CO-OCCURRENCE OF OPPOSITIONAL DEFIANT DISORDER WITH GENERALIZED AND SEPARATION ANXIETY DISORDERS AMONG INNER-CITY CHILDREN

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ABSTRACT

Co-occurrence of Oppositional Defiant Disorder with Generalized and Separation Anxiety Disorders Among Inner-City Children

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There is a paucity of research that has examined co-occurring oppositional defiant disorder and generalized anxiety disorder (ODD+GAD) symptoms and oppositional defiant disorder and separation anxiety disorder (ODD+SAD) symptoms among children. To address this gap, I investigated multiple explanations for the co-occurrence of ODD+GAD and ODD+SAD. Specifically, I investigated whether (a) GAD symptoms prospectively predicted ODD symptoms and SAD symptoms prospectively predicted ODD symptoms (Explanation 1), (b) ODD symptoms prospectively predicted GAD symptoms and ODD symptoms prospectively predicted SAD symptoms (Explanation 2), and (c) shared risk processes accounted for the co-occurrence of ODD+GAD and ODD+SAD (Explanation 3). Participants were an ethnic minority, inner-city sample of first through fourth grade children (N = 88, 51% male) and their primary caregivers. I used data collected at the baseline and 1-year follow-up assessments of the Child Health and Behavior Study, a longitudinal survey of families residing in North Philadelphia. Findings provided support for Explanation 2 and Explanation 3 in the development of co-occurring ODD+GAD symptoms and support for Explanation 3 in the development of co-occurring ODD+SAD symptoms. This study contributes to the extant literature by providing the first empirical examination of these multiple explanations in an ethnic minority, inner city sample of children.

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TABLE OF CONTENTS

Page

LIST OF TABLES

LIST OF FIGURES

Figure Page

CHAPTER 1
INTRODUCTION

Oppositional defiant disorder (ODD), defined by a pattern of negativistic, hostile, and oppositional behaviors toward adults, significantly co-occurs with both generalized anxiety disorder (GAD) and separation anxiety disorder (SAD; Angold, Costello, & Erkanli, 1999a; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Drabick, Gadow, & Loney, 2008; Garland & Garland, 2001; Kendall, Brady, & Verduin, 2001; Loeber & Keenan, 1994; Verduin & Kendall, 2003). GAD is characterized by excessive anxiety and worry in multiple domains of the child's life, whereas SAD is characterized by developmentally inappropriate, excessive, and persistent worry about separation from attachment figures (APA, 2000). Previous results suggest that GAD and SAD are equally likely to co-occur with ODD (Verduin & Kendall, 2003). This observed co-occurrence of ODD with GAD (ODD+GAD) and ODD with SAD (ODD+SAD) has important ramifications with regard to level of impairment, course of the symptoms and disorders, and treatment.

Generally, research has found that children with comorbid problems exhibit greater levels of impairment and are at higher risk of developing more severe psychopathology than children with single diagnoses (Kendall et al., 2001). For example, the presence of ODD and overanxious disorder, which was replaced by GAD in the *Diagnostic and Statistical Manual of Mental Disorders – 4th edition* (*DSM-IV*; APA, 1994), predicts subsequent depression among adolescent boys (Burke, Loeber, Lahey, & Rathouz, 2005). In terms of treatment, ODD likely alters the course and treatment response for children with GAD or SAD (Ferguson, 2002; Flannery-Shroeder, Suveg, Safford, Kendall, & Webb, 2004; Garland & Garland, 2001; Kendall, Panichelli-Mindel, Sugarman, & Callahan, 1997).

When examined independently, ODD, GAD, and SAD have similar negative outcomes and correlates, such as impairment in peer relations and academic performance (Frankel & Feinberg, 2002; Grover, Ginsburg, & Ialongo, 2005; Last, Hansen, & Franco, 1997; Matthys, Cuperus, & van England, 1999; Shaffer, Fisher, Dulcan, & Davies, 1996); elevated levels of substance use (Dishion, French, & Patterson, 1995; Kandel et al., 1997); and elevated levels of depression (Angold & Costello, 1993; Brady & Kendall, 1992). Despite these negative outcomes and significant covariation, little research has examined ODD+GAD and ODD+SAD symptoms, and research that has examined these comorbidities tends to rely on clinic-based samples (Drabick et al., 2008; Garland & Garland, 2001; Verduin & Kendall, 2003). In addition, much of the literature examining these disorders to date has been descriptive in nature, and/or has combined anxiety disorders (e.g., SAD, GAD, and social phobia) and externalizing disorders (e.g., ODD, conduct disorder (CD); Boylan, Vaillancourt, Boyle, & Szatmari, 2007; Gregory, Eley, & Plomin, 2004; Kolko, Baumann, Bukstein, & Brown, 2007; Mireault, Rooney, Kouwenhoven, & Hannan, 2008), which limits the understanding of specific relations between disorders. Furthermore, research has failed to consider potential mechanisms and shared processes that may contribute to the development of comorbid ODD+GAD, as well as ODD+SAD. Given these notable gaps in the literature, I examined multiple explanations for the co-occurrence of ODD+GAD and ODD+SAD symptoms in an ethnic minority, inner-city sample of children. The framework used to conceptualize possible explanations for the development of co-occurring ODD+GAD and ODD+SAD is presented next.

Possible Explanations for the Co-occurrence of ODD+GAD and ODD+SAD

Various authors have suggested frameworks for conceptualizing comorbidity of psychological syndromes among children (Angold et al., 1999a; Caron & Rutter, 1991; Drabick,

2009b; Klein & Riso, 1993; Seligman & Ollendick, 1998). There are at least three explanations stemming from these frameworks that may be useful for conceptualizing the development of co-occurring ODD+GAD and ODD+SAD. These explanations include the following: (a) GAD and/or SAD symptoms confer risk for ODD symptoms, (b) ODD symptoms confer risk for GAD and/or SAD symptoms, and (c) shared risk factors account for the co-occurrence of ODD+GAD and ODD+SAD. Despite support for each of these explanations garnered from separate studies, I am unaware of any research that has concurrently examined each of these explanations for the co-occurrence of ODD and anxiety disorders in the same sample of children. In the current study, I propose to examine these three potential explanations to evaluate which explanation best accounts for the co-occurrence of ODD+GAD and ODD+SAD symptoms among inner-city children, as well as which variables may be associated with symptom co-occurrence (e.g., as mediators or potential shared risk factors).

Explanation 1: GAD and/or SAD Symptoms Confer Risk for ODD Symptoms

Evidence for the explanation that GAD and SAD symptoms may confer risk for ODD is limited. Foley, Pickles, Maes, Silberg, and Eaves (2004) examined SAD and ODD, and found that 19% of children with transient SAD (i.e., only at initial interview) and 15% of children with persistent SAD (i.e., at initial interview and at follow up approximately 18 months later) met diagnostic criteria for ODD at follow up. Given limited research examining the co-occurrence of these diagnostic categories, another way to consider the relations among these disorders is to examine the symptoms associated with GAD/SAD and ODD. Though not equivalent to a diagnosis, the symptoms associated with these disorders often cause significant impairment, even at subthreshold levels (Angold, Costello, Farmer, Burns, & Erkanli, 1999b) and these symptoms may represent important precursors to their respective syndromes. Using this approach, studies

examining anxiety and externalizing symptoms (e.g., aggression) dimensionally, rather than categorically, have found a positive relationship between anxiety in the fall of first grade and aggression in the spring of first grade (Ialongo, Edelsohn, Werthamer-Larsson, & Kellam, 1994).

One mechanism that may lead an anxious child to exhibit externalizing behaviors (e.g., ODD symptoms) involves the child's information processing. Specifically, researchers have posited that anxious children exhibit aggressive behavior as a result of fearing both their environment and the people with whom they must interact (Kashani, Deuser, & Reid, 1991). As such, the anxious child may be sensitized to react aggressively in response to even minor provocations, in turn eliciting aggression or other negative consequences from peers, parents, and/or teachers (Ialongo, Edelsohn, Werthamer-Larsson, Crockett, & Kellam, 1996). These negative consequences may reinforce the child's perception of the environment as threatening, which in turn could perpetuate a cognitive bias. Indeed, research has shown that both anxious and aggressive children tend to view the world and others as hostile (Dodge, Bates, & Pettit, 1990; Dodge, Price, Bachorowski, & Newman, 1990; Hoglund & Leadbeater, 2007; Muris et al., 2000a; Muris, Luermans, Merckelbach, & Mayer, 2000b; Orbio de castro, Veerman, Koops, Bosch, & Monshouwer, 2002). Therefore, hostile attribution biases may be one mechanism by which anxiety symptoms contribute to the onset of future externalizing problems.

Explanation 2: ODD Symptoms Confer Risk for GAD and/or SAD Symptoms

Relatively more evidence has been found for the explanation that ODD confers risk for GAD and/or SAD than for the explanation that GAD and/or SAD confer risk for ODD. Among community-based samples, children with persistent ODD in preschool are at risk for developing comorbid anxiety in later childhood years (Lavigne et al., 2001). Similarly, Burke and colleagues (2005) found that ODD in childhood predicts the presence of anxiety in adolescence and

adulthood in a clinic-based sample. Among adolescents, community-based studies indicate that the diagnosis of an anxiety disorder is more likely given a diagnosis of ODD (Kashani et al., 1987). Although there are various possibilities, two mechanisms that may account for the development of anxiety subsequent to the expression of externalizing behaviors include peer rejection and poor academic skills. Children with externalizing disorders are often rejected by their peers (Little & Garber, 1995), perhaps because of the social skills deficits often associated with externalizing symptoms (Frankel & Feinberg, 2002) that lead to social failures. This peer rejection, in turn, can lead to the development of anxiety (Bell-Dolan, 1995; Bell-Dolan, Foster, & Christopher, 1995). In support of this possibility, Bell-Dolan and colleagues found that peer-rejected girls exhibited higher levels of social anxiety symptoms as compared to their non-rejected peers. Moreover, among boys, experiencing peer rejection can lead to subsequent anxiety about expected peer rejection in the future (London, Downey, Bonica, & Paltin, 2007). Though speculative, if children become anxious about their social interactions as a result of peer rejection, their anxiety then may generalize to include excessive worry in multiple domains of their lives (e.g., other interpersonal interactions), which eventually could be manifested as GAD.

In terms of basic academic skills, school-related difficulties among children with externalizing problems include poor grades and academic deficits (Hinshaw, 1992). An association between anxiety and poor academic skills also has been documented (Ialongo, Edelsohn, Werthamer-Larsson, Crockett, & Kellam, 1995; Rapport, Denney, Chung, & Hustace, 2001). For instance, children who experience academic failures in the first grade exhibit higher levels of anxiety at 6-month follow up (Grover et al., 2005). Therefore, children with externalizing behaviors who are underachieving academically may develop an awareness of their academic difficulties, which could contribute to anxiety and worry about poor academic

performance. In this way, poor academic skills may be a potential mechanism linking externalizing behaviors with the subsequent development of anxiety symptoms.

Explanation 3: Shared Risk Factors Account for the Co-occurrence of ODD+GAD and ODD+SAD

A third possibility involves the shared risk factors explanation for comorbidity, which suggests that anxiety symptoms and ODD symptoms are associated with overlapping and unique factors, and that comorbidity stems from shared risk factors (Angold et al., 1999a; Caron & Rutter, 1991; Klein & Riso, 1993). For example, literature suggests that ODD, GAD, and SAD symptoms are associated with indices of difficult temperament, but that specific temperamental processes may differentially predict ODD, GAD, and SAD symptoms. Few studies concurrently have examined potential overlapping and/or unique risk factors for ODD+GAD and ODD+SAD. Based on the available literature, I selected the following risk factors that have been linked to both anxiety and externalizing symptoms: difficult temperament, autonomic functioning, stressful life events, and neighborhood danger. These particular constructs were chosen to provide a range of factors that would either overlap (i.e., shared process) or provide unique prediction to ODD, GAD, and SAD symptoms. Moreover, from a developmental psychopathology perspective, it is critical to consider both child-specific and contextual variables as risk processes (e.g., Drabick, 2009b; Steinberg & Avenevoli, 2000). Thus, these processes include variables that are operationalized at each of these levels. Evidence for each of these processes is considered next.

Difficult temperament. Difficult temperamental styles, which include features of negative emotionality (i.e., proneness to experience feelings of anger, frustration, sadness, anxiety, and fearfulness; Rothbart & Bates, 2006) and lack of self regulation (i.e., poor attentional control,

impulsivity, and poor inhibitory control; Muris & Ollendick, 2005), have been linked to both anxiety and externalizing symptoms (Bates, Pettit, Dodge & Ridge, 1998; Carrasco Ortiz & del Barrio Gandara, 2006; Caspi, Henry, McGee, Moffitt, & Silva, 1995; Guerin, Gottfried, & Thomas, 1997; Keenan, Shaw, Delliquadri, Giovannelli, & Walsh, 1998; Shaw, Keenan, Vondra, Delliquadri, & Giovannelli, 1997). Evidence suggests temperament is linked to externalizing and anxiety disorders through negative emotionality (Derryberry & Rothbart, 1997; Rettew & McKee, 2005; Rothbart & Bates, 2006). In particular, temperamental anger and frustration are strongly linked to externalizing behaviors (Burke, Loeber, & Birmaher, 2002; Cole, Zahn-Waxler, & Smith, 1994; Eisenberg et al., 2001), whereas temperamental fearfulness typically precedes anxiety symptoms (Guerin et al., 1997; Rothbart & Bates, 1998). In terms of self-regulation processes, impulsivity, or speed of response initiation, has been linked to externalizing behaviors (Ackerman, Brown, & Izard, 2003; Olson, Schilling, & Bates, 1999). Furthermore, inhibitory control, which is important for the development of effortful control (Rothbart, Ahadi, Hershey, & Fisher, 2001) and children's ability to regulate their emotions (Carlson & Wang, 2007), has been linked to both internalizing and externalizing symptoms (Lemery, Essex, & Smider, 2002; Rettew & McKee, 2005). Based on these findings, I would hypothesize that certain aspects of difficult temperament, such as inhibitory control, may be related to comorbid anxiety and ODD symptoms, whereas others may be specifically associated with anxiety symptoms (e.g., fearfulness) or ODD behaviors (e.g., anger, impulsivity).

Autonomic functioning. Autonomic functioning also has been associated with anxiety and externalizing symptoms in childhood; however, the relation of autonomic functioning to comorbid anxiety and externalizing symptoms is less clear. As a brief overview, the autonomic nervous system (ANS) controls basic visceral functions of the body, such as cardiovascular

activity and metabolism, and is composed of two divisions, the parasympathetic (PNS) and sympathetic (SNS) nervous systems. Generally speaking, the SNS regulates involuntary reactions to stress (e.g., increased heart and breathing rates, stimulation of sweat glands) and prepares the body for action in the context of stressors. The PNS, in contrast, promotes growth and restorative processes. Sympathetic activation is indexed by pre-ejection period (PEP), and shorter PEPs are associated with sympathetic activation. Parasympathetic cardiac influences are indexed by respiratory sinus arrhythmia (RSA), or the waxing and waning of heart rate across the respiratory cycle (Porges, 1995). RSA typically is used as an estimate of vagal tone because it is a proxy for regulatory processes that cannot readily be measured non-invasively. In terms of the relation between RSA and vagal tone, RSA results from *decreases* in vagal efference during inhalation, which increase heart rate, and *increases* in vagal efference during exhalation, which decreases heart rate (Beauchaine, 2001). Measurement of PEP and RSA at rest appear to reflect temperamental reactivity and emotionality (Beauchaine, 2001), whereas measurement during challenge or in response to a stressor indexes reactivity and self regulation (Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996).

Work by Beauchaine and colleagues has shown that children diagnosed with ODD exhibit sympathetic and parasympathetic deficiencies (i.e., decreased PEP and RSA at baseline and lower levels of PEP and RSA modulation), though associations differ depending on the age period considered (Beauchaine, Gatzke-Kopp, & Mead, 2007; Beauchaine, Katkin, Strassberg, & Snarr, 2001). For instance, preschool children with ODD exhibit attenuated SNS, but not PNS, activity (Beauchaine et al., 2007; Crowell et al., 2006). In middle childhood and adolescence, ODD is associated with both attenuated SNS and PNS activity (Beauchaine et al., 2001, 2007), which suggests that PNS activity may come “online” during middle childhood and SNS

deficiencies may emerge before reductions in PNS activity. At this point, the exact timing of the emergence of PNS deficiencies is unclear. However, previous research in this sample suggests that PNS deficiencies associated with ODD symptoms may not be present in middle childhood among impoverished, ethnic minority children residing in the inner city (Bubier & Drabick, 2008).

In terms of identifying potential shared autonomic processes for ODD and anxiety symptoms, research suggests that children with externalizing problems and ODD, specifically, become hyperaroused when provoked and in stressful situations (Hubbard et al., 2002; Tyson, 1998; van Goozen et al., 1998; van Lang et al., 2007). Coupled with the work of Beauchaine and colleagues, these findings suggest that children with externalizing symptoms may exhibit dysregulated levels of autonomic functioning, such that they are underaroused in most situations and hyperaroused when provoked or under stress. In contrast, anxiety among children consistently has been linked to autonomic hyperarousal, as measured by increases in heart rate and skin conductance level and decreases in RSA at baseline, and does not appear to differ across developmental periods (Greaves-Lord et al., 2007; Roth et al., 2008; van Lang et al., 2007). Given these findings, it may be that children with externalizing symptoms and those with anxiety exhibit similar physiological hyperarousal under specific conditions that induce negative emotions (e.g., when under stress). One notable study addressing this issue found that boys with comorbid disruptive behavior disorders and anxiety problems had stronger autonomic responses to a difficult computer task intended to induce frustration than boys with only externalizing problems (van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van England, 2000). This finding suggests that boys with co-occurring anxiety and externalizing symptoms may exhibit greater autonomic hyperarousal when experiencing frustration. Therefore, it is possible that the tendency

to become autonomically hyperaroused in certain situations may confer risk for the development of both anxiety and externalizing symptoms. It remains unclear, however, whether anxiety and externalizing symptoms are associated with hyperarousal under conditions that induce negative emotions other than frustration, such as fear.

In sum, it is likely that certain autonomic processes are unique in their association with either ODD symptoms or anxiety symptoms, whereas other processes that predict ODD and anxiety symptoms may overlap. For example, attenuated autonomic functioning may confer risk for ODD symptoms, whereas autonomic hyperarousal may confer risk for anxiety symptoms. In terms of a shared process, autonomic hyperarousal under conditions that induce negative emotions may confer risk for both ODD and anxiety symptoms. In addition to child-specific variables (i.e., difficult temperament and autonomic processes), various contextual level variables also have been shown to confer risk for ODD, GAD, and SAD. Two such contextual factors were chosen for examination in the present study, namely (a) neighborhood danger and (b) stressful life events.

Neighborhood danger. Disadvantaged urban communities often are characterized by high rates of violence, and neighborhood violence exposure is associated with numerous childhood problems, including anxiety and externalizing behaviors (Attar, Guerra, & Tolan, 1994; Gorman-Smith & Tolan, 1998; Guerra, Huesmann, & Spinder, 2003; Martinez & Richters, 1993; Singer, Anglin, Song, & Lunghofer, 1995; Youngstrom, Weist, & Albus, 2003). Externalizing behaviors among children exposed to neighborhood violence may develop because children exposed to neighborhood violence model aggressive behavior. This modeling of aggressive behavior may lead the child to develop positive outcome and efficacy beliefs for aggression, as well as the perception that aggression is an appropriate response to ambiguous peer provocation (Schwartz

& Proctor, 2000). In addition, exposure to neighborhood violence may induce fear among children and result in anxiety symptoms (Chorpita & Barlow, 1998). Regardless of the temporal ordering of exposure to violence and child symptoms, it likely that a child's perception of neighborhood danger is associated with risk for ODD and/or anxiety symptoms.

Stressful life events. Life events that are construed as stressful, such as a family move, change in a family's financial state, and death of a close family member, also may confer risk for childhood anxiety and externalizing problems (Goodwin, Fergusson, & Horwood, 2004; Morales & Guerra, 2006). For example, there is a large literature linking economic disadvantage to greater risk for adjustment difficulties in childhood (Ackerman, Brown, & Izard, 2004; McLoyd, 1998). In addition, increases in family disruption and transitions have been linked to both externalizing and internalizing problems (Forehand, Biggar, & Kotchik, 1998; Shaw & Emery, 1988). Despite associations among specific stressors, externalizing, and anxiety symptoms, it is clear that stressors rarely occur in isolation. Thus, it may be the combination or number of stressors, as opposed to the specific type of stressor, that is associated with internalizing and externalizing symptoms (Morales & Guerra, 2006; Rutter, Cox, Tupling, Berger, & Yule, 1975). This combination or number of stressors may be especially important to consider among children growing up in impoverished communities, where exposure to multiple stressors is more likely. In support of this possibility, research has documented a relation between numbers of stressful life experiences and internalizing and externalizing problems. For example, Boer et al. (2002) found that parents of anxiety-disordered children endorsed significantly more negative life events (e.g., death of a grandparent, relocation of family, marital conflict) than parents of control children. It may be that an increased number of stressful life events leads to decreased structure and routine in the home, which in turn may lead to increased behavior problems and/or anxiety. Therefore, I

would hypothesize that among children living in the inner city, the number of stressful life events may confer risk for both anxiety and ODD symptoms.

It is important to note that, despite these hypothesized associations, the usefulness of the above mentioned shared risk factors for understanding co-occurring ODD+GAD and ODD+SAD is somewhat limited. First, these risk factors are not specific to co-occurring ODD+GAD or ODD+SAD, which limits their utility for etiological and intervention models for these co-occurring symptoms (Steinberg & Avenevoli, 2000). Second, the labeling of these correlates as "shared" risk factors is arbitrary because these factors have not been tested effectively in other viable roles, such as mediators or moderators (Drabick, 2009a, 2009b). For example, many of these risk factors could be included in explanations 1 and 2 above as the mechanisms that facilitate development of the co-occurring condition (e.g., autonomic hyperarousal when experiencing negative emotions may account for the prospective relation between ODD and SAD symptoms). Thus, it is plausible that these factors each play a role in the development of co-occurring symptoms, but their effects differ depending on when they are experienced. Taken together, these limitations suggest that researchers must use caution when choosing potential shared risk factors for inclusion in comorbidity models, and provide a further impetus to develop and test conceptual models that hypothesize distinct roles for factors (i.e., moderation vs. mediation) thought to be involved in the onset or maintenance of childhood psychopathology.

In sum, authors have offered useful frameworks for conceptualizing comorbidity of psychological syndromes among children (Angold et al., 1999a; Caron & Rutter, 1991; Drabick, 2009b; Klein & Riso, 1993; Seligman & Ollendick, 1998). Three explanations that can be specified from these frameworks have received some support when applied to co-occurring ODD+GAD and ODD+SAD: (a) GAD and/or SAD symptoms confer risk for ODD symptoms,

(b) ODD symptoms confer risk for GAD and/or SAD symptoms, and (c) shared risk factors account for the co-occurrence of ODD+GAD and ODD+SAD. Potential candidates for the shared risk processes associated with ODD+GAD and ODD+SAD include difficult temperament, autonomic functioning, neighborhood danger, and stressful life events. At this point in time, identifying the most optimal explanation requires concurrent examination of each alternative across time within the same sample, something that has not been accomplished to date.

Gaps in the Literature

Currently, there are a number of gaps in the literature that examines ODD in relation to GAD and SAD. First, there has been a relative dearth of literature that examines co-occurring ODD+GAD and ODD+SAD symptoms in the same study. Furthermore, there is limited research that has examined anxiety symptoms separately (e.g., GAD vs. SAD symptoms) and that has examined ODD independently of other disruptive behavior disorder symptoms (e.g., attention-deficit/hyperactivity disorder and CD). The lack of explicit attention to co-occurring ODD, GAD, and SAD symptoms is a critical gap in the literature given that children with comorbid problems exhibit greater levels of impairment and are at higher risk of developing more severe psychopathology than children with single diagnoses (Kendall et al., 2001). Second, it is unclear whether one disorder (e.g., ODD) places a child at risk for another (e.g., GAD) or if shared risk factors account for their co-occurrence. It is important to note, however, that multiple explanations likely account for the co-occurrence of ODD+GAD and ODD+SAD. Therefore, I examined these explanations concurrently to determine which explanations best account for co-occurrence among these disorders. Ultimately, tests of these explanations can lead research closer to recognizing etiological mechanisms, shared processes, differential distal outcomes, as

well as more homogeneous subgroups of children with anxiety and/or ODD that may have different courses and responses to intervention (Drabick, 2009a, 2009b).

Third, sex differences may be important to consider in the relation between ODD+GAD and ODD+SAD; unfortunately, this is often a neglected issue. Previous studies have found that anxiety disorders are generally more common among girls, whereas externalizing disorders are more common among boys (Simonoff et al., 1997). When examining comorbid ODD+GAD specifically, results suggest that males are at greater risk for their co-occurrence (Garland & Garland, 2001; Marmorstein, 2007), though significant associations between ODD and GAD also have been reported among girls (Marmorstein, 2007). In terms of ODD+SAD, one study found that girls were at greater risk for later ODD symptoms given persistent SAD than boys (Foley et al., 2004). In contrast, another study found that boys had a greater likelihood of ODD+SAD as compared to girls (Marmorstein, 2007). It is likely that these discrepancies in the literature are due, in part, to differences in diagnostic criteria. For instance, Marmorstein (2007) created diagnoses based on the *Diagnostic and Statistical Manual of Mental Disorders – 3rd edition-Revised* criteria, whereas *DSM-IV* criteria were used in Foley et al. (2004), as well as Garland and Garland (2001). Sex differences also may exist in terms of the prevalence rates of risk factors and the relation of these risk factors to comorbid ODD+GAD and ODD+SAD (Rutter, Caspi, & Moffitt, 2003). For example, levels of inhibitory control are higher among girls than boys (Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006), which may better enable girls to inhibit impulses to be oppositional in adult-child interactions compared to boys. In sum, concurrent examination of the levels of relevant risk factors, levels of symptom severity, and magnitude of symptom associations among boys and girls may be particularly fruitful for understanding how these potential sex differences contribute to these comorbid conditions.

Fourth, there is limited research examining co-occurring ODD+GAD and ODD+SAD among impoverished, high risk samples. This is a crucial limitation given that children residing in disadvantaged neighborhoods may be exposed to higher rates of potential shared risk factors that could increase the risk of developing co-occurring anxiety and externalizing behaviors. Indeed, disadvantaged neighborhoods often are characterized by high levels of crime, residential mobility, environmental stressors (e.g., noise, overcrowding), and delinquent peer groups; poor social cohesion; and low quality schools (Leventhal & Brooks-Gunn, 2004; Sampson, 1997; Wandersman & Nation, 1998), each of which has been linked to physical and psychological adjustment, including psychophysiological factors (Evans & English, 2002; Kliewer, Wilson, & Plybon, 2002; Wilson, Kliewer, Plybon, & Sica, 2000). Given the adverse impact of living in an impoverished neighborhood, testing the relations among these processes and psychological symptoms among children residing in an inner-city environment could be useful for informing early intervention and prevention efforts.

Fifth, there is a paucity of research that concurrently addresses contextual and child-specific factors and potential child × context interactions in the prediction of ODD, GAD, and SAD symptoms. This is unfortunate given that contextual factors confer non-specific risk for various types of psychopathology and that children likely differ in their sensitivity to contextual factors based on biological and cognitive processes (Belsky, 2005; Boyce & Ellis, 2005; Drabick, 2009a; Steinberg & Avenevoli, 2000). For example, previous studies have found evidence of temperament × parenting interactions in the prediction of externalizing behaviors (Bates et al., 1998; Colder, Lochman, & Wells, 1997). Specifically, an impulsive/unmanageable temperament has been found to interact with parental restrictive control in the prediction of externalizing symptoms (Bates et al., 1998). In addition, poorly monitored active boys who

experience harsh parental discipline have been shown to exhibit elevated levels of aggression (Colder et al., 1997). There is, however, less research that has examined additional contextual processes, such as neighborhood danger and stressful life events, which likely moderate child specific processes in the prediction of both anxiety and externalizing symptoms. Notable exceptions include studies documenting that low positive affect among children interacts with neighborhood danger in the prediction of internalizing symptoms (Colder, Lengua, Fite, Mott, & Bush, 2006), and temperamental inattention interacts with parenting stress in the prediction of social adjustment (Coplan, Boweker, & Cooper, 2003). In addition, research has indicated that autonomic functioning may interact with neighborhood characteristics in the prospective prediction of externalizing problems and anxiety (Bubier, Drabick, & Breiner, in press; Hill, Ross, & Angel, 2005). These findings have begun to offer important insights into why contextual factors lead to negative outcomes among some, but not all, children, and further illustrate the importance of examining child-specific and contextual factors concurrently in the prediction of childhood psychopathology (Steinberg & Avenevoli, 2000).

Overview of the Current Study

To address these gaps in the literature, I compared multiple explanations for co-occurring ODD+GAD and ODD+SAD symptoms in a sample of ethnic minority children residing in the inner city. Examination of these processes among younger children is essential given that anxiety and externalizing disorders tend to be more strongly associated in younger, compared to older, youth (Russo & Beidel, 1994; Marmorstein, 2007). I used data collected at the baseline (Time 1) and 1-year follow-up (Time 2) assessments of the Child Health and Behavior Study (CHBS), a longitudinal survey of approximately 90 families residing in North Philadelphia. CHBS was designed to understand children's social, physical, and psychological adjustment, and examined

risk factors specific to the child (e.g., temperament) and the child's many contexts (e.g., family, neighborhood) in relation to the child's behavior. Specific constructs drawn from the CHBS that were used for this study include parent-reported ODD, GAD, and SAD symptoms; child hostile attribution biases; peer rejection; academic skills; difficult temperament; autonomic (sympathetic and parasympathetic) functioning; neighborhood danger; stressful life events; and demographic (control) variables.

In the current study, I first investigated whether (a) GAD symptoms prospectively predicted ODD symptoms, and (b) SAD symptoms prospectively predicted ODD symptoms (Explanation 1). Based on previous research suggesting that anxious and aggressive children tend to view the world and others as hostile, I also investigated whether hostile attribution biases in social interaction with peers mediated the relations of (a) GAD to ODD and (b) SAD to ODD. Second, I examined whether ODD symptoms prospectively predicted (a) GAD symptoms and (b) SAD symptoms (Explanation 2). Furthermore, I investigated whether peer rejection and poor academic skills mediated the relations between ODD and anxiety, as both are documented sequelae of ODD symptoms and potential risk factors for anxiety symptoms. Third, I examined whether shared risk factors account for the co-occurrence of ODD+GAD and ODD+SAD (Explanation 3). In terms of shared processes, I evaluated both child-specific and contextual factors that may confer risk for co-occurring ODD+GAD and ODD+SAD symptoms. I also explicitly tested for the presence of child × context interactions, as contextual influences likely have the greatest effect on children who are vulnerable (i.e., biologically predisposed) to their impact (Steinberg & Avenevoli, 2000). Last, given sex differences in the likelihood of developing ODD, GAD, SAD, and their respective comorbidities, I also examined whether

symptom associations, symptom severity, and levels of risk factors differed among boys and girls.

Hypotheses

Given power issues (see discussion on p. 41), hypotheses were broken into component parts so that statistical analyses could proceed in a step-wise manner.

Hypothesis 1: Concurrent Relations

Given previous findings suggesting significant associations among ODD, GAD, and SAD, I hypothesized that ODD symptoms would be concurrently correlated with both GAD and SAD symptoms at Time 1 and Time 2.

Hypothesis 1a. ODD symptoms were expected to be positively correlated with GAD symptoms and with SAD symptoms at Time 1.

Hypothesis 1b. ODD symptoms were expected to be positively correlated with GAD symptoms and with SAD symptoms at Time 2.

Hypothesis 2: Explanation 1

Given that previous research suggests that anxiety predicts ODD symptoms when measured dimensionally, I expected that anxiety symptoms would prospectively predict ODD symptoms. In terms of variables that may account for this prospective prediction, I hypothesized that hostile attribution biases would mediate the relation between anxiety symptoms and ODD symptoms.

Hypothesis 2a. Time 1 GAD symptoms were expected to be positively associated with Time 2 ODD symptoms.

Hypothesis 2b. Hostile attribution biases were expected to partially mediate the relation between Time 1 GAD symptoms and Time 2 ODD symptoms.

Hypothesis 2c. Time 1 SAD symptoms were expected to be positively associated with Time 2 ODD symptoms.

Hypothesis 2d. Hostile attribution biases were expected to partially mediate the relation between Time 1 GAD symptoms and Time 2 ODD symptoms.

Hypothesis 3: Explanation 2

Given the finding that children with ODD in preschool are at risk for developing comorbid anxiety in later childhood years, I predicted that ODD symptoms would prospectively predict anxiety symptoms. Furthermore, I hypothesized that peer rejection and poor academic skills would partially mediate the relation between ODD and anxiety symptoms.

Hypothesis 3a. Time 1 ODD symptoms were expected to be positively associated with Time 2 GAD symptoms.

Hypothesis 3b. I expected that peer rejection and poor academic skills at Time 1 would partially mediate the relation between Time 1 ODD symptoms and Time 2 GAD symptoms.

Hypothesis 3c. Time 1 ODD symptoms were expected to be positively associated with Time 2 SAD symptoms.

Hypothesis 3d. I expected that peer rejection and poor academic skills at Time 1 would partially mediate the relation between Time 1 ODD symptoms and Time 2 SAD symptoms.

Hypothesis 4: Explanation 3

Four constructs were considered as potential shared risk factors in the present study: (a) difficult temperament, (b) autonomic nervous system activity, (c) stressful life events, and (d) neighborhood danger. These constructs were chosen to examine both child-specific and contextual risk factors that have been shown to be associated with ODD and anxiety symptoms.

Hypothesis 4a. Measures of difficult temperament (assessed at Time 1) were expected to be associated with Time 2 ODD, GAD, and SAD symptoms, even after controlling for child age, sex, and Time 1 symptoms. Specifically, I hypothesized that fearful temperament would be associated with Time 2 GAD symptoms and Time 2 SAD symptoms, whereas temperamental anger/frustration and impulsivity would be associated with Time 2 ODD symptoms. In addition, I hypothesized that measures of inhibitory control would be associated with Time 2 ODD, GAD, and SAD symptoms.

Hypothesis 4b. Autonomic nervous system activity (assessed at Time 1) was expected to be associated with Time 2 ODD, GAD, and SAD symptoms, even after controlling for child age, sex, and Time 1 symptoms. More specifically, I expected that longer PEP scores at baseline and decreased PEP reactivity would be associated with higher levels of Time 2 ODD symptoms, whereas shorter PEP scores at baseline and increased PEP reactivity would be associated with Time 2 GAD and SAD symptoms. I hypothesized that shorter PEP scores during the emotion task would be positively associated with Time 2 ODD, GAD, and SAD symptoms, given research suggesting that children with anxiety and ODD symptoms become hyperaroused when experiencing negative emotions. In terms of RSA variables, I expected that RSA deficiencies (i.e., decreased RSA at baseline, RSA reactivity, and RSA during the emotion task) would be related to Time 2 ODD symptoms. I also expected that decreased RSA at baseline would be associated with Time 2 GAD and SAD symptoms.

Hypothesis 4c. I hypothesized that neighborhood danger (assessed at Time 1) would be positively associated with Time 2 ODD, GAD, and SAD symptoms, and these associations would be maintained after controlling for child age, sex, and Time 1 symptoms.

Hypothesis 4d. I predicted that the number of stressful life events experienced in the past year would be positively associated with Time 2 ODD, GAD, and SAD symptoms, and that these associations would remain significant after controlling for child age, sex, and Time 1 symptoms.

Hypothesis 5: Child × Context Interactions

There is a paucity of research that examines child × context interactions in the prediction of ODD, GAD, and SAD symptoms. Therefore, I examined whether child-specific risk factors (i.e., difficult temperament and autonomic nervous system activity) interacted with contextual risk factors (i.e., neighborhood danger and stressful life events) to prospectively predict Time 2 ODD, GAD, and SAD symptoms.

Hypothesis 5a. I hypothesized that measures of difficult temperament would interact with neighborhood danger in the prediction of Time 2 ODD, GAD, and SAD symptoms.

Hypothesis 5b. I hypothesized that measures of difficult temperament would interact with stressful life events in the prediction of Time 2 ODD, GAD, and SAD symptoms.

Hypothesis 5c. I hypothesized that measures of autonomic functioning would interact with neighborhood danger in the prediction of Time 2 ODD, GAD, and SAD symptoms.

Hypothesis 5d. I hypothesized that measures of autonomic functioning would interact with stressful life events in the prediction of Time 2 ODD, GAD, and SAD symptoms.

Hypothesis 6: Sex Differences

Hypothesis 6a. I hypothesized that boys and girls would differ in terms of their levels of ODD, GAD, and SAD symptoms. Severity of GAD and SAD symptoms was hypothesized to be greater among girls, whereas severity of ODD symptoms was hypothesized to be greater among boys.

Hypothesis 6b. Given suggestions that different rates of risk factors may differentially contribute to sex differences in symptoms, it was hypothesized that boys and girls would differ in terms of the level of some risk factors. Specifically, parent ratings of inhibitory control were expected to be higher among girls than boys, and parent-reported impulsivity was expected to be greater among boys than girls. However, no differences were hypothesized for temperamental anger/frustration or fear (Else-Quest, et al., 2006). Perception of neighborhood danger was expected to be higher among girls, as compared to boys (Zalot, Jones, Forehand, & Brody, 2007). It also was hypothesized that there would be no sex differences for autonomic functioning or parent ratings of stressful life events.

Hypothesis 6c. I expected that there would be sex differences in the bivariate associations between symptoms (e.g., ODD and GAD, ODD and SAD). However, given inconsistencies in the literature, no directional hypotheses were made for sex differences among symptom associations.

CHAPTER 2

METHOD

Participants

Participants were 88 children (M = 7.74 ±1.06 years old; 51% male, 94% African-American, 6% Latino/a) and their primary caregivers (84% biological mothers) drawn from three elementary schools in North Philadelphia. Based on census data, the neighborhoods from which families were drawn can be characterized as an inner city area, with high levels of crime, poverty, and homogeneity in terms of ethnic minority status. In terms of family configurations, 52.3% of children lived in single-parent households, 31.8% in intact (i.e., two-biological parent) households, 9.1% in blended homes, and 6.8% in other family configurations (grandparental, adoptive). In terms of annual family income, 63% of families reported income less than $20,000, 17% reported income from $20,000-$30,000, and 15% reported income over $30,000. Sixty-seven percent of the children lived in families receiving public assistance. Fifty-three percent of the primary caregivers completed high school, 29% less than high school, and 18% beyond high school. These families thus represent a predominantly impoverished group, and given significant contextual stressors, a high-risk sample.

Children and their families were assessed at two time points approximately one year apart. Preliminary analyses suggest that the children for whom both time points have been completed (n = 62) did not differ (all ps > .05) from those who completed only the Time 1 visit (N = 88) in terms of age, $t(86) = .21$, Cohen's $d = .00$; sex, $\chi^2(1) = .11$, w = .00; ethnicity, $\chi^2(1) = 1.24$, w = .01; family configuration, $\chi^2(4) = 1.14$, w = .01; or income, $\chi^2(1) = .14$, w = .00 (Cohen, 1988).

Procedure

The present study is part of a larger research program (CHBS) designed to follow at-risk children and their parents over time. The CHBS was approved by Temple University's Institutional Review Board. The project director obtained permission from the principals of three elementary schools to send information regarding the project to primary caregivers (hereafter "parents") of first- through third-grade children. The families were mailed a description of the study, parental consent form, and a self-addressed, stamped postcard. The description stated that we were interested in children's social, physical, and emotional adjustment, as well as what might place children at risk for emotional or behavioral problems. Parents interested in participating in the project either returned a self-addressed stamped postcard or called to make an appointment. Approximately 21% of families responded to the information sent, which is consistent with other research using high-risk samples with similar ethnic and SES compositions (e.g., Sessa, Avenevoli, Steinberg, & Morris, 2001; Silk, Sessa, Morris, Steinberg, & Avenevoli, 2004). The sample characteristics (i.e., ethnicity, sex, family SES) reflect the schools from which the families were drawn; nevertheless, due to confidentiality requirements, no information was available to compare those who self-selected into the project and those that did not.

At the initial time point (Time 1), parents and their children were invited to the research lab for 2 visits, each lasting approximately 2.5 hours. Parents and children provided consent and assent, respectively, prior to participation. Following consent and assent, the parent completed questionnaires related to the child and family, and the child reported on factors related to his or her neighborhood and participated in a protocol designed to measure autonomic functioning. Parents were paid for their participation and reimbursed for transportation. Children received a small gift. In addition, a donation was made to the school for each child that participated.

Approximately 9 months after their initial visit, parents who had participated at Time 1 were sent a letter inviting them to participate in another assessment, a consent form, and self-addressed stamped postcard. As was the case with the initial visit, parents could either return the postcard or call to make an appointment. Parents and their children were invited to the research lab for 1 visit, lasting approximately 2.5 hours (Time 2). Parents completed questionnaires regarding parenting and their child's anxiety and externalizing behaviors, while children participated in a researcher-led interview designed to assess children's social information processing. The timing of the invitation was intended to allow the follow-up visit to occur approximately one year after the initial assessment. On average, families came in 10.7 ± 1.3 months after the Time 1 visit. As with the initial visit, parents were compensated for their participation and transportation. Children received a small gift.

Measures

Demographics

Parents provided information about their child's age and sex at Time 1.

ODD, GAD, and SAD symptoms

At both Time 1 and 2, parents rated child ODD, GAD, and SAD symptoms using the Child Symptom Inventory-4 (CSI-4; Gadow & Sprafkin, 1994, 2002), which contains the behavioral symptoms of most childhood disorders described in the *DSM-IV* (APA, 1994). Individual items bear one-to-one correspondence with *DSM-IV* symptoms (i.e., high content validity). Because the goal of the present study was to examine processes associated with ODD, GAD, and SAD symptoms, as opposed to the diagnostic categories, and given research demonstrating that the symptoms associated with these disorders often cause significant impairment (Angold et al., 1999b), these symptoms were examined dimensionally. Items were

scored on a scale from 0 (*never*) to 3 (*very often*). Responses to individual items were summed to create a Symptom Severity score for three symptom categories: ODD (8 items; Time 1 α = .92, Time 2 α = .93); GAD (7 items; Time 1 α = .75, Time 2 α = .78); and SAD (8 items; Time 1 α = .83, Time 2 α = .85).

Compared to scores derived from the community-based samples used to norm the CSI-4 (Gadow & Sprafkin, 2002), the present sample of inner-city children was consistently rated as exhibiting higher levels of ODD, GAD, and SAD symptoms for boys and for girls (Table 1). An ancillary way to facilitate comparisons between the present sample and other samples is to use the CSI-4 to derive Symptom Count (categorical) scores, 0 = *never/sometimes*, 1 = *often/very often*. For Symptom Count scores, a specific symptom is considered to be a clinically relevant problem if it is rated as occurring "often" or "very often." When the total Symptom Count score equals or exceeds the number of symptoms specified by *DSM-IV* as necessary for a diagnosis, the child receives a Screening Cutoff score of "yes." The number of children at Time 1 who received parent-rated Screening Cutoff scores for ODD (≥ 4 symptoms), SAD (≥ 3 symptoms), and GAD (≥ 4 symptoms), compared to the total number of children in the present sample (*N* = 88), was ODD (*n* = 11, 13%); SAD (*n* = 9, 10%); and GAD (*n* = 3, 3%). For Time 2, the number of children who received parent-rated Screening Cutoff scores for ODD (≥ 4 symptoms), SAD (≥ 3 symptoms), and GAD (≥ 4 symptoms), compared to the total number of children for whom Time 2 information was obtained (*n* = 62), was ODD (*n* = 7, 11%); SAD (*n* = 7, 11%); and GAD (*n* = 3, 5%).

Potential Mediators and Risk Factors

*Social information processing***.** The Social Cognitive Assessment Profile (SCAP) was administered at Time 2 as a measure of social information processing (Hughes, Meehan, &

Table 1. Means (SD) for Parent-reported Symptoms in the Normative vs. Current Sample at Time 1

	Boys		Girls	
Symptom Category	Normative	Current	Normative	Current
ODD	5.4 (4.0)	7.1 (4.97)	4.4 (3.9)	5.5 (5.3)
GAD	3.0 (3.1)	2.9 (2.31)	2.4(2.7)	2.6 (3.4)
SAD	1.7 (2.9)	3.2 (4.06)	1.3 (2.3)	3.1 (4.1)

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder, SAD = separation anxiety disorder.

Cavell, 2004). The SCAP is a brief and easily administered interview that presents children with 7 hypothetical provocation situations. Four of the 7 original provocation situations were used in the CHBS. The vignettes were chosen to represent a range of peer-child events, settings, number of people present, and type of aggression (relational vs. physical). These provocations included the child being hit with a ball in the back, being bumped with a tray in the lunchroom, being left out of a classroom project with his/her friends, and having his/her best friend ignore him/her on the playground. For each vignette, children were presented with line drawings that illustrated a peer provocation described in a vignette that the experimenter verbally provided. Children were asked to pretend that they were the individual in the vignette. The experimenter then asked the children a series of questions about the provocation situations, including the children's goals, attributions, solutions, and outcome expectancies, which represent four of the six stages of Crick and Dodge's (1994) social information processing model. Children's responses were recorded

verbatim.

In the present study, I examined children's hostile attributions given that children with anxiety and/or externalizing behaviors have been found to attribute hostile intent in ambiguous situations. Specifically, after each vignette, children were asked to explain what happened and the intention of the peer provocateur (i.e., why the peer acted in a specific way). Children were able to generate multiple solutions. A score of 1 was given if any of the responses were hostile, and a score of 0 was given if all the responses were non-hostile. A hostile response referred to peer intent to cause harm (e.g., "she meant to hit me because she doesn't like me"). A non-hostile response included responses referring to accidental or prosocial intent and/or statements of fact. Responses across the 4 vignettes were summed to create a Hostile Attribution score (possible range = 0 to 4).

Peer rejection. Parents reported on children's peer rejection at Time 1 using the Peer Acceptance/Rejection scale of the MacArthur Health and Behavior Questionnaire (HBQ; Ablow et al., 1999). The Peer Rejection/Acceptance scale consists of 8 items ($\alpha = .85$) rated on a scale from 1 (*not at all like*) to 4 (*very much like).* Sample items include, "Is often left out by other children" and "Has lots of friends at school" (reverse scored). In the present study, items were scored so that higher scores indicated higher levels of peer rejection.

Academic skills. The Letter-Word Identification and Calculation subtests of the Woodcock-Johnson-III Tests of Achievement (Woodcock, McGrew, & Mather, 2001) were administered to children at Time 1 to index basic academic skills. The Letter-Word Identification subtest assesses the child's basic reading skills and requires children to read basic sight words out loud to the administrator. The Calculation subtest measures the child's ability to perform mathematical computations and requires the child to solve basic math problems using a pencil

and paper. Items are scored as 0 (*incorrect*) or 1 (*correct*) and summed to create the subtest raw score. Scaled scores, which are based on a mean of 100 and *SD* of 15, were used in the current study.

Difficult temperament. Parents rated child temperament using the Children's Behavior Questionnaire (CBQ; Rothbart et al., 2001), which assesses 15 dimensions of temperament. Parents endorse items describing children's reactions on a scale from 1 *(extremely untrue)* to 7 *(extremely true of your child).* Four subscales that are purported to index two primary components of difficult temperament (i.e., negative emotional reactivity and self-regulatory capacities) were examined. These subscales were Anger/Frustration (6 items, $\alpha = .79$); Fear (6 items, $\alpha = .57$); Impulsivity (6 items, $\alpha = .76$); and Inhibitory Control (6 items, $\alpha = .59$). The Anger/Frustration and Fear scales are thought to index negative emotional reactivity, whereas the Impulsivity and Inhibitory Control scales are thought to index self-regulatory capacities. The Anger/Frustration scale measures negative affectivity related to interrupting of ongoing tasks or goal blocking (sample item: "Gets frustrated when prevented from doing what he/she wants to do"). The Fear scale measures negative affectivity related to unease, worry, or nervousness (sample item: "Is afraid of the dark"). The Impulsivity scale measures speed of response initiation (sample item: "Usually rushes into an activity without thinking about it"), and Inhibitory Control measures the capacity to plan and suppress inappropriate approach responses under instructions or in novel or uncertain situations (sample item: "Can lower his/her voice when asked to do so").

Autonomic functioning. Autonomic functioning was measured at Time 1 using Bio-Impedance Technology's HIC-2000 (Chapel Hill, NC, n.d.), a noninvasive instrument for detecting and monitoring bioelectric impedance signals. An external electrocardiographic (ECG)

cable was added to the HIC-2000 to increase the flexibility for electrode positioning and ease of detecting the ECG signal. The HIC-2000 recorded RSA and PEP with a constant 5 V potential across 7 electrodes that were pre-gelled and have a circular contact area with a 1cm diameter. Disposable spot electrodes were attached to the child's neck, back, stomach, and shoulder (Qu, Zhang, Webster, & Tompkins, 1986). Cardiac signals were monitored by and interfaced to a PC-based computer.

Both RSA and PEP were measured during tasks chosen to provide a range of stressors (i.e., social, cognitive, physical, and emotional; Alkon et al., 2003; Bubier & Drabick, 2008; Bubier et al., in press). The protocol has been shown to be a reliable and valid method for examining sympathetic and parasympathetic responses to challenge among children (Alkon et al., 2003). For each child, the order of the tasks was as follows: social (3 min), cognitive (3 min), physical (1 min), and emotional (3 min). The social challenge was designed to engage the child in conversation and included questions about the child's school, family, and interests. In the cognitive challenge, the child repeated a list of 2 to 6 numbers presented orally by the experimenter. In the physical challenge, the child was asked to taste and identify several drops of lemon juice that were placed on his or her tongue by the experimenter. The emotional challenge consisted of two brief video clips designed to evoke emotional reactions (i.e., fear and sadness). The "fear" video depicts a young boy who is frightened during a thunderstorm and the "sadness" video depicts a child and her mother addressing the loss of the child's pet bird (Alkon et al., 2003; Eisenberg et al., 1988). Age-appropriate books were read to the child before and after the challenge tasks to obtain baseline measures of resting autonomic activity. This standardized protocol took approximately 20 min to administer. For all of the tasks, the child's behavior and physiological reactions (i.e., heart rate, RSA, and PEP) were monitored.

Sympathetic-linked cardiac activity was indexed by PEP, measured as the time between the ECG Q wave (onset of ventricular depolarization) and the impedance cardiographic B wave (onset of left ventricular ejection). Waveforms were collected using the spot electrode configuration described above (Qu et al., 1986). PEP data were ensemble-averaged in Cop-Win 6.0 H software, in 30 s epochs. It is important to note that shortened PEP scores are consistent with activation of the sympathetic nervous system.

Parasympathetic cardiac activity was assessed using spectral analysis via Nevrokard's Long-Term Heart Rate Variability (LT-HRV) software (Ljubljana, Slovenia, n.d.), which separates heart rate variability time series into component frequencies using fast-Fourier transformations (Berntson et al., 1997). High frequency spectral power (>.15 Hz) was extracted to measure RSA. This high frequency band is believed to better index cardiac vagal control than low frequency (< .04 Hz) or midfrequency (.04 - .15 Hz) variability (Houtveen & Molenaar, 2001; Mezzacappa, Kindlon, Earls, & Saul, 1994). Spectral densities were calculated in 30 s epochs. The log of RSA was used to index parasympathetic functioning, which is a common transformation used to normalize spectral analytic data (Crowell et al., 2006).

Mean scores for PEP and RSA were calculated during the baseline and across each of the four tasks (social, cognitive, physical, emotional) (Alkon et al., 2003; Bubier & Drabick, 2008). Participants were included in the analyses if they had at least 50% scorable epochs within each task and during baseline. This decision was made to maximize the number of participants included while maintaining an adequate number of epochs. Variables of interest for this study included parasympathetic (RSA) baseline and parasympathetic reactivity (RSA averaged across the four tasks minus RSA baseline), and sympathetic (PEP) baseline and reactivity (PEP averaged across the four tasks minus PEP baseline). RSA and PEP during the emotional

challenge also were examined because the emotional challenge was hypothesized to most accurately index autonomic activation for negative emotions.

Neighborhood danger. Given that a child's perception of contextual factors influences context-outcome relations (Boyce et al., 1998), a puppet interview was used at Time 1 to obtain children's reports on neighborhood danger and decay (7 items, $\alpha = .73$). These items are part of a larger puppet interview that has been shown to be a reliable and valid assessment instrument for children (Morris et al., 2002; Sessa et al., 2001). The puppet interview is administered to children individually, and children's responses are videotaped for later coding by trained research assistants (κs range from .98 to 1.00). In the interview, children are presented with two puppets that offer opposing statements and are asked to pick which puppet is more like them (Silk et al., 2004). Responses were scored as 0 or 1, where 1 indicated the presence of danger or decay. The mean of the summed items was used to create a neighborhood danger and decay score. Sample items include, "People in my neighborhood get in trouble with the police/People in my neighborhood do not get in trouble with the police" and "Some people in my neighborhood steal things/People in my neighborhood do not steal things."

Stressful life events. Parents rated stressful life experiences at Time 2 using an abbreviated version of the original Social Readjustment Rating Scale (SRRS; Holmes & Rahe, 1967), a widely used measurement of stress (Scully, Tosi, & Banning, 2000). The abbreviated version of the SRRS used in the present study is a checklist that includes 19 stressors that the family may have experienced in the past 12 months. Responses were scored as 0 or 1, where 1 indicated the presence of a stressor. The average number of stressors was used to create a stressful life events score. It is important to note that the abbreviated scale consists of only negative life events and does not include positive stressors from the original measure. Sample

items include divorce, being fired at work, and moving to a new location.

Statistical Analyses

Before testing specific hypotheses, preliminary descriptive analyses were conducted. Specifically, means, standard deviations, skewness, kurtosis, and outliers were examined. To minimize multicollinearity, independent variables were centered ($M = 0$) before inclusion in the regression equations.

Concurrent Relations

Hypothesis 1a. Bivariate correlations were conducted to examine relations among symptom categories at Time 1.

Hypothesis 1b. Bivariate correlations were conducted to examine relations among symptom categories at Time 2.

Explanation 1: GAD and/or SAD Symptoms Confer Risk for ODD Symptoms

Hypothesis 2a. A bivariate correlation was conducted to determine whether Time 1 GAD symptoms were positively associated with Time 2 ODD symptoms. If the correlation was significant, a hierarchical multiple regression analysis was conducted for which child age and child sex were entered in the first step, and Time 1 GAD symptoms were entered in the second step. If Time 1 GAD symptoms predicted Time 2 ODD symptoms, a second multiple regression analysis was conducted to determine whether Time 1 GAD symptoms prospectively predicted Time 2 ODD symptoms above and beyond Time 1 ODD symptoms. In this second hierarchical regression, child age, child sex, and Time 1 ODD symptoms were entered in the first step and Time 1 GAD symptoms were entered in the second step.

Hypothesis 2b. Tests of mediation were conducted to examine whether hostile attribution biases partially mediated the relation between Time 1 GAD symptoms and Time 2 ODD

symptoms. This analysis only was conducted if multiple regression analyses indicated that Time 1 GAD symptoms significantly predicted Time 2 ODD symptoms above and beyond Time 1 ODD symptoms (as described with Hypothesis 2a). Tests of mediation were conducted using the ordinary least squares regression procedure suggested by Baron and Kenny (1986) and a bootstrap procedure (Shrout & Bolger, 2002). Bootstrap procedures have been used to develop more accurate estimates of the indirect and direct effects when testing mediation, and may be more powerful when using smaller sample sizes (Shrout & Bolger, 2002). The following steps were conducted to test mediation: First, Time 1 ODD symptoms were entered into two equations, predicting (a) Time 2 GAD symptoms (i.e., the outcome) and (b) peer rejection (i.e., the hypothesized mediating variable). Next, peer rejection was entered simultaneously with Time 1 ODD symptoms to examine whether peer rejection accounted for the relation between Time 1 ODD symptoms and Time 2 GAD symptoms. If peer rejection was significant and the prediction from Time 1 ODD symptoms was reduced when these variables were entered simultaneously, this finding would suggest that peer rejection is a mechanism through which Time 1 ODD symptoms are related to Time 2 GAD symptoms (Baron & Kenny, 1986). The bootstrap procedure described by Shrout and Bolger was conducted using AMOS 5.0 (Arbuckle, 2003). This procedure was used to determine the significance of the indirect effects and whether the direct effect is equivalent to zero, after controlling for the proposed mediator.

Hypothesis 2c. A bivariate correlation was conducted to determine whether Time 1 SAD symptoms were positively associated with Time 2 ODD symptoms. If the correlation was significant, a hierarchical multiple regression analysis was conducted for which child age and child sex were entered in the first step, and Time 1 SAD symptoms were entered in the second step. If Time 1 SAD symptoms predicted Time 2 ODD symptoms, a second multiple regression

analysis was conducted to determine whether Time 1 SAD symptoms prospectively predicted Time 2 ODD symptoms above and beyond Time 1 ODD symptoms. In this second hierarchical regression, child age, child sex, and Time 1 ODD symptoms were entered in the first step and Time 1 SAD symptoms were entered in the second step.

Hypothesis 2d. Tests of mediation were conducted to examine whether hostile attribution biases partially mediated the relation between Time 1 SAD symptoms and Time 2 ODD symptoms. This analysis only was conducted if multiple regression analyses indicated that Time 1 SAD symptoms significantly predicted Time 2 ODD symptoms above and beyond Time 1 ODD symptoms (as described with Hypothesis 2c). As described in Hypothesis 2b above, tests of mediation were conducted using the ordinary least squares regression procedure suggested by Baron and Kenny (1986) and a bootstrap procedure (Shrout & Bolger, 2002).

Explanation 2: ODD Symptoms Confer Risk for GAD and/or SAD Symptoms

Hypothesis 3a. A bivariate correlation was conducted to determine whether Time 1 ODD symptoms were positively associated with Time 2 GAD symptoms. If the correlation was significant, a hierarchical multiple regression analysis was conducted for which child age and child sex were entered in the first step, and Time 1 ODD symptoms were entered in the second step. If Time 1 ODD symptoms predicted Time 2 GAD symptoms, a second multiple regression analysis was conducted to determine whether Time 1 ODD symptoms prospectively predicted Time 2 GAD symptoms above and beyond Time 1 GAD symptoms. In this second hierarchical regression, child age, child sex, and Time 1 GAD symptoms were entered in the first step and Time 1 ODD symptoms were entered in the second step.

Hypothesis 3b. Tests of mediation were conducted to examine whether peer rejection and poor academic skills partially mediated the relation between Time 1 ODD symptoms and Time 2

GAD symptoms. This analysis only was conducted if multiple regression analyses indicated that Time 1 ODD symptoms significantly predicted Time 2 GAD symptoms above and beyond Time 1 GAD symptoms. As described in Hypothesis 2b above, tests of mediation were conducted using the ordinary least squares regression procedure suggested by Baron and Kenny (1986) and a bootstrap procedure (Shrout & Bolger, 2002).

Hypothesis 3c. A bivariate correlation was conducted to determine whether Time 1 ODD symptoms were positively associated with Time 2 SAD symptoms. If the correlation was significant, a hierarchical multiple regression analysis was conducted for which child age and child sex were entered in the first step, and Time 1 ODD symptoms were entered in the second step. If Time 1 ODD symptoms predicted Time 2 SAD symptoms, a second multiple regression analysis was conducted to determine whether Time 1 ODD symptoms prospectively predicted Time 2 SAD symptoms above and beyond Time 1 SAD symptoms. In this second hierarchical regression, child age, child sex, and Time 1 SAD symptoms were entered in the first step and Time 1 ODD symptoms were entered in the second step.

Hypothesis 3d. Tests of mediation were conducted to examine whether peer rejection and poor academic skills partially mediated the relation between Time 1 ODD symptoms and Time 2 SAD symptoms. This analysis only was conducted if multiple regression analyses indicated that Time 1 ODD symptoms significantly predicted Time 2 SAD symptoms above and beyond Time 1 SAD symptoms. As described in Hypothesis 2b above, tests of mediation were conducted using the ordinary least squares regression procedure suggested by Baron and Kenny (1986) and a bootstrap procedure (Shrout & Bolger, 2002).

Explanation 3: Shared Risk Factors Account for the Co-occurrence of ODD+GAD and ODD+SAD

Hypothesis 4a. First, I conducted bivariate correlations to examine whether measures of difficult temperament were associated with Time 2 ODD, GAD, and SAD symptoms. Second, to determine whether measures of difficult temperament prospectively predicted Time 2 ODD, GAD, and SAD symptoms, three separate regression analyses were conducted for which Time 2 ODD, Time 2 GAD, and Time 2 SAD symptoms were the dependent variables. Given issues related to power and concerns with multicollinearity, only those measures of difficult temperament that were found to be significantly associated ($p < .05$) with a particular symptom category were included in the regression analyses. For each analysis, the child's age and sex were entered in the first step, and indices of difficult temperament were entered in the second step. If indices of difficult temperament were found to prospectively predict Time 2 symptoms, a second set of regression analyses were conducted to determine whether difficult temperament indices predicted Time 2 symptoms above and beyond Time 1 symptoms. In this second series of hierarchical regressions, child age, child sex, and Time 1 symptoms were entered in the first step and difficulty temperament indices were entered in the second step. To be considered a shared risk factor, the same measure of difficult temperament would be required to prospectively predict Time 2 ODD and Time 2 GAD symptoms, or Time 2 ODD and Time 2 SAD symptoms.

Hypothesis 4b. First, I conducted bivariate correlations to examine whether measures of autonomic functioning were associated with Time 2 ODD, GAD, and SAD symptoms. Second, to determine whether measures of autonomic functioning prospectively predicted Time 2 ODD, GAD, and SAD symptoms, three separate regression analyses were conducted for which Time 2 ODD, Time 2 GAD, and Time 2 SAD symptoms were the dependent variables. Again, to minimize the number of predictors that were included in this second set of analyses, only those measures of autonomic functioning found to be significantly associated with a particular

symptom category were included in the regression analyses. For each analysis, the child's age and sex were entered in the first step and autonomic functioning variables were entered in the second step. If autonomic functioning variables were found to prospectively predict Time 2 symptoms, a second series of regression analyses were conducted to determine whether autonomic functioning variables predicted Time 2 symptoms above and beyond Time 1 symptoms. In this second series of hierarchical regressions, child age, child sex, and Time 1 symptoms were entered in the first step and autonomic functioning variables were entered in the second step. To be considered a shared risk factor, the same measure of autonomic functioning would be expected to prospectively predict Time 2 ODD and Time 2 GAD symptoms, or Time 2 ODD and Time 2 SAD symptoms.

Hypothesis 4c. First, bivariate correlations were conducted to examine whether neighborhood danger was associated with Time 2 ODD, GAD, and SAD symptoms. Second, in cases where the correlations between neighborhood danger and Time 2 symptom categories were significant, separate regression analyses were conducted for which the respective Time 2 symptoms were the dependent variables. Child age and sex were entered in step 1 of these regressions. If neighborhood danger was found to prospectively predict Time 2 symptoms, a second set of regression analyses were conducted to determine whether neighborhood danger predicted Time 2 symptoms above and beyond Time 1 symptoms. In this second series of hierarchical regressions, child age, child sex, and Time 1 symptoms were entered in the first step and neighborhood danger was entered in the second step. To be considered a shared risk factor, neighborhood danger would be expected to prospectively predict Time 2 ODD and Time 2 GAD symptoms, or Time 2 ODD and Time 2 SAD symptoms.

Hypothesis 4d. First, bivariate correlations were conducted to examine whether the number of stressful life events in the past year was associated with Time 2 ODD, GAD, and SAD symptoms. Second, in cases where the associations between the number of stressful life events and symptom categories were significant, separate regression analyses were conducted for which the appropriate Time 2 symptoms were the dependent variables. Child age and sex were entered in step 1 of these regressions. If the number of stressful life events was found to prospectively predict Time 2 symptoms, a second set of regression analyses were conducted to determine whether number of stressful life events predicted Time 2 symptoms above and beyond Time 1 symptoms. In this second series of hierarchical regressions, child age, child sex, and Time 1 symptoms were entered in the first step and number of stressful life events was entered in the second step. To be considered a shared risk factor, stressful life events would be expected to prospectively predict Time 2 ODD and Time 2 GAD symptoms, or Time 2 ODD and Time 2 SAD symptoms.

Child × Context Interactions

Hypothesis 5a. It was hypothesized that measures of difficult temperament would interact with neighborhood danger in the prediction of Time 2 ODD, GAD, and SAD symptoms. Therefore, analyses examining whether neighborhood danger moderated the relation between temperament and Time 2 symptom categories were conducted. To examine moderation, the independent variables were centered ($M = 0$) and a child × context cross-product interaction term was created using the centered predictors (Aiken & West, 1991). Next a series of regression equations were conducted. Each regression equation involved the following steps: (a) child age and sex, and (b) a temperament variable, neighborhood danger, and the temperament × neighborhood danger cross-product interaction term. Given difficulty in identifying interactions

with smaller sample sizes, for all interaction terms that at least tended to be significant ($p < .10$), procedures outlined by Holmbeck (2002) were followed for post-hoc probing of moderational effects. Consistent with Holmbeck (2002), two new conditional moderator variables (± 1 *SD* from the mean) were created, as well as two new interaction terms that incorporated the conditional variables. Two post-hoc regressions, each of which involved simultaneous entry of the child-specific variable (i.e., temperament), the contextual variable (i.e., neighborhood danger), and the child specific × conditional contextual variable (i.e., temperament × neighborhood danger) were then conducted (Holmbeck, 2002). From these analyses, unstandardized betas (slopes) and a regression equation for children reporting high (1 *SD* above the mean) and low (1 *SD* below the mean) levels of neighborhood danger can be computed. If the unstandardized betas associated with the child variable are significant, this would suggest that neighborhood danger moderates the relation of temperament and Time 2 symptom categories.

Hypothesis 5b. As noted in the statistical analyses for Hypothesis 5a, procedures outlined by Aiken and West (1991) and Holmbeck (2002) were used to examine whether stressful life events moderated the relation between temperament and Time 2 symptom categories. To examine moderation, the independent variables were centered ($M = 0$) and a child × context cross-product interaction term was created using the centered predictors (Aiken & West, 1991). Then a series of multiple regression analyses were conducted, which included the following steps: (a) child age and sex, and (b) a temperament variable, stressful life events, and the temperament × stressful life events cross-product interaction term. As described for Hypothesis 5a, procedures outlined by Holmbeck (2002) were followed for post-hoc probing of moderational effects that at least tended to be significant ($p < .10$).

Hypothesis 5c. To examine whether neighborhood danger moderated the relations between measures of autonomic functioning and Time 2 symptom categories, a series of regression equations were conducted (Aiken & West, 1991). Each multiple regression analysis included the following steps: (a) child age and sex, and (b) an autonomic functioning variable, neighborhood danger, and the autonomic functioning × neighborhood danger cross-product interaction term. Procedures outlined by Holmbeck (2002) were followed for post-hoc probing of moderational effects that at least tended to be significant ($p < .10$).

Hypothesis 5d. Last, multiple regression analyses (Aiken & West, 1991) were conducted to examine whether stressful life events moderated the relation between autonomic functioning and Time 2 ODD, GAD, and SAD symptoms. Each multiple regression analysis included the following steps: (a) child age and sex, and (b) an autonomic functioning variable, stressful life events, and the autonomic functioning × stressful life events cross-product interaction term. Procedures outlined by Holmbeck (2002) were followed for post-hoc probing of moderational effects that at least tended to be significant ($p < .10$).

Sex Differences

Hypothesis 6a. To examine whether boys and girls differed in terms of the level of ODD, GAD, and SAD symptoms, independent sample *t*-tests (boys vs. girls) were conducted.

Hypothesis 6b. To determine whether boys and girls differed in the level of risk factors, independent sample *t*-tests (boys vs. girls) were conducted with difficult temperament, autonomic functioning, neighborhood danger, and stressful life events as dependent variables.

Hypothesis 6c. To examine whether there were sex differences in the magnitude of associations among Time 1 and Time 2 ODD and GAD symptoms, and among Time 1 and Time

2 ODD and SAD symptoms, correlation coefficients were compared by transforming correlations to z scores.

Power Analysis

A power analysis was conducted to determine the sample size necessary to test the various explanations for the co-occurrence of ODD+GAD and ODD+SAD. With four predictors, as was needed to test Explanations 1, 2, and 3, sufficient power (.80; $\alpha = .05$) to detect moderate effects is attained with a sample size of 65 (Faul & Erdfelder, 1992). For child × context moderation analyses, using a medium effect size as an estimate, with 3 predictors (child-specific variable, contextual variable, and child-specific × context interaction term), a minimum sample of 55 is needed to attain sufficient power (.80; $\alpha = .05$) when the predictors are reliable (Aiken & West, 1991). Therefore, the present sample provides adequate power for examining explanations for the co-occurrence of ODD+GAD and ODD+SAD.

CHAPTER 3

RESULTS

Means, standard deviations, and number of participants for all study variables are presented in Table 2. Skewness and kurtosis were examined and no outliers were identified. Given the results of these analyses, the proposed analyses were conducted without transforming or otherwise modifying any of the variables.

Concurrent Relations

Bivariate correlations between Time 1 and Time 2 symptom categories are presented in Table 3.

Hypothesis 1a: ODD symptoms were expected to be positively correlated with GAD symptoms and with SAD symptoms at Time 1.

As expected, bivariate correlations revealed significant concurrent associations between Time 1 symptoms. Specifically, at Time 1, ODD symptoms were positively associated with GAD symptoms ($r = .50$, $p < .01$) and with SAD symptoms ($r = .28$, $p < .01$). Comparison of the correlation coefficients at Time 1 suggests that the relation between ODD and GAD symptoms was significantly greater than the relation between ODD and SAD symptoms (z score = 2.42, $p < .05$).

Hypothesis 1b: ODD symptoms were expected to be positively correlated with GAD symptoms and with SAD symptoms at Time 2.

At Time 2, concurrent associations between symptoms were also significant; ODD was positively associated with GAD ($r = .66$, $p < .01$). However, at Time 2, ODD symptoms were not significantly related to SAD symptoms ($r = .20$, $p > .05$). Comparison of the correlation coefficients at Time 2 suggests that the relation between ODD and GAD symptoms was

Table 2. Means, Standard Deviations, and Number of Participants for Study Variables

Variable	*M*	*SD*	*n*
Time 1 ODD Symptoms	6.32	5.17	86
Time 1 GAD Symptoms	2.75	2.89	85
Time 1 SAD Symptoms	3.17	4.07	85
Time 2 ODD Symptoms	4.73	5.05	61
Time 2 GAD Symptoms	2.67	2.62	57
Time 2 SAD Symptoms	2.72	3.91	58
Age	7.74	1.06	88
PEP Baseline	97.04	12.22	68
RSA Baseline	3.97	.66	61
PEP Reactivity	.97	2.92	67
RSA Reactivity	.44	.74	59
PEP Emotion	97.97	12.82	68
RSA Emotion	3.73	.76	64
CBQ Anger/Frustration	4.63	1.38	87
CBQ Fear	3.90	1.29	87
CBQ Impulsivity	4.30	.70	86
CBQ Inhibitory Control	4.50	1.11	87

Table 2. (continued)

Variable	*M*	*SD*	*n*
WJ Letter-Word Identification	94.32	17.58	81
WJ Calculation	90.58	26.25	81
HBQ Peer Rejection	3.14	.67	87
Neighborhood Danger	.49	.30	87
Stressful Life Events	.10	.09	64
Hostile Bias	2.18	1.54	55

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder, SAD = separation anxiety disorder, PEP = pre-ejection period, RSA = respiratory sinus arrhythmia, CBQ = Child Behavior Questionnaire, WJ = Woodcock-Johnson Tests of Achievement, HBQ = MacArthur Health and Behavior Questionnaire.

Table 3. Bivariate Correlations among Symptoms

Variable	1	2	3	4	5	6
1. Time 1 ODD Symptoms	-					
2. Time 1 GAD Symptoms	.50**	-				
3. Time 1 SAD Symptoms	.28**	.49**	-			
4. Time 2 ODD Symptoms	.59**	.41**	.16	-		
5. Time 2 GAD Symptoms	.53**	.62**	.39**	.66**	-	
6. Time 2 SAD Symptoms	.28*	.29*	.65**	.20	.49**	-

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder, SAD = separation anxiety disorder.
** $p < .01$, * $p < .05$.

significantly greater than the relation between ODD and SAD symptoms (z score = 4.28, $p < .01$).

Explanation 1- GAD and/or SAD Symptoms Confer Risk for ODD Symptoms

Hypothesis 2a: Time 1 GAD symptoms were expected to be positively associated with Time 2 ODD symptoms.

Consistent with hypotheses, Time 1 GAD symptoms were positively associated with Time 2 ODD symptoms ($r = .41$, $p < .01$). Because of the significant relation, multiple regression analyses were conducted. Multiple regression analyses revealed that, after controlling for child age and child sex, Time 1 GAD symptoms prospectively predicted Time 2 ODD symptoms (β =

.35, $p < .01$). When Time 1 ODD symptoms were entered into the equation, Time 1 GAD symptoms no longer predicted Time 2 ODD symptoms (see Table 4; $\beta = .18$, $p > .05$).

Table 4. Multiple Regression Analysis Summary for Time 1 GAD Symptoms Predicting Time 2 ODD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.39	.39**	.64
Age	.04	.57	.01			
Sex	.80	.54	.16			
Time 1 ODD Symptoms	3.31	.65	.57**			
Step 2				.42	.03	.03
Time 1 GAD Symptoms	1.21	.78	.18			

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder.
** $p < .01$.

Hypothesis 2b: Hostile attribution biases were expected to partially mediate the relations between Time 1 GAD symptoms and Time 2 ODD symptoms.

Given that Time 1 GAD symptoms did not significantly predict Time 2 ODD symptoms after controlling for Time 1 ODD symptoms, multiple regression analyses examining hostile attribution biases as a possible mediator of the relation between Time 1 GAD and Time 2 ODD were not conducted.

Hypothesis 2c: Time 1 SAD symptoms were expected to be positively associated with Time 2 ODD symptoms.

Contrary to prediction, bivariate correlations between Time 1 SAD and Time 2 ODD symptoms were nonsignificant (r = .16, p >.05). As a result of the nonsignificant finding, multiple regression analyses examining this prospective relation were not conducted.

Hypothesis 2d: Hostile attribution biases were expected to partially mediate the relations between Time 1 GAD symptoms and Time 2 ODD symptoms.

Again, given that Time 1 SAD symptoms did not significantly predict Time 2 ODD symptoms, multiple regression analyses examining hostile attribution biases as a possible mediator of the relation between Time 1 SAD and Time 2 ODD were not conducted.

Explanation 2- ODD Symptoms Confer Risk for GAD and/or SAD Symptoms

Hypothesis 3a: Time 1 ODD symptoms were expected to be positively associated with Time 2 GAD symptoms.

Consistent with hypotheses, Time 1 ODD symptoms were positively associated with Time 2 GAD symptoms (r = .53, p < .01). Because of the significant relation, multiple regression analyses were conducted. Multiple regression analyses revealed that, after controlling for child age and child sex, Time 1 ODD symptoms prospectively predicted Time 2 GAD symptoms (β = .43, p < .01). When Time 1 GAD symptoms were entered into the first step, Time 1 ODD symptoms continued to predict Time 2 GAD symptoms (see Table 5; β = .27, p < .05).

Hypothesis 3b: I expected that peer rejection and poor academic skills at Time 1 would partially mediate the relations between Time 1 ODD symptoms and Time 2 GAD symptoms.

To examine peer rejection as a potential mediator, a series of multiple regression analyses were conducted. Time 1 ODD symptoms were entered into two equations, predicting (1) Time 2 GAD symptoms and (2) peer rejection. Next, peer rejection was entered simultaneously with

Table 5. Multiple Regression Analysis Summary for Time 1 ODD Symptoms Predicting Time 2 GAD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.45	.45	.81
Age	.65	.27	.25*			
Sex	.38	.27	.15			
Time 1 GAD Symptoms	1.86	.37	.53**			
Step 2				.51	.06*	.06
Time 1 ODD Symptoms	.80	.34	.27*			

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder.
** $p < .01$, * $p < .05$.

Time 1 ODD symptoms to examine whether peer rejection accounts for the relation between Time 1 ODD symptoms and Time 2 GAD symptoms.

Results for these multiple regression analyses are presented in Table 6. Consistent with hypotheses, peer rejection predicted Time 2 GAD symptoms when entered simultaneously with Time 1 ODD symptoms ($\beta = .27$, $p < .05$). Furthermore, the relation between Time 1 ODD and Time 2 GAD symptoms remained significant when peer rejection was entered into the equation ($\beta = .39$, $p < .01$), suggesting that peer rejection is a partial mediator of this prospective relation.

To further examine the role of peer rejection as a potential mediator of the relation between Time 1 ODD and Time 2 GAD symptoms, bootstrap analyses were conducted. Results from the bootstrap samples (Shrout & Bolger, 2002) indicated that Time 1 ODD symptoms

Table 6. Multiple Regression Analysis Summary for Time 1 ODD Symptoms and Peer Rejection Predicting Time 2 GAD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.20**	.20	.25
Age	.98	.32	.37**			
Sex	.69	.32	.26*			
Step 2				.43**	.23	.30
HBQ Peer Rejection	.66	.27	.27*			
Time 1 ODD Symptoms	1.16	.34	.39**			

Note. HBQ = MacArthur Health and Behavior Questionnaire, ODD = oppositional defiant disorder.
** $p < .01$, * $p < .05$.

tended to be associated with peer rejection, indirect effect = .03, $p < .10$, 95% CI (.01, .06). Peer rejection was significantly associated with Time 2 GAD symptoms, indirect effect = 1.12, $p < .01$, 95% CI (.42, 1.95). Last, the relation between Time 1 ODD symptoms and Time 2 GAD symptoms remained significant after controlling for peer rejection, direct effect = .27, $p < .01$, 95% CI (.13, .41). Because ODD at Time 1 did not significantly predict peer rejection, these results do not support the hypothesis that peer rejection mediates the relation between Time 1 ODD and Time 2 GAD symptoms. As noted previously, bootstrap procedures have been used to develop more accurate estimates of the indirect and direct effects when testing mediation; therefore, the nonsignificant bootstrap finding was interpreted as the more reliable result, as compared to the significant multiple regression mediation analysis.

Multiple regression analyses examining whether poor academic skills mediated the relation between Time 1 ODD and Time 2 GAD symptoms indicated that Time 1 ODD symptoms did not predict WJ Letter-Word Identification scores ($\beta = -.16$, $p > .05$) or WJ Calculation scores ($\beta = .06$, $p > .05$). Therefore, remaining tests of mediation for academic skills were not conducted.

Hypothesis 3c: Time 1 ODD symptoms were expected to be positively associated with Time 2 SAD symptoms.

Consistent with hypotheses, Time 1 ODD symptoms were positively correlated with Time 2 SAD symptoms ($r = .28$, $p < .05$). Multiple regression analyses revealed that, after controlling for child age and child sex, Time 1 ODD symptoms tended to prospectively predict Time 2 SAD symptoms ($\beta = .25$, $p < .10$). When Time 1 SAD symptoms were entered into the first step, Time 1 ODD symptoms no longer predicted Time 2 SAD symptoms (see Table 7; $\beta = .12$, $p > .05$).

Hypothesis 3d: I expected that peer rejection and poor academic skills at Time 1 would partially mediate the relations between Time 1 ODD symptoms and Time 2 SAD symptoms.

Because Time 1 ODD symptoms did not predict Time 2 SAD symptoms above and beyond Time 1 SAD symptoms, mediation analyses were not conducted.

Explanation 3 - Shared Risk Factors Account for the Co-occurrence of ODD+GAD and ODD+SAD

Bivariate correlations among Time 2 ODD, GAD, and SAD symptoms with potential shared risk factors are presented in Table 8.

Table 7. Multiple Regression Analysis Summary for Time 1 ODD Symptoms Predicting Time 2 SAD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.45	.45**	.82
Age	.68	.40	.18†			
Sex	.20	.40	.05			
Time 1 SAD Symptoms	2.62	.41	.66**			
Step 2				.47	.02	.02
Time 1 ODD Symptoms	.51	.48	.12			

Note. SAD = separation anxiety disorder, ODD = oppositional defiant disorder.
** $p < .01$, $^{\dagger}p < .10$.

Hypothesis 4a: Measures of difficult temperament (assessed at Time 1) were expected to be associated with Time 2 ODD, GAD, and SAD symptoms, even after controlling for child age and sex.

Consistent with hypotheses, CBQ Anger/Frustration ($r = .51$, $p < .01$) and CBQ Inhibitory Control ($r = -.47$, $p < .01$) were significantly associated with Time 2 ODD symptoms, and CBQ Impulsivity tended to be associated with Time 2 ODD symptoms ($r = .24$, $p < .10$). In addition, CBQ Inhibitory Control was significantly associated with Time 2 GAD symptoms ($r = -.39$, $p < .01$) and CBQ Fear was associated with Time 2 SAD symptoms ($r = .41$, $p < .01$). Contrary to hypotheses, CBQ Inhibitory Control was not related to Time 2 SAD symptoms ($r = -.03$, $p > .05$), and CBQ Fear was not related to Time 2 GAD symptoms ($r = .06$, $p > .05$). Also

Table 8. Bivariate Correlations Among Potential Shared Risk Factors and Time 2 Symptoms

Variable	1	2	3	4	5	6	7	8	9
1. Time 2 ODD Symptoms	-								
2. Time 2 GAD Symptoms	.66**	-							
3. Time 2 SAD Symptoms	.20	.49**	-						
4. Age	.19	.36**	.08	-					
5. CBQ Anger	.51**	.46**	.38**	.00	-				
6. CBQ Fear	-.04	.06	.41**	-.01	.17	-			
7.CBQ Impulsivity	.24†	.01	-.09	-.16	.43**	-.09	-		
8. CBQ Inhibitory Control	-.47**	-.39**	-.03	-.17	-.33**	-.04	-.27*	-	
9. PEP Baseline	.28†	.38*	.12	.35**	.26*	-.04	.00	-.21†	-
10. RSA Baseline	-.08	-.12	-.08	.19	.15	.13	-.08	-.09	-.09
11. PEP Reactivity	.04	-.17	-.11	-.08	-.14	-.04	.04	.06	-.01
12. RSA Reactivity	.06	.03	-.04	-.16	-.07	.04	.14	.05	.18
13. PEP Emotion	.30*	.38*	.13	.33**	.20	-.05	-.02	-.22†	.96**
14. RSA Emotion	.04	.03	-.13	.02	.23†	-.02	.28*	-.13	.23†
15. Neighborhood Danger	.03	.04	.04	.30**	-.04	.21†	-.06	-.12	.13
16. Stressful Life Events	.22†	.38**	.07	.30*	.12	.04	.07	-.14	.45**

Table 8. (continued)

Variable	10	11	12	13	14	15	16
1. Time 2 ODD Symptoms							
2. Time 2 GAD Symptoms Time 2							
3. Time 2 SAD Symptoms							
4. Age							
5. CBQ Anger							
6.CBQ Fear							
7. CBQ Impulsivity							
8. CBQ Inhibitory Control							
9. PEP Baseline							
10. RSA Baseline	-						
11. PEP Reactivity	.13	-					
12. RSA Reactivity	-.79**	.06	-				
13. PEP Emotion	-.12	.18	.19	-			
14. RSA Emotion	.21†	.00	.04	.20	-		
15. Neighborhood Danger	.06	-.12	-.00	.12	-.07	-	
16. Stressful Life Events	-.17	-.27†	-.12	.34*	.07	.19	-

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder, SAD = separation anxiety disorder disorder, CBQ = Child Behavior Questionnaire, PEP = pre-ejection period, RSA = respiratory sinus arrhythmia.
**p <.01, *p <.05, $^{\dagger}p$ < .10.

contrary to hypotheses, CBQ Anger/Frustration was associated with Time 2 GAD (r = .46, p < .01) and Time 2 SAD (r = .38, p < .01) symptoms.

Multiple regression analyses indicated that, after controlling for child age and sex, CBQ Anger/Frustration (β = .44, p < .01) and CBQ Inhibitory Control (β = -.36, p < .01) significantly predicted Time 2 ODD symptoms. When Time 1 ODD symptoms were entered into the equation, CBQ Inhibitory Control continued to predict Time 2 ODD symptoms (β = -.30, p < .05) and CBQ Anger/Frustration tended to predict Time 2 ODD symptoms (β = .26, p < .10; see Table 9).

Table 9. Multiple Regression Analysis Summary for Time 1 Temperament Measures Predicting Time 2 ODD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.36	.36**	.56
Age	.17	.57	.03			
Sex	.71	.55	.14			
Time 1 ODD Symptoms	3.16	.66	.55**			
Step 2				.46	.10*	.11
CBQ Anger/Frustration	1.37	.72	.26†			
CBQ Inhibitory Control	-1.49	.57	-.30*			

Note. CBQ = Child Behavior Questionnaire.
** p < .01, * p < .05, † p < .10.

After controlling for child age and sex, CBQ Anger/Frustration (β = .41, p < .01) significantly predicted Time 2 GAD symptoms; however, CBQ Inhibitory Control (β = -.20, p < .10) only

tended toward significance. When Time 1 GAD symptoms were entered into the equation, prediction from CBQ Anger/Frustration continued to be significant (β = .26, $p < .05$) whereas CBQ Inhibitory Control was no longer significant (β = -.12, $p > .05$; see Table 10). Last, after controlling for child age and sex, CBQ Anger/Frustration (β = .29, $p < .05$) and CBQ Fear (β = .37, $p < .01$) predicted Time 2 SAD symptoms. When Time 1 SAD symptoms were included, CBQ Anger/Frustration (β = .20, $p < .10$) and CBQ Fear (β = .18, $p < .10$) tended to predict Time 2 SAD symptoms (see Table 11).

Table 10. Multiple Regression Analysis Summary for Time 1 Temperament Measures Predicting Time 2 GAD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.45	.45**	.82
Age	.65	.27	.25**			
Sex	.38	.27	.15			
Time 1 GAD Symptoms	1.86	.37	.53**			
Step 2				.52	.07*	.08
CBQ Anger/Frustration	.72	.31	.26*			
CBQ Inhibitory Control	-.31	.28	-.12			

Note. CBQ = Child Behavior Questionnaire.
** $p < .01$, * $p < .05$, $^{\dagger}p < .10$.

Table 11. Multiple Regression Analysis Summary for Time 1 Temperament Measures Predicting Time 2 SAD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.45	.45**	.82
Age	.68	.40	.18†			
Sex	.20	.40	.05			
Time 1 SAD Symptoms	2.62	.41	.66**			
Step 2				.52	.07*	.08
CBQ Anger/Frustration	.82	.43	.20†			
CBQ Fear	.72	.43	.18†			

Note. CBQ = Child Behavior Questionnaire.
** $p < .01$, * $p < .05$, † $p < .10$.

Hypothesis 4b: Autonomic nervous system activity (assessed at Time 1) was expected to be associated with Time 2 ODD, GAD, and SAD symptoms.

Consistent with hypotheses, longer PEP scores (i.e., decreased sympathetic activity) at baseline were positively associated with Time 2 ODD symptoms ($r = .28$, $p < .05$). Contrary to hypotheses, longer PEP scores at baseline were positively associated with Time 2 GAD symptoms ($r = .38$, $p < .05$), and longer PEP scores during the emotion task were positively associated with both Time 2 ODD ($r = .30$, $p < .01$) and Time 2 GAD ($r = .38$, $p < .01$) symptoms. Also contrary to hypotheses, RSA measures were not related to Time 2 ODD symptoms, RSA at baseline was not related to Time 2 GAD or SAD symptoms, PEP measures

were not related to Time 2 SAD symptoms, and PEP reactivity measures were not related to Time 2 GAD and Time 2 ODD symptoms (all *r*s < .17, all *p*s > .05; see Table 8).

After controlling for child age and sex, multiple regression analyses indicated that PEP scores during the emotion task (β = .17, $p > .05$) did not significantly predict Time 2 ODD symptoms (see Table 12). Similarly, PEP scores at baseline (β = .19, $p > .05$) and PEP scores during the emotion task (β = .18, $p < .10$) did not significantly predict Time 2 GAD symptoms (see Table 13). As a result of the nonsignificant findings, multiple regression analyses examining whether autonomic functioning variables predicted Time 2 symptoms above and beyond Time 1 symptoms were not conducted.

Table 12. Multiple Regression Analysis Summary for Time 1 PEP Emotion Predicting Time 2 ODD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.15	.15*	.18
Age	1.24	.71	.25†			
Sex	1.60	.75	.30*			
Step 2				.17	.02	.02
PEP Emotion	.80	.79	.17			

Note. PEP = pre-ejection period.
* $p < .05$, $^{\dagger}p < .10$.

Table 13. Multiple Regression Analysis Summary for Time 1 PEP Baseline and PEP Emotion Predicting Time 2 GAD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.26	.26**	.35
Age	.98	.34	.41**			
Sex	1.66	.79	.30*			
Step 2				.31	.05	.05
PEP Baseline	.04	.03	.19			
PEP Emotion	.04	.04	.18			

Note. PEP = pre-ejection period.
** $p < .01$, * $p < .05$.

Hypothesis 4c: I hypothesized that neighborhood danger (assessed at Time 1) would be positively associated with Time 2 ODD, GAD, and SAD symptoms, and these associations would be maintained after controlling for child age and sex.

Contrary to prediction, neighborhood danger was not significantly correlated with Time 2 ODD ($r = .03$, $p > .05$), GAD ($r = .04$, $p > .05$), or SAD ($r = .04$, $p > .05$) symptoms. Therefore, multiple regression analyses examining the prediction of neighborhood danger to Time 2 symptoms were not conducted.

Hypothesis 4d: I predicted that the number of stressful life events experienced in the past year would be positively associated with Time 2 ODD, GAD, and SAD symptoms, and that these associations would remain significant after controlling for child age and sex.

Consistent with hypotheses, the number of stressful life events experienced in the past year were significantly associated with Time 2 GAD symptoms (r = .38, p <.05) and tended to be significantly associated with Time 2 ODD symptoms (r = .22, p <.10). Contrary to hypotheses, stressful life events were not significantly correlated with Time 2 SAD symptoms (r = .07, p >.05). After controlling for child age and sex, multiple regression analyses indicated that stressful life events significantly predicted Time 2 GAD symptoms (β = .27, p < .05). Stressful life events continued to predict Time 2 GAD symptoms after controlling for Time 1 symptoms (β = .25, p < .05; see Table 14).

Table 14. Multiple Regression Analysis Summary for Stressful Life Events Predicting Time 2 GAD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.45	.45**	.82
Age	.65	.27	.25*			
Sex	.38	.27	.15			
Time 1 GAD Symptoms	1.86	.37	.53**			
Step 2				.51	.06*	.06
Stressful Life Events	.67	.28	.25*			

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

Child × Context Interactions

Hypothesis 5a: I hypothesized that measures of difficult temperament would interact with neighborhood danger in the prediction of Time 2 ODD, GAD, and SAD symptoms.

Difficult temperament × neighborhood danger interactions did not significantly predict Time 2 ODD, GAD, or SAD symptoms (see Table 15).

Hypothesis 5b: I hypothesized that measures of difficult temperament would interact with stressful life events in the prediction of Time 2 ODD, GAD, and SAD symptoms.

Difficult temperament × stressful life events interactions did not significantly predict Time 2 ODD, GAD, or SAD symptoms (see Table 16).

Hypothesis 5c: I hypothesized that measures of autonomic functioning would interact with neighborhood danger in the prediction of Time 2 ODD, GAD, and SAD symptoms.

Multiple regression analyses, controlling for child age and sex, indicated that the RSA baseline × neighborhood danger interaction tended ($p < .10$) to predict Time 2 ODD symptoms (see Table 17). To explore this relation, post hoc probing procedures were conducted based on the guidelines of Aiken and West (1991) and Holmbeck (2002). Specifically, two new conditional moderator variables (± 1 *SD* from the mean of neighborhood danger) and new interactions that incorporated the conditional variables were computed. Two post-hoc regressions, each of which involved simultaneous entry of RSA baseline, one of the conditional neighborhood danger variables, and the RSA baseline × conditional neighborhood danger variable were then conducted (Holmbeck, 2002). From these analyses, unstandardized betas (slopes) and a regression equation for children experiencing high (1 *SD* below the mean) and low (1 *SD* above the mean) neighborhood danger were derived. Post hoc probing indicated that the slope was not significantly different from zero for high ($\beta = .-.01$, $t(37) = -.07$, $p > .05$, Cohen's *d*

Table 15. Regression Beta Weights for Nonsignificant Temperament × Neighborhood Danger Interaction Terms

Outcome	Interaction Term	*B*	*SE B*	β
Time 2 ODD				
	CBQ Anger × Neighborhood Danger	.43	.68	.07
	CBQ Fear × Neighborhood Danger	.11	.74	.02
	CBQ Impulsivity × Neighborhood Danger	-.37	.66	-.07
	CBQ Inhibitory Control × Neighborhood Danger	-.37	.63	-.08
Time 2 GAD				
	CBQ Anger × Neighborhood Danger	.21	.34	.07
	CBQ Fear × Neighborhood Danger	.04	.37	.01
	CBQ Impulsivity × Neighborhood Danger	.56	.37	.20
	CBQ Inhibitory Control × Neighborhood Danger	-.13	.33	-.05
Time 2 SAD				
	CBQ Anger × Neighborhood Danger	.77	.59	.17
	CBQ Fear × Neighborhood Danger	.70	.54	.16
	CBQ Impulsivity × Neighborhood Danger	.34	.72	.07
	CBQ Inhibitory Control × Neighborhood Danger	-.50	.56	-.14

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder, SAD = separation anxiety disorder, CBQ = Child Behavior Questionnaire.

Table 16. Regression Beta Weights for Nonsignificant Temperament × Stressful Life Events Interaction Terms

Outcome	Interaction Term	*B*	*SE B*	β
Time 2 ODD				
	CBQ Anger × Stressful Life Events	.83	.79	.13
	CBQ Fear × Stressful Life Events	-.63	.69	-.11
	CBQ Impulsivity × Stressful Life Events	-.46	.74	-.08
	CBQ Inhibitory Control × Stressful Life Events	-.73	.71	-.12
Time 2 GAD				
	CBQ Anger × Stressful Life Events	.38	.39	.11
	CBQ Fear × Stressful Life Events	-.18	.34	-.07
	CBQ Impulsivity × Stressful Life Events	-.09	.37	-.03
	CBQ Inhibitory Control × Stressful Life Events	-.11	.36	-.04
Time 2 SAD				
	CBQ Anger × Stressful Life Events	-.45	.68	-.09
	CBQ Fear × Stressful Life Events	.43	.51	.10
	CBQ Impulsivity × Stressful Life Events	-.76	.62	-.18
	CBQ Inhibitory Control × Stressful Life Events	.28	.62	.06

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder, SAD = separation anxiety disorder, CBQ = Child Behavior Questionnaire.

Table 17. Multiple Regression Analysis Summary for the RSA Baseline × Neighborhood Danger Interaction Term in the Prediction of Time 2 ODD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.17	.17*	.20
Age	1.52	.75	.20†			
Sex	1.72	.83	.31*			
Step 2				.26	.09	.10
RSA Baseline	-.22	.85	-.04			
Neighborhood Danger	.45	.89	.08			
RSA Baseline × Neighborhood Danger	1.56	.84	.28†			

Note. RSA = respiratory sinus arrhythmia.
* $p < .05$, $^{\dagger}p < .10$.

= .02) or for low (β =.15, $t(37) = .96$, $p > .05$, Cohen's $d = .32$) neighborhood danger, indicating that the impact of RSA baseline levels on Time 2 ODD symptoms did not differ for children experiencing low vs. high levels of neighborhood danger (see Figure 1). Examination of Figure 1 suggests that children with low RSA baseline, who have experienced low levels of neighborhood danger, tend to have the lowest levels of Time 2 ODD symptoms.

Multiple regression analyses, controlling for child age and sex, indicated that RSA reactivity × neighborhood danger tended to predict Time 2 ODD symptoms (see Table 18). Post-hoc probing indicated that the slope was not significantly different from zero for high (β = .002, $t(36) = .01$, $p > .05$, Cohen's $d = .00$) or for low (β = .09, $t(36) = .52$, $p > .05$, Cohen's d =.17)

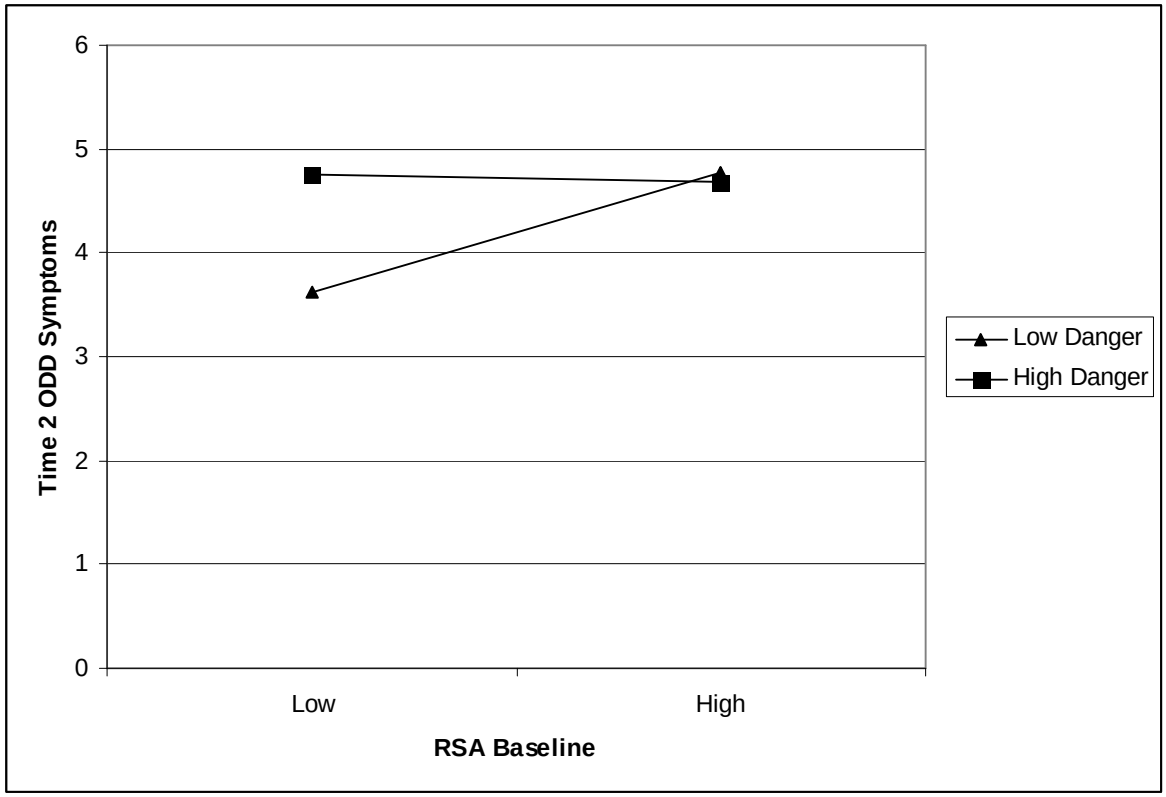

Figure 1. Relation between RSA baseline and Time 2 ODD symptoms among children experiencing high (1 *SD* above mean) vs. low (1 *SD* below mean) levels of neighborhood danger.

neighborhood danger, suggesting that the impact of RSA reactivity levels on Time 2 ODD symptoms did not differ among children experiencing low vs. high levels of neighborhood danger (see Figure 2). Examination of Figure 2 suggests that children with low RSA reactivity, who have experienced low levels of neighborhood danger, tend to have the lowest levels of Time 2 ODD symptoms.

After controlling for child age and sex, multiple regression analyses indicated that the RSA reactivity × neighborhood danger interaction term also tended to predict Time 2 SAD symptoms (see Table 19). Post-hoc probing indicated that the slope was not significantly different from zero for high (β = .18, $t(36) = .97$, $p > .05$, Cohen's $d = .32$) or for low (β = -.05,

Table 18. Multiple Regression Analysis Summary for the RSA Reactivity × Neighborhood Danger Interaction Term in the Prediction of Time 2 ODD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.17	.17*	.20
Age	1.53	.77	.30†			
Sex	1.71	.85	.30†			
Step 2				.25	.08	.08
RSA Reactivity	-.04	.92	-.01			
Neighborhood Danger	.42	.92	.07			
RSA Reactivity × Neighborhood Danger	-1.62	.93	-.29†			

Note. RSA = respiratory sinus arrhythmia
* $p < .05$, † $p < .10$.

$t(36) = -.33$, $p > .05$, Cohen's $d = .11$) neighborhood danger, suggesting that the impact of RSA reactivity levels on Time 2 SAD symptoms did not differ among children experiencing low vs. high levels of neighborhood danger (see Figure 3). Examination of Figure 3 suggests that among children experiencing high levels of neighborhood danger, those with high RSA reactivity tend to exhibit more Time 2 SAD symptoms compared to those with low RSA reactivity.

Interaction terms between autonomic functioning variables and neighborhood danger that did not predict Time 2 symptoms are presented in Table 20.

Hypothesis 5d: I hypothesized that measures of autonomic functioning would interact with stressful life events in the prediction of Time 2 ODD, GAD, and SAD symptoms.

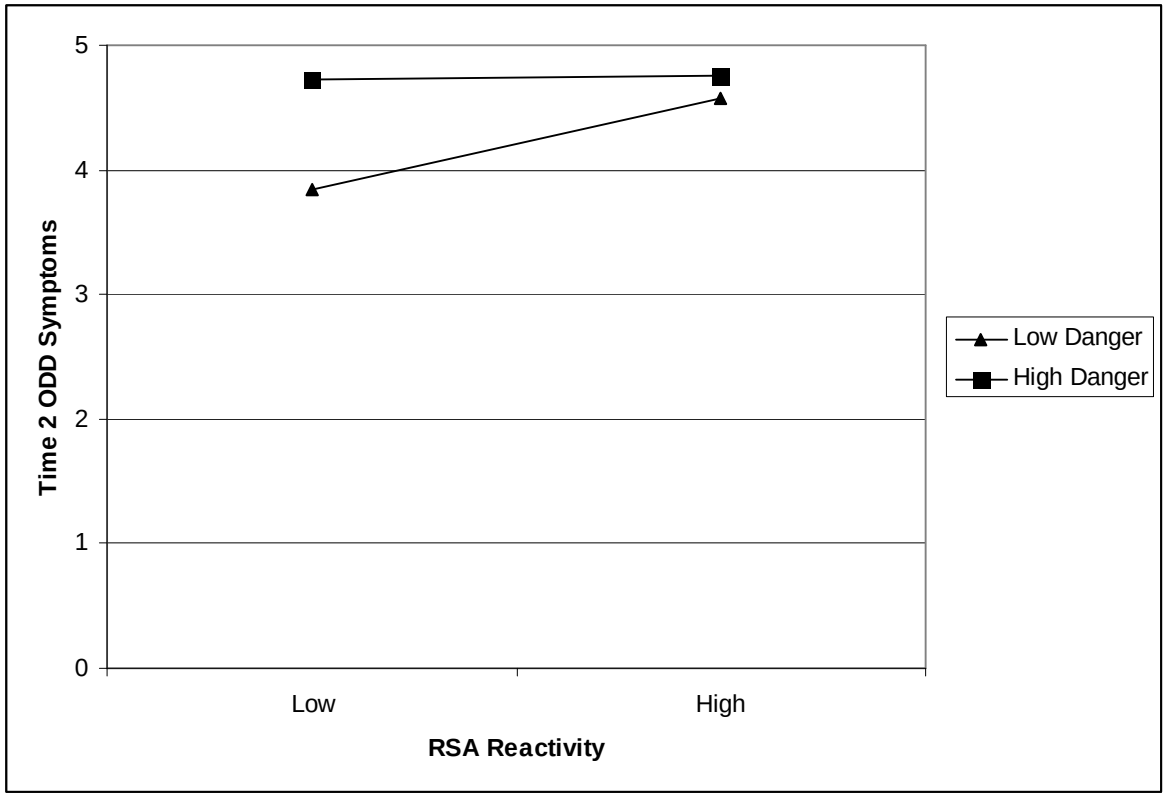

Figure 2. Relation between RSA reactivity and Time 2 ODD symptoms among children experiencing high (1 SD above mean) vs. low (1 SD below mean) levels of neighborhood danger.

Multiple regression analyses indicated that, after controlling for child age and sex, the RSA reactivity × stressful life events interaction term predicted Time 2 ODD symptoms (Table 21). Post-hoc probing indicated that the slope was not significantly different from zero for high (β = -.10, $t(36)$ = -.59, p > .05, Cohen's d = .20) or for low (β = -.03, $t(36)$ = -.18, p > .05, Cohen's d = .06) stressful life events, suggesting that the impact of RSA reactivity levels on Time 2 ODD symptoms did not differ among children experiencing low vs. high levels of stressful life events (see Figure 4). Examination of Figure 4 suggests that, among children with low RSA reactivity, those experiencing higher levels of stress tended to be rated as having higher levels of Time 2 ODD symptoms, as compared to children experiencing lower levels of stress.

Table 19. Multiple Regression Analysis Summary for the RSA Reactivity × Neighborhood Danger Interaction Term in the Prediction of Time 2 SAD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.10	.10	.11
Age	.37	.67	.09			
Sex	1.37	.75	.30†			
Step 2				.20	.10	.11
RSA Reactivity	-.01	.79	-.00			
Neighborhood Danger	-.24	.84	-.05			
RSA Reactivity × Neighborhood Danger	1.51	.79	.34†			

Note. RSA = respiratory sinus arrhythmia.
†$p < .10$.

Interaction terms between autonomic functioning variables and stressful life events that did not predict Time 2 symptoms are presented in Table 22.

Sex Differences

Hypothesis 6a: I hypothesized that boys and girls would differ in terms of their levels of ODD, GAD, and SAD symptoms. Severity of GAD and SAD symptoms was hypothesized to be greater among girls, whereas severity of ODD symptoms was hypothesized to be greater among boys.

Results of independent *t*-tests are located in Table 23. Consistent with hypotheses, boys tended to be rated as having higher levels of Time 2 ODD symptoms as compared to girls, $t(60) = -1.96$, $p < .10$, Cohen's $d = .51$. Contrary to prediction, however, boys also tended to be rated

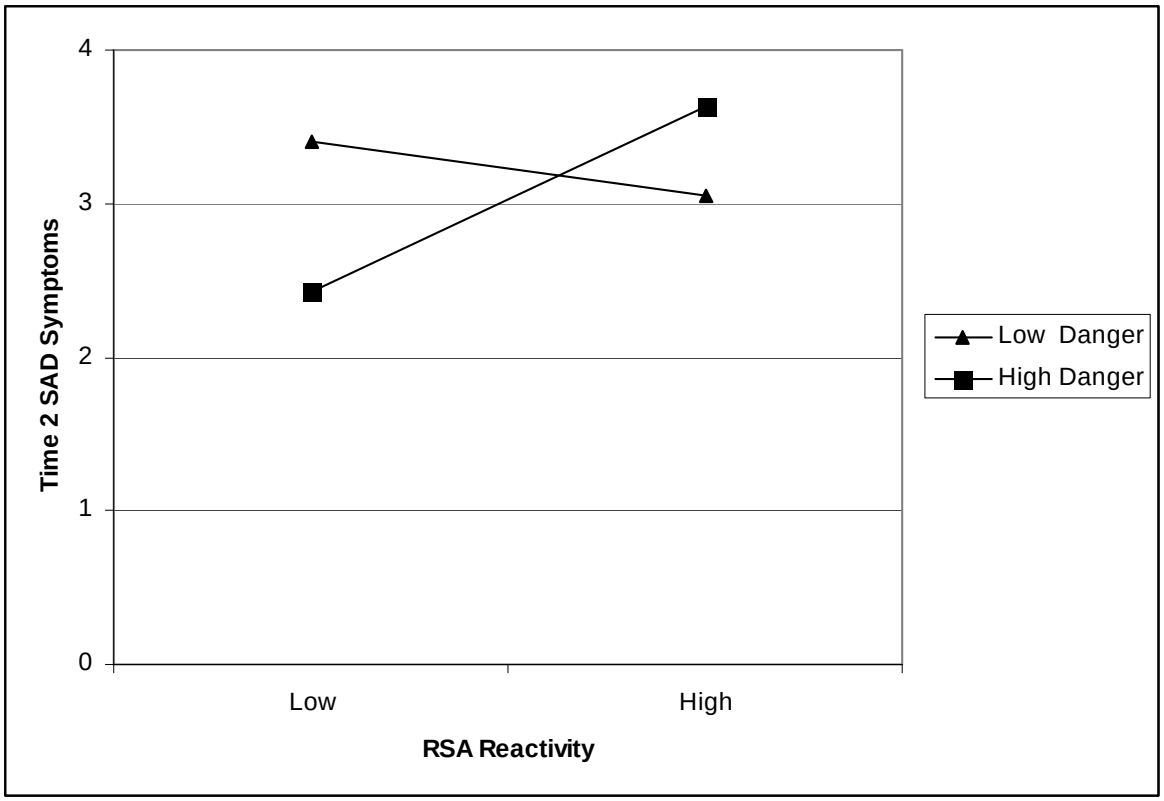

Figure 3. Relation between RSA reactivity and Time 2 SAD symptoms among children experiencing high (1 SD above mean) vs. low (1 SD below mean) levels of neighborhood danger.

as having higher levels of Time 2 GAD symptoms, as compared to girls, $t(60) = -1.86$, $p < .10$, Cohen's $d = .50$. Boys and girls did not differ in terms of Time 1 ODD symptoms, Time 1 GAD symptoms, or Time 1 and Time 2 SAD symptoms.

Hypothesis 6b: I hypothesized that boys would evidence lower levels of parent-reported inhibitory control and higher levels of parent-reported impulsivity compared to girls. Perception of neighborhood danger was expected to be higher among girls than boys. No sex differences were hypothesized for temperamental anger/frustration or fear, autonomic functioning, or number of stressful life events reported by parents.

Table 20. Regression Beta Weights for Nonsignificant Autonomic Functioning × Neighborhood Danger Interaction Terms

Outcome	Interaction Term	*B*	*SE B*	β
Time 2 ODD				
	PEP Baseline × Neighborhood Danger	.46	.89	.08
	PEP Reactivity × Neighborhood Danger	-1.02	1.05	-.14
	PEP Emotion × Neighborhood Danger	.57	.86	.11
	RSA Emotion × Neighborhood Danger	-.55	.94	-.09
Time 2 GAD				
	PEP Baseline × Neighborhood Danger	-.13	.44	-.05
	PEP Reactivity × Neighborhood Danger	-.12	.53	-.03
	PEP Emotion × Neighborhood Danger	-.12	.44	-.04
	RSA Baseline × Neighborhood Danger	-.05	.43	-.02
	RSA Reactivity × Neighborhood Danger	.22	.45	.08
	RSA Emotion × Neighborhood Danger	.12	.42	.04
Time 2 SAD				
	PEP Baseline × Neighborhood Danger	-.40	.71	-.10
	PEP Reactivity × Neighborhood Danger	-.04	.79	-.01

Table 20. (continued)

Outcome	Interaction Term	*B*	*SE B*	β
Time 2 SAD				
	PEP Emotion × Neighborhood Danger	-.46	.69	-.12
	RSA Baseline × Neighborhood Danger	-.88	.77	-.20
	RSA Emotion × Neighborhood Danger	.67	.78	.14

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder, SAD = separation anxiety disorder, PEP = pre-ejection period, RSA = respiratory sinus arrhythmia.

Table 21. Multiple Regression Analysis Summary for the RSA Reactivity × Stressful Life Events Interaction Term in the Prediction of Time 2 ODD Symptoms

Step and Variable	*B*	*SE B*	β	R^2	ΔR^2	f^2
Step 1				.17	.17*	.20
Age	1.53	.77	.30†			
Sex	1.71	.85	.30†			
Step 2				.38	.21*	.27
RSA Reactivity	-.68	.85	-.12			
Stressful Life Events	1.92	1.00	.29†			
RSA Reactivity × Stressful Life Events	-3.06	1.16	-.39*			

Note. RSA = respiratory sinus arrhythmia.
*$p < .05$, $^{\dagger}p < .10$.

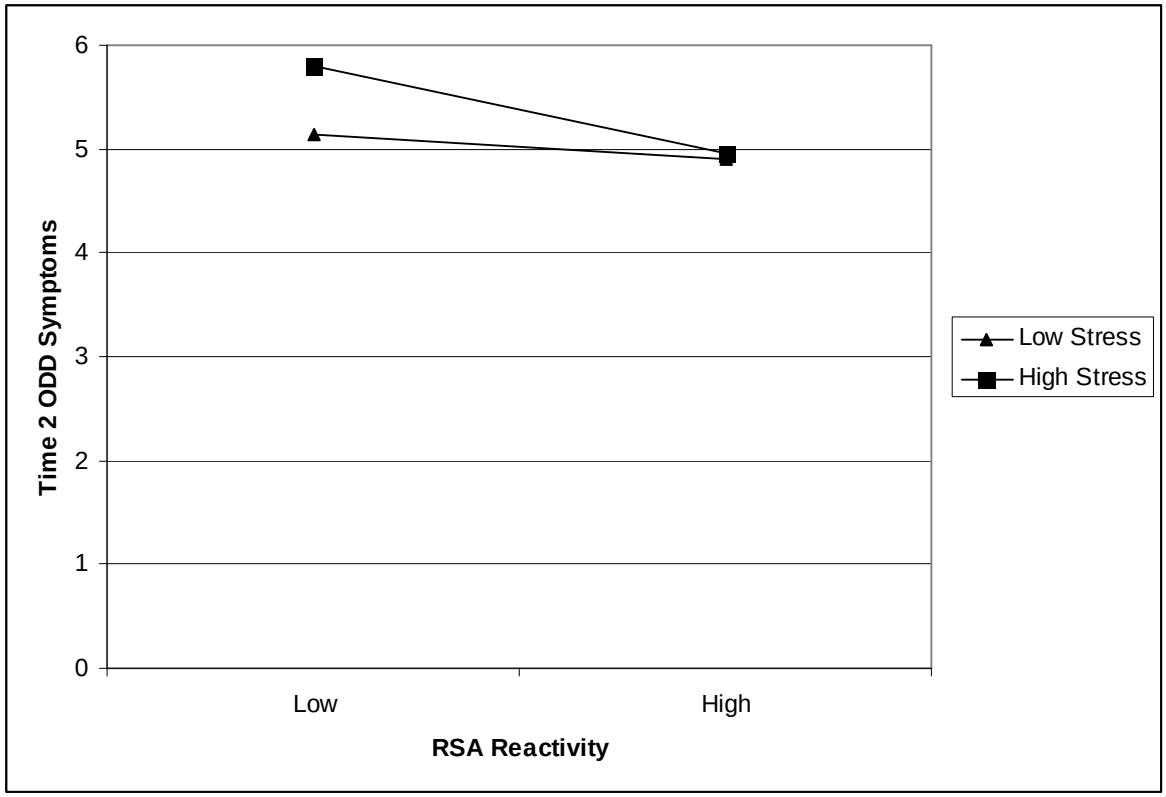

Figure 4. Relation between RSA reactivity and Time 2 ODD symptoms among children experiencing high (1 SD above mean) vs. low (1 SD below mean) levels of stressful life events.

Consistent with hypotheses, results indicated that boys were rated as having lower levels of CBQ Inhibitory Control, as compared to girls, $t(85) = -2.20$, $p < .05$, Cohen's $d = .48$, and perception of neighborhood danger was greater among girls as compared to boys, $t(85) = -2.88$, $p < .01$, Cohen's $d = .63$. In addition, temperamental anger/frustration and fear, autonomic functioning measures, and stressful life events were not found to differ among boys and girls (see Table 23). Contrary to prediction, temperamental impulsivity was not greater among boys than girls, $t(85) = -.54$, $p > .05$, Cohen's $d = .12$.

Table 22. Regression Beta Weights for Nonsignificant Autonomic Functioning × Stressful Life Events Interaction Terms

Outcome	Interaction Term	*B*	*SE B*	β
Time 2 ODD				
	PEP Baseline × Stressful Life Events	-.69	.70	-.15
	PEP Reactivity × Stressful Life Events	-.66	1.47	-.08
	PEP Emotion × Stressful Life Events	-.30	.70	-.06
	RSA Baseline × Stressful Life Events	1.52	.99	.22
	RSA Emotion × Stressful Life Events	-1.15	.89	-.22
Time 2 GAD				
	PEP Baseline × Stressful Life Events	-.06	.36	-.03
	PEP Reactivity × Stressful Life Events	.13	.66	.03
	PEP Emotion × Stressful Life Events	.11	.37	.04
	RSA Baseline × Stressful Life Events	-.12	.44	-.04
	RSA Reactivity × Stressful Life Events	-.17	.57	-.04
	RSA Emotion × Stressful Life Events	-.60	.40	-.24
Time 2 SAD				
	PEP Baseline × Stressful Life Events	-.55	.60	-.16

Table 22. (continued)

Outcome	Interaction Term	*B*	*SE B*	B
Time 2 SAD (cont.)				
	PEP Reactivity × Stressful Life Events	-.29	1.09	-.05
	PEP Emotion × Stressful Life Events	-.40	.59	-.11
	RSA Baseline × Stressful Life Events	-.106	.87	-.21
	RSA Reactivity × Stressful Life Events	1.32	1.06	.22
	RSA Emotion × Stressful Life Events	.28	.71	.07

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder, SAD = separation anxiety disorder, PEP = pre-ejection period, RSA = respiratory sinus arrhythmia.

Hypothesis 6c: I expected that there would be sex differences in the bivariate associations between symptoms (e.g., ODD and GAD, ODD and SAD). However, given inconsistencies in the literature, no directional hypotheses were made for sex differences among symptom associations.

Bivariate correlations among symptoms categories for boys and girls are presented in Table 24. Contrary to prediction, comparison of correlation coefficients among symptom categories between boys and girls indicates that the magnitude of several associations did not differ based on sex. Specifically, correlation coefficients between Time 1 ODD and Time 1 SAD symptoms (z score = 1.12, $p > .05$), Time 1 ODD and Time 2 SAD symptoms (z score = -.19, $p > .05$), Time 1 SAD and Time 2 ODD symptoms (z score = -.98, $p > .05$), and Time 1 SAD and Time 2 ODD symptoms (z score = -.11, $p > .05$) were not significantly greater among girls than boys. Similarly, the magnitude of the association between Time 1 ODD and Time 1 GAD (z score = .47, $p > .05$), Time 1 ODD and Time 2 GAD (z score = .19, $p > .05$), Time 1 GAD and

Table 23. Means (SD) and Comparison Statistics for Boys vs. Girls

Variable	Boys	Girls	*t*	Effect Size
Time 1 ODD Symptoms	7.10 (4.97)	5.51 (5.30)	1.43	.31
Time 1 GAD Symptoms	2.93 (2.31)	2.57 (3.41)	.58	.13
Time 1 SAD Symptoms	3.23 (4.06)	3.12 (4.13)	.12	.03
Time 2 ODD Symptoms	6.00 (4.59)	3.51 (5.26)	1.96†	.51
Time 2 GAD Symptoms	3.33 (2.75)	2.07 (2.41)	1.86†	.50
Time 2 SAD Symptoms	3.48 (4.90)	1.97 (2.44)	1.49	.39
Age	7.75 (.99)	7.73 (1.13)	.04	.00
PEP Baseline	97.44 (14.48)	96.69 (10.15)	.25	.06
RSA Baseline	3.88 (.66)	4.05 (.65)	-1.02	.27
PEP Reactivity	1.69 (2.76)	.34 (2.94)	1.94†	.48
RSA Reactivity	.47 (.84)	.41 (.65)	.33	.09
PEP Emotion	99.83 (14.2)	96.22 (11.29)	1.16	.29
RSA Emotion	3.77 (.83)	3.68 (.70)	.46	.12
CBQ Anger/Frustration	4.91 (1.20)	4.34 (1.51)	1.94†	.42
CBQ Fear	3.83 (1.31)	3.98 (1.27)	-.55	.12
CBQ Impulsivity	4.25 (.63)	4.34 (.76)	-.54	.12
CBQ Inhibitory Control	4.24 (1.05)	4.75 (1.13)	-2.20*	.48
Neighborhood Danger	.40 (.27)	.58 (.29)	-2.88**	.63

Table 23. (continued)

Variable	Boys	Girls	*t*	Effect Size
Stressful Life Events	.10(.09)	.10 (.09)	.29	.07

Note. ODD = oppositional defiant disorder, GAD = generalized anxiety disorder, SAD = separation anxiety disorder, PEP = pre-ejection period, RSA = respiratory sinus arrhythmia, CBQ = Child Behavior Questionnaire, effect size = Cohen's *d.*
** $p < .01$, * $p < .05$, $^{\dagger} p < .10$.

Time 2 ODD (z score = .26, $p > .05$), and Time 2 ODD and Time 2 GAD (z score = 1.59, $p > .05$) symptoms were not greater among boys than girls.

Table 24. Bivariate Correlations for Time 1 and Time 2 Symptoms Among Boys and Girls

Variable	1	2	3	4	5	6
1. Time 1 ODD Symptoms	-	**.40****	**.53****	**.58****	**.23**	**.53****
2. Time 1 SAD Symptoms	.17	-	**.65****	**.01**	**.30**	**.28**
3. Time 1 GAD Symptoms	.45**	.27†	-	**.41***	**.11**	**.54****
4. Time 2 ODD Symptoms	.55**	.27	.35†	-	**.15**	**.76****
5. Time 2 SAD Symptoms	.28	.75**	.37*	.19	-	**.32**†
6. Time 2 GAD Symptoms	.50**	.42**	.66**	.51**	.57**	-

Note. ODD = oppositional defiant disorder, SAD = separation anxiety disorder, GAD = generalized anxiety disorder. Correlations among boys are below the diagonal. Correlations among girls are in bold and above the diagonal.
** $p < .01$, * $p < .05$, $^{\dagger} p < .10$.

CHAPTER 4

DISCUSSION

Despite having important implications for etiological and intervention models, little research has focused on understanding the development of co-occurring ODD+GAD and ODD+SAD among children. Therefore, the primary goal of this study was to examine multiple explanations for the co-occurrence of ODD+GAD and ODD+SAD. Specifically, in the present study, I investigated whether (1) GAD symptoms prospectively predicted ODD symptoms, and SAD symptoms prospectively predicted ODD symptoms; (2) ODD symptoms prospectively predicted GAD symptoms, and ODD symptoms prospectively predicted SAD symptoms; and (3) shared risk factors accounted for the co-occurrence of ODD+GAD and ODD+SAD. Findings provide support for explanation 2 and explanation 3 in the development of co-occurring ODD+GAD symptoms and support for explanation 3 in the development of co-occurring ODD+SAD symptoms. Support for these different explanations suggests that the investigation of specific symptom categories (e.g., GAD and SAD), rather than a combination of symptoms (e.g., anxiety symptoms), may be particularly useful for developing conceptual models that attempt to outline the etiological pathways of comorbid disorders. This study furthermore contributes to the extant literature by providing the first empirical examination of these multiple explanations in an ethnic minority, inner city sample of children.

Concurrent Relations

Consistent with study hypotheses and previous research, findings demonstrated concurrent associations between ODD, GAD, and SAD symptoms (Verduin & Kendall, 2003). However, these findings also extend previous research because there has been a limited amount of work that has examined anxiety symptoms, as well as the co-occurrence of ODD+GAD and

ODD+SAD symptoms, among predominantly impoverished, ethnic minority children living in the inner city (Neal-Barnett, 2004). These results indicate that anxiety symptoms are important to consider among ethnic minority youth (Neal-Barnett & Smith, 1997) and may be helpful for understanding the developmental pathways of disruptive behavior disorders, such as ODD.

Explanation 1

The hypothesis that GAD symptoms would prospectively predict ODD symptoms, after controlling for Time 1 ODD symptoms, was not supported in this sample of children. Similarly, results did not support the hypothesis that SAD symptoms would prospectively predict ODD symptoms. These findings are inconsistent with previous research suggesting a positive and prospective relation between anxiety and externalizing symptoms (Foley et al., 2004; Ialongo et al., 1994). The lack of support for explanation 1 may be a function of the current sample; that is, the current sample's age and cultural background and the way in which families were recruited into the study (i.e., self selected) may account for the nonsignificant findings. For instance, the average age of children in the present study was approximately one year older than the Ialongo et al. (1994) sample. In addition, the current sample was significantly different in terms of the age and cultural background from the Foley et al. (2004) study sample, which was composed of Caucasian twin pairs aged 8 to 17 who were enrolled in private schools. Thus, it may be that the age range of the present sample was not appropriate for the identification of a relation between Time 1 anxiety and Time 2 ODD symptoms. Contextual factors also may be important. It may be that children from disadvantaged urban communities, who are subject to increased risk for neighborhood violence exposure (Attar et al., 1994; Gorman-Smith & Tolan, 1998), are more likely to exhibit externalizing behaviors before the onset of anxiety symptoms. Finally, it may be that families self-select into the CHBS because their children are exhibiting higher levels of

externalizing symptoms. In this way, a higher level of initial externalizing symptoms would limit the ability to identify a prospective relation between anxiety and ODD symptoms. To determine whether this study's findings are specific to the current sample, future research will need to examine whether GAD and/or SAD symptoms confer risk for ODD symptoms among children with varying levels of initial externalizing symptoms and among children of varying ages, ethnicities, and SES.

Another possible explanation for the nonsignificant relation between Time 1 anxiety and Time 2 ODD symptoms is the way in which symptoms were measured. For example, previous research has indicated that parents and teachers report higher levels of ODD than do youth (Loeber, Burke, Lahey, Winters, & Zera, 2000) and that parents may have difficulty identifying anxiety symptoms that are less easy to observe, such as worry (Comer & Kendall, 2004). Furthermore, relations between symptom categories depend on the informant used to identify symptoms (e.g., parent vs. teacher; Drabick et. al., 2008). Indeed, informants likely differ in their ratings of child behaviors because of some combination of the child's characteristics, context (i.e., where the ratings occur), and individual rater's perspectives (Kraemer et al., 2003). It would be useful, therefore, for future research to examine the prospective relation between anxiety and ODD symptoms using multiple informants to determine whether particular informants are more likely to identify this prospective relation. It may be that child self-reports of anxiety result in the endorsement of a wider range of anxiety symptoms, which are needed to document significant relations between anxiety and ODD symptoms.

Given that a significant relation was not found between Time 1 anxiety and Time 2 ODD symptoms, analyses examining the potential meditational role of hostile attribution biases were not conducted. Thus, whether hostile attribution biases in peer situations can account for

prospective relations between anxiety and ODD symptoms is a question that awaits future research. Given the lack of associations between Time 1 anxiety and Time 2 ODD symptoms, it may be better to conceptualize hostile attribution biases as a mediator of the relation between ODD and anxiety symptoms or a shared risk process in the co-occurrence of ODD+GAD and ODD+SAD. However, suggestions regarding the role of hostile attribution biases in the relation between anxiety and ODD symptoms remain tentative. Future research will be necessary to examine these alternatives suggestions, as well as the possibility that hostile attribution biases are specific to one symptom category (e.g., ODD) and not the other (e.g., anxiety).

Explanation 2

Findings demonstrating that ODD symptoms were prospectively associated with GAD symptoms are consistent with study hypotheses and extend previous literature demonstrating prospective relations between ODD and anxiety disorders (Burke et al., 2005). Furthermore, ODD and GAD symptoms were significantly associated at Time 2, which is consistent with previous research documenting a relation between ODD in preschool and subsequent comorbid ODD and anxiety disorders (Lavigne et al., 2001). It is important to note that this pathway from ODD to GAD symptoms is also consistent with the average age of onset for each symptom category, and thus this association might be expected from a developmental perspective. Specifically, ODD symptoms typically emerge during the preschool period (Lavigne et al., 1996), whereas the average age of onset for GAD symptoms is during the middle childhood period (Wagner, 2001). The later emergence of GAD symptoms is likely dependent on the development of cognitive processes, which increases the possibility for worry to emerge (Muris, Merckelbach, Meesters, & van den Brand, 2002).

Results were less clear in identifying the mechanisms that may link ODD and GAD symptoms. Multiple regression analyses suggested that peer rejection may account for the relation from ODD to GAD; however, further examination of this relation via bootstrap analyses, which tend to compute more accurate estimates of the indirect and direct effects when testing mediation, suggested that the indirect relation between ODD and peer rejection was not statistically significant. Therefore, peer rejection was not found to be a mediator of the relation from ODD to GAD. Before the construct of peer rejection can be considered an unlikely mechanism by which ODD leads to GAD, future research should examine whether the relation between ODD and peer rejection depends on how peer rejection is measured. For instance, the present study used parent report as an index of peer rejection. However, some research suggests that direct observation of peer rejection may be particularly useful for understanding the relation between peer rejection and externalizing behaviors (Arnold, Homrok, Ortiz, & Stowe, 1999). Other studies have noted the importance of the child's perception of peer relationships and proposed the use of narrative accounts of peer behaviors as an important measure of peer status (Xie, Li, Boucher, Hutchins, & Cairns, 2006). Therefore, parent report of peer rejection may have obscured the relations between peer rejection and child symptoms in the present study.

It also will be important for future research to examine other potential mediators, such as parental behaviors, that may account for the prospective relation between ODD and GAD symptoms. Previous research has indicated that mothers of children who are oppositional and/or anxious display more intrusive involvement with their children than mothers of non-clinical children (Hudson & Rapee, 2001) and display more overinvolvement with their children than other non-clinical children (Hudson, Doyle, & Gar, 2009). Based on these findings, it may be that oppositional children elicit and experience intrusive involvement from their mothers and that

this intrusive involvement may preclude children's exposure to certain anxiety-provoking situations (e.g., social interactions). Over time, frequent avoidance of a particular situation or combination of situations may lead to the development of significant anxiety and worry. It also may be that intrusive involvement hinders the development of emotion regulation, which results in oppositional children relying on less adaptive strategies for negotiating anxiety-provoking situations, and in turn may lead to significant anxiety (Suveg et al., 2008).

Contrary to hypotheses, Time 1 ODD symptoms were unrelated to Time 2 SAD symptoms, after controlling for Time 1 SAD symptoms. Coupled with the findings that (a) Time 1 ODD and SAD symptoms were significantly correlated, but (b) Time 2 ODD and SAD symptoms were not significantly related, these results suggest that ODD and SAD symptoms may be more likely to co-occur during preschool to early school age years, as opposed to middle childhood. This possibility is consistent with evidence that ODD symptoms typically emerge during the preschool period (Lavigne et al., 2001) and that the average age of onset for SAD is approximately 4 years of age (Doerfler, Toscano, & Connor, 2008). Thus, explanations for the co-occurrence of ODD+SAD may be better studied in a younger sample of children.

Another potential explanation is that parents may have difficulty determining whether their children's behaviors are indicative of separation anxiety vs. oppositionality and defiance and may only endorse symptoms of ODD or SAD. For example, if a child refuses to follow a parent's instruction to get out of a vehicle and enter a daycare center, it may be unclear whether the child's refusal stems from fear/anxiety about being separated from one's caregiver, defiance of the parental request, or both. Using multiple informants could address this issue, given that consideration of multiple informants would improve our ability to determine how contextual factors may affect ODD and SAD behaviors. The inclusion of teachers as informants may be

particularly helpful given previous research suggesting that teachers are better able to differentiate symptom outcomes and associations with impairment (Drabick, Gadow, & Loney, 2007). Using multiple informants also allows for the identification of cross-informant, mixed comorbidity (i.e., the child meets criteria for multiple disorders, but informants endorse symptoms that are consistent with different disorders; Drabick et al., 2008). Future research examining the co-occurrence of ODD+SAD in various developmental periods, particularly in the preschool age, and examining issues related to source specificity and rater biases will be necessary to tease apart alternative explanations.

Explanation 3

Examination of the shared risk factors explanation revealed that child-specific risk factors significantly predicted Time 2 symptom categories. Specifically, negative emotional reactivity, as indexed by child anger/frustration, predicted Time 2 ODD, GAD, and SAD symptoms. As expected, child fearfulness, a different index of negative emotional reactivity, predicted Time 2 SAD symptoms, suggesting that temperamental fearfulness may be a more specific risk factor for SAD. Furthermore, limited self-regulatory capacity, as indexed by child inhibitory control, predicted Time 2 ODD symptoms and tended to predict Time 2 GAD symptoms. Temperamental impulsivity, a second index of self-regulatory capacity, also tended to be associated with Time 2 ODD symptoms. Therefore, consistent with previous literature (Bates et al., 1998; Carrasco Ortiz & del Barrio Gandara, 2006; Caspi et al., 1995; Guerin et al., 1997; Keenan et al., 1998; Shaw et al., 1997), the present study's findings indicate that difficult child temperament may be a shared correlate of ODD, GAD, and SAD symptoms among impoverished, ethnic minority children. After controlling for Time 1 symptoms, analyses suggested some specificity with these temperamental variables, such that temperamental inhibitory control may confer risk for ODD

symptoms, temperamental anger/frustration may confer risk for GAD symptoms, and temperamental fearfulness may confer risk for SAD symptoms. Future research should examine whether specific combinations of temperamental variables place children at risk for the development of co-occurring ODD+GAD or ODD+SAD. For example, it may be that high levels of temperamental anger/frustration, in combination with high levels of poor inhibitory control, are associated with ODD+GAD. In contrast, high levels of temperamental anger/frustration in combination with high levels of fearfulness may confer risk for ODD+SAD.

Consistent with study hypotheses and previous research, autonomic variables were significantly associated with Time 2 ODD and GAD symptoms. However, contrary to prediction, longer (rather than shorter) PEP scores during the emotion task were correlated with Time 2 ODD and GAD symptoms. Therefore, findings were not consistent with previous research suggesting that children with anxiety and externalizing symptoms become hyperaroused under conditions that induce negative emotions (van Goozen et al., 2000). This discrepancy may stem from the particular emotion challenge used in the present study. Indeed, previous research identifying autonomic hyperarousal among children with anxiety and disruptive behavior disorders used a protocol that induced significant frustration, whereas the emotion challenge in this study was designed to induce fear and sadness. The results of the present study therefore cannot rule out the possibility that autonomic hyperarousal resulting from frustration is a shared risk process in anxiety and externalizing symptoms. Future research that considers multiple methodologies for assessing autonomic activity during emotion-inducing tasks would be useful for clarifying these discrepancies in the literature.

Also contrary to hypotheses, after controlling for child age and sex, PEP variables did not prospectively predict Time 2 symptoms. Examination of bivariate correlations suggests that PEP

at baseline and PEP during the emotional challenge were positively associated with age; in other words, older age was associated with decreased sympathetic activation at baseline and during the emotion challenge. In addition, independent *t*-tests suggested that boys tended to be rated as having higher levels of ODD and GAD symptoms. Taken together, these findings suggest that age and sex differences may be important to consider in the relations among PEP, ODD, and GAD, though disentangling the nature of these relations awaits future research.

In addition, contrary to hypotheses, RSA deficiencies were not related to Time 2 ODD symptoms. This finding may be explained by previous research indicating that parasympathetic deficiencies associated with externalizing symptoms come "online" in middle childhood (Beauchaine et al., 2007). Therefore, if RSA deficiencies were assessed in this sample at a later age, associations between RSA measures and ODD symptoms may have been significant. Ultimately, this is an empirical question that will need to be examined in a similar sample of children.

In terms of contextual risk factors, results did not support the hypothesis that neighborhood danger would be correlated with Time 2 symptom categories. The lack of a relation between neighborhood danger and Time 2 symptoms may result from limited variability in the types of neighborhoods in which participating children reside. That is, the sample is recruited from lower income neighborhoods that are likely similar in terms of violence levels. The lack of findings for neighborhood danger may also be the result of significant sex differences. Independent *t*-tests suggested that, compared to boys, girls perceived higher levels of neighborhood danger. Therefore, future research may benefit from examining the interaction of sex and neighborhood danger in the prediction of ODD, GAD, and SAD symptoms. With regard to stressful life events, the number of stressful life events experienced in the past year

prospectively predicted Time 2 GAD symptoms, but not Time 2 ODD or SAD symptoms, suggesting some specificity to GAD symptoms among impoverished, ethnic minority youth, rather than a shared risk process for multiple symptom categories. Consideration of alternative contextual risk factors (e.g., specific types of stressors rather than number of stressors and/or emotion socialization practices in families) may help to identify other overlapping and/or unique risk factors in the development of ODD, GAD, and SAD symptoms.

Although the limited findings for contextual risk factors were unexpected, they are not necessarily inconsistent with previous research. For instance, there is a literature to suggest that contextual factors place some, but not all, children at risk for the development of psychopathology (Ellis, Essex, & Boyce, 2005). To examine this possibility of differential susceptibility, various researchers have recommended examining both endogenous biological factors and contextual factors, as well as child ×context interactions (Belsky, 2005; Boyce & Ellis, 2005; Steinberg & Avenevoli, 2000).

Child × Context Interactions

Exploratory multiple regression analyses examining child × context interactions provided a more in-depth analysis of the relation between child-specific and contextual risk factors in the prediction of symptom categories. Significant findings were specific to parasympathetic functioning and revealed that RSA reactivity interacted with stressful life events in the prediction of Time 2 ODD symptoms. However, post hoc probing of this interaction suggested that impact of RSA reactivity levels on Time 2 ODD symptoms did not differ among children experiencing low vs. high levels of stressful life events. This finding is likely the result of being underpowered to detect results and suggests that future research will need to replicate this finding in larger samples. Graphical representation of the interaction indicates that, among children with low RSA

reactivity, those experiencing higher levels of stressful life events exhibited higher levels of Time 2 ODD symptoms, as compared to those experiencing low levels of stress. These tentative results extend the work of Beauchaine (2001) and suggest that RSA deficiencies coupled with stressful life events may confer greater risk for the development of ODD symptoms, above and beyond RSA deficiencies alone.

Results also suggested that a number of RSA × context interactions tended to be significant in the prediction of Time 2 symptoms; however, post hoc probing indicated that the slopes were not significantly different from zero. These findings are counter to previous research supporting the presence of child ×context interactions in the prediction of externalizing and anxiety symptoms (Bates et al., 1998; Bubier et al., in press; Colder et al., 1997, 2006). As noted, these nonsignificant findings may be the result of limited power. It also may be that child × context interactions are better predictors of broader classes of behavior (e.g., externalizing symptoms as opposed to ODD only). Future research should examine these possibilities.

Sex Differences

Results investigating sex differences in symptom severity, levels of risk factor, and associations between ODD, GAD, and SAD partially supported study hypotheses. As predicted, perception of neighborhood danger was greater among girls as compared to boys (Zalot et al., 2007), and boys were rated as having less inhibitory control than girls (Else-Quest et al., 2006). Previous research has proposed that higher levels of particular risk factors among boys or girls may differentially contribute to symptom development (Rutter et al., 2003). For example, because levels of inhibitory control are lower among boys than girls, boys may be less able to inhibit impulses to be oppositional in adult-child interactions compared to girls. This explanation

is consistent with previous literature (Simonoff et al., 1997) and the present study's finding that boys tended to be rated as having higher levels of Time 2 ODD symptoms, as compared to girls.

Contrary to prediction, however, boys and girls did not differ in terms of their levels of GAD or SAD symptoms. Despite epidemiological evidence suggesting that girls exhibit higher levels of anxiety as compared to boys (Simonoff et al., 1997), other evidence suggests that sex differences in nonclinical anxiety symptoms are not evident among young children (Mireault et al., 2008), but rather emerge with increasing age (Spence, Rapee, McDonald, & Ingram, 2001). Furthermore, the lack of sex differences in GAD and SAD symptoms is consistent with findings from clinical samples of children aged 9 to 13 (Treadwell, Flannery-Shroeder, & Kendall, 1995). Therefore, these findings suggest that sex differences in anxiety symptoms may only be evident starting in early adolescence and that future research should identify specific developmental processes that may mark the onset of sex differences in anxiety symptoms.

Also contrary to hypotheses, the magnitude of the association between ODD and SAD was not greater among girls, as compared to boys, nor was the magnitude of the association between ODD and GAD greater among boys, compared to girls. Given inconsistencies in the literature with regard to sex differences and co-occurring ODD and GAD (Garland & Garland, 2001; Marmorstein, 2007), results suggest that ODD+GAD symptoms may be equally likely to co-occur among boys and girls residing in the inner city, perhaps because children in this sample are exposed to similar contextual influences that may affect boys and girls similarly. At the same time, the nonsignificant finding may be due to a reduced ability to detect small to medium differences in relations, stemming from the relatively sample size. Moreover, because children in this sample are prepubescent, sex differences among symptom associations may not be apparent in this particular developmental period. It will be important for future studies to examine sex

differences in associations among ODD, GAD, and SAD across various developmental periods and concurrently examine reasons for why particular sex differences may occur.

Clinical Implications

The present study's findings have important implications for assessment, prevention, and intervention of ODD, GAD, and SAD symptoms among children. In terms of assessment, these findings suggest that clinicians would do well to assess for ODD, GAD, and SAD symptoms concurrently given their significant covariation. A comprehensive conceptualization of the processes underlying and maintaining a child's behaviors also must consider the interplay among different sets of problems. For example, a child's oppositional behavior may stem from ODD symptoms and/or avoidance associated with separation anxiety. Thus, the inclusion of a functional analysis when assessing a child's behaviors would be useful to determine the antecedents and consequences of particular behaviors, in addition to possible maintaining or exacerbating roles of the co-occurring symptoms. Evaluating information from multiple informants who can speak to the child's behavior in various settings is also important, as alternative perspectives may help to differentiate among ODD, GAD, and SAD symptoms and diagnoses.

From a prevention perspective, early identification of ODD symptoms may enable clinicians to target children who are at significant risk for developing GAD. The present study suggests that children with a difficult temperament, particularly those with high levels of temperamental anger/frustration and low levels of inhibitory control, may be at significant risk for developing both ODD and GAD. Another risk factor that will be important to consider with regard to the development of GAD symptoms is the number of stressors that the child and his/her family have experienced in the past year. Early identification of these risk factors and symptom

correlates may facilitate recognition of children who could benefit from early intervention, potentially preclude or mitigate these syndromes and co-occurring conditions, minimize the significant impairment that is often associated with comorbid disorders, and aid clinicians in selecting treatment strategies and targets for intervention (Kendall et al., 2001).

With regard to intervention, the present study's findings suggest that research would do well to investigate treatment outcomes for youth with co-occurring ODD+GAD and ODD+SAD. Several previous studies have examined treatments for youth with comorbid anxiety and disruptive behavior disorders (Chase & Eyberg, 2008; Flannery-Schroeder et al., 2004; Kendall et al., 2001; Levy, Hunt, & Heriot, 2007; Rapee, 2003). Flannery-Schroeder et al. (2004), Kendall et al. (2001), and Rapee (2003) all documented significant improvements as a result of participation in anxiety-based treatments among anxious children with and without comorbid disruptive behavior disorders. In addition, Kendall et al. (2001) noted that the rate of ODD post-treatment was 1.8% (down from 9.2%). Similarly, Chase and Eyberg (2008) reported that among children with comorbid ODD and SAD, parent-child interaction therapy led to significant declines in both ODD and SAD symptoms following intervention. Levy et al. (2007) explicitly examined a treatment designed to address both anxiety and externalizing behaviors. In this study, strategies targeting anger and aggression were added to an anxiety treatment program (Rapee, Wignall, Hudson, & Schniering, 2000). Comparisons involved the original anxiety program with the combined anxiety +anger/aggression program. Strategies for anger and aggression included self-management, self-reflection, and self-monitoring skills. Both programs led to significant improvements (symptom decrements) on measures of child internalizing symptoms and parent-reported externalizing problems, indicating that children's internalizing and externalizing problems decreased even when they received a treatment program targeting anxiety only (Levy

et al., 2007). Taken together, these findings suggest that treatments for anxiety disorders can be successful in reducing co-occurring externalizing symptoms among children and, similarly, treatments for externalizing or disruptive behavior disorders can reduce the frequency of co-occurring anxiety symptoms. It will be beneficial for future research to examine the components of treatment that lead to change in ODD, GAD, and SAD symptoms among children to identify factors associated with successful treatment of ODD+GAD and ODD+SAD.

Strengths, Limitations, and Future Directions

The present study has numerous strengths. First, I addressed the dearth of literature examining ODD+GAD and ODD+SAD symptoms concurrently, and tested multiple explanations for the co-occurrence of ODD+GAD and ODD+SAD, which has not been accomplished to date. Second, I examined sex differences in the levels of risk factors, severity of symptoms, and symptom associations in an effort to better understand differences between boys and girls in the development of ODD+GAD and ODD+SAD. Third, I examined processes among an impoverished, inner city sample of children, who are likely at an increased risk of developing co-occurring anxiety and externalizing behaviors. Finally, I used objective indicators of autonomic functioning, as well as multiple methods and informants for measuring risk factors.

Despite these strengths, this investigation has several potential limitations. First, ODD, GAD, and SAD symptoms were defined using a rating scale, not a diagnostic interview; thus, the *DSM-IV* symptoms examined were not be equivalent to the diagnostic categories of ODD, GAD, and SAD. Nevertheless, the *DSM*-referenced rating scale used in this study has been included in numerous studies of ODD (e.g., Drabick, Gadow, Carlson, & Bromet, 2004; Drabick, et al., 2007) and anxiety (Drabick et al., 2008; Weisbrot, Gadow, DeVincent, & Pomeroy, 2005). Moreover, the symptoms associated with these disorders often cause significant impairment,

even at subthreshold levels (Angold et al., 1999b). Therefore, this study provides a first step to understanding sex differences and explanations of co-occurring anxiety and ODD symptoms.

Although the merits of examining these processes in an inner city sample are notable, the sampling method (i.e., self-selection) introduces the possibility of sampling biases. For example, the method of self selection may have biased this sample to higher functioning families (e.g., families that were able to organize transportation and child care, experienced lower levels of stress or parental psychological difficulties, etc.). Therefore, it might be expected that the children in this sample exhibit lower levels of psychological symptoms than their counterparts living in similar contextual disadvantage. It is important to note, however, that the children in the present investigation exhibited higher mean levels of ODD, GAD, and SAD symptoms compared to normative children. As such, it is also feasible that families who self-selected into the present study may have done so because their children exhibited significant symptoms. Given these competing accounts, it will be necessary for future work to examine the generalizability of this study's findings. If results do generalize to varying SES and ethnic groups, findings of the present study generally would be characteristic of children with ODD, GAD, and SAD symptoms. If the findings do not generalize, results may suggest that contextual factors, such as poverty, may exert an important influence on the development of co-occurring ODD+GAD and ODD+SAD.

Although a power analysis suggested adequate power for examining explanations for the co-occurrence of ODD+GAD and ODD+SAD, the relatively small sample size suggests that I may have been underpowered to detect effects, particularly for the examination of shared risk factors and child × context interactions. In addition, the step-wise procedure used to test the shared risk factors explanation was a limitation. Because of the sample size and resultant power-

related issues, I used *a priori* steps to determine which potential shared risk factors should be included in subsequent analyses. Specifically, the potential shared risk factor variables had to be significantly correlated with the dependent variable before inclusion in the multiple regression analyses. Future research will be necessary to determine the usefulness of this procedure. Unfortunately, the sample size limited the ability to use more complex statistical analyses, such as structural equation modeling, which would have been optimal for testing multiple variables concurrently, consistent with the shared risk factors explanation. However, findings provide preliminary evidence for pathways that can be further validated in larger, longitudinal studies.

In sum, findings suggest that the co-occurrence of ODD+GAD is best explained by (1) the prospective prediction from ODD symptoms to GAD symptoms and (2) the presence of shared risk factors. In addition, there was support for shared risk processes, particularly indices of difficult temperament, in the co-occurrence of ODD+SAD. Future research would do well to examine explanations for the co-occurrence ODD+GAD and ODD+SAD symptoms among children of various ages, ethnicities, and SES to determine the generalizability of the present study's findings. Such knowledge could inform both etiological and intervention models for ODD+GAD and ODD+SAD, and thus improve our efforts to intervene and mitigate the negative correlates and sequelae associated with these co-occurring conditions.

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