (5%), 13 some college (23%), 17 college (30%), 7 some graduate school (13%), 15 graduate school (27%), and 1 did not report education (2%). In the comparison group, 1 mother did not complete high school (2%), 6 completed high school (14%), 4 some college (9%), 18 college (42%), 6 some graduate school (14%), 6 graduate school (14%), and 2 did not report (5%). Groups did not differ, χ^2 (5) = 8.87, p = .11. Regarding paternal education, in the HFA group, 1 father did not complete high school (2%), 6 fathers completed high school (11%), 12 some college (21%), 9 college (16%), 5 some graduate school (9%), 18 graduate school (32%), and 5 parents did not report education (9%). In the comparison group, 8 fathers completed high school (19%), 9 some college (21%), 8 college (19%), 4 some graduate school (9%), 10 graduate school (23%), and 4 did not report (9%). Groups did not differ on paternal education, χ^2 (5) = 2.62, p = .76.

The diagnostic groups significantly differed on reported annual household income, χ^2 (5) = 12.38, p = .03, with the families in the HFA group reporting higher income. In the HFA group income was reported as follows: 7 in the $10-24, 000 range (13%), 5 in the $25-49, 000 range (9%), 11 in the $50-74, 000 range (20%), 12 in the $75-99, 000 range (21%), 17 in the greater than $100, 000 range (30%), and 4 parents did not report (7%). In the comparison group, income was reported as follows: 1 in the less than $10, 000 range (2%), 3 in the $10-24, 000 range (7%), 13 in the $25-49, 000 range

(30%), 3 in the \$50-74, 000 range (7%), 5 in the \$75-99, 000 range (12%), 15 in the greater than \$100, 000 range (35%), and 3 parents did not report (7%).

The groups were also significantly different on medication usage, $\chi^2 (1) = 8.27$, $p < .001$; more children in the HFA group were taking medication than in the comparison group (HFA group "yes" $n = 19$ (34%), Comparison group "yes" $n = 4$ (9%). Medication usage was entered as a covariate in subsequent analyses.

Finally, with respect to symptom confirmation, consistent with their diagnosis, children in the HFA group had higher Total scores on the SCQ, $F (1, 97) = 179.57$, $p < .001$, and ASSQ, $F (1, 97) = 199.64$, $p < .001$. Regarding performance on the ADOS, the participants in the HFA group showed greater deficit in the three areas (i.e., core deficits) measured, ADOS Communication Total, $F (1, 94) = 80.20$, $p < .001$; Social Interaction Total, $F (1, 94) = 101.18$, $p < .001$; Stereotyped Behaviors/Restricted Interests Total, $F (1, 94) = 18.55$, $p < .001$; See Table 2.

Preliminary Analyses

Social, emotional, and behavioral functioning. Measures of child externalizing problems (i.e., BASC Attention Problems Hyperactivity, Aggression), internalizing problems (i.e., BASC Withdrawal, Anxiety, Depression), and social responsiveness (i.e., SRS Total) were significantly correlated with each other, with the exception of BASC Withdrawal and Aggression, see Table 3.

Expressed emotion. Diagnostic groups did not differ on overall expressed emotion group membership (high/low) on the FMSS, $\chi^2 (1) = 2.02$, $p = .16$). Within each diagnostic group, few families were classified as high EE. Specifically, in the HFA

group, only 11 families (19.64%) were classified as high EE and in the comparison group only 4 families (9.30%) were classified as high EE.

Individuals were not evenly distributed within diagnostic groups across high/low EE groups on the FMSS (χ^2 (1) = 48.09, p < .001). These classification frequencies suggested that in this sample, as in preliminary work with a subsample of the current sample (Zahka, 2005), the narrow categorization of presence or absence of expressed emotion yielded limited variability in classification, limiting the power to detect EE group differences on the dependent variables of interest. To address this issue, a broader categorical scheme was utilized. Expressed emotion membership was recoded into *higher* EE (vs. *high)* and low EE groups. The *Higher EE* group included individuals coded as either high or borderline EE. Chi-square analyses examining classification rates across the diagnostic groups revealed a more even distribution, χ^2 (1) = .25, p = .62, with 33 higher EE HFA families (58.92%) and 19 higher EE comparison families (44.18%); See Table 4.

In terms of analyses of individual items of the FMSS, the diagnostic groups did not differ on ratings of negative (HFA n = 2, Comparison n = 1), neutral (HFA n = 39, Comparison n = 22), or positive (HFA n = 15, Comparison n = 20) relationship, FMSS Relationship, χ^2 (2) = 4.16, p = .13, the presence (HFA n = 9, Comparison n = 3) or absence (HFA n = 47, Comparison n = 40) of dissatisfaction, FMSS Dissatisfaction, χ^2 (1) = 1.89, p = .17), the presence (HFA n = 4, Comparison n = 1) or absence (HFA n = 52, Comparison n = 42) of self-sacrificing attitudes, FMSS Self Sacrificing, χ^2 (2) = 1.38, p = .50, the presence (HFA n = 6, Comparison n = 1) or absence (HFA n = 50, Comparison n = 42) of excess detail, FMSS Excess Detail, χ^2 (1) = 2.61, p = .11, the

number of critical statements, FMSS Number of Critical Statements, F (1, 96) = 2.33, p = .19, or the number of highly positive statements of attitude, FMSS Statements of Attitude, F (1, 96) = .87, p = .36. There was a trend toward significant diagnostic group differences on negative (HFA n = 2, Comparison n = 0), neutral (HFA n = 22, Comparison n = 26), or positive (HFA n = 32, Comparison n = 17) ratings of the initial statement, FMSS Initial Statement, χ^2 (2) = 5.31, p = .07 and presence (HFA n = 4, Comparison n = 0) or absence (HFA n = 52, Comparison n = 43) of crying during the sample, FMSS Emotional Display, χ^2 (1) = 3.20, p = .07. Parents in the HFA group more frequently displayed crying during the FMSS and made more positive initial statements than did parents in the Comparison group. Groups were significantly different on the number of positive remarks made during the speech sample, F (1, 96) = 5.98, p = .02; see Table 5, with the parents of children in the HFA group making fewer positive remarks.

Family cohesion. In order to assess normality of the FES, an analysis of skew and kurtosis was conducted, as well as a boxplot analysis. The FES Cohesion variable was not normally distributed. There was significant negative skew and kurtosis (skew = -1.64, kurtosis = 2.52) and an examination of the distribution using a boxplot analysis revealed four data points that were significant outliers toward a lower score. Three occurred in the comparison group and one occurred in the HFA group. In order to normalize the distribution without removing data points, data was modified using a cube transformation that resulted in a normal distribution (skew = -0.54, kurtosis = -0.83). The diagnostic groups did not differ on this transformed measure, F (1, 91) = .08, p = .78. In order to examine the relations between cohesion and the FMSS categorical grouping, mean levels

of cohesion were compared across groups; no significant difference was found on the raw, $F(1, 91) = .85$, $p = .36$, or transformed, $F(1, 91) = 1.95$, $p = .17$, variable.

Hypothesis Testing

Hypothesis 1. Hypothesis 1 stated that diagnostic groups will differ on externalizing behavior problems, with the HFA group displaying greater externalizing problems. This relation is also predicted to be moderated by family factors (i.e., expressed emotion and family cohesion) such that diagnostic group differences in externalizing behaviors will be particularly pronounced in the presence of higher EE or lower cohesion. Based on the literature, the relation of family factors is predicted to be unique to externalizing behaviors, and less so to internalizing symptoms or social symptoms.

Expressed emotion in relation to child externalizing problems. To examine this hypothesis MANCOVA analyses were conducted to determine the effect of diagnostic group and EE group (Higher versus Lower groupings) on parent reported externalizing symptoms on the BASC-2. WISC-IV VCI and medication usage were entered as covariates for all analyses.

There was a significant effect of diagnostic group on externalizing symptoms, $F(3, 90) = 11.20$, $p < .01$; Wilks' $\Lambda = .73$; partial $\eta^2 = .27$. The effect of EE group membership was not significant, $F(3, 90) = .14$, $p = .93$; Wilks' $\Lambda = .99$; partial $\eta^2 = .01$, nor was the interaction between diagnosis and EE, $F(3, 90) = 1.89$, $p = .14$; Wilks' $\Lambda = .94$; partial $\eta^2 = .06$.

In order to explore the possibility that EE was related to individual dimensions within the larger externalizing cluster, post hoc univariate analyses were conducted for

each dimension (aggression, attention problems, hyperactivity). Post hoc univariate

analyses, using a Bonferroni correction of .017, revealed significant diagnostic group

differences for hyperactivity, $F(1, 92) = 12.07$, $p < .001$, partial $\eta^2 = .12$, and attention

problems, $F(1, 92) = 33.43$, $p < .001$, partial $\eta^2 = .27$, and a trend for group differences

in aggression, $F(1, 92) = 5.13$, $p = .03$, partial $\eta^2 = .05$. Children in the HFA group were

rated as showing greater impairment on attention and hyperactivity, and tended to be

rated higher in aggression.

In addition, the interaction of diagnostic and EE groups approached significance

in the prediction of hyperactivity, $F(1, 92) = 4.78$, $p = .03$, partial $\eta^2 = .05$, but did not

reach corrected significance with respect to parent-reported levels of attention problems,

although means were in the expected direction, $F(1, 92) = 3.61$, $p = .06$, partial $\eta^2 = .04$.

Post hoc follow-up analyses were used to examine the interaction involving

hyperactivity. Within the higher EE group, HFA children were rated as more hyperactive

than children in the comparison group, $t(50) = 4.12$, $p < .001$. In contrast, within the

lower EE group, HFA children and children in the comparison group did not differ in

ratings of hyperactivity, see Table 6 and Figure 2.

Family cohesion in relation to child externalizing problems. To examine the

effects of diagnostic group and the continuous FES Cohesion scores on parent-reported

BASC2 Hyperactivity, Aggression and Attention Problems, a series of hierarchical

multiple regression analyses were computed.

The covariates (i.e., WISC-IV VCI and medication usage) were entered first but

did not predict variability in Hyperactivity. However, both diagnostic group and FES

Cohesion, accounted for unique variance in Hyperactivity ratings in the full model, $F(1,$

87) = 4.60, $p < .001$; Adjusted $R^2 = .16$; see Table 9. Parents of children in the HFA group reported higher levels of hyperactivity. Across both diagnostic groups, parents who reported higher family cohesion also reported lower levels of hyperactivity in their children. The diagnostic group x FES interaction term did not account for a significant portion of unique variability in Hyperactivity, see Table 7.

The covariates (i.e., WISC-IV VCI and medication usage) were not significantly associated with BASC-2 Aggression. When diagnostic group, FES Cohesion, and the interaction term were entered into the equation the model was significant and both diagnostic group and diagnostic group X FES interaction term accounted for significant unique variance, see Table 8. Parents of children in the HFA group reported higher levels of aggression in their children. This effect was qualified, however, by levels of FES cohesion. Following procedures outlined by Aiken and West (1991) and Holmbeck (2002) for probing and graphing significant interactions, two new conditional moderator variables were computed (± 1 *SD* from the mean of FES Cohesion), as were their interaction variables. Two post-hoc regressions were conducted that included simultaneous entry of diagnostic group, one of each conditional cohesion variable, and the group x cohesion variable (Holmbeck, 2002). Unstandardized betas and regression equations were derived for parents reporting high (1 *SD* above the mean) and low (1 *SD* below the mean) cohesion; see Figure 3. For high cohesion, the slope was not significantly different from zero, $B = 1.92$, $t(89) = .70$, $p = .49$. For low cohesion, the slope was significant, $B = 9.50$, $t(89) = 3.42$, $p = .01$, indicating that the impact of low family cohesion on aggression differed for diagnostic groups. Examination of Figure 3 indicated that in the comparison group, low family cohesion was associated with lower

aggression, whereas in the HFA group, low family cohesion was associated with higher aggression (HFA mean = 52.33, Comparison mean = 42.86).

The covariates (i.e., WISC-IV VCI and medication usage) were significantly associated with BASC-2 Attention Problems, F (1, 90) = 5.61, p = .01, see Table 10. However, when diagnostic group was entered into the equation, the covariates were no longer significant and diagnostic group accounted for significant variability in BASC-2 Attention Problems in the full model, F (1, 87) = 10.76, p < .001; Adjusted R^2 = .35. Parents of children in the HFA group reported greater attention problems than did parents of children in the comparison group. The effects of FES Cohesion and the interaction between diagnostic group and FES Cohesion were not significant; see Table 9.

Expressed emotion in relation to child internalizing problems. To determine whether the effects of diagnostic group, family factors, and importantly, their interaction, were specific to externalizing symptoms, comparable analyses were conducted with internalizing symptoms (i.e., BASC-2 PRS Anxiety, Depression, and Withdrawal).

WISC-IV VCI and medication usage were entered as covariates for the MANCOVA of this set of three variables, including BASC Depression, Anxiety, and Withdrawal. There was a significant effect of diagnostic group on internalizing symptoms, F (3, 90) = 21.33, p < .001; Wilks' Λ = .58; partial η^2 = .42. However, neither the effect of EE group, F (3, 90) = .49, p = .69; Wilks' Λ = .98; partial η^2 = .02, or the effect of the Diagnostic Group X EE Group interaction, F (3, 90) = 1.56, p = .20; Wilks' Λ = .95; partial η^2 = .05, were significant.

Post hoc exploratory univariate analyses were conducted, using a Bonferroni adjusted alpha level of .017. Diagnostic groups differed in terms of anxiety, F (1, 92) =

29.79, $p < .001$, partial $\eta^2 = .25$, depression, $F(1, 92) = 26.53$, $p < .001$, partial $\eta^2 = .22$, and withdrawal, $F(1, 92) = 54.23$, $p < .001$, partial $\eta^2 = .37$, with children in the HFA group experiencing more impairment on all variables. These analyses revealed that EE and the interaction of EE and diagnostic group were unrelated to any of the internalizing dimensions, with the exception of a trend toward significance, for the effect of the interaction term on anxiety, $F(1, 92) = 4.72$, $p = .03$, partial $\eta^2 = .05$, See Table 10.

Post hoc follow-up analyses were used to probe this interaction. With regard to anxiety, within the higher EE group, HFA children were rated as more anxious than children in the comparison group, $t(50) = 5.74$, $p < .001$. In contrast, within the lower EE group, HFA children and children in the comparison group did not differ in ratings of anxiety, see Table 10 and Figure 4.

Family cohesion in relation to child internalizing problems. Hierarchical multiple regression analyses were used to explore the relation of diagnostic group and family cohesion on the same set of internalizing symptoms (i.e., BASC-2 PRS Anxiety, Depression, Withdrawal).

The covariates (i.e., WISC-IV VCI and medication usage) were not associated with BASC-2 PRS Anxiety scores. Diagnostic group predicted unique variance in BASC-2 PRS Anxiety in the full model, $F(1, 87) = 5.74$, $p < .001$; Adjusted $R^2 = .21$. However, the ability to predict BASC-2 PRS Anxiety was not significantly improved after adding FES Cohesion or the interaction term (Diagnostic group x FES) into the regression equation, see Table 11. Parents of children in the HFA group reported higher levels of anxiety in their children than did parents of children in the comparison group.

The covariates (i.e., WISC-IV VCI and medication usage) were not associated

with significant effects regarding BASC-2 PRS Depression. Diagnostic group predicted

BASC-2 PRS Depression significantly in the full model, $F(1, 87) = 7.51$, $p < .001$,

Adjusted $R^2 = .26$. There was not a main effect of FES, however, the interaction of FES

cohesion and diagnostic group was significant, see Table 12. Following procedures

outlined by Aiken and West (1991) and Holmbeck (2002) for probing and graphing

significant interactions, two new conditional moderator variables were computed (± 1 SD

from the mean of FES Cohesion), as were the interaction variables. Two post-hoc

regressions, which each included simultaneous entry of diagnostic group, one of

conditional cohesion variables, and the group x cohesion variable (Holmbeck, 2002).

Unstandardized betas and regression equations were derived for parents reporting high (1

SD above the mean) and low (1 *SD* below the mean) cohesion. For high cohesion, the

slope was significantly different from zero, $B = 9.63$, $t(89) = 2.38$, $p = .02$, indicating that

the impact of high cohesion on depression differed for diagnostic groups. For low

cohesion, the slope was significant, $B = 22.91$, $t(89) = 5.60$, $p < .001$, indicating that the

impact of low family cohesion on depression differed for diagnostic groups. Examination

of Figure 5 indicated that in the comparison group, low family cohesion was associated

with lower depression (Mean = 33.21), whereas in the HFA group, low family cohesion

was associated with higher depression (Mean = 56.04). In the comparison group, high

family cohesion was associated with higher depression (Mean = 44.93), whereas in the

HFA group, higher FES was associated with lower depression (Mean = 54.53), see

Figure 5.

The covariates (i.e., WISC-IV VCI and medication usage) were not associated with BASC-2 Withdrawal scale scores. However, diagnostic group was associated with BASC-2 Withdrawal in the full model, $F(1, 87) = 11.61$, $p < .001$; Adjusted $R^2 = .37$. The model was not significantly improved by adding FES or the interaction term into the regression equation; see Table 13. Parents of children in the HFA group reported their children to be more socially withdrawn than children in the comparison group.

Expressed emotion in relation to child social symptoms. ANCOVA analyses were conducted to determine the effect of diagnostic group and EE group (Higher versus Lower groupings) on parent reported social symptoms from the SRS. WISC-IV VCI and medication usage were entered as covariates for the analysis. There was a significant effect of diagnostic group on social symptoms, $F(1, 93) = 177.20$, $p < .001$; partial $\eta^2 = .66$. The effect of EE group membership was not significant, $F(1, 93) = .54$, $p = .46$; partial $\eta^2 = .01$, nor was the interaction between diagnosis and EE, $F(1, 93) = .25$, $p = .62$; partial $\eta^2 < .001$; see Table 14.

Family cohesion in relation to child social symptoms. Finally, in terms of analyses of social symptoms and FES Cohesion, the covariates (i.e., WISC-IV VCI and medication usage) were not significantly associated with SRS Total. When diagnostic group was entered into the equation, the ability to predict SRS Total significantly improved in the full model, $F(1, 87) = 43.27$, $p < .001$; Adjusted $R^2 = .71$. The model was not significantly improved by adding FES or the interaction term into the regression equation; see Table 15. Parents of children in the HFA group reported their children to be less socially responsive than children in the comparison group.

Overall, the analyses for this hypothesis indicated a main effect for diagnostic group, but not EE group, or the interaction term, on externalizing problems. There was a trend toward a significant interaction effect for the individual analyses of inattention and hyperactivity. Analyses of the cohesion variable indicated an association of higher cohesion and lower hyperactivity regardless of diagnostic group membership, and an interaction effect for cohesion and diagnostic group for aggression only. As expected, there was a main effect for diagnostic group on the internalizing variables, but no effects were significant for EE group or the interaction. Analyses of the cohesion variable revealed an interaction effect for depression. There was a main effect for diagnostic group on the social symptoms variable, and no main effects for EE group or the interaction term.

Hypothesis 2. It was hypothesized that groups will differ on parents' perceptions of their child's behavior, including perceptions of the child's level of control over his/her behavior and types of attributions about their child's behavior. Parents of children in the HFA group are predicted to report lower behavioral controllability. The relation of diagnostic group and perception of the child's behavior is expected to be moderated by family factors (i.e., expressed emotion and family cohesion). It is predicted that diagnostic group differences in parental perceptions will be particularly pronounced in the presence of higher EE or lower cohesion.

Expressed emotion in relation to behavioral controllability. Parent perception of a child's ability to control their behavior was assessed via the Controllability of Behavior Questionnaire, which was designed for this study. Child Symptom Control (CSC) was correlated with Parent Symptom Control (PSC), r (83) = .85, $p < .001$ for the whole

sample, but not with positive, negative or neutral attributions. PSC was correlated with

positive attributions, $r(84) = .32$, $p < .001$, but not with negative or neutral attributions.

MANCOVA analyses were conducted to determine the effect of diagnostic group

status and family factors on measures of parent perception of child behavior control (i.e.,

behavior controllability and attributions about behavior). The dependent variables were

CSC, PSC, and FMSS negative, neutral and positive attributions. There was a significant

effect for WISC-IV VCI, $F(5, 73) = 2.81$, $p = .02$; Wilks' $\Lambda = .84$; partial $\eta^2 = .16$. There

was also a main effect for diagnostic group, $F(5, 73) = 9.84$, $p < .001$; Wilks' $\Lambda = .60$;

partial $\eta^2 = .40$, and EE group, $F(5, 73) = 2.73$, $p = .03$; Wilks' $\Lambda = .84$; partial $\eta^2 = .16$

on perceptions of child behavioral controllability. However, the effect of the interaction

term (diagnostic group x EE group) was not significant, $F(2, 73) = 1.16$, $p = .34$; Wilks'

$\Lambda = .93$; partial $\eta^2 = .07$.

Analysis of each individual dependent variable, using a Bonferroni adjusted alpha

level of .01, showed that the diagnostic groups differed in terms of parent-reported

perceptions of child behavior control, CSC, $F(1, 77) = 46.28$, $p < .001$, partial $\eta^2 = .38$,

parent control of child behavior, PSC, $F(1, 77) = 35.58$, $p < .001$, partial $\eta^2 = .32$.

Parents of children in the HFA group reported their children had less control and they as

parents had less control over their children's behavior than did parents of children in the

comparison group. These analyses revealed that EE and the interaction of EE and

diagnostic group were unrelated to CSC or PSC. There were additionally no significant

effects for diagnostic group, EE group, or the interaction on the number of positive or

negative attributions. However, EE groups differed in terms of the number of neutral

attributions, $F(1, 77) = 10.54$, $p < .001$, partial $\eta^2 = .12$, regardless of diagnostic group.

Parents classified as High EE made fewer neutral attributions about their children's behavior; see Table 16.

Family cohesion in relation to behavioral controllability. Hierarchical multiple regression analyses were used to explore the relations between diagnostic group and family cohesion on parent perceptions of child behavior control, including CSC, PSC, and FMSS positive, negative, and neutral attributions.

The covariates (i.e., WISC-IV VCI and medication usage) were not significantly associated with CSC. When diagnostic group was entered into the equation, the ability to predict CSC was significantly improved in the full model, $F(1, 74) = 12.91$, $p < .001$; Adjusted $R^2 = .43$. The model was not significantly improved by adding FES or the interaction term into the regression equation. Parents of children in the HFA group reported their children to have less control over their own behavior than did parents of children in the comparison group; see Table 17.

The covariates (i.e., WISC-IV VCI and medication usage) were not significantly associated with PSC. When diagnostic group was entered into the equation, the ability to predict PSC was significantly improved in the full model, $F(1, 74) = 8.16$, $p < .001$; Adjusted $R^2 = .31$. The model was not significantly improved by adding FES or the interaction term into the regression equation. Parents of children in the HFA group reported they had less control over their child's behavior than did parents of children in the comparison group; see Table 18.

When the covariates (i.e., WISC-IV VCI and medication usage) were entered in the first block, they did not significantly improve the ability to predict positive attributions. When diagnostic group and FES Cohesion were entered into the equation,

the ability to predict positive attributions was significantly improved in the full model, F (1, 74) = 2.26, p = .05; Adjusted R^2 = .07. The model was not significantly improved by adding the interaction term into the regression equation. Parents of children in the HFA group made fewer positive attributions than did parents of children in the comparison group. Regardless of group status, parents who reported higher family cohesion also made more positive attributions; see Table 19.

When WISC-IV VCI was entered in the first block it significantly predicted negative attributions in the first step, F (1, 78) = 4.63, p = .04; Adjusted R^2 = .04. Adding medication usage, diagnostic group, FES, or the interaction term did not significantly improve the model, F (1, 74) = 1.61, p = .17, Adjusted R^2 = .04. Parents made fewer negative attributions about their children when the children had higher scores on WISC-IV VCI; see Table 20.

When WISC-IV VCI was entered in the first block, there was a trend toward significance in predicting neutral attributions in the first step of the model, F (1, 78) = 3.46, p = .07; Adjusted R^2 = .03. Adding medication usage, diagnostic group, FES, or the interaction term did not significantly improve the model, F (1, 74) = .94, p = .46, Adjusted R^2 = -.00. Parents made a greater number of neutral attributions about their children when the children had higher scores on WISC-IV VCI; see table 21.

Hypothesis 3. It was hypothesized that diagnostic groups will differ on measures of parents' experiences of stress, with the parents of children in the HFA group reporting higher stress. The relation between diagnostic group and report of stress is predicted to be moderated by family factors (i.e., expressed emotion and family cohesion). It is predicted

that diagnostic group differences in parent stress will be particularly pronounced in the presence of higher EE or lower cohesion.

Expressed emotion in relation to parent stress. MANCOVA analyses were conducted to determine the effect of group status and family factors on measures of parent endogenous and exogenous stress. Variables included SCL-90 R GSI, LISRES-A, Negative Life Events, EMAS Social Evaluation, and Leyton Trait. After controlling for covariates (i.e., WISC-IV VCI and medication usage), there was no significant effect of diagnostic group, $F(4, 74) = 1.71$, $p = .16$; Wilks' $\Lambda = .92$; partial $\eta^2 = .09$, EE group ($F(4, 74) = 1.56$, $p = .20$; Wilks' $\Lambda = .92$; partial $\eta^2 = .08$, or the interaction term, $F(4, 74) = .73$, $p = .58$; Wilks' $\Lambda = .96$; partial $\eta^2 = .04$, on parent reported stress, see Table 22.

Analysis of each individual dependent variable, using a Bonferroni adjusted alpha level of .01, did not provide significant diagnostic group differences, EE group differences, or effects of the interaction term.

Family cohesion in relation to parent stress. Hierarchical multiple regression analyses were used to explore the relation of diagnostic group and family cohesion on parent endogenous and exogenous stress. None of the entered variables significantly predicted SCL-90GSI, see Table 23, LISRES-NLE, see Table 24, EMAS Social Evaluation, see Table 25, or Leyton Obsessional Inventory Trait Total, See Table 26.

Chapter 4: Discussion

The awareness and knowledge of autism spectrum disorders has grown in breadth and depth, as autism moves to the forefront in the field of research on child development. The study of the role of family factors is relatively new and is in the beginning stages of empirical investigation. A primary goal of the current study was to clarify the role of family factors in the prediction of variation in individual differences in child and parent outcome in families with a child with higher functioning autism. Of particular interest were family factors (i.e., expressed emotion and cohesion) and their role as modifiers of the individual differences in children with HFA's social and emotional development, parental beliefs about their children's behavior, and parental stress. Overall, as expected, children with autism were consistently rated higher than their typically-developing peers on internalizing problems, externalizing problems, and social impairment. In addition, as expected, parents of children with autism perceived their child's behaviors to be less controllable. The effects of expressed emotion and family cohesion on individual differences in child and family adjustment were more nuanced. Family factors were associated with individual differences in comorbid symptoms including attention problems, hyperactivity, and aggression, and anxiety, depression, and withdrawal. In contrast, family factors were unrelated to variations in social symptoms.

Family factors played a less powerful modifier role with regard to parental perceptions of their children's behavioral control; parents of both typically developing children and children with HFA who were classified as higher EE made fewer neutral attributions, but did not differ from lower EE parents on negative or positive attributions,

or on parents' perceptions of behavioral control. Higher family cohesion, regardless of diagnosis, predicted positive attributions.

In contrast to child adjustment and parent perceptions of controllability, parental stress, as measured in the current study, was unrelated to either diagnostic status or family factors.

Family Factors in Relation to Child Behavior Problems

One of the most interesting findings in this study was the effect of family factors on child behavior problems. Based on the literature, it was expected that family factors would be a better predictor of externalizing versus internalizing problems; however, family factors predicted both internalizing and externalizing problems in children.

Groups differed with regard to externalizing and internalizing problems, even with the inclusion of children with ADHD and anxiety into the comparison sample; children with HFA were rated as having higher levels of hyperactivity and attention problems, but not aggression. Children with HFA were also rated as higher in anxiety, depression and social withdrawal compared to typically developing children.

Relative levels of expressed emotion were unrelated to externalizing and internalizing problems overall. However, there were some interesting trends for interactions supporting the idea that family factors may be associated with individual differences in behavioral expression among children with autism. A trend toward significance was observed in the interaction of diagnosis and EE ratings in predicting hyperactivity. Specifically, the diagnostic group difference on hyperactivity was conditional with respect to EE such that, children with HFA had higher hyperactivity than did typically developing children only in the comparison of the higher EE rather than

lower EE subgroups. In contrast, HFA and typically developing children did not differ in hyperactivity among lower EE families. Peris and Hinshaw (2003) similarly found that High EE differentially predicted ADHD symptoms from families categorized as Low EE, but not behavior associated with Conduct Disorder/Oppositional Defiant Disorder (i.e., aggression). The current investigation also found that aggression was not predicted by EE, but was related only to lower levels of family cohesion. Higher cohesion was not related to aggression in either group and it may be that higher cohesion and/or lower expressed emotion has less of an impact than less positive family factors (i.e., low cohesion, high expressed emotion). It may also be that in this case, children who are typically developing are less impacted by the family environment with regard to behavioral self-regulation and are more resilient to the impacts of less positive family environments, given the multiple systems in which children typically develop (Parke and Ladd, 1992). It may be that children with HFA are more limited in the systems in which they observe and learn social and behavioral regulation abilities, and as a result, the family system is much more important, and high levels of EE lead to poorer behavior regulation.

Regardless of diagnostic group, higher family cohesion was associated with lower levels of parent-reported hyperactivity, which is in line with other research indicating higher levels of conflict were reported by parents of children with ADHD (Schroeder & Kelley, 2009). Family cohesion and expressed emotion did not appear to predict inattention directly and this may be due to the fact that inattention causes relatively less "disruptive behavior," while hyperactivity and aggression are more external and may impact the family system directly (i.e., predicting more negative feelings about parenting;

Donenberg & Baker, 1993). Another explanation for the finding could be that comparable family processes are at play across the externalizing problem domains but that it is relatively difficult for parents to report on inattention compared to more observable behaviors. It also might be worthwhile to investigate whether parents do not notice inattention as much at home, but perhaps teachers would rate the same child as having greater attention problems in a classroom setting.

Similar to the trend of the impact of EE on children's hyperactivity was observed in the prediction of anxiety, which was not expected; children with HFA in the higher EE group displayed greater anxiety than typically developing children. However, HFA and typically developing children did not differ in ratings of anxiety for lower EE families. Based on these results, it may be that family factors pose a specific risk for development of anxiety in children with higher functioning autism, but do not provide the same risk, or have the same level of impact for a child who is not diagnosed with autism.

With regard to internalizing problems, higher family cohesion was associated with lower depression in the HFA group but higher depression in the typically developing children. Lower family cohesion was associated with higher depression in the HFA group and lower depression in the typically developing children. This finding runs in parallel to the pattern discussed above with regard to the role of family cohesion in child aggression, with family factors relating in an intuitively predictable way for children in the HFA group, but in the opposite direction for typically developing children and adolescents. This pattern of results provides some validity for the trends found with regard to expressed emotion, and suggests that expressed emotion and family cohesion, and possibly by extension, family support and structure may function differently and

therefore have different developmental consequences for children with HFA relative to typically developing children. Children and adolescents with HFA may be especially dependent upon their families for some level of extrinsic structure, particularly for help in regulation of behavior. For example, parents of children with Asperger Syndrome report providing higher levels of care than parents of a typically developing child, including structuring routines, social interactions, education, and overall relationships (Portway & Johnson, 2005). This same level of increased structure and overcontrol can have a negative affect on typically developing children and has been associated with increased risk for anxiety (Hudson and Rapee, 2001).

In families of typically developing children, those who were classified as higher EE reported *less* hyperactivity and *less* anxiety as compared to families of a typically developing child who were classified as lower EE. It may be that in families of a typically developing child, or at the very least a child who does not have an autism spectrum disorder, that high EE, which includes high emotional overinvolvement, actually functions less as a risk and more as a protective factor, or as a factor for resiliency. Much of the research in expressed emotion is based on adults with fairly significant psychopathology (e.g., schizophrenia) and the negative impact of emotional overinvolvement is understandable. However, as suggested by Wamboldt et al. (2000), if emotional overinvolvement, and "positivity" actually represents developmentally normative emotional attitudes toward a child or adolescent, then we would expect that high EE, and hence higher positivity toward a child, would lead to fewer behavioral problems, as it does in this study with regard to hyperactivity and anxiety only. It may be that (a) looking more closely at whether the impact of EE changes with regard to the

child's and (b) investigating EE over the course of development (i.e., a longitudinal study) will elucidate the relation between EE and child behaviors. This is especially true given the reliance of children on their families for emotional/behavior regulation early on, and then later in development turning toward their peers and other social systems (Morris, Silk, Steinberg, Myers & Robinson, 2007).

Family Factors in Relation to Social Symptoms

In contrast to the findings for behavior problems, children's social symptoms were predicted only by diagnostic status. That is, family factors did not appear to confer additional risk or resilience for children with HFA or typically developing children. This finding is consistent with the Mundy et al. (2007) model that family factors could act as a modifier independent of diagnosis and symptom severity, but rather affect other aspects of secondary symptom presentation. In this investigation, secondary symptoms (i.e., comorbidity) appear to be more impacted by the family system than core symptoms of autism. This is a relatively understudied area of autism spectrum disorders. Research in other areas, such as ADHD, indicates the impact of the family system on comorbidity is meaningful; comorbid anxiety was associated with maternal overprotectiveness, lack of positive parenting, and maternal anxiety (Pfiffner & McBurnett, 2006).

Family Factors in Relation to Behavioral Controllability

A secondary aim of this investigation was to examine the relations between family factors and parent perceptions of the degree to which a child's behavior could be regulated or controlled by (a) the child or (b) the parent. In this study, parents of children with HFA perceived both they and their children to have less control over the child's behavior than did parents of typically developing children and adolescents. Although this

investigation aimed to elucidate the role of family factors in parental perceptions of child behavioral control, expressed emotion and family cohesion did not predict variations in perceptions of controllability on the CBQ questionnaire. It may be that these relationships were not significant due to questionnaire design or measurement error. The questionnaire was designed to assess levels of control related to core systems of autism as well as behavioral and emotional comorbidities. However, a factor analysis indicated that all items loaded onto one factor and it may be that the questionnaire was not effective in differentiating behavior control associated with core symptoms of autism, and control associated with comorbidity symptoms. The main variables were, however, negatively associated with a child self-report of locus of control, and indicated higher parent-ratings of control were associated with a more perceived internal locus of control by child self-report, supporting the validity of the measure as an index of controllability. Given this was the first use of this questionnaire, it will be important to continue to collect data on it to determine if a large sample size has any impact on more consistently identifying the proposed two-factor model.

In contrast to perceptions of controllability, family factors were associated with parents' attributions for their children's behaviors. Higher EE was associated with the tendency to make fewer neutral attributions during the FMSS for both typically developing children and children with HFA. That is, parents categorized as higher EE regardless of diagnostic group made fewer neutral attributions. Given the common methodology (i.e., the FMSS) from which EE and attributions were coded, this association is not surprising. However, the association between independently coded constructs provides theoretical and methodological validity to the FMSS, in that one

would expect higher EE families to be either more intensely negative or positive, and thus, less neutral. Further validity was found in the finding that higher family cohesion predicted a greater number of positive attributions, regardless of diagnostic group status.

The finding that parents of children with HFA perceive themselves and their children to have less control over child behaviors and symptoms overall was in the direction we predicted. This finding bears further investigation to determine if the perceived lack of control is related to perception about the causality or controllability of symptoms and behaviors associated with HFA.

Although child verbal IQ was unrelated to child behavioral and emotional functioning, it was predictive of the types of attributions parents made on the FMSS. Regardless of diagnostic status, parents made fewer negative and more neutral attributions about children with a higher verbal comprehension index. It may be that for children who are more intelligent, at least with regard to verbal abilities, parents are more likely to attribute some level of control to their behavior. This finding however, is unclear, and VCI was not a major variable in these analyses. It may be that in future investigations, it will be important to look more closely at intellectual abilities with regard to parental perceptions of behavior control.

Family Factors in Relation to Parent Stress

Family factors did not appear to amplify or diminish parental stress for either group. This was contradictory to the hypothesis family factors would moderate the prediction of parental psychopathology and stressful life events. One reason for these findings might have to do with the operationalization and measurement of parent stress in the current study. The lack of group differences on the negative life events measure

makes more sense, as one would not expect these aspects of stress to be related to family factors (i.e., a car accident, although stressful, is not necessarily directly related to family relationships). However, it was surprising that there were no significant findings for parent-reported psychological distress, especially given the literature on higher rates of affective disorders and social anxiety (Bailey, et al., 1998; Bolton et al., 1998; Ghaziuddin & Greden, 1998; Micali, et al., 2004; J. Piven & Palmer, 1999). It may be that looking more closely at the SCL-90 scales component scales assessing more specific areas of psychopathology would have been more fruitful. However, the EMAS social evaluation scale and Leyton Obsessional Inventory Trait Total variables specifically assessed aspects of anxiety and obsessive compulsive behaviors, and were not significant. It may be that in this investigation, we did not consider coping strategies used by parents to reduce the stress experienced, such as social or partner support (Heiman & Berger, 2008) and this in an area that could be investigated in the future.

A similar lack of significant effect for the association of stress and family factors was found earlier in research conducted in this lab (Zahka, 2005) and it may be that measuring *parenting*-related stress instead of more global stress will be important. Rao and Beidel (2009), for example, found that parents of a child with HFA differed from the parents of typically developing comparison children on a measure of total parenting stress. Upon further exploration, they found the significant difference was due to the child behavioral factors' contribution and not parent factors. In the current investigation, only stress related to parent factors was utilized. In future investigation, it will be important to add a parenting-stress related component.

Limitations

There were some limitations to this study. First, the sample size was smaller than anticipated, which likely reduced the power to detect many of the hypothesized effects. For example, several hypothesized effects were at the level of a trend and did not reach conventional levels of statistical significance. In addition, there was limited variability in EE status using the traditional high versus low classification systems. As a result, participants were reclassified so that the higher EE group included participants who were identified originally as borderline EE. This same procedure was followed by Shimodera et al. (1999) in a study of families of an adult with schizophrenia. This method improved the sensitivity of the measure when compared to the results using the more time-intensive Camberwell Family Interview (CFI). However, it did also add to the concern identified by Wamboldt (2000), who suggested collapsing criticism and EOI into high EE (or higher EE in this case) may be masking an important difference in expressed emotion. That is, critical attitudes and emotionally overinvolved attitudes may have different impacts on child outcome. It is certainly possible that our higher EE group here was driven more by EOI than criticism, given a qualitative assessment of the number of families who were emotionally overinvolved versus critical or both. It may be more informative in the future to begin to investigate EE, at least in this population, from the standpoint of "positive" versus "negative" EE, as opposed to high versus low. Liljestrand, Willoughby, Lam, & Braaten (2010) found that the positive remarks scale of the FMSS was a reliable and valid marker of parental positivity in a group of clinic referred children and their families. Parental positivity (i.e., positive remarks) was negative related to parent psychopathology and child externalizing problems, and positively correlated with

the parent's initial statement on the FMSS. It may be that looking more closely at just the positive remarks subscale could be informative in future studies.

A third limitation is the exclusive reliance on parent-report measures for the assessment of both the moderating family factors variables as well as all dependent variables. To some extent, these findings may be harder, although not impossible, to generalize past maternal perceptions, given the vast majority of the reporters in this study were mothers. However, Hastings et al. (2005) did not find differences in maternal and paternal reports of stress or behavior problems. Although parents have been found to be the best reporters for externalizing problems, children and adolescents may be better reporters of their own internal states and emotional including anxiety and depression (LaGreca & Lemanek, 1996).

This study was a concurrent design, rather than a longitudinal study and as a result, we were not able to fully clarify the direction of the effects.

Future Directions

It will likely be very informative to investigate family factors as a modifier of individual differences, in both children with autism and typically-developing children, via a causal model through a longitudinal study in the future. At this point, we are not able to determine whether, for example, higher aggression in children with HFA leads to a less cohesive family style because more time is spent monitoring the child's behavior and less is spent in family activities (Gray, 1997) or whether lower cohesion provides less of a protective environment for children who are at risk for developing aggressive behaviors. From an alternate perspective, it may be that for children with HFA, depression arises when family cohesion is lower because there is such a reliance on the family for

structuring social interactions and emotional regulation. Altiere and von Kluge (2009) found that a highly positive family cohesion, actually defined as enmeshed within their investigation, might actually be protective for a child with autism. We additionally did not investigate family factors over time, and are unable to make predictions about the relative differences for children and adolescents in this sample. It may be that the needs of the child, or even the needs of the family, change as the child ages and reaches developmental levels which typically expect a higher level of independence. This should be examined in both typically developing children and children with higher functioning autism.

In addition, in this study, our comparison group was diverse in terms of diagnoses; children with ADHD and anxiety were included to attempt to match variability in comorbid symptom presentation relative to the HFA sample. With separate comparison groups as opposed to one mixed group, it may clarify the finding of higher EE and lower cohesion association with lower ratings of impairment in the comparison group. For example, it may be that family factors are associated with child behavioral problems overall in families with a child with any clinical diagnosis, and this investigation was not able to fully explain this relationship with a mixed comparison group.

Overall, the findings of this investigation indicate that family factors could lead to differences in hyperactivity and anxiety in children with HFA versus controls, or that higher EE tends to emerge and complicate the quality of life in families with ASD in response to hyperactivity and/or anxiety in children with ASD.

If family factors lead to hyperactivity and/or anxiety in children, then family therapy may be expected to lead to reductions in these aspects of comorbidity in higher EE HFA families. Conversely, if hyperactivity and/or anxiety in children leads to higher EE than effective treatment of those (e.g., medication, CBT) should lead to improved family factors.

Placing these findings in context of clinical care suggests that we need to be mindful of the family system when considering intervention strategies for children with higher functioning autism. Given the impact of the family system on parent-report of internalizing and externalizing problems, it is important to assess the climate of the family, before proceeding with interventions. This is particularly true as parents are increasingly required to act as an in-home therapist (Sanders, 1999) to address problematic behaviors in clinically referred children with a range of presenting problems. This increased need for structured has been found to impact coping abilities, and activate anxiety, overprotection and rigidity in parents of a child with a disability (Lardieri, Blacher, & Swanson, 2000) Given the level of extrinsic support children with higher functioning autism need from their family, it is important that any interventions requiring home support are carefully crafted to avoid disrupting the family system and potentially putting children with HFA at greater risk for developing comorbid psychopathology

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Appendix A

Figure 1. Conceptual Model
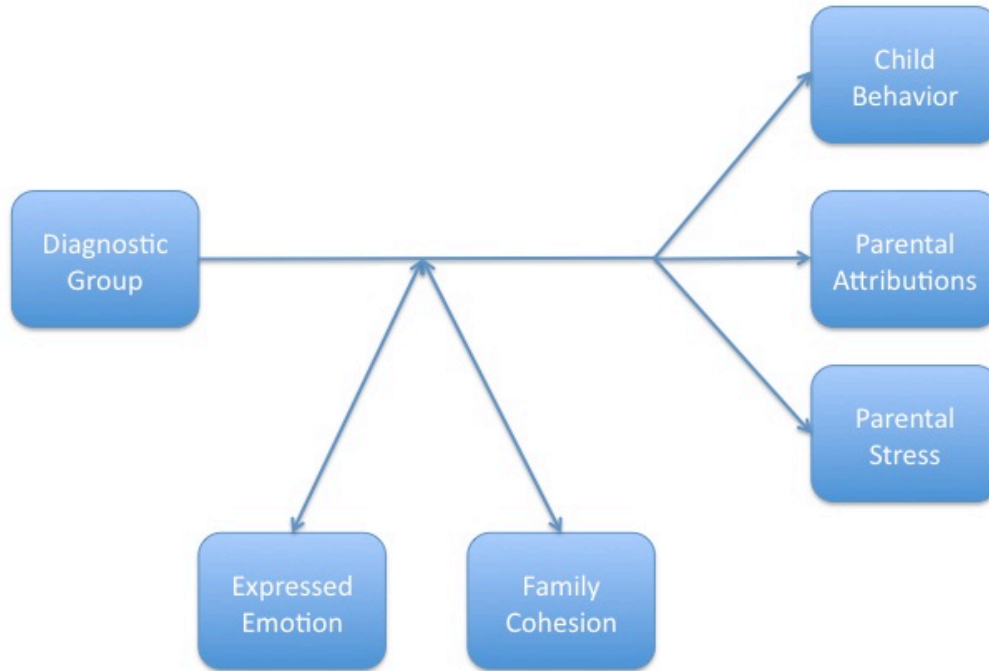
Figure 2. BASC2 PRS Hyperactivity Predicted by Expressed Emotion x Diagnostic Group
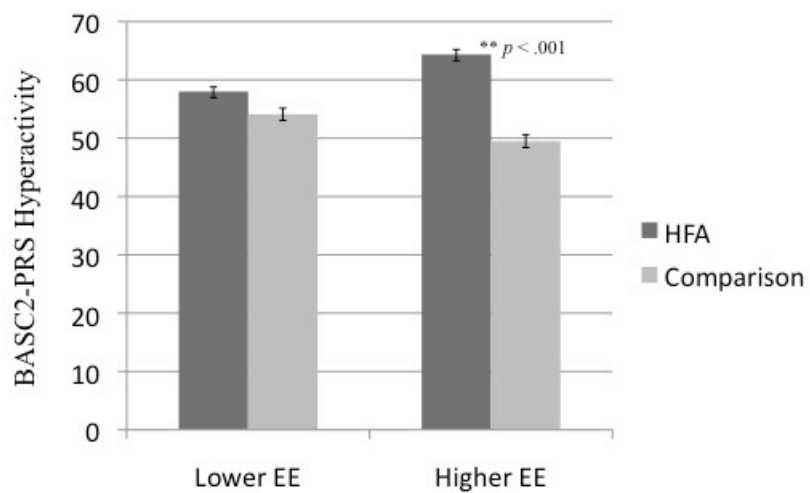
Figure 3. BASC2 PRS Aggression Predicted by FES Cohesion x Diagnostic Group
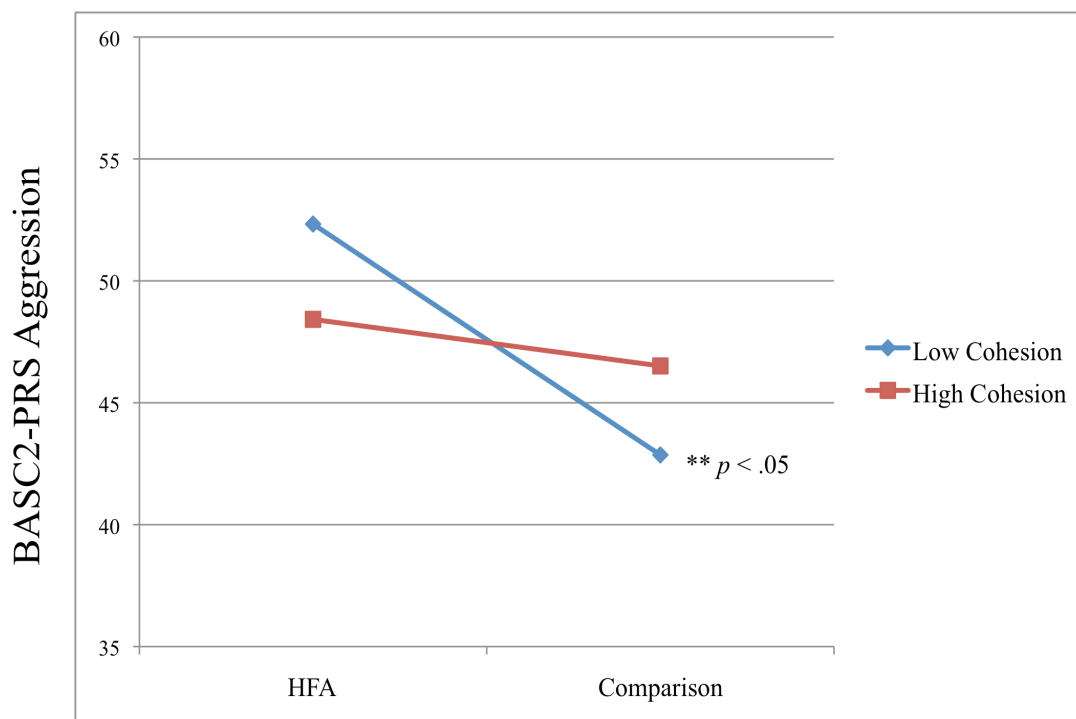
Figure 4. BASC2 PRS Anxiety Predicted by Expressed Emotion x Diagnostic Group
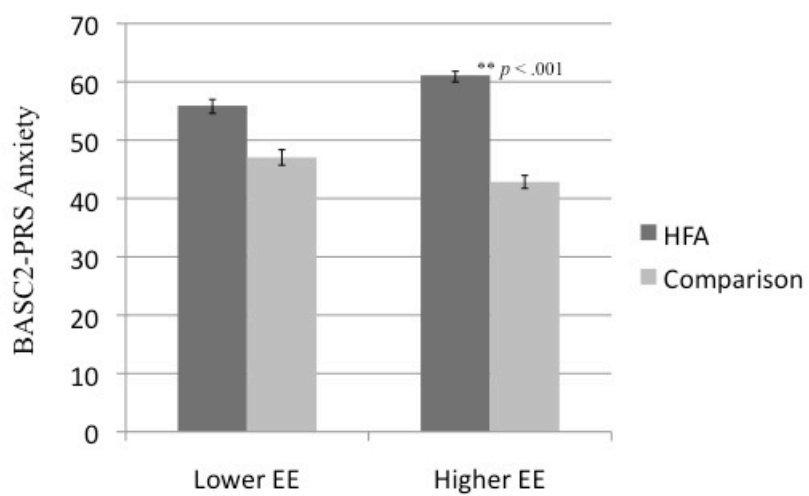
Figure 5. BASC2 PRS Depression Predicted by FES Cohesion x Diagnostic Group
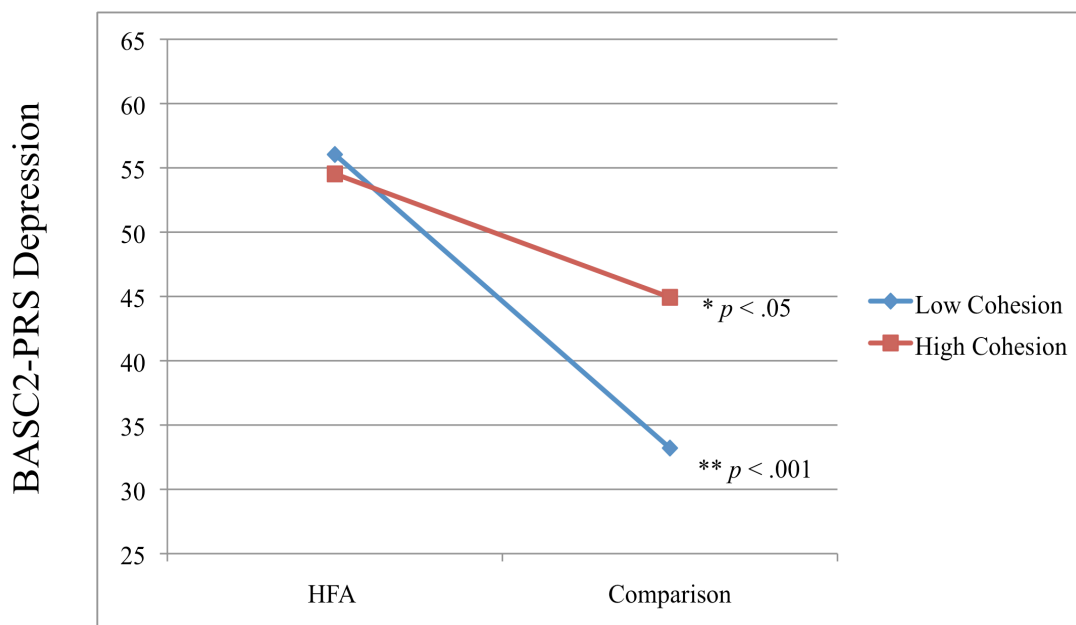
Appendix B

Table 1

Group Descriptions

	HFA	Comparison
	n = 56	*n* = 43
Male	50 (89.3%)	37 (86.0%)
ADHD	-	4 (9.3%)
Anxiety	-	2 (4.7%)
Female	6 (10.7%)	6 (14.0%)
ADHD	-	2 (4.7%)
Anxiety	-	-

Table 2

Group Comparisons on Age, Diagnostic Measures, and Intellectual Functioning

Measures	HFA		Comparison		*F* value	*p* value
	M	*SD*	*M*	*SD*		
	n = 56		*n* = 43			
Age (months)	151.98	29.15	158.93	24.10	1.60	ns
ASSQ[a]	26.71	8.62	5.28	5.66	199.64	<.01
SCQ[b]	19.95	6.39	5.58	3.32	179.57	<.01
ADOS Soc. Int.[c]	8.23	2.99	2.00	3.00	101.18	<.01
ADOS Comm.[d]	4.00	1.61	1.20	1.36	80.20	<.01
ADOS Stereo.[e]	.96	1.41	.00	.00	18.55	<.01
Vocabulary[f]	9.63	2.84	11.63	2.41	13.77	<.01
Similarities[f]	10.88	2.70	11.33	2.74	.67	ns
Estimated VCI[g]	101.55	14.39	108.35	13.73	5.65	<.05
Block Design[f]	10.29	3.46	10.29	3.01	.00	ns
Matrix Reason[f]	10.38	2.85	11.00	2.63	1.25	ns
Estimated PRI[h]	103.31	16.57	104.79	15.13	.20	ns

[a] Autism Spectrum Screening Questionnaire, [b] Social Communication Questionnaire, [c] Autism Diagnostic Observation Schedule (ADOS) Social Interaction, [d] ADOS Communication, [e] ADOS Stereotyped Behaviors, [f] from Wechsler Intelligence Scale for Children-4th Ed. (WISC-IV), [g] WISC-IV Verbal Comprehension Index, [h] WISC-IV Perceptual Reasoning Index

Table 3

Correlations of Social, Emotional, and Behavioral Variables

Variable	1	2	3	4	5	6	7
1. Attn Probs [a]	-						
2. Hyper [b]	.59**	-					
3. Aggress [c]	.44*	.58**	-				
4. Anxiety [d]	.32**	.40**	.26*	-			
5. Depress [e]	.53**	.47**	.48**	.63**	-		
6. Withdraw [f]	.47**	.23*	.12	.54**	.58**	-	
7. SRS Total [g]	.68**	.56**	.40**	.57**	.66**	.75**	-

* *p* < .05

** *p* < .01

[a] Behavior Assessment System for Children-2nd Edition (BASC2) Parent Rating Scale Attention Problems, [b] BASC2 Hyperactivity, [c] BASC2 Aggression, [d] BASC2 Anxiety, [e] BASC2 Depression, [f] BASC2 Withdrawal, [g] Social Responsiveness Scale Total

Table 4

FMSS Group Distribution

	HFA	Comparison	χ^2 value	p value
	$n = 56$	$n = 43$		
Low EE	45	39	48.09	<.01
High EE	11	4		
Lower EE	23	24	.25	ns
Higher EE	33	19		

Table 5

Group Comparisons on Family Factors

Measures	HFA		Comparison		*F* value	*p* value
	M	*SD*	*M*	*SD*		
	n = 56		*n* = 43			
FES Cohesion [a]	53.65	12.06	54.41	13.33	.079	ns
Critical Statem. [b]	.05	.227	0	0	2.33	ns
State. Of Att. [c]	.23	.504	.14	.417	0.87	ns
Pos. Remarks [d]	2.29	2.24	3.93	4.34	5.98	<.05

[a] Family Environment Scale-Cohesion, [b] Five Minute Speech Sample (FMSS) Critical

Statements, [c] FMSS Statements of Attitude, [d] FMSS Positive Remarks

Table 6

MANCOVA of Diagnostic Group x EE Group on Parent Report of Child Externalizing

Behaviors

	Lower EE				Higher EE			
	HFA		Comparison		HFA		Comparison	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
	n = 23		*n* = 22		*n* = 33		*n* = 19	
Hyper [ac]	58.00	8.87	54.09	11.58	64.36	13.68	49.47	10.25
Aggress [b]	50.96	7.73	48.83	7.42	54.91	12.83	47.53	7.23
Attn Probs [a]	60.30	9.47	50.48	11.89	64.79	8.33	46.74	12.16

[a] Effect for diagnostic group

[b] Trend for diagnostic group effect

[c] Trend for diagnostic X EE group effect

Hyper = Behavior Assessment System for Children-2nd Edition (BASC2) Parent Rating

Scale Hyperactivity, Attn Probs = BASC2 Attention Problems, Aggress = BASC2

Aggression

Table 7

Hierarchical Regression Analysis for Variables Predicting BASC-2 PRS Hyperactivity (n = 93)

	Model 1			Model 2			Model 3			Model 4		
Variable	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β
VCI	-.11	.09	-.13	-.05	.09	-.06	-.07	.09	-.08	-.08	.09	-.08
Meds.	5.60	3.03	.19	2.89	2.97	.10	2.73	2.91	.09	2.82	2.95	.10
Diagnostic Group				9.22	2.66	.36**	8.96	2.61	.35**	7.66	5.97	.30
Cohesion							-.00003	.00	-.20*	-.00003	.00	-.23
Group x Cohesion										.000007	.00	.06
Adjusted R^2		.04			.14			.17			.16	
F		2.65			5.98**			5.79**			4.60**	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 8

Hierarchical Regression Analysis for Variables Predicting BASC-2 PRS Aggression (n = 93)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β
VCI	-.04	.07	-.06	-.004	.07	-.01	-.02	.07	-.03	-.03	.07	-.01
Meds.	2.32	2.42	.10	.66	2.43	.03	.53	2.38	.02	-.002	2.36	.00
Diagnostic Group				5.62	2.18	.28*	5.39	2.13	.27*	13.45	4.78	.67**
Cohesion							-.00003	.00	-.23*	-.0000009	.00	-.01
Group x Cohesion										-.00004	.00	.48
Adjusted R^2		-.01			.05			.10			.12	
F		.67			2.69			3.40*			3.50**	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 9

Hierarchical Regression Analysis for Variables Predicting BASC-2 PRS Attention Problems (n = 93)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*
VCI	-.21	.09	-.24*	-.12	.08	-.14	-.13	.08	-.14	-.11	.08	-.12
Meds.	6.06	2.90	.21*	2.03	2.55	.07	2.00	2.57	.07	1.55	2.57	.05
Diagnostic Group				13.68	2.29	.54**	13.63	2.30	.53**	20.46	5.20	.80**
Cohesion							-.000006	.00	-.04	.00002	.00	.11
Group x Cohesion										-.00004	.00	-.32
Adjusted R^2		.09			.34			.34			.35	
F		5.61**			17.08**			12.75**			10.76**	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 10

MANCOVA of Diagnostic Group x EE Group on Parent Report of Child Internalizing Problems

	Lower EE				Higher EE			
	HFA		Comparison		HFA		Comparison	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
	n = 23		*n* = 22		*n* = 33		*n* = 19	
Anxiety [ab]	55.91	12.35	47.04	14.46	61.12	11.30	42.84	10.62
Depression [a]	60.30	14.87	47.22	13.65	65.91	14.89	47.00	8.84
Withdraw. [a]	69.43	13.20	50.39	13.00	70.52	14.23	48.16	11.01

[a] Effect for diagnostic group

[b] Trend for diagnostic group x EE group effect

Anxiety = Behavior Assessment System for Children-2nd Edition (BASC2) Parent Rating

Scale Anxiety, Depression = BASC2 Depression, Withdraw = BASC2 Withdrawal

Table 11

Hierarchical Regression Analysis for Variables Predicting BASC-2 PRS Anxiety (n = 93)

Variable	Model 1 B	Model 1 $SE\ B$	Model 1 β	Model 2 B	Model 2 $SE\ B$	Model 2 β	Model 3 B	Model 3 $SE\ B$	Model 3 β	Model 4 B	Model 4 $SE\ B$	Model 4 β
VCI	-.12	.10	-.12	-.02	.09	-.02	-.02	.09	-.02	-.02	.10	-.02
Meds.	.04	3.45	.001	-4.30	3.14	-.13	-4.26	3.16	-.13	-4.26	3.20	-.13
Diagnostic Group				14.72	2.82	.51**	14.78	2.83	.51**	14.72	6.48	.51*
Cohesion							.000007	.00	.04	.000007	.00	.04
Group x Cohesion										.0000003	.00	.003
Adjusted R^2		-.01			.22			.21			.21	
F		.71			9.71**			7.26**			5.74**	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 12

Hierarchical Regression Analysis for Variables Predicting BASC-2 PRS Depression (n = 93)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*
VCI	-.11	.11	-.10	-.001	.10	-.001	-.01	.11	-.01	.02	.10	.02
Meds.	6.92	3.82	.19	2.28	3.52	.06	2.21	3.52	.06	1.28	3.47	.04
Diagnostic Group				15.73	3.15	.49**	15.61	3.16	.48**	26.70	7.03	.92**
Cohesion							-.00001	.00	-.08	.00003	.00	.16
Group x Cohesion										-.00007	.00	-.52*
Adjusted R^2		.03			.23			.23			.26	
F		2.22			10.18**			7.78**			7.51**	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 13

Hierarchical Regression Analysis for Variables Predicting BASC-2 PRS Withdrawal (n = 93)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β
VCI	-.07	.12	-.06	.08	.10	.07	.09	.10	.08	.10	.10	.08
Meds.	7.00	3.97	.18	.76	3.30	.02	.84	3.30	.02	.61	3.33	.02
Diagnostic Group				21.19	2.96	.63**	21.33	2.96	.63**	24.75	6.75	.74**
Cohesion							.00002	.00	.09	.00003	.00	.15
Group x Cohesion										-.00002	.00	-.12
Adjusted R^2		.02			.37			.37			.37	
F		1.79			18.98**			14.54**			11.61**	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 14

MANCOVA of Diagnostic Group x EE Group on Parent Report of Social Symptoms

	Lower EE				Higher EE			
	HFA		Comparison		HFA		Comparison	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
	n = 23		*n* = 22		*n* = 33		*n* = 19	
SRS Total [a]	78.65	11.86	48.00	9.69	81.61	10.26	48.21	12.17

[a] Effect for diagnostic group

SRS Total = Social Responsiveness Scale Total

Table 15

Hierarchical Regression Analysis for Variables Predicting Social Responsiveness Scale Total (n = 93)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	*SE B*	β	B	*SE B*	β	B	*SE B*	β	B	*SE B*	β
VCI	-.24	.13	-.18	-.03	.08	-.02	-.03	.08	-.03	-.02	.08	-.01
Meds.	10.72	4.48	.24*	1.17	2.67	.03	1.11	2.67	.03	.71	2.68	.02
Diagnostic Group				32.39	2.39	.83**	32.38	2.40	.83**	32.38	5.43	.98**
Cohesion							-.00001	.00	-.06	.000006	.00	.03
Group x Cohesion										-.00003	.00	-.18
Adjusted R^2		.08			.70			.70			.70	
F		4.86*			70.86*			53.39**			43.27**	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 16

MANCOVA of Diagnostic Group x EE Group on Behavioral Controllability

	Lower EE				Higher EE			
	HFA		Comparison		HFA		Comparison	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
	n = 18		*n* = 19		*n* = 29		*n* = 17	
CSC [a]	-.53	.53	.84	.79	-.61	.71	.67	.98
PSC [a]	-.53	.67	.67	.89	-.49	.82	.68	.91
Pos. Attrib.	.17	.38	.47	.70	.28	.53	.47	.80
Neg. Attrib.	.72	1.32	.42	1.02	.76	1.15	.41	.80
Neut. Attrib.[b]	1.00	1.18	1.32	.95	.83	.76	.24	.44

[a] Effect for diagnostic group

[b] Effect for EE group

CSC = CBQ Child Symptom Control, PSC = CBQ Parent Symptom Control, Pos. Attrib.

= FMSS Positive Attributions, Neg. Attrib. = FMSS Negative Attributions, Neut. Attrib.

= FMSS Neutral Attributions

Table 17

Hierarchical Regression Analysis for Variables Predicting CBQ Child Symptom Control (n = 80)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β
VCI	.02	.01	.25*	.01	.01	.12	.01	.01	.14	.01	.01	.14
Meds.	-.42	.25	-.19	-.09	.20	-.04	-.07	.20	-.03	-.10	.21	-.05
Diagnostic Group				-1.26	.18	-.62**	-1.25	.18	-.62**	-.90	.43	-.45*
Cohesion							.0000009	.00	.08	.000002	.00	.18
Group x Cohesion										-.000002	.00	-.20
Adjusted R^2		.09			.43			.43			.43	
F		4.91*			21.04**			15.98**			12.91**	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 18

Hierarchical Regression Analysis for Variables Predicting CBQ Parent Symptom Control (n = 80)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β
VCI	.01	.01	.11	-.001	.01	-.02	.00	.01	.004	.001	.01	.01
Meds.	-.31	.26	-.14	-.002	.22	-.001	.03	.22	.01	-.002	.23	-.001
Diagnostic Group				-1.19	.20	-.58**	-1.17	.20	-.58**	-.83	.47	-.41
Cohesion							.000001	.00	.12	.000002	.00	.22
Group x Cohesion										-.000002	.00	-.20
Adjusted R^2		.01			.31			.32			.31	
F		1.39			12.84**			10.08**			8.16**	

$* p < .05, ** p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 19

Hierarchical Regression Analysis for Variables Predicting FMSS Positive Attributions (n = 93)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β
VCI	.002	.01	.04	.00	.01	-.01	.002	.01	.04	.002	.01	.05
Meds.	.13	.16	.10	.21	.16	.15	.25	.16	.18	.22	.16	.16
Diagnostic Group				-.31	.15	-.25*	-.29	.14	-.23*	.09	.33	.07
Cohesion							.000002	.00	.23*	.000003	.00	.40*
Group x Cohesion										.000002	.00	-.36
Adjusted R^2		-.02			.03			.07			.07	
F		.38			1.70			2.42			2.26	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 20

Hierarchical Regression Analysis for Variables Predicting FMSS Negative Attributions (n = 93)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	*SE B*	β	B	*SE B*	β	B	*SE B*	β	B	*SE B*	β
VCI	-.02	.01	-.21	-.02	.01	-.19	-.02	.01	-.18	-.02	.01	-.19
Meds.	.46	.28	.18	.40	.29	.17	.43	.29	.17	.46	.30	.18
Diagnostic Group				.14	.26	.06	.15	.26	.07	-.16	.61	-.07
Cohesion							.0000006	.00	.05	-.0000004	.00	-.03
Group x Cohesion										.000002	.00	.16
Adjusted R^2		.07			.06			.05			.04	
F		3.73*			2.56			1.95			1.61	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 21

Hierarchical Regression Analysis for Variables Predicting FMSS Neutral Attributions (n = 93)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*
VCI	.02	.01	.22*	.02	.01	.23*	.02	.01	.23	.02	.01	.22
Meds.	.23	.24	.11	.21	.24	.10	.20	.25	.10	.23	.25	.11
Diagnostic Group				.07	.22	.04	.07	.23	.04	-.18	.52	-.10
Cohesion							-.0000003	.00	-.03	-.000001	.00	-.10
Group x Cohesion										.000001	.00	.16
Adjusted R^2		.03			.02			.01			-.004	
F		2.21			1.49			1.12			.94	

* *p* < .05, ** *p* < .001

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 22

MANCOVA of Diagnostic Group x EE Group on Parent Report of Stress

	Lower EE				Higher EE			
	HFA		Comparison		HFA		Comparison	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
	n = 21		*n* = 18		*n* = 31		*n* = 13	
SCL GSI	50.95	10.97	50.00	10.82	55.23	11.76	48.00	8.88
Neg. Ev.	52.29	11.34	52.28	11.48	53.55	11.36	46.23	8.69
Soc.Eval.	51.05	7.433	48.67	6.03	48.74	10.57	44.31	8.85
Leyton Tr.	7.10	4.69	6.56	4.36	7.65	4.83	6.77	3.83

SCL GSI = Symptom Checklist 90-Revised Global Severity Index, Neg. Ev. = Life Stress and Resources Inventory Negative Life Events Subscale, Soc. Eval. = Endler Multidimensional Anxiety Scale Social Evaluation Subscale, Leyton Tr. = Leyton Obsessional Inventory Trait Subscale

Table 23

Hierarchical Regression Analysis for Variables Predicting Symptom Checklist 90-R Global Severity Index (n = 83)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β
VCI	-.01	.09	-.01	.02	.05	.02	.02	.09	.002	-.01	.09	-.01
Meds.	-1.38	2.84	-.06	-2.93	2.90	-.12	-3.36	2.90	-.13	-3.10	2.94	-.12
Diagnostic Group				5.14	2.63	.23	5.14	2.61	.23	1.40	6.25	.06
Cohesion							-.00002	.00	-.16	-.00003	.00	-.25
Group x Cohesion										.00002	.00	.20
Adjusted R^2		-.02			.01			.03			.02	
F		.12			1.36			1.53			1.30	

$* p < .05, ** p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 24

Hierarchical Regression Analysis for Variables Predicting Life Stress and Resources Inventory Negative Life Events (n = 83)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β
VCI	-.07	.09	-.10	-.06	.09	-.08	-.06	.09	-.08	-.08	.09	-.10
Meds.	2.01	2.82	.08	1.18	2.93	.05	1.09	2.96	.04	1.42	3.00	.06
Diagnostic Group				2.74	2.66	.12	2.75	2.67	.12	-1.94	6.38	-.09
Cohesion							-.000004	.00	-.03	-.00002	.00	.25
Group x Cohesion										.00002	.00	.25
Adjusted R^2		-.01			-.01			-.02			-.02	
F		.65			.79			.61			.61	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 25

Hierarchical Regression Analysis for Variables Predicting Endler Multidimensional Anxiety Scale Social Evaluation (n = 83)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*
VCI	.01	.07	.02	.03	.07	.05	.02	.07	.03	.03	.07	.05
Meds.	-2.46	2.17	-.13	-3.63	2.22	-.19	-4.01	2.21	-.21	-4.26	2.24	-.22
Diagnostic Group				3.87	2.02	.22	3.87	2.00	.22	7.40	4.77	.42
Cohesion							-.00002	.00	-.18	-.000006	.00	-.06
Group x Cohesion										-.00002	.00	-.24
Adjusted R^2		-.01			.03			.04			.04	
F		.67			1.69			1.93			1.67	

* *p* < .05, ** *p* < .001

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage

Table 26

Hierarchical Regression Analysis for Variables Predicting Leyton Obsessional Inventory Trait Total (n = 83)

Variable	Model 1			Model 2			Model 3			Model 4		
	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β	B	$SE\ B$	β
VCI	-.02	.03	-.08	-.02	.03	-.06	-.02	.03	-.08	-.02	.04	-.08
Meds.	-2.19	1.12	-.21	-2.60	1.16	-.25*	-2.74	1.16	-.27*	-2.71	1.18	-.26*
Diagnostic Group				1.35	1.05	.15	1.35	1.05	.15	.99	2.52	.11
Cohesion							-.000007	.00	-.13	-.000008	.00	-.15
Group x Cohesion										.000002	.00	.05
Adjusted R^2		.03			.03			.04			.03	
F		2.11			1.96			1.82			1.45	

* $p < .05$, ** $p < .001$

VCI = Wechsler Intelligence Scale for Children-4th Edition (WISC-IV) Verbal Comprehension Index, Meds = Medication usage