Linking Temperament and Parenting Dimensions to the Co-occurrence of Internalizing and Externalizing Disorders in Childhood and Adolescence

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ABSTRACT

Linking temperament and parenting dimensions to the co-occurrence of internalizing and externalizing disorders in childhood and adolescence

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Child psychological disorders co-occur at a much greater rate than would be expected by chance. Comorbidity is known to occur both within and across diagnostic classes, such that even disorders lying at opposite ends of the diagnostic spectrum (i.e., internalizing and externalizing disorders) commonly co-occur. Although patterns of concurrent comorbidity have been widely studied, relatively little is know about how internalizing and externalizing disorders co-occur and influence one another over time.

The overall aim of this research was to investigate patterns of sequential comorbidity in a community-based sample of children, followed longitudinally from age 3 to age 16. Data was drawn from the Concordia Longitudinal Risk Project, a prospective, intergenerational study of children from disadvantaged neighborhoods (Study 1: $N = 154$; Study 2: $N = 143$). Three pathways to comorbidity were considered: 1) that symptoms of internalizing and externalizing disorders influence one another over time; 2) that common temperament risk factors are implicated in the co-development of internalizing and externalizing problems; and 3) that disorders co-occur over time because of the high degree of symptom overlap across disorders. Study 1 examined longitudinal patterns of internalizing and externalizing problems, finding an inhibitory effect of internalizing problems on the development of future externalizing problems. Results demonstrated that the presence of common risk factors (i.e., temperamental negative emotionality) best explained the co-occurrence between internalizing and externalizing problems and additionally, unique temperamental risk factors (i.e., sociability, activity) predicted their differentiation. Study 2 examined patterns of sequential comorbidity within the diagnostic class of internalizing disorders (i.e., anxiety and depression). Results from this study indicated a sequential progression from anxiety to depression, but that this association was primarily attributed to symptom overlap among questionnaires used to assess anxiety and depressive

symptoms. Instead, the co-occurrence between anxiety and depression (at both the symptom and diagnostic level) was best explained by a common temperamental style characterized by high negative emotionality and low positive emotionality. Additionally, both studies revealed significant interactions between child characteristics (gender and temperament) and parenting in the prediction of children's internalizing and externalizing problems.

The present studies build on previous work linking temperament and parenting practices to the development of specific childhood psychopathologies. Findings of common temperamental styles predicting the co-development of internalizing and externalizing problems across time, however, are novel. More broadly, these findings suggest that temperamental styles explain a large portion of the co-variation in symptomatology, both within and across diagnostic classes. Additionally, other temperamental styles and parenting practices, both separately and in combination, predict the differentiation of problems. These findings highlight the need for future studies to consider both concurrent and longitudinal patterns of comorbidity, as well as multiple potential pathways to comorbidity. This line of research has implications for etiology, intervention, prevention, and classification of child psychopathology.

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CONTRIBUTIONS OF THE AUTHORS

Danielle Kingdon developed the research questions, designed, performed, and interpreted the statistical analyses, and wrote and edited all chapters included in the current thesis. Drs. Lisa Serbin and Dale Stack provided commentary on these manuscripts and Dr. Serbin edited the final draft of the dissertation. Drs. Serbin and Stack direct the Concordia Longitudinal Risk Project (Concordia Project) and are integrally involved in all aspects of the Project (e.g., collaboration in the conceptualization, design, analysis, and development of the protocols for the intergenerational studies). The Concordia Project originated in 1976 under the direction of Jane Ledingham and Alex Schwartzman. Drs. Schwartzman and Ledingham were responsible for the original design and data collection. Finally, Drs. Serbin and Stack designed the waves of data collection from which the outcomes examined in this thesis were drawn.

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CHAPTER 1: GENERAL INTRODUCTION

In child psychopathology, comorbidity is the rule rather than the exception. In the 1990's, the National Comorbidity Survey, the first nationally representative epidemiological mental health survey in the United States, established that among individuals with at least one lifetime mental health disorder, 79% had two or more co-occurring conditions (Kessler et al., 1994). Moreover, the youngest age group in this survey, 15 to 24-year-olds, was at greatest risk for multiple disorders. Comorbidity, defined as the occurrence of two or more forms of psychopathology in the same person, has been the subject of a growing body of research in the child psychopathology literature (Angold, Costello, & Erkanli, 1999). Over the past 30 years, researchers have made increasing efforts to trace the etiology of comorbid conditions back to childhood and adolescence.[1]

Childhood psychopathology has been popularly classified as falling into two general forms of symptomatology: internalizing and externalizing problems (Krueger, Caspi, Moffitt, & Silva, 1998, Krueger, 1999). The term internalizing problems refers to a wide range of problems characterized by disordered mood or emotion, which appear in the form of anxiety, fearfulness, depression, and social withdrawal (Achenbach, 1991; 1992). In contrast, externalizing problems are characterized by dysregulation in behaviour, including hyperactivity, inattention, aggression, defiance, and destructive behavior (Achenbach, 1991; 1992). Factor analytic studies have generally found support for two factors, internalizing and externalizing, underlying psychopathology in both adults and youth (Krueger, 1999; Krueger, McGue, & Iacono, 2001). Despite the distinctiveness of these classes of psychopathology, it has also been noted that psychological syndromes tend to be positively correlated with one another, such that individuals who have high symptom levels in one domain also tend to present with elevated symptoms in other domains (Achenbach, 1991; Achenbach, Conners, Quay, Verhulst, & Howell, 1989).

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[1] *As an index of interest in comorbidity research, I conducted a PsycINFO search on the stem ``comorb'' appearing in the title or abstract fields and the stems ``child '' or ``adolescent'' appearing in any field. A search of the PsycINFO data base reveals that prior to 1986, no studies were conducted on the topic of comorbidity in youth, but in the following years, the number of citations on this topic increased substantially: 1986 - 1990: *235*; 1991 – 1995: *1,536*; 1996 – 2000: *3,387*; 2001 - 2005: *6,010*; 2006 – 2010: *10,238*; 2011 – 2015: *12,872*.

Children with co-occurring symptomatology are found in both clinic and community-based samples (Lilienfeld, 2003) at rates far greater than expected by chance (Caron & Rutter, 1991). Children with co-occurring symptoms have an earlier age of onset (Newman, Moffitt, Caspi, & Silva, 1998), more severe and chronic course of illness (Kovacs & Devlin, 1998; Newman et al., 1998), higher rates of mental health service usage (Costello et al., 1996), and worse developmental outcomes (Karlsson et al., 2006; Keiley, Lofthouse, Bates, Dodge, & Petit, 2003; Lewinsohn, Rohde, & Seeley, 1995) than children with only a single disorder. In addition, clinical interventions that are designed for particular disorders are often less efficacious when children have comorbid problems (Chase & Eyberg, 2008). Increasingly, researchers have acknowledged that an understanding of comorbidity is necessary for explaining the etiology and course of psychopathology in youth (Angold & Costello, 1993; Caron & Rutter, 1991; Hinshaw, 2002; Lilienfeld, 2003). Studies of comorbidity also hold important implications for intervention, prevention, and nosology. Despite the importance of research in this area, studies have largely failed to consider the developmental pathways to co-occurring disorders. Accordingly, a thorough understanding of co-occurring symptom patterns, as well as the factors underlying these patterns, is the next step for research attempting to elucidate the etiology of childhood psychopathology (see Rutter, 1997; Angold et al., 1999 for review).

The purpose of the present research was to examine how developmental patterns of internalizing and externalizing problems emerge among a community sample of youth, followed longitudinally from preschool to late adolescence. Adopting a developmental psychopathology approach, the studies in this dissertation examined patterns of continuity and discontinuity in symptoms over time. In addition, each study considered whether symptom trajectories were predicted by risk factors identified in early childhood. A central goal of each study was to examine whether some risk factors were relatively specific to either internalizing or externalizing conditions, whereas other risk factors were common to multiple, co-occurring problems. In the first study, broad symptoms types (i.e., internalizing and externalizing problems) were considered, while the second study examined how different disorders within a symptom class tend to co-vary (i.e., anxiety and depression). The specific and general discussions emphasize the importance of examining longitudinal patterns of co-occurring internalizing and externalizing problems across childhood and adolescence and testing multiple theoretical pathways to co-occurrence. These papers aim to integrate findings regarding the etiology of different classes of

psychopathology, which historically have been studied separately and have lacked consideration of how one problem may influence the expression of another. It was anticipated that the findings from this dissertation would guide future studies by clarifying the relative importance of various methodological and substantive explanations of comorbidity. These results, in turn, were expected to shed light on the etiology, course, nosology, and treatment of child psychopathology.

First, the theoretical approach guiding the present study will be discussed. Next, definitions of comorbidity will be presented, followed by a description of the epidemiology of comorbidity across a variety of symptom classes. Finally, several proposed pathways to comorbidity will be presented, including a review of previous research on common and specific predictors of internalizing and externalizing disorders, along with an outline of the general questions addressed by the two studies.

Theoretical Approach

According to the developmental psychopathology framework, psychopathology results from complex interactions among multiple factors representing characteristics of individuals and their context over time (Cicchetti & Toth, 2009). Integrating research and theory from multiple disciplines, including developmental psychology and clinical psychology, the developmental psychopathology approach is concerned with individual differences in the origins, course, and outcomes of normative and psychopathological developmental processes (Cicchetti & Toth, 2009; Rutter & Sroufe, 2000). A guiding tenant of this theoretical approach is to go beyond a description of the characteristics or events that precede the onset of psychopathology, to an understanding of the mechanisms by which risk factors lead to their development. By considering multiple risk factors and causal pathways involving a transactional process between the child and his/her environment, the developmental psychopathology framework can capture the complex processes involved in the etiology of comorbidity. In particular, this model accounts for the development and maintenance of symptomatology, the interconnectedness of both common and unique risk factors, and the direct and indirect relations between disorders over time.

Terminology

Comorbidity typically refers to the presence of two or more distinct, co-occurring disorders in one person simultaneously (*concurrent comorbidity*) or over time (*sequential comorbidity*; Angold, Costello, & Erkanli, 1999). When considering patterns of sequential

comorbidity, disorders may co-occur over a relatively short time period, or may have occurred widely separated in time (e.g., over the course of a lifetime). Acknowledgment of sequential comorbidity is important because prevalence studies covering relatively brief periods of time are likely to underestimate the true extent to which an individual experiences two or more disorders. Moreover, as the prevalence rates of various disorders are known to change across age, patterns of concurrent comorbidity are likely to vary by age group. By examining patterns of sequential comorbidity, researchers can more fully describe and understand how comorbidity develops over time. For example, if one disorder regularly precedes another, the first disorder may be a risk factor for the second disorder. Conversely, if two disorders are highly comorbid across all stages of development, they may represent different manifestations of the same disorder. Definitive inferences about patterns of sequential comorbidity cannot yet be made because most studies in this area have relied on the retrospective ascertainment of past psychological symptoms (Lilienfeld, 2003; Moffitt et al., 2007). Despite efforts by researchers to promote accurate respondent recall, retrospective studies are widely known to result in the underreporting of past symptoms/disorders and inaccurate reporting regarding the time of onset (Simon & VonKorff, 1994). Thus, researchers have highlighted the need for prospective, longitudinal designs so that patterns of comorbidity occurring across various developmental periods can be accurately characterized (Lilienfeld, 2003; Krueger & Markon, 2006).

Studies of comorbidity may consider two types of comorbidity: comorbidity occurring between disorders within the same diagnostic class, such as the co-occurrence of major depression and dysthymia (*homotypic comorbidity*); and comorbidity occurring between different diagnostic classes, such depression and conduct disorder (*heterotypic comorbidity*; Angold et al., 1999). Both patterns of homotypic and heterotypic comorbidity are widely cited in the literature. For instance, depressed adolescents have been found to be more likely than non-depressed adolescents to experience a major depressive episode in adulthood, demonstrating the homotypic continuity of mood disorders (Birmaher et al., 1996). Other research has found evidence of heterotypic comorbidity, such that externalizing problems in children predict the development of depression several years later (Wolff & Ollendick, 2006). When discussing longitudinal outcomes of early child symptomatology, homotypic and heterotypic comorbidity provide a theoretical framework for discussing the continuity (or discontinuity) in symptoms over time.

While the term comorbidity has generally been reserved for the description of two or more categorically defined and distinct co-existing diagnoses, the term *co-variation* has been used in the context of a dimensional approach to psychopathology and refers to the statistical degree to which one syndrome correlates with another syndrome (Capaldi, 1992; Keiley et al., 2003). The term *co-occurrence* has been used more generally to describe an individual with two or more psychopathological conditions, including diagnoses, symptoms, or syndromes (Keiley et al., 2003). In the developmental psychopathology literature, researchers have highlighted the importance of assessing symptom patterns, rather than diagnoses alone, given consistent findings that the clinical significance of symptomatology in youth does not depend on crossing the diagnostic threshold. For example, Gotlib, Lewinsohn, and colleagues found that youth with elevated depressive symptoms closely resembled youth with a diagnosis of major depressive disorder on several measures of psychosocial functioning, and also presented with the same increased risk for future mood disorders (Gotlib, Lewinsohn, & Seeley, 1995; Lewinsohn et al., 2000). In addition, the early manifestations of emotional and behavioral problems in children are often accompanied by negative consequences (e.g., academic, social, and familial impairments) and increases in other symptom domains (e.g., Capaldi, 1991; 1992). Given the clinical significance of elevated symptomatology in childhood and its potential to affect the course of adjustment across several domains of developmental functioning, the incorporation of dimensional measures of psychopathology is necessary to fully capture how comorbidity emerges in youth.

Epidemiology

Epidemiological studies have shown that a substantial proportion of youth suffer from a psychological disorder. At any given time, approximately 1 in 5 youth have a psychological disorder, with 1 in 10 having a disorder which results in significant interference in the child's life and merits clinical intervention (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Ford, Goodman, Meltzer, 2003). Internalizing disorders (including anxiety disorders and depression) as well as externalizing disorders (i.e., ADHD and disruptive behavior disorders, including oppositional-defiant disorder and conduct disorder) are among the most prevalent childhood disorders. Three-month prevalence rates of about 5% are found for both types of problems (Costello et al., 2003; Ford et al., 2003).

Research indicates that rates of concurrent comorbidity in youth are high both between and within diagnostic classes. Comorbidity is particularly evident between anxiety and depression, between ADHD and disruptive behaviour disorders, and between depression and some disruptive behaviour disorders. To illustrate, in reviewing patterns of comorbidity in a meta-analysis of 21 population-based studies, Angold and colleagues (1999) found the highest odds ratios between ADHD and conduct disorder (odds ratio = 10.7), between depression and anxiety (odds ratio = 8.2) and between conduct disorder and depression (odds ratio 6.6). Epidemiological research has produced a similar pattern of results. For example, more than 65% of children with depression also meet criteria for another disorder (most commonly an anxiety disorder or a disruptive behavior disorder), while at least 25% of children with an anxiety disorder also meet criteria for comorbid depression or a disruptive behavior disorder (Angold et al., 1999; Costello et al., 2003; Ford et al., 2003).

Moreover, dimensional models of psychopathology, such as the Achenbach System of Empirically Based Assessment (ASEBA; Achenbach, 1991), have established that even among factor-analytically derived syndromes of internalizing and externalizing problems, high rates of co-occurrence exist (Gilliom & Shaw, 2004; McConaughy & Achenbach, 1994; Oland & Shaw, 2005; Overbeek et al., 2006). Although patterns of concurrent comorbidity have been estimated, at present the extant literature lends itself to speculation regarding the sequential patterns and causes of comorbid psychopathologies. Specifically, few prospective, longitudinal studies have been conducted to trace patterns of sequential comorbidity that develop across childhood and adolescence and to investigate the mechanisms underlying co-occurrence.

Pathways to Comorbidity

In general, the literature suggests three possible explanations for comorbidity, which are either methodological or substantive in nature. First, it may be that comorbidity is a methodological artifact, emerging from referral biases, use of multiple informants, or problems with the measurement and classification of psychopathology. Alternatively, the presence of one disorder may place an individual at increased risk for developing a second disorder. Finally, two disorders may co-occur because they share an underlying risk factor (Caron & Rutter, 1991; Krueger & Markon, 2006; Wolff & Ollendick, 2006).

Researchers have considered the possibility that comorbidity may be a methodological artifact, rather than a real psychological phenomenon. Previous studies of comorbidity were

based heavily on clinic samples, which provide inaccurate estimates of comorbidity in the general population due to referral biases (Caron & Rutter, 1991; Costello et al., 2003; Lilienfeld, 2003). In particular, individuals presenting to treatment settings have more severe symptomatology, experience greater functional impairment, and come from families that feel more burdened by their children's symptoms, compared to individuals with psychological disorders who do not present for treatment (Angold, Messer, et al., 1998; Costello et al., 1996). However, recent community-based and epidemiological studies confirm that comorbidity rates in general population samples are also much higher than would be expected by chance, indicating that comorbidity is a real psychological phenomenon in need of future study (Wolff & Ollendick, 2006).

Other methodological issues, including flaws in current nosologies and overlapping diagnostic criteria, may over-estimate rates of comorbidity. Researchers and clinicians have pointed out that certain non-specific symptoms are shared by different diagnoses, even those representing separate classes of psychopathology. For example, with regards to childhood depression and conduct disorder, irritability is a core feature represented in the diagnostic criteria for both disorders (APA, 2013). In an effort to rule out such methodological explanations for comorbidity, Biederman and colleagues (1995), controlled for overlapping symptoms of depression and conduct disorder in a sample of clinically referred children, finding that the elimination of such overlapping symptoms did not reduce rates of diagnostic comorbidity. Yet, other diagnoses, especially those within the same diagnostic class (e.g., anxiety and depression), share even greater symptom overlap. To illustrate, generalized anxiety disorder (GAD) and depression in childhood are both characterized by irritability, worry, fatigue, somatic complaints, difficulty concentrating, and sleep disturbance (APA, 2013). To date, there is little research addressing how overlapping symptoms among depression and anxiety may contribute to the high rates of comorbidity that are cited in the literature (e.g., Axelson & Birmaher, 2001).

A second possible explanation for comorbidity is that the experience of one disorder causes or puts an individual at risk for developing another disorder. These effects could be directional or reciprocal, such that different conditions may cause another, or such that different conditions influence one another in a transactional process. An examination of the age onset for different classes of psychopathology reveals that certain problems (e.g., separation anxiety, specific phobias, generalized symptoms of anxiety, ADHD, ODD) show peak periods of onset in

early-to-middle childhood, while other problems (e.g., depression, conduct disorder) show an average age of onset in the early adolescent years (Merikangas, Nakamura, & Kessler, 2009). From a dimensional perspective, internalizing symptoms are known to emerge in early childhood and to show stable or increasing symptom trajectories (on average), while externalizing symptoms are known to peak in the preschool years, and show decreasing trajectories across development (Fanti & Henrich, 2010; Keiley, Bates, Dodge & Pettit, 2000; Leve, Kim, & Pears, 2005; Miner & Clarke-Stewart, 2008). These data have led some researchers to hypothesize causal relations between different classes of psychopathology over time. For example, many clinical studies have found that disruptive behaviour problems precede rather than follow the development of depression, suggesting that externalizing disorders are causally implicated in the development of depression (Burke, Hipwell & Loeber, 2010; Calpaldi, 1991; 1992; Wolff & Ollendick, 2006). There is also research indicating that in most cases of comorbidity, anxiety precedes the development of depression, rather than the converse (Axelson & Birmaher, 2001). This data has lead researchers to posit that the experience of anxiety increases an individual's risk for developing depression.

The studies presented in the current dissertation were designed to examine, in depth, the temporal relations between internalizing problems and externalizing problems more generally, and anxiety and depression more specifically. In order for such causal assumptions to be supported, longitudinal data must be used, showing that one disorder temporally precedes the development of the other and is associated with an increased risk of developing a second condition, controlling for baseline levels of each problem. The studies included in the present dissertation are novel in this regard.

A final theoretical explanation for comorbidity involves the presence of common risk factors underlying the development of two or more disorders. To be defined as a common risk factor, the risk factor must be shown to significantly relate to both types of psychopathology under investigation. To be defined as a unique risk factor, the variable must discriminate between classes of psychopathology, showing a significant correlation with one of the disorders, but not the other. As noted by Caron and Rutter (1991), common risk factors may lead to the parallel development of two or more disorders, while unique risk factors serve to differentiate between two disorders. Comorbidity is likely to involve a dynamic interplay between a number of common genetic, biological, physiological, psychological, environmental, and social factors,

including specific child characteristics, family factors, and aspects of the environmental context in which development takes place. While an abundance of possibilities exist, the present dissertation focuses on how dimensions of temperament may represent common risk factors for the development of co-occurring internalizing and externalizing problems. Simultaneously, this work considers how other dimensions of temperament may distinguish among different types of psychopathology.

Associations with temperament have been frequently highlighted as a promising avenue for understanding the roots of developmental psychopathology more generally, and comorbidity in particular (Clark, 2005). Temperament is generally defined as the biologically-based emotional and behavioral consistencies that appear early in life and predict (often in conjunction with contextual factors) outcomes in numerous domains such as psychopathology and personality (Rothbart, 2007). Temperament is widely believed to play an important role in the etiology and maintenance of internalizing and externalizing disorders in children (Lonigan & Phillips, 2001; Nigg, 2006). Several definitions and models of classifying temperament have been proposed (e.g., the Behavioral Style Approach of Thomas and Chess, 1977; the Criterial Approach of Buss and Plomin, 1984; the Psychobiological Approach of Rothbart, 1981), but these approaches all include a number of common elements. Temperament is considered to have a strong genetic or neurobiological basis, be manifested early in life (from infancy onward), and show relative consistency across situations and time (Goldsmith et al., 1987; Rothbart & Bates, 2006). There is also a general consensus that the expression of temperament and its effects on developmental outcomes depend on environmental or contextual factors, such as the quality of parenting. Few studies, however, have focused on the relation between temperament (and its interaction with characteristics of the environmental context) and co-occurring problems in children and adolescents.

In the past decade, there has been increasing consensus as to what the most important dimensions of temperament are. Most theoretical models of temperament focus on individual differences in emotional processing (e.g., negative emotionality), approach/withdrawal (e.g., activity and sociability), and regulatory processes (e.g., effortful control), although there are differences in the specific aspects of these processes that are the focus of each model (Rothbart & Jones, 1998; Shiner & Caspi, 2003). Emotion processing differences are usually identified by the term negative emotionality. Negative emotionality in childhood is consistently associated

with the adult personality trait of neuroticism and includes dimensions such as emotional reactivity, sadness, difficultness, and distress (Muris & Ollendick, 2005). Individual differences in approach/withdrawal are characterized by the tendency to be outgoing, expressive, and energetic. Linked to the personality trait of extraversion, dimensions of activity, approach, sociability, and shyness (negatively loaded) are used to describe children who experience the world with vigor, activity, and positive emotion (Muris, Meesters, & Blijlevens, 2007). Although not included as a specific dimension of temperament in the temperament survey employed in the present studies (EAS Temperament Survey; Buss & Plomin, 1984), some theories also consider differences in regulation processes, or conscientiousness, including attention focusing and purposeful shifting, perceptual sensitivity, persistence, and inhibitory control. Table 1 provides on overview of the three most influential theoretical models of temperament and their common dimensions (Thomas & Chess, 1977; Buss & Plomin, 1984; Rothbart, 1981; Shiner & Caspi, 2003).

As greater consensus on the definition and important facets of temperament has emerged, researchers have begun to focus on how temperament is related to a child's risk for psychopathology (Lahey & Waldman, 2003). There is emerging evidence that negative emotionality represents a risk factor for the development of both internalizing and externalizing problems across the lifespan (Calkins & Fox, 2002; Clark, Watson, & Mineka, 1994; Kotov, Gamez, Schmidt, & Watson, 2010; Lahey, 2009; Lengua, West, & Sandler, 1998; Lonigan & Phillips, 2001; Muris & Ollendick, 2005). Other dimensions of temperament have been related to psychopathology in conceptually coherent ways and may distinguish between different classes and subclasses of psychopathology. For example, greater activity and lower regulatory abilities have been found to be associated with higher prospective levels of externalizing problems, while separate investigations have found that low approach behaviors (i.e., low activity and low sociability) to be associated with higher levels of internalizing problems, especially depression (Mesman & Koot, 2000; Rubin, Coplan, & Bowker, 2009).

Investigating the relationship between temperament traits and psychopathology in children and adolescents is not uncommon. However, the majority of research in this area has focused on how a particular personality trait (e.g., negative emotionality) is related to a particular form of psychopathology (e.g., depression), rather than employing a multivariate perspective. Because temperament correlates of internalizing or externalizing problems have primarily been

conducted through separate analyses (e.g., Leve et al., 2005; Williams et al., 2009) we know relatively little about the extent to which temperament variables act as shared risk factors for the co-development of internalizing and externalizing problems. We also know little about how temperament variables are related to the co-occurrence of disorders within diagnostic classes (e.g., anxiety and depression). The extant research lends itself largely only to speculation as to how temperament factors affect the co-development of mental health problems and conversely, the factors that differentiate them.

Developmental psychopathologists highlight the need to consider the means by which temperament might lead to co-occurring internalizing or externalizing problems. First, temperament may directly influence a child's likelihood of displaying internalizing or externalizing problems. For example, while negative emotionality (the tendency to react to stressful situations with unpleasant emotions) may represent a general risk for psychopathology, a child who is also rated high on activity/impulsivity may be quick to show externalizing problems such as anger and aggressiveness. In contrast, a child rated as high on social withdrawal may be quick to internalize feelings of sadness and to withdraw from his/her social environment. Alternatively, temperament may affect the development of psychopathology through indirect means (Wolff & Ollendick, 2006). Specifically, children's own characteristics can modify, or moderate, how they respond to environmental experiences such as parenting, with consequences for their mental health and adjustment (Davidov, Knafo-Noam, Serbin, & Moss, 2015). Goodness-of-fit between temperamental style and parenting are an example of this form of influence (Kuczynski, 2003). In such models, the consequences of parenting depend on whether parenting matches the behavioral tendencies and needs of the child. Child x environment interactions can examine levels of susceptibility to environmental inputs, and also active child influences. For example, children with certain temperamental styles may be strongly influenced by the quality of parenting received, for better or for worse (Belsky, 1997; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007) or children may elicit different kinds of parenting as a function of their temperament.

In terms of parental characteristics that potentially contribute to poor goodness-of-fit between parent and child, maternal depression and negative parenting styles have been commonly investigated. In general, depressed mothers engage in more negative parenting practices, including the use of more physical punishment, less effective monitoring and

supervision, and less warmth and sensitivity than non-depressed mothers (Lovejoy, Graczyk, O'Hare & Neuman, 2000). Such parenting practices have been consistently linked to child internalizing and externalizing problems (Goodman et al., 2011). The use of harsh and inconsistent discipline is theorized to contribute to coercive interactions between parent and child, resulting in increases in externalizing problems (Patterson, Reid, & Dishion, 1992; Serbin, Kingdon, Ruttle, & Stack, 2015). Similarly, reduced parental warmth and responsiveness to offspring's internalizing problems may be associated with lowered child emotion regulation abilities, which in turn may lead to increases in internalizing problems (Yap, Allen, & Sheeber, 2007). Although research has investigated how temperament and parenting operate in a transactional manner to produce psychopathology, an important next step is to investigate the mechanisms through which temperament, parenting, and other contextual factors operate to place a child at risk for comorbid psychopathology.

Sex and Gender Differences

Sex and gender differences in the rates of childhood psychopathology are consistently cited in the literature and have been suggested as potentially important in the etiology of comorbid conditions. Specifically, early-onset disorders (e.g., conduct problems, ADHD, and other neurodevelopmental disorders) show a marked male preponderance, whereas adolescent-onset emotional disorders (e.g., depression, some types of anxiety disorders) show a marked female preponderance (Zahn-Waxler, Shirtcliff, & Marceau, 2008). Some studies have indicated that rates of comorbidity may be higher among girls versus boys. Specifically, the comorbidity between depressive and anxiety disorders appears to be more common in girls than in boys (Breslau, Schultz, & Peterson, 1995; Lewinsohn et al., 1995; Zahn-Waxler et al., 2008). However, rates of comorbidity for conduct problems and depression are more inconsistent, with some researchers indicating higher rates in girls versus boys (Loeber & Keenan, 1994) and others indicating the reverse (Miller-Johnson, Lochman, Coie, Terry, & Hyman, 1998). Even less is known about whether the pathways to comorbidity differ for boys and girls. For example, little is known about whether gender differences in risk factors for internalizing and externalizing problems (e.g., temperament) may contribute to gender differences in pathways to co-occurrence.

Additionally, child gender has been hypothesized as an important factor that may moderate the relation between parenting and other family factors in the development of

internalizing and externalizing problems. Several longitudinal studies have demonstrated interactions between family adversities and gender such that only one sex is affected, one sex is affected more than the other is, or both sexes are affected but in opposite ways (Zahn-Waxler et al., 2008). To illustrate, some research has indicated that girls' socialization for greater sensitivity to interpersonal relationships may increase their vulnerability to internalizing problems, especially when interpersonal supports are lacking (Hyde, Mezulis, Abramson, 2008; Zahn-Waxler et al., 2008). Additional research is required to understand how parenting and other family factors may interact with child gender to differentiate trajectories of co-occurring internalizing and externalizing problems in youth.

Goals of the Thesis

The present series of studies examined the sequential relations between internalizing and externalizing problems across childhood and adolescence, using developmental psychopathology as a conceptual framework. The goal of the current dissertation project was to test multiple potential pathways to co-occurrence in order to determine the risk factors and processes that best characterize the development of comorbidity in childhood. Specifically, the studies investigated whether symptoms in one domain put a child at risk for developing symptoms in other domains later in life. They also examined whether specific temperament factors (i.e., negative emotionality) act as shared etiological risk factors for the development of comorbid internalizing and externalizing problems. In addition to identifying common risk factors for comorbidity, it was hypothesized that other dimensions of temperament (e.g., activity and sociability) may act as specific risk factors that contribute to the differentiation of internalizing and externalizing problems across development. A secondary goal of this work was to identify contextual factors that moderate the temperament-psychopathology relationship and explain why some temperamentally "at-risk" children develop psychopathology while others do not. This information has the potential to identify pathways to specific forms of psychopathology, as well as shared etiological risk factors that are associated with multiple forms of psychopathology.

The Current Studies

The present thesis involves two studies. Data for these studies were drawn from the Concordia Longitudinal Risk Project, a project initiated between 1976-1978 that involved over 4000 children in schools sampled from neighborhoods in working class areas of Montreal, Québec. Children in grades 1, 4 and 7 were peer-nominated on levels of aggression, social

withdrawal and likeability. The final sample included an oversampling of children with elevated ratings on dimensions of aggression, withdrawal, or both, as well as a comparison sample of typically developing children. These individuals have now reached adulthood and have offspring of their own, who are the primary focus of the following two studies. Between 1999 and 2003, the original participants and their preschool-age children were contacted and invited to participate in the present studies. Parents and children completed a number of measures examining their psychological, physical, and social functioning (see Serbin et al., 2011 for a more in-depth description of the present sample). At this time, trained research assistants visited the family at home to gather observations of the home environment, including parenting quality, and to make observations of the child's temperament in a naturalistic setting. Children and their families were followed throughout the child's development, at regular 3-year intervals (ages 7, 10, 13, and 16). At each of these 3-year interviews, information was obtained regarding children's mental health symptomatology and additional observational data was collected on parenting and other family processes. Finally, in adolescence, children and their parents completed clinical interviews with trained clinical psychology graduate students in order to obtain information regarding mental health diagnoses. The sub-samples used for each of these follow-up studies are representative of the original sample in terms of gender, family SES, neighborhood, and behavioral characteristics in childhood.

To address the previously identified gaps in the literature, several methodological considerations were integrated into the design of the proposed studies. These studies utilized a longitudinal, multi-method, multisource design. That is, different combinations of observational data, along with child, mother, and father report data were used to assess parenting, temperament, and mental health problems. This is an important contribution to the existing literature as few studies have used both preschool characteristics and environmental factors to predict adolescent outcomes using multiple methods within a prospective, long-term time frame.

The purpose of each study was to identify how early childhood symptoms were related to adolescent psychopathology outcomes, both within and across diagnostic classes. Four questions were examined in each of the studies: (1) whether the experience of symptoms of one disorder was associated with the development of future symptoms of another disorder, controlling for baseline symptom levels in each domain; (2) whether common temperament risk factors predicted the co-development of internalizing and externalizing problems, and conversely,

whether certain temperament dimensions predicted their differentiation; (3) whether other contextual variables, such as the quality of parenting, predicted children's mental health outcomes, either separately or in combination with temperament factors (i.e., moderation); (4) whether parenting and other family factors interacted with child gender to differentiate trajectories of internalizing and externalizing problems in boys and girls. Study 1 examined the longitudinal associations across diagnostic classes (i.e., between internalizing and externalizing problems), while Study 2 was designed to focus on how comorbidity within a diagnostic class (i.e., anxiety and depression) emerged over time. Furthermore, Study 2 was designed to extend Study 1 by exploring whether methodological factors contributed to comorbidity and how findings differed when examining symptom versus diagnostic level data.

First, it was anticipated that the results would support previous findings linking early childhood symptoms to increases in those symptoms across development. Second, it was anticipated that early symptomatology would predict increases in symptoms in other domains over time, but also that these associations would be reduced once common risk factors (e.g., temperament, parenting) and methodological factors were accounted for. This pattern of results would support the common risk factor model of co-occurrence. Specifically, results from Study 1 were anticipated to demonstrate that negative emotionality represents a general risk factor for both internalizing and externalizing problems, but that combinations of sociability and activity predict the differentiation of internalizing and externalizing problems. Extending the results from Study 1, Study 2 was anticipated to highlight that a particular combination of temperament traits (i.e., high negative emotionality and low positive emotionality) would predict the co-development of anxiety and depression. Parenting quality was expected to moderate the relation between temperament and its impact on the development of internalizing and externalizing problems, such that poor goodness-of-fit between child temperamental traits and parenting would predict worse developmental outcomes. Owing to the lack of research on the topic, no specific predictions were made regarding sex and gender differences in pathways to comorbidity, although it was anticipated that gender would moderate some of the relations between family risk factors and psychopathology.

Table 1

Influential Theoretical Models of Temperament and their Common Dimensions

	Definition of temperament	Negative Emotionality	Positive Emotionality	Effortful Control
Thomas and Chess (1977)	Stylistic aspects of behavior	Negative emotionality	Activity Approach Mood	Distractibility Task persistence
Buss and Plomin (1984)	Early-appearing, heritable aspects of personality	Emotionality	Activity Sociability Shyness	Attention span
Rothbart (1981)	Reactive and self-regulatory aspects of behavior	Negative affectivity	Surgency	Effortful control

Note. This table is based on the work of Shiner and Caspi (2003).

CHAPTER 2: STUDY 1

The Impact of Temperament and Parenting on the Co-Development of Internalizing and Externalizing Problems in Boys and Girls

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Abstract

Internalizing and externalizing problems among youth co-occur at a much higher rate than would be expected by chance. We investigated the development of co-occurring mother-reported internalizing and externalizing behaviors in youth aged 7 to 16, using parallel process latent growth curve analysis and structural equation modeling. Two hypothesized pathways to co-occurrence were investigated: 1) that internalizing and externalizing problems influence each other over time (causal model hypothesis); and 2) that common temperament risk factors are implicated in the co-development of internalizing and externalizing problems (shared risk factor hypothesis). Participants (*N*=154) were part of the Concordia Longitudinal Risk Project, a prospective community-based study of lower-income families. Results indicated a progression in which high levels of internalizing problems predicted declines in externalizing problems over time. Child and family risk factors accounted for a substantial amount of the shared variance between these problems over time, as well as their differentiation. Specifically, negative emotionality measured during early childhood was related to the development of both internalizing and externalizing problems, while low sociability and high activity differentiated internalizing from externalizing problems, respectively. In addition, temperament moderated the impact of parenting practices (low responsiveness and harsh discipline) on the development of internalizing and externalizing behaviors, such that children with difficult temperament styles were most impacted by negative parenting. Results most strongly support the shared risk factor theory of co-occurrence, while also indicating that specific temperament and parenting factors predict their differentiation.

The Impact of Temperament and Parenting on the Co-Development of Internalizing and Externalizing Problems

Internalizing and externalizing problems co-occur at a much higher rate than chance, at both clinical and subclinical levels of symptomatology, across all stages of development (Achenbach, Howell, Quay, & Conners, 1991; Beyers & Loeber, 2003; Caron & Rutter, 1991; Gilliom & Shaw, 2004; Lilienfeld, 2003; Oland & Shaw, 2005). Internalizing problems are characterized by withdrawal, anxiety, fearfulness, or depression, whereas externalizing problems take the form of hyperactivity, conduct problems, aggression, or delinquency (Achenbach, 1991). Children with co-occurring disorders have an earlier age of onset, more severe and chronic course of illness, higher rates of mental health service usage, and worse developmental outcomes, than children with only a single disorder (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Keiley, Lofthouse, Bates, Dodge, & Petit, 2003). Less clear than their tendency to co-occur is the nature and sequence of the association between internalizing and externalizing problems across childhood and adolescence, and the factors that produce co-occurrence.

Several theoretical models have been proposed to explain the high co-occurrence between internalizing and externalizing problems. One popular model of co-occurrence suggests that internalizing and externalizing problems change together over time and may even be causally implicated in the development of one another (Beyers & Loeber, 2003; Gilliom & Shaw, 2004; Lilienfeld, 2003; Oland & Shaw, 2005). Evidence supporting the causal model hypothesis includes findings that children who display conduct problems are at increased risk for later depression (Capaldi, 1992; Beyers & Loeber, 2003). From this perspective, externalizing problems may be associated with adverse life consequences such as family conflict, academic problems, and social difficulties (i.e., peer rejection), which contribute to the development of depression and anxiety. On the other hand, there is also evidence for a progression from internalizing problems to externalizing problems such that children who display high levels of depression are at increased risk for later conduct problems (Capaldi, 1992; Beyers & Loeber, 2003). In this view, children suffering from internalizing problems are more likely to experience social failure and become involved in deviant peer groups, which may lead to increased delinquency. However, other research has shown that anxiety plays a protective role among children with disruptive behaviour disorders, such that internalizing problems may decrease risk for future externalizing problems (see Cunningham & Ollendick, 2010).

Although interest in comorbidity has fueled a growing research literature, most studies have used designs in which symptoms (or diagnoses) are used to predict future symptomatology. From these studies it remains unclear how *changes* in internalizing problems affect changes in externalizing problems or vice versa. With recent advances in longitudinal data analytic methods (e.g., parallel process growth curve modeling; Cheong, MacKinnon, & Khoo, 2003) researchers are now able to simultaneously estimate developmental trajectories in several domains and to model whether one predicts the other longitudinally, as well as cross-sectionally. A handful of recent studies have examined developmental trajectories of internalizing and externalizing problems simultaneously, providing mixed results regarding the nature of their co-occurrence (see Gilliom & Shaw, 2004; Keiley, Bates, Dodge & Pettit, 2000; Lee & Bukowski, 2012; Leve, Kim, & Pears, 2005). Some studies have shown that externalizing problems are predictors of worsening internalizing problems (Keiley et al., 2000; Gilliom & Shaw, 2004), whereas other studies have found that internalizing problems predict changes in externalizing problems or have bi-directional associations (for better or for worse) that occur concurrently and prospectively (Lee & Bukowski, 2012; Sheidow et al., 2008). Although this research has provided important insights into the temporal patterns of co-occurrence between internalizing and externalizing problems, it does not explain the mechanisms underlying their developmental progression.

Another popular theoretical model of comorbidity argues that co-occurrence may be largely explained by common risk factors that contribute to the development of both types of problem behaviour (Caron & Rutter, 1991; Krueger & Markon, 2006). The developmental psychopathology literature has consistently identified overlapping risk factors for internalizing and externalizing problems, representing aspects of the child, family, and environmental context. Risk variables in these domains commonly include the child's gender, temperament, parenting quality, socio-economic status, stress, and familial psychopathology (e.g., Leve et al., 2005; Mesman & Koot, 2000). Although a large number of studies exist that report upon various risks for externalizing and internalizing problems, most have focused on only one type of problem rather than employing a multivariate perspective. Consequently, we know relatively little about the extent to which these variables may operate as common risks for the co-development of internalizing and externalizing problems. Conversely, we also know relatively little about how risk factors may distinguish between them.

Children's temperament has been widely cited as a key influence on the development of

both internalizing and externalizing problems and may represent a pathway to comorbidity (Lilienfield, 2003; Oland & Shaw, 2005). Modern theories posit that temperament comprises several dimensions of behavior, most commonly negative emotionality, activity, and social approach (or conversely, withdrawal; Rothbart & Bates, 2006; Shiner & Caspi, 2003). The temperament trait of negative emotionality (i.e., the tendency to react to stressors with high-intensity negative reactions, including irritability, anger, negative mood, unsoothability, fear, or sadness) has been related to internalizing and externalizing problems across the lifespan (Kotov, Gamez, Schmidt, & Watson, 2010; Lonigan & Phillips, 2001; Muris & Ollendick, 2005; Rothbart & Bates, 2006) and is considered to represent a general risk factor for psychopathology (Clark, 2005; Muris & Ollendick, 2005). However, it is not well understood how other aspects of temperament may distinguish the two types of psychopathology, either separately or in combination with negative emotionality. For example, temperamental activity has been found to be associated with higher prospective levels of externalizing problems, while separate investigations have shown social withdrawal as a risk factor for clinical elevations in internalizing problems, such as anxiety and depression (Mesman & Koot, 2000; Rubin, Coplan, & Bowker, 2009).

Gender differences. Gender has also been identified as an important predictor of developmental trajectories of internalizing and externalizing problems (Miller, Malone, & Dodge, 2010; Zahn-Waxler et al., 2008). In general, males develop higher levels of externalizing problems than females, whereas females are at greater risk for internalizing problems (Achenbach et al., 1991; Zahn-Waxler et al., 2008). These gender differences seem to vary across developmental stages. During the early preschool years, boys and girls show similar rates of behavior problems, whereas after the age of 4, boys display significantly higher rates of externalizing problems than girls (Zahn-Waxler et al., 2008). Rates of internalizing problems remain similar across the sexes until adolescence, when girls' rates of internalizing disorders (particularly depression) begin to increase and exceed those of boys (Zahn-Waxler et al., 2008). Additional research is necessary to determine whether risk factors for internalizing and externalizing problems (e.g., temperamental factors, parenting) differentially contribute to boys and girls' co-occurring internalizing and externalizing trajectories.

Negative parenting and other risk factors. Other family factors and contextual variables may be important for understanding the co-development of internalizing and

externalizing problems. Negative parenting, characterized by a lack of emotional responsiveness and warmth and a high degree of harsh discipline and demandingess (reflective of the authoritarian parenting style described by Baumrind; 1971), has been frequently related to children's internalizing and externalizing problems (Galambos, Barker, & Almeida, 2003; Serbin, Kingdon, Ruttle, & Stack, 2015; Owens & Shaw, 2003). In addition, numerous studies have linked parental depression and lower socioeconomic status with the development of both internalizing and externalizing problems (Achenbach et al., 1991; Goodman et al., 2011; Owens & Shaw, 2003) as well as negative parenting (Burke, 2003). Additional research is required to understand whether these risk factors are common to both internalizing and externalizing problems, or contribute to the differentiation of internalizing and externalizing trajectories across development, either separately or in combination with child characteristics.

Children's gender has been hypothesized as an important factor that may moderate the relation between parenting and the development of internalizing and externalizing problems. Recent reviews have suggested that girls' socialization for greater sensitivity to interpersonal relationships and reliance on support from parents and peers for coping may increase their vulnerability to internalizing problems, especially when interpersonal supports are lacking (Hyde, Mezulis, & Abramson, 2008; Zahn-Waxler et al., 2008). In contrast, socialization practices that emphasize assertiveness and underemphasize self-regulation in boys, combined with temperamental differences in activity and impulsivity, may put boys at higher risk for externalizing problems (Chaplin, Cole, Zahn-Waxler, 2005; Zahn-Waxler et al., 2008). Additional research is required to understand how parenting factors may interact with child gender to differentiate trajectories of internalizing and externalizing problems in youth.

Interactions Between Child and Family Factors

Despite widespread recognition by developmental psychopathologists of the importance of understanding transactional processes between children and their environments, research typically considers only main effects of child and parent contributions to child psychopathology (Crouter & Booth, 2003). Developmental psychopathologists acknowledge the likelihood that complex, interactive effects between parents and children may more closely resemble real-world relations and processes (Crockenberg & Leerkes, 2003). Research investigating parenting x temperament interactions is beginning to show that children's temperamental characteristics may make them more "vulnerable" to developmental inputs (e.g., low quality parenting). For

example, a number of studies have shown that the effects of negative parenting, such as unresponsiveness, low sensitivity, lack of contingent responding, and harsh punishment on child behavior outcomes are accentuated among children with high levels of negative emotionality or difficult temperaments (Hasting, Rubin, & DeRose, 2005; Lengua & Kovacs, 2005; Morgan, Shaw, & Olino, 2012; Rothbart & Bates, 2006). Results from these studies suggest that children's temperament and family environment characteristics jointly contribute to children's emotional and behavioral problems through either a stress-diathesis or differential susceptibility model of development (Belsky & Pluess, 2009). Parenting x temperament interactions have been examined primarily in cross-sectional studies, which examine either internalizing *or* externalizing outcomes. To our knowledge, no study has investigated the specificity of early temperament and parenting interaction effects in predicting internalizing and externalizing problems within a multivariate model. Furthermore, such interaction effects have been rarely investigated in models that examine *change* in behavior problems over time.

The Current Study

The present project extends previous research by simultaneously considering two hypothesized pathways to co-occurrence: 1) that internalizing and externalizing problems influence one another over time; and 2) that specific temperamental characteristics and family/environmental risk factors are implicated in the co-development of internalizing and externalizing problems. To test these hypotheses, trajectories of mother-rated internalizing and externalizing problems were examined among a community-based study of boys and girls from lower-income backgrounds, using parallel process latent growth curve modeling (LGM; Cheong et al., 2003), controlling for family socio-economic status and maternal depression. Gender differences in the effects of negative parenting, and parenting x temperament interactions were also explored.

Based on the literature reviewed above, we expected that bi-directional effects would be observed, such that initial levels of externalizing problems would predict developmental trajectories of internalizing problems and vice versa. However, we also expected that temperament, parenting, and other family factors assessed in the early childhood years would predict, in part, co-occurring trajectories of internalizing externalizing problems. Specifically, we predicted that temperamental negative emotionality would be related to co-developing problems, whereas other factors would differentiate domains of behavioral difficulty (e.g., social

withdrawal predicting internalizing problems; activity predicting externalizing problems). We also hypothesized that temperament would moderate the impact of parenting (e.g., harsh discipline, responsiveness) on the development of behavior problems, in support of the transactional theories of child psychopathology. If these predictors (and their interactions) could adequately characterize the temporal covariation among children's internalizing and externalizing symptoms, this would be interpreted as support for a common risk factor explanation of symptom co-occurrence. Finally, although boys were expected to display greater levels of externalizing problem behaviors than girls, no predictions were made regarding whether common and specific risk factors would differ for boys and girls, owing to a lack of research on this topic.

Method

Participants

The participants in the current study consisted of 154 parent–child dyads, recruited from the Concordia Longitudinal Risk Project (Concordia Project), an intergenerational, community-based research project that examines the processes that are associated with positive versus negative social and health outcomes across the life course. The original longitudinal study began in 1976 with screening of the school populations at 22 inner-city schools and the selection for follow-up of over 1,700 elementary-school French-speaking children living in disadvantaged neighborhoods of Montreal, Quebec. The original participants have now reached adulthood and have children of their own. Representative subsets of these original participants and their offspring have been screened at 3-year intervals on observational, interview-based, and questionnaire measures of health, educational, and social functioning (Serbin et al., 2011).

A sub-sample of original participants in the Concordia Project who had preschool-aged children (2 to 5 years; M=3.54, SD=1.57) were identified and invited to participate in the present study. This report focuses on the 154 families who participated in the initial wave of data collection at preschool age, and at least one of four follow-up assessments between the ages of 7 to 16 years. Approximately 88% of invited families (eligible N=175) participated during the period of the current study. The included participants did not differ significantly (all p's >.05) from the non-included participants or from the larger sample in terms of family income, maternal education, occupational prestige, family structure, or parental psychopathology. Families were primarily (i.e., over 95%) of French-Canadian descent and spoke French at home, and the

children attended French language schools. Mothers were on average 29.65 years of age (*SD*=2.66) and fathers were 31.93 years (*SD*=4.45) at the first wave of data collection used for the trajectory analyses. Approximately 26% of mothers and 23% of fathers left school without completing their secondary education (equivalent to Grade 11 in the Quebec system). Mean annual income of families at that time was $40,460 CAD (SD=$24,807 CAD), with approximately 16% of families receiving government assistance. This was well below the median family income in Quebec and across Canada (CAD $50,242 and CAD $55,016, respectively; Statistics Canada, 2003, p. 20). Rates of father absence in the home increased from 27% to 54% across the study period.

Procedure

Informed consent and demographic information was obtained from the participants during an initial telephone interview. Multiple study procedures were implemented at various phases of data collection, including observational data, semi-structured interviews, and parent-report questionnaires. At preschool age, two visits were made to the participants' homes, lasting 2 to 3 hours each. During these home visits, a senior research assistant (M.A. in psychology) completed a semi-structured interview and observation of the child in the home environment in order to complete ratings of the quality of parenting and the child's temperament. At this time, both mothers and fathers completed questionnaire measures assessing the child's temperament. Children who participated in the first phrase of data collection were subsequently assessed for internalizing and externalizing problems at approximately 3-year intervals across childhood and adolescence using questionnaire-based measures that were mailed to the children's mothers (Wave 2 middle childhood: age 6-9; *M*=7.61, *SD*=0.92; Wave 3 pre-adolescence: age 9-12; *M*=10.85, *SD*=0.92; Wave 4 early adolescence: age 12-15; *M*=13.45, *SD*=0.90; Wave 5 late adolescence: age 15-18; *M*=16.61, *SD*=0.89). Families and children were compensated at each wave of data collection with an honorarium of CAD $50 (family) and small gift (child). These procedures were approved by the university's Institutional Review Board.

Measures

Mother-reported internalizing and externalizing behavior symptoms. Internalizing and externalizing problem behaviors were assessed by mother's ratings on a French translation of the Child Behavior Checklist (CBCL 4-18/parent form; Achenbach, 1991). Fathers' reports were also requested, but the reply rate across each of the waves of data collection was not

sufficient to include in the present analyses. The CBCL is one of the most commonly used and well-validated questionnaire measures in child and adolescent psychiatric research (Achenbach, 1991). At each of the assessments (age 7, 10, 13, 16), mothers indicated if the 112 problem behaviors listed on the CBCL were observed by them "often" (2), "sometimes" (1), or "rarely/never" (0) in the past six months. For the present study, we analyzed total (raw) scores of the internalizing and externalizing broad-band scales. The internalizing scale is composed of 32 items in the CBCL, consisting of items measuring Anxious/Depressed, Withdrawn/Depressed, and Somatic Complaints. The externalizing scale is composed of 33 items in the CBCL, consisting of items measuring Delinquent and Aggressive behavior. Reliability coefficients for the internalizing and externalizing scales in this sample ranged from 0.84 to 0.94 across each time point.

Temperament. Temperament was measured using Buss and Plomin's EAS Temperament Survey (EAS; Buss & Plomin, 1984), assessed at preschool age. Mothers and fathers independently completed this questionnaire and the experimenter completed a parallel version of this questionnaire after observing the child in his or her home. The EAS is composed of 20 questions, which are rated on a five-point Likert scale. Questions are grouped into three subscales that correspond to temperament dimensions of (negative) Emotionality, Activity, and Sociability. The factor structure of the EAS has been consistently replicated and the stability (i.e., test-retest reliability) for each of the three EAS subscales is high among preschool-aged children (Mathiesen & Tambs, 1999). Mothers, fathers and experimenters' ratings of children's negative emotionality, activity and sociability on the EAS were transformed to z-scores and averaged to create a multi-informant scale. If only one parent rating was available, the scale was calculated using the experimenter's and the available parent's rating (97% of mothers and 82% of fathers completed the questionnaires). Each temperament dimension demonstrated acceptable internal consistency (Cronbach's α: negative emotionality = 0.82; activity = 0.74; sociability = 0.66).

Socio-economic Status (SES). When children were of preschool age, parents' occupational prestige was assessed via the Standard International Occupational Prestige Scale (Ganzeboom & Treiman, 1996), an international measure of occupational codes and prestige criteria. The highest scores are given to professionals (e.g., lawyers, physicians, and chief executive officers), while the lowest scores are given to those who hold such occupations as

domestic laborers, manufacturers, and farmhands. The mean level of occupational prestige in this sample was 38.69 (*SD*=11.07), equivalent to the level of occupational prestige represented by cashiers, receptionists, welders, and agricultural workers.

Maternal Depression. When the child was of preschool age, mothers completed the SCL-90 (Derogatis & Lipman, 1977) to assess their depressive symptoms. The SCL-90 consists of 90 items used to assess psychiatric symptomatology among adults in the general population. In this study, we used the depression subscale (13 items rated on a 5-point Likert-type scale) to assess mother's symptoms of dysphoric mood and affect during the past week. Scores were converted to standard T-scores with a mean of 50 and a standard deviation of 10. Internal consistency for the depression subscale was good (Cronbach's $\alpha = 0.88$).

Parenting. The Home Observation for Measurement of the Environment (HOME; Caldwell & Bradley, 1984) was administered to each participating family as a measure of the quality of parenting. The HOME is a widely used observational and interview instrument that has been shown to have excellent reliability and validity in predicting children's development over time (Caldwell & Bradley, 1984). The Infant/Toddler HOME version was administered to children between the ages of 1 and 3 years, while the Early Childhood HOME version was administered to children between the ages of 3 and 6 years. In the current study, we utilized the parental responsiveness subscale as a measure of the verbal and affective communications between the parent and child, and the acceptance of child behavior scale as a measure of parental acceptance of the child's negative behavior and avoidance of undue restriction and harsh punishment (reverse coded, referred to subsequently as "harsh punishment"). The responsiveness scale includes parallel items across the Infant/Toddler and Early Childhood versions, such as "parent vocalizes to [converses with] child at least twice during visit." The harsh punishment scale includes parallel items across the Infant/Toddler and Early Childhood versions, such as "caregiver does not shout at child during visit," "no more than one instance of physical punishment occurred during the past week" and "caregiver does not express overt annoyance with or hostility to the child" (reverse coded). Raw scores on the responsiveness and harsh punishment scales were transformed into standardized scores.

Analytic Strategy

The overarching goal of the current study was to increase our understanding of how changes in internalizing problems are associated with changes in externalizing problems over

time and the factors that produce this co-occurrence. To fully understand this phenomenon, we employed parallel process latent growth curve modeling (LGM; Cheong et al., 2003) to investigate the growth of internalizing and externalizing behaviors and an autoregressive cross-lagged panel model (ARCL) to evaluate the direction of associations over time. All models were estimated via Mplus Version 7 (Muthén & Muthén, 2012). For the parallel process model, data analysis was conducted in three steps. In the first step, we began by separately modeling the individual unconditional growth model that best represented the change in internalizing and externalizing behaviors, separately. In the second step, after examining the individual growth curves, separately, we then modeled the internalizing and externalizing growth trajectories simultaneously in the same analysis. In the third and final step, after finding the parallel process growth trajectories that fit the data best, we added covariates to the model. Because these models estimated the growth in internalizing and externalizing problems simultaneously, all results correct for the large correlations between these two domains. Interaction terms were added to the final conditional model in order to determine whether temperament and gender moderated the relations between parenting and trajectories of internalizing and externalizing problems. The predictor and moderator variables were entered first, followed by a single interaction term, the interaction between one of the key parenting and child variables (temperament or gender). For the interpretation of statistically significant interactions, regression lines were plotted for high (+1 SD) and low (-1 SD) moderator values, as recommended by Aiken and West (1991). For the ARCL model, we allowed the residuals of internalizing and externalizing problems to covary within each time period and set the magnitude of similar path coefficients to be equal over the four measurement points.

Missing Data. In the present study, there was minimal missing data (0-4%) on the predictor variables. The amount of missing data for the outcome measures (internalizing total scores, externalizing total scores) ranged between 16% and 42%, reflecting increasing missing data over time due to attrition. Most mothers (65%) completed assessment measures on at least 3 of 4 waves of data collection. All available observations were included in the analysis, for which the full information maximum likelihood (FIML) approach of Mplus was used, which is a robust estimation method when data are missing at random or completely at random (Enders & Bandalos, 2001). We conducted a missing-value analysis using SPSS software version 22 and Little's MCAR test. Results of this analysis supported the missing completely at random

assumption $\chi^2(263)=287.88$, $p=.14$, suggesting that missingness in our dataset did not likely impact our results and permitting the application of FIML procedures (Enders & Bandalos, 2001).

Testing Model Fit. Model fit was assessed by examining the chi-square statistic test, the comparative fit index (CFI), the root mean square error of approximation (RMSEA), and the standardized root mean square residual (SRMR). Good-fitting models yield non-significant chi-square goodness-of-fit tests. CFI values greater than .95 indicate good fit, and values between .90 and .95 indicate adequate fit. Generally, RMSEA valued less than .05 are considered to indicate good fit, values between .05 and .08 indicate adequate fit, and values larger than .10 indicate poor fit. SRMR values range from 0 to 1.0, with values less than 0.09 considered adequate (Muller & Hancock, 2008).

Results

Descriptive Analyses

Table 1 presents the means, standard deviations, and inter-correlations for all study variables. The descriptive statistics for the CBCL raw scores, which were used for analysis as a requirement of the latent growth curve design, showed a pattern of decreasing internalizing and externalizing problems across time. As expected, there was a high degree of co-occurrence between internalizing and externalizing problems at each time point (r = .38 to 78). Table 2 presents the means and standard deviations for the CBCL raw scores (and corresponding T-scores), separated by sex. Figure 1 presents the average internalizing and externalizing problems from age 7 to 16, and variation in these trajectories, separately for each sex.

Trajectories of Internalizing and Externalizing Problems

In the first stage of data analysis, we estimated an unconditional latent growth model (omitting the predictor variables) to establish the growth function that best captured growth in internalizing and externalizing problems. Visual inspection of the plots indicated that changes in both domains were linear, suggesting that linear growth models would fit the data well. Thus, the growth model contained two individual growth parameters: (1) an intercept parameter representing initial status, and (2) a slope parameter representing rate of change. Each child's intercept and slope terms were estimated, with time centered at the first time point in the trajectory analysis, age 7 (intercept). The unconditional model also allowed us to estimate average trajectories and heterogeneity in individual trajectories and to estimate correlations

among intercept and slope parameters. We allowed the error variances of the outcome measures to covary within time points based on the assumption that mothers' measurement errors for externalizing problems were related to those for internalizing problems.

The linear model of internalizing and externalizing problems fit the data well: $\chi 2(18)=37.320$, $p<.01$; CFI=0.953; RMSEA=0.083; SRMR=0.066. To confirm the linear pattern, we tested whether a curvilinear pattern fit the data better by adding quadratic terms to the baseline model. The mean of the quadratic term did not differ significantly from zero and did not improve model fit. Based on these results, we concluded that growth was linear in both domains. We then tested the adequacy of the equal residual variances over time and found that there was a significant decrement in model fit associated with this restriction. We removed this restriction and allowed the residual variances associated with externalizing problems measured at Time 2 and Time 3 to be freely correlated. The final unconditional model ($\chi 2(17)=20.278$, $p=.26$; CFI=0.992; RMSEA=.035; SRMR=0.056) showed good fit to the data.

Table 3 presents the parameter estimates for the latent factors for trajectories of mother-rated internalizing and externalizing problems. On average, children's internalizing problems decreased across the four time points ($\mu=-0.812$, $p<.01$). There was statistically significant variation in the initial level of internalizing problems among children ($\psi=26.001$, $p<.001$) and non-statistically significant variation in the growth rate of internalizing problems ($\psi= 1.787$, $p=0.33$), indicating that individuals had a similar rate of growth. The correlation between the initial level and growth rate of internalizing problems was negative and statistically significant ($\beta=-0.435$, $p<.05$), indicating that children with a relatively high internalizing intercept (initial status) experienced a steeper decline in internalizing problems across childhood and adolescence. A similar pattern emerged for externalizing problems: on average children's externalizing problems decreased across the four measurement points ($\mu=-1.621$, $p<.001$). There was statistically significant variation in the initial level of externalizing problems among children ($\psi=43.038$, $p<.001$) and non-statistically significant variation in the growth rate of externalizing problems ($\psi= 1.514$, $p=0.44$).

Relations Between Trajectories. Table 3 presents the correlations between the intercept and slope parameters from the final unconditional model. The positive correlation between the initial level of internalizing and externalizing problems ($\beta=0.643$, $p<0.001$), indicated that children who were perceived by mothers as anxious, sad, and withdrawn also tended to be

perceived as aggressive, defiant, and hyperactive. In terms of cross-domain relationships, the correlation between the initial level of externalizing problems and growth in internalizing problems was negative and statistically significant (β=-0.317, p<0.05), suggesting that children who had high early levels of disruptive and aggressive behavior decreased more rapidly on internalizing problems across childhood and adolescence than children who lacked these characteristics.

Given that regression to the mean could produce these negative correlations, in which extremely high (and low) intercept values are likely to be closer to the sample mean at repeat assessment, these associations were investigated in more detail via an autoregressive cross-lagged panel model (see Figure 2). The cross-lagged model produced adequate model fit: $\chi 2$ (20)=42.14, p=.01, RMSEA=.08, SRMR=.11. These results indicated that internalizing problems showed strong stability throughout childhood and adolescence (β = .56-.62, p < .01), as did externalizing problems (β = .77 -.83, p < .01). Internalizing problems and externalizing problems were positively concurrently related at each time point, indicating that more internalizing problems were related to more externalizing problems. Examination of cross-lagged paths indicated that higher internalizing problems at each wave were related to *lowered* externalizing problems at the next wave of data collection, controlling for contemporaneous associations between constructs, and the stability of each construct over time. Cross-lagged paths from externalizing to internalizing problems were all non-significant. These results are consistent with the hypothesis that internalizing problems buffer against the development of externalizing problems, as children's emotional and behavioural problems become increasingly differentiated across development.

Results of Conditional Model: Effects of Common and Unique Risk Factors

In the final stage of the growth analysis, we predicted individual differences in internalizing and externalizing trajectories using child temperament and parenting variables, while controlling for the effects of child gender, family occupational prestige, and mother's depression (see Figure 3 for the path diagram of this model). The parallel process model of internalizing and externalizing problems, including predictors, fit the data well: $\chi 2(56)=61.164$, p=.30; CFI=0.990; RMSEA=.024; SRMR=0.051. The early childhood risk factors were related to internalizing and externalizing trajectories in a conceptually coherent manner (see Table 4). As hypothesized, child temperament variables measured at preschool age, including higher levels

of negative emotionality and lower levels of sociability, predicted internalizing problems at age 7 (initial status). Child temperament was also related to the rate of change in this domain: children who rated higher on sociability experienced weaker decreases in internalizing trajectories, while children with higher activity levels were more likely to experience steeper declines in internalizing problems over time. Family characteristics including higher levels of maternal depression were also related to higher initial status on internalizing problems.

For externalizing problems, as hypothesized, higher levels of emotionality and activity predicted higher initial levels of externalizing problems. Temperament factors were not statistically significantly related to the rate of change. Gender was related to externalizing problems such that boys had higher initial levels of externalizing problems than girls. Although boys sustained higher levels of externalizing problems than girls across all four waves, the rate of change in externalizing problems did not differ across gender.

Overall, child temperament and family factors assessed in preschool predicted 37% of the stability and 36% of the linear changes in internalizing problems and 33% of the stability and 24% of the linear changes in externalizing problems. When the predictor variables were included in the final conditional model, the correlation between initial status of internalizing and initial status of externalizing problems remained large and statistically significant (β=0.651, p<0.001). However, the correlation between initial status of externalizing problems and change in internalizing problems was reduced to non-significance. Temperament emerged as the strongest predictor of internalizing and externalizing trajectories: the three temperament dimensions predicted 13% of the stability and 15% of the linear changes in internalizing problems and 9% of the stability and 5% of the linear changes in externalizing problems, over and above the other demographic, family, and environmental variables included in the model.

Gender. As shown in Table 1, bivariate correlations revealed that male gender was significantly related to higher levels of externalizing problems at each time point, as well as to greater activity level. In the final conditional model, male gender predicted greater externalizing problems at age 7 (intercept), controlling for the other predictors in the model. Although sample size limitations precluded the ability to test the full model separately for each gender, preliminary analyses revealed that the slope of internalizing problems for boys was negative (μ =-1.30, p<.001), while the slope for girls was flat (μ =-0.30, p=.44). Models were also run to test whether gender moderated the effect of the predictor variables on internalizing and externalizing

outcomes by adding interaction terms for gender and each of the predictor variables in the final step. The interaction effects of child gender and maternal responsiveness were significant predictors of the intercept of both internalizing (β=-0.796, $p<0.05$) and externalizing problems (β=-0.673, $p<0.05$). Examination of the interaction effects revealed that high maternal responsiveness predicted lower age 7 internalizing and externalizing problems in girls, but *higher* levels of internalizing and externalizing problems in boys (internalizing: b=-3.69, $p<0.05$; externalizing: b=-6.16, $p<0.01$). None of the other gender interaction terms were statistically significant.

Parenting x temperament interactions. Examination of interaction effects revealed that temperament significantly moderated the effects of maternal responsiveness and harsh discipline on the prediction of internalizing and externalizing problems (see Table 4 for all interaction effects). The interpretation of these significant interaction effects, inferred from the plotted regression lines for children with high (+1 SD) and low (-1 SD) temperament, showed that harsh discipline was differentially related to age 7 internalizing problems among more sociable (b=-2.63, $p<0.05$), versus less sociable children (b=2.94, $p<0.05$). Specifically, among children with low levels of sociability, harsh discipline predicted greater levels of age 7 internalizing problems. Among highly sociable children, harsh discipline predicted lower levels of internalizing problems. In addition, results revealed a statistically significant interaction between negative emotionality and maternal responsiveness in the prediction of age 7 internalizing problems. Follow-up analyses showed that negative emotionality did not predict age 7 internalizing problems among children with responsive mothers (b=0.85, p=0.44), whereas it did significantly predict internalizing problems among children with less responsive mothers (b=4.73, $p<0.01$). Specifically, among children with less responsive mothers, negative emotionality predicted greater levels of internalizing problems.

The significant association between negative emotionality and children's initial levels of externalizing problems was moderated by a statistically significant two-way interaction of emotionality x harsh discipline. Specifically, harsh discipline did not significantly predict age 7 externalizing problems among the high emotionality group (b=0.23, p=0.90), however it did predict externalizing problems for the low negative emotionality group (b=2.63, $p<0.01$). Specifically, among children with lower levels of negative emotionality, lower levels of harsh discipline predicted fewer age 7 externalizing problems. Although statistically significant

interactions between temperament and parenting variables emerged in predicting the slope of internalizing and externalizing problems, in each of these analyses, regression lines did not statistically differ from one another.

Discussion

It is commonly speculated that early externalizing problems contribute to later emerging internalizing problems (Beyers & Loeber, 2003; Calpaldi, 1992). Conversely, others argue that early internalizing problems lead to later acting out behaviors (Gilliom & Shaw, 2004; Keiley et al., 2003). These speculations are based on a large literature showing a high level of co-occurrence between internalizing and externalizing problems across development (Costello et al., 2003; Lilienfeld, 2003; Oland & Shaw, 2005). However, this work often fails to longitudinally track patterns of sequential comorbidity and also to examine risk factors that might represent an underlying mechanism for co-occurrence. The primary purpose of the current investigation was to clarify the co-development of internalizing and externalizing problems in a community sample of lower-income children, by modeling trajectories simultaneously within a parallel process growth-modeling framework and by examining potential bi-directional relations via structural equation modeling.

Progression from externalizing to internalizing problems. Consistent with previous work (Achenbach et al., 1991), we found a high degree of co-occurrence between internalizing and externalizing problems cross-sectionally (r = .38 to .78). When we examined the co-occurrence longitudinally, results indicated that early internalizing problems predicted a *decreased* level of externalizing problems in subsequent years. Although internalizing and externalizing problems are highly correlated contemporaneously and are often theorized to increase the risk for one another over time, our results are consistent with other work indicating that internalizing problems may actually attenuate the severity and course of externalizing problems (see Cummingham & Ollendick, 2010). Given our findings that early internalizing problems decreased risk for future externalizing problems, rather than increasing risk over time, a causal model may not be the best explanation for their co-occurrence. Instead, our results suggest that common risk factors precede the development of both types of problems. In fact, as a set, child temperament, gender, and family factors assessed in preschool predicted between 24 and 37% of the stability and linear changes in internalizing and externalizing problems across the study period. When including these important risk factors in our model, the correlation between

the intercepts and slopes of internalizing and externalizing problem behaviors was no longer statistically significant. Thus, conflicting results regarding the temporal relations between internalizing and externalizing problems may be best understood by identifying common risk factors (e.g., temperament), that predict their co-development.

Common and unique temperament predictors of internalizing and externalizing trajectories. Ratings of temperamental characteristics assessed in early childhood emerged as the strongest predictors of internalizing and externalizing problems, as evaluated over a 9-year period, at ages 7, 10, 13, and 16. As expected, some risk factors contributed uniquely to problems in each domain, while other factors acted as shared etiological factors. Mothers reported wide variations in their children's internalizing and externalizing behavior at age 7, with levels differing according to the temperamental characteristics of their children. In general, children who were rated at preschool age as having high levels of negative emotionality showed greater levels of both internalizing and externalizing problems at age 7. While negative emotionality was consistently related to children's initial levels of internalizing and externalizing problems, it was not related to change in behavioral trajectories. This may reflect the fact that emotionality is a general risk factor for psychopathology (i.e., distress proneness) and does not differentiate between trajectories of internalizing and externalizing problems over time.

Our results add to the understanding of the etiology of co-occurring internalizing and externalizing problems by showing that other facets of temperament (i.e., activity and sociability) have specific risks in predicting how trajectories of internalizing and externalizing problems differentiate themselves over time. As predicted, boys and those children with high activity levels were rated as having greater levels of externalizing, but not internalizing problems, at age 7. This finding is consistent with previous reports that activity levels assessed in early childhood predict future disruptive behavior disorders, such as ADHD and conduct problems, which are highly prevalent among boys (Harvey, Youngwirth, Thakar, & Errazuriz, 2009). In early childhood, temperamental activity is linked to impulsivity and the tendency to become easily frustrated (De Pauw, Mervielde, & Van Leeuwen, 2009; Rothbart & Bates, 2006). Thus, the combination of negative emotionality and activity may place children (especially boys who tend to have higher activity levels) at risk for increased rates of externalizing problems. These children are more likely to experience a high degree of distress/frustration and due to deficits in impulse control, may act out their feelings by displaying aggressive or rule-breaking

behavior. Although preschool activity was identified as a powerful predictor of children's externalizing problems, it did not predict the rate of change in externalizing trajectories. This finding may indicate that the relationship between temperament and externalizing problems is fairly stable across development. Alternatively, temperament factors may set into motion a series of environmental events that mediate the association between early childhood characteristics and later externalizing behaviours.

Also consistent with our hypotheses, lower levels of sociability predicted greater internalizing problems at age 7, while the relation between sociability and externalizing problems was not statistically significant. The children who were rated in preschool as having high levels of negative emotionality and low levels of sociability had relatively high levels of internalizing symptoms at age 7. These results are consistent with other research indicating that children who experience high levels of distress/frustration and who have a tendency to withdraw socially, may be more likely to internalize their negative emotional states and feel increasingly inhibited, anxious, isolated, and depressed over time (Rubin et al., 2009). Perhaps consistent with the 'law of initial values', children who rated higher on sociability at preschool age experienced weaker decreases in internalizing trajectories over time than children with low sociability scores. Finally, children with higher activity levels were more likely to experience a faster declining rate of change in internalizing problems over time. Taken together, these results may indicate that although internalizing and externalizing problems often co-occur, they represent two distinct types of behavioral problems that differ somewhat with regard to the types of temperament profiles that characterize them. Furthermore, internalizing and externalizing problems appear to become increasingly differentiated across development, as their rate of co-occurrence decreases across time and specific risk factors for one domain of behavioural difficulty (i.e., activity level, which represents a specific risk factor for externalizing problems) weaken growth in the other domain (i.e., internalizing problems).

Gender differences. Recognizing that very limited research has examined gender differences in developmental trajectories of co-occurring behaviour problems, we tested whether the risk factors identified in the present study were differentially related to boys' and girls' internalizing and externalizing behaviours. Although findings were generally consistent across boys and girls, we did find evidence of a sex difference in the effect of parental responsiveness on risk for both age 7 internalizing and externalizing problems. Whereas high maternal

responsiveness predicted lower levels of internalizing and externalizing problems in girls, it predicted *higher* levels of internalizing and externalizing problems in boys. It has been suggested that beginning in toddlerhood, mothers express greater levels of responsiveness and affection with daughters, as compared to sons, consistent with socialization practices that encourage interpersonal closeness during girls' development (Leaper, 2002). Thus, mothers' culturally-defined, sex-appropriate responsive parenting towards girls may be a protective factor. On the other hand, boys who elicit "culturally inappropriate" high levels of responsiveness may have characteristics which indicate vulnerability or need of protection from their mothers, which may be a forerunner of emotional and behavioral problems later on. Additional research is needed to explore whether this sex difference can be replicated in other samples.

Goodness-of-fit between child and context. Results of the current study indicated that early temperamental traits exerted strong independent effects on the development of later internalizing and externalizing problems, whereas parenting effects were significant largely in their interaction with child temperament traits. Taken together, our results suggest that children's temperament affects child outcomes directly and also interactively, by strengthening the nature of the associations between temperament and parenting on developmental outcomes (Belsky & Pluess, 2009). Numerous theorists have discussed the importance of goodness-of-fit, or compatibility between a child's temperament and the parenting styles they are exposed to in predicting children's emotional and behavioral problems (Belsky & Pluess, 2009; Lengua & Kovacs, 2005, Leve et al., 2005). These researchers have often found that infants and young children with difficult temperaments (i.e., those that are easily distressed, highly reactive, or fearful) are particularly susceptible to the effects of negative parenting (Crockenberg & Leerkes, 2003; Morgan et al, 2012; Pluess & Belsky, 2010). Consistent with these results, we also found evidence for "dual risk" effects such that children with relatively high levels of negative emotionality and low levels of sociability had poor developmental outcomes (i.e., more internalizing problems), but even more so when exposed to negative parenting (e.g., low responsiveness and harsh discipline). These results extend previous findings, which have primarily focused on cross-sectional data, by demonstrating that parent x child interactions have relatively long-lasting effects such that they predict future levels of internalizing and externalizing problems.

Recent research has suggested that not only may temperamentally vulnerable children be most adversely affected by negative parenting, they may also reap the most benefit from positive parenting and other environmental supports (differential susceptibility model; Belsky & Pluess, 2009; Pluess & Belsky, 2010). Although we did not find direct support for this hypothesis, our results did indicate that children with lower levels of negative emotionality had low levels of externalizing problems, but these children had even fewer problems when exposed to positive parenting (e.g., low levels of harsh discipline). Such a result may indicate a "dual benefit" effect (Poehlmann et al., 2011) that warrants additional research within the differential susceptibility framework.

Strengths and Limitations

The study had some notable and unique strengths. First, few studies have used both children's early temperamental characteristics and environmental factors to predict adolescent outcomes using multiple methods and done so within a prospective, long-term time frame. The use of an observed measure of parenting and temperament (along with both mother and father ratings of temperament) reduced mother-reporter bias and common method variance, which are often limitations within this research literature. In addition, we used flexible modeling techniques that not only estimate growth patterns in internalizing and externalizing problem behaviors simultaneously, but also determine person-level factors that influence growth patterns. By examining these processes in a multivariate model, we were able to examine the bidirectional relations between internalizing and externalizing problems, as well as the common and specific risk factors influencing their development. To our knowledge, no other study has used latent growth modeling to examine the effects of temperament and family factors on changes in both internalizing and externalizing problems from early and middle-childhood through mid-adolescence.

Despite these strengths, the study also has limitations. First, although designed to identify both reciprocal and unidirectional effects over time, our analyses remain correlational. Because correlational designs can only suggest causality, an experimental design would be necessary to confirm that change in either child temperament or parent behavior during the periods identified in the present study will be associated with future behavior problems. The present study is not so comprehensive as to involve all plausible risk factors that are involved in the development of internalizing and externalizing problems over time, reflected in the fact that the set of risk factors

identified in the present study explained, on average, approximately one quarter to one half of the variance in internalizing and externalizing problems. Further research on mediating risk factors (e.g., peer relations, cognitive styles, environmental stress, pubertal timing; Mesman & Koot, 2000) would provide a deeper understanding of the developmental mechanisms of comorbidity. Another limitation of the present design was the relatively small sample of mother-child dyads eligible for inclusion. Power to detect significant interactive effects may have been compromised by having a relatively smaller sample size. In addition, we did not find statistically significant variation in the rate of growth among internalizing and externalizing trajectories, which limited our ability to detect statistically significant predictors of change in behavioural trajectories. Inclusion of a larger sample of youth with clinically elevated symptomatology may have enhanced the power to detect such effects.

Conclusions and Implications

In integrating two competing models of co-occurrence, results from the present study demonstrate that the developmental mechanisms of internalizing and externalizing problems are best understood when their co-development is considered, along with the risk factors that underlie their co-occurrence. This study has important implications for clinical assessments and interventions with youth and their families. In particular, temperament and parenting seem to be the most important target variables for prevention and intervention efforts because these were most consistently related to both adolescent internalizing and externalizing psychopathology. Temperament is a very powerful predictor of children's internalizing and externalizing problems, with long-term effects on children's behaviour trajectories. The fact that some child temperament factors had selective impact on the two domains of behavior problems has relevance for prevention and intervention. Finally, the impact of temperament on internalizing and externalizing behavior problems also depended on the quality of parenting received. These results suggest that clinicians must address child characteristics, parenting style, and their goodness of fit, in order to successfully reduce childhood emotional and behavioral problems.

Table 1

Correlation Matrix and Descriptive Statistics

Variable	7-INT	10-INT	13-INT	16-INT	7-EXT	10-EXT	13-EXT	16-EXT	Gender	Emo	Act	Soc	SES	Dep	Harsh	Resp
1. Age 7 INT																
2. Age 10 INT	**0.40**															
3. Age 13 INT	**0.26**	**0.55**														
4. Age 16 INT	**0.49**	**0.48**	**0.68**													
5. Age 7 EXT	**0.78**	**0.27**	**0.18**	**0.36**												
6. Age 10 EXT	**0.30**	**0.47**	**0.35**	**0.29**	**0.56**											
7. Age 13 EXT	**0.27**	**0.29**	**0.50**	**0.29**	**0.46**	**0.76**										
8. Age 16 EXT	**0.35**	**0.26**	**0.25**	**0.38**	**0.60**	**0.70**	**0.71**									
9. Gender (female)	-0.11	-0.12	0.05	0.08	**-0.27**	**-0.32**	**-0.21**	**-0.23**								
10. Emotionality	0.16	**0.29**	**0.23**	**0.28**	**0.21**	**0.27**	0.14	**0.25**	-0.12							
11. Activity	0.07	**-0.20**	**-0.18**	-0.13	**0.37**	**0.19**	0.11	**0.27**	**-0.21**	0.10						
12. Sociability	-0.06	-0.14	-0.12	0.06	0.09	0.04	-0.00	0.13	0.01	**0.16**	**0.47**					
13. SES	**-0.17**	-0.10	-0.11	-0.14	-0.09	-0.11	-0.14	-0.12	-0.05	0.01	0.02	0.02				
14. Depression	**0.19**	**0.35**	**0.31**	**0.31**	0.10	0.13	0.15	0.12	-0.01	**0.25**	-0.05	0.09	-0.12			
15. Harsh discipline	0.02	0.14	0.15	0.10	0.11	0.18	0.09	**0.17**	-0.02	**0.30**	0.07	0.03	-0.03	0.13		
16. Responsiveness	-0.04	0.05	0.00	0.02	-0.02	0.01	-0.06	-0.11	0.12	0.05	-0.03	0.10	0.12	-0.01	-0.04	
Mean	8.80	9.28	7.52	6.93	12.03	10.56	8.31	7.93	1.54	0.01	0.00	0.01	38.69	55.09	-0.02	0.0
SD	8.86	6.98	6.03	5.59	8.97	7.99	7.75	7.05	0.50	0.71	0.73	0.70	11.07	9.77	1.04	0.9

Note. INT = internalizing problems; EXT = externalizing problems; Emo = negative emotionality; Act = activity; Soc = Sociability; Dep = maternal depression; Harsh = harsh discipline, Resp = maternal responsiveness. Statistically significant values at $p < .05$ are marked in **bold**.

Table 2

Internalizing and Externalizing Problems Raw and T-scores, Separated by Sex

	Boys			
	Internalizing mean (*SD*)		Externalizing mean (*SD*)	
	Raw scores	T-scores	Raw scores	T-scores
Age 7	9.73 (9.03)	55.78 (11.39)	14.67 (9.43)	55.60 (9.94)
Age 10	9.81 (7.41)	56.86 (10.77)	13.35 (9.59)	53.67 (10.98)
Age 13	7.11 (5.89)	52.83 (9.99)	9.97 (9.69)	53.03 (10.35)
Age 16	6.69 (5.21)	51.92 (9.26)	9.10 (8.25)	50.28 (7.82)
	Girls			
	Internalizing mean (*SD*)		Externalizing mean (*SD*)	
	Raw scores	T-scores	Raw scores	T-scores
Age 7	7.67 (8.76)	52.10 (10.95)	9.55 (8.02)	51.73 (9.70)
Age 10	8.50 (6.43)	53.77 (10.10)	8.28 (5.73)	50.52 (8.80)
Age 13	7.66 (6.18)	52.24 (9.75)	6.39 (4.97)	50.86 (8.40)
Age 16	7.42 (6.09)	51.22 (9.39)	6.12 (4.58)	48.96 (9.37)

Note. Boys $N = 71$; Girls $N = 83$.

Table 3

Parameter Estimates for Unconditional Models of Internalizing and Externalizing Problems

	(*N* = 154)	
Latent Factor Means (SE)	Mean	(S.E.)
INT intercept	9.26**	(0.69)
INT slope	-0.81**	(0.27)
EXT intercept	12.28**	(0.75)
EXT slope	-1.62**	(0.27)
Covariance of Latent Factors	*B*	(S.E.)
INT-I with EXT-I	0.64**	(0.10)
INT-I with INT-S	-0.44*	(0.19)
INT-I with EXT-S	-0.25	(0.31)
EXT-I with EXT-S	-0.39	(0.24)
EXT-I with INT-S	-0.32*	(0.15)
INT-S with EXT-S	-0.03	(0.74)

Note. *B* = standardized coefficient. INT = internalizing problems; EXT = externalizing problems; I = intercept; S = slope. *p<.05 **p <.01. $\chi 2(17)=20.278$, p=.26; CFI=0.992; RMSEA=.035; SRMR=0.056.

Table 4

Parameter Estimates for Mother-Rated Models of Internalizing and Externalizing Problems with Predictors

	INT intercept		INT slope		EXT intercept		EXT slope	
	b (S.E.)	*B*	*b* (S.E.)	*B*	*b* (S.E.)	*B*	*b* (S.E.)	*B*
Gender	-1.33 (1.16)	-0.13	0.66 (0.41)	0.25	-3.55 (1.25)**	-0.27**	0.51 (0.45)	0.19
Emotionality	2.15 (0.85)*	0.31*	-0.17 (0.37)	-0.10	1.93 (0.82)*	0.21*	-0.12 (0.33)	-0.06
Activity	0.59 (0.91)	0.09	-0.78 (0.39)*	-0.44*	3.59 (0.97)**	0.41**	-0.76 (0.40)	-0.41
Sociability	-2.48 (1.04)*	-0.35*	1.02 (0.42)*	0.55*	-1.76 (1.04)	-0.19	0.63 (0.36)	0.32
SES	-0.10 (0.05)	-0.21	0.02 (0.02)	0.18	-0.06 (0.05)	-0.10	-0.00 (0.02)	-0.03
Depression	0.15 (0.06)*	0.30*	-0.00 (0.03)	-0.02	0.08 (0.06)	0.13	-0.01 (0.02)	-0.07
Harsh discipline	0.23 (0.57)	0.05	-0.01 (0.25)	-0.01	0.81 (0.51)	0.13	-0.14 (0.19)	-0.10
Responsiveness	0.36 (0.66)	0.07	-0.12 (0.22)	-0.09	0.60 (0.71)	0.09	-0.34 (0.24)	-0.24
Step 2								
Emotionality X Harsh discipline	-0.67 (0.57)	-0.14	-0.09 (0.25)	-0.07	-1.20 (0.57)*	-0.19*	0.18 (0.21)	0.14
Activity X Harsh discipline	-0.21 (0.87)	-0.03	0.28 (0.39)	0.16	0.26 (0.72)	0.03	0.32 (0.32)	0.17
Sociability X Harsh discipline	-2.78 (1.28)*	-0.29*	0.85 (0.53)	0.34	-2.07 (1.25)	-0.17	1.04 (0.47)*	0.39*
Emotionality X Responsiveness	-1.94 (1.00)*	-0.28*	0.73 (0.34)*	0.38*	-0.89 (0.97)	-0.10	0.43 (0.32)	0.23
Activity X Responsiveness	-0.012 (1.07)	-0.00	0.06 (0.41)	0.03	-0.15 (1.02)	-0.15	1.09 (0.42)**	0.48**
Sociability X Responsiveness	-0.97 (1.16)	-0.12	0.74 (0.40)	0.34	-1.80 (1.07)	-0.17	1.02 (0.44)*	0.46*

Note. *b* = unstandardized coefficient; *B* = standardized coefficient. INT = internalizing problems; EXT = externalizing problems. *p<.05 **p <.01.

$\chi 2(56)=61.164$, $p=.30$; CFI=0.990; RMSEA=.024; SRMR=0.051.

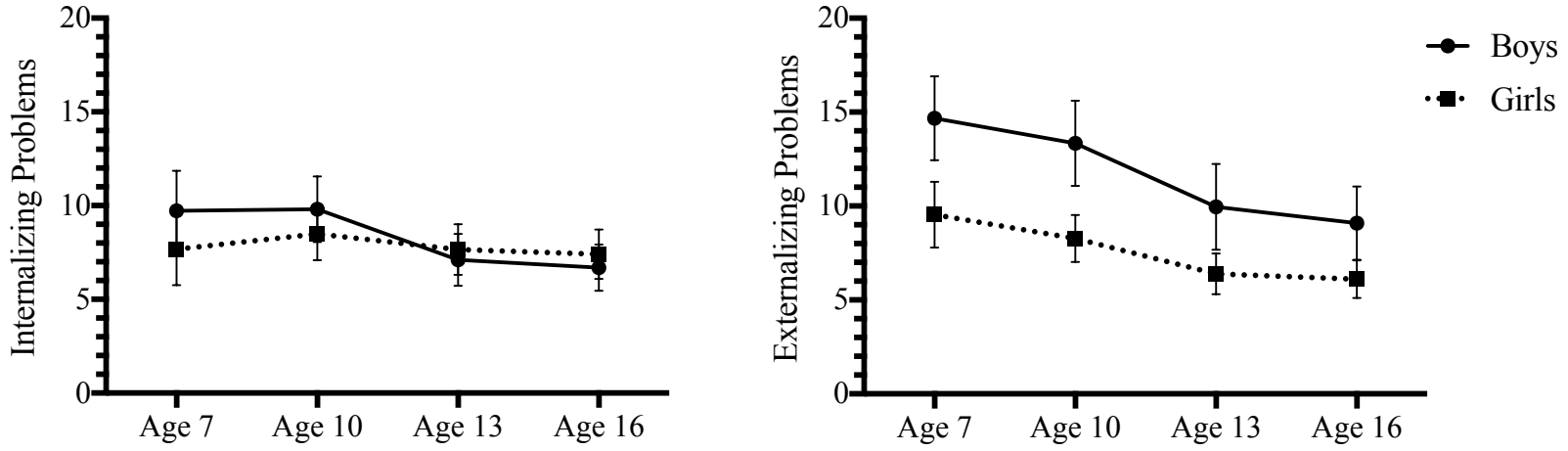

Figure 1. Estimated means and 95% confidence intervals for mother-rated internalizing and externalizing problems from age 7 to 16.

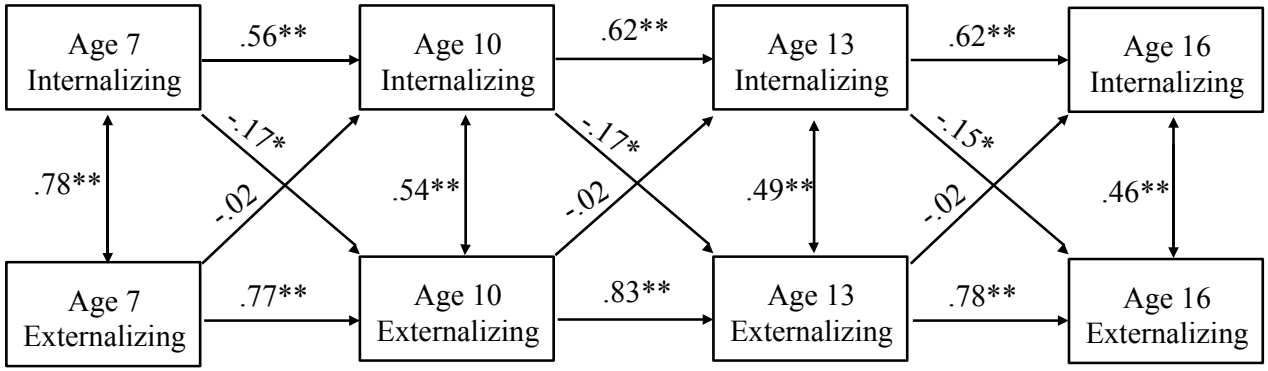

Figure 2. Standardized path coefficients for the reciprocal effects between children's mother-reported internalizing and externalizing problems, without predictors. *p<.05 **p <.01. $\chi 2$ (20)=42.14, *p*=.01, RMSEA=.08, SRMR=.11. *N*=154.

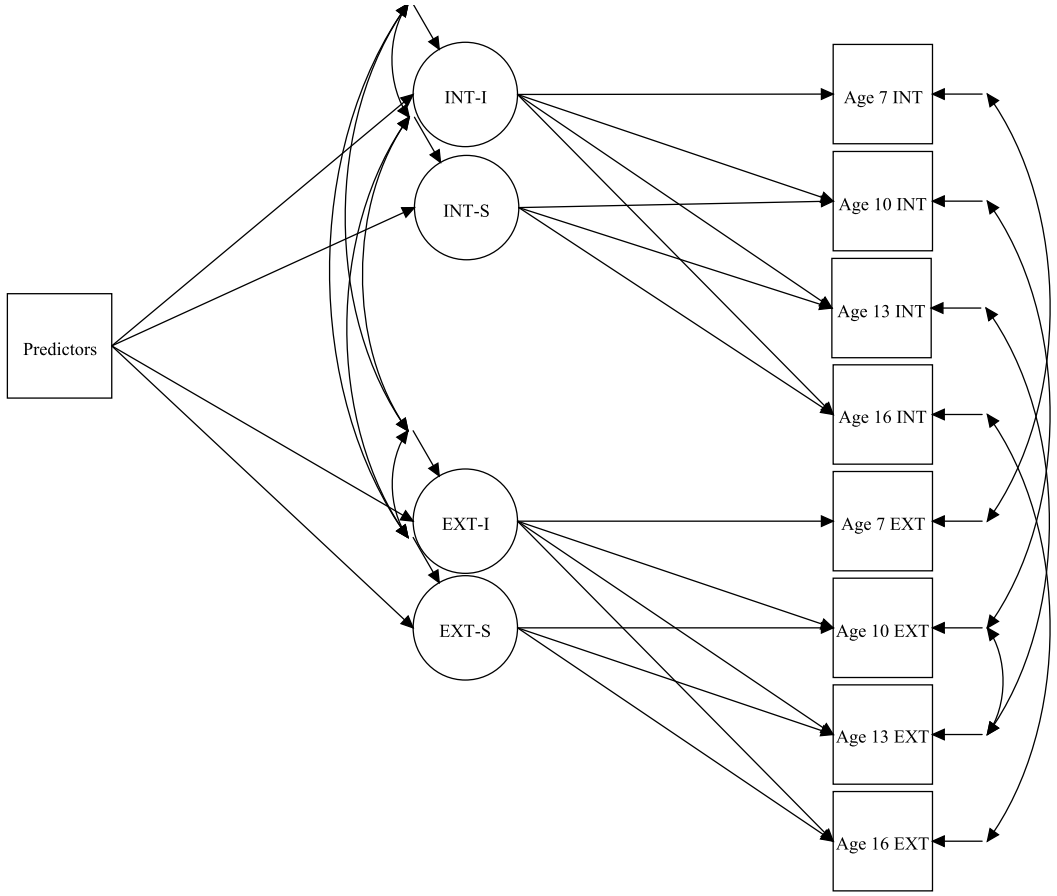

Figure 3. Path diagram illustrating the parallel process latent growth model for ages 7 to 16, including slopes and intercepts. INT = internalizing problems; EXT = externalizing problems; I = intercept; S = slope.

CHAPTER 3: DISCUSSION OF STUDY 1 AND RATIONALE FOR STUDY 2

Results from Study 1 contributed to the current literature by expanding our understanding of the sequential relations between internalizing and externalizing problems. The associations between internalizing and externalizing problems across time provided evidence of how common risk factors predict their co-development, and also how unique risk factors predict their differentiation. Extending the findings and methodology employed in Study 1, Study 2 was designed to focus on how comorbidity within a diagnostic class (i.e., anxiety and depression) develops over time. It has also been suggested that research should consider the degree to which the covariation in symptoms reflects methodological artifacts, rather than the presence of true comorbidity. To this end, in addition to examining sequential patterns between these expressions of psychopathology (controlling for baseline symptoms in each domain) and their common and unique risk factors, Study 2 was designed to extend Study 1 by exploring the whether methodological factors contributed to comorbidity. Specifically, it was examined whether the overlap in symptomatology among questionnaires assessing anxiety and depressive symptoms contributed to their longitudinal associations. Additionally, Study 2 aimed to test how patterns of comorbidity and the identified common and unique risk factors for disorders varied across symptom versus diagnostic outcomes.

While much of the current child psychopathology literature focuses on maternal ratings of temperament, parenting, and psychopathology, the present studies utilized multi-method, multi-informant ratings to reduce the bias that results from shared-rater variance. Both Study 1 and Study 2 utilized multi-method measures of temperament (observational data, mother and father ratings), assessed at age 3. Study 1 focused on internalizing and externalizing outcomes, based on maternal ratings of child behaviour at ages 7, 10, 13, and 16. Study 2 focused on anxiety and depression outcomes at ages 10, 13, and 16. Given that youth show the ability to reflect on their internalizing states during the late-childhood years (Myers & Winters, 2002), self-ratings and diagnostic interviews with youth were incorporated in the assessment procedures in Study 2. Although different parenting measures were used in Studies 1 and 2 (relevant parenting measures were selected based on the age and developmental level of the children), both studies utilized observed measures of parenting. These parenting measures were intended to assess mothers' levels of warmth/responsiveness (Study 1: HOME Inventory; responsiveness; Study 2: EAS scales; sensitivity, nonhostility) and control/restrictiveness (Study 1: HOME

Inventory; avoidance of restriction and punishment; Study 2: EAS scales; structuring), which tap key aspects of the authoritative parenting style that has been consistently linked to positive child mental health outcomes (Chaudhuri, Easterbrooks, & Davis, 2009).

Using a comprehensive, in-depth approach, Study 2 was designed to address gaps in the literature by extending our understanding of the development of comorbidity within a diagnostic class (i.e., internalizing disorders). Using innovative methodologies and statistical applications, Study 2 was designed to provide more detail on the potential role of methodological explanations of co-occurrence, as well as how patterns of co-occurrence and associated risk factors varied across symptom and diagnostic outcomes.

CHAPTER 4: STUDY 2

Longitudinal Associations Between Anxiety and Depression in Youth: A Manifestation of Underlying Temperament?

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Abstract

Anxiety and depression frequently co-occur in children and adolescents, with the onset of anxiety symptoms commonly preceding depression. It is unknown whether: 1) anxiety is a risk factor for later depression; 2) anxiety and depression co-occur over time because of the high degree of symptom overlap; or 3) the association between early anxiety and later depression is explained by a shared underlying temperament. These competing explanations were evaluated using data from a longitudinal, community-based study. Youth's symptomatology was assessed at 3 time-points (ages 10, 13, 16), using questionnaire-based measures and semi-structured diagnostic interviews. Results revealed that changes in depressive symptoms were most reliably and strongly predicted by depressive symptoms measured in the previous wave. Changes in anxiety and depression symptoms were not predicted by changes in the other domain, when using measures with discriminant validity. Instead, preschool temperament characterized by low positive emotionality and high negative emotionality predicted the development of anxiety and depression, controlling for the effects of family income, gender, parental history of internalizing disorders, and parenting quality. Gender differences in pathways to anxiety and depression were found, with gender moderating the impact of parenting on internalizing symptomatology. Implications for intervention and prevention are discussed.

Longitudinal Associations Between Anxiety and Depression in Youth: A Manifestation of Underlying Temperament?

The comorbidity between anxiety disorders and depression is one the most frequently occurring comorbidities in developmental psychopathology. In epidemiological and clinic-referred samples, between 15% and 75% of youth identified as anxious or depressed report experiencing comorbid internalizing disorders (Angold et al., 1999; Avenevoli, Stolar, Dierker, & Merikangas, 2001; Costello et al., 2003). At the symptom level, anxiety and depression also show a high degree of overlap, as evidenced by high inter-correlations on common self-report measures of anxiety and depression across diverse populations (e.g., *r*'s=.50 - .70; Brady & Kendall, 1992; Stark and Laurent, 2001). Experiencing comorbid anxiety and depression predicts greater symptom severity, poorer response to treatment and a more chronic and severe course of illness compared to experiencing anxiety or depression alone (Ollendick, Jarrett, Grills-Taquechel, Hovey, & Wolff, 2008; Starr & Davila, 2008).

Several theoretical models (some methodological, some substantive) have been proposed as explanations for the high co-occurrence between anxiety and depression in youth: (1) anxiety directly causes or puts youth at risk for future depression; (2) comorbidity is due to an overlap in symptoms and items used to assess these different disorders; and (3) common etiological risk factors lead to the development of both disorders (e.g., Avenevoli et al., 2001; Cummings, Caporino, & Kendall, 2014; Mathew, Pettit, Lewinsohn, Seeley, & Roberts, 2011; Seligman & Ollendick, 1998). However, few longitudinal studies have explicitly tested these different models in order to understand how anxiety and depression develop over time in childhood and adolescence (Cohen, Young, Gibb, Hankin, & Abela 2014). A developmental psychopathology perspective (Cicchetti & Toth, 2009), which attempts to identify the multiple developmental pathways, precursors, and protective factors that lead to development of depression and anxiety may be particularly helpful for illuminating the etiological processes that lead to comorbidity (Cummings et al., 2014).

An area of intense speculation is whether there is a temporal or causal link between pediatric anxiety and depression. In reviewing research on the temporal sequence of anxiety and depression, clinical and epidemiological studies have consistently shown that the mean age of onset of anxiety disorders is earlier than that of depressive disorders and that anxiety precedes depression in most cases of comorbidity (Ollendick, Shortt, & Sander, 2005; Pine, Cohen,

Gurley, Brook, & Ma, 1998; Wittchen, Kessler, Pfister, Höfler, & Lieb, 2000). This phenomenon seems to be especially true for adolescent girls (Keenan & Hipwell, 2005), who show elevated levels of anxiety relative to boys, beginning in early childhood, and who are at a two- to three-fold increased risk for developing depression during the adolescent period (Zahn-Waxler et al., 2008). These findings have led some researchers to suggest that anxiety is causally implicated in the development of depression, especially among girls (Avenevoli et al., 2001; Fichter, Quadflieg, Fischer, & Kohlboeck, 2010). Consequently, clinicians and researchers have proposed that treating childhood anxiety disorders could prevent the development of depression in adolescence and adulthood (Flannery-Shroeder, 2006).

However, several methodological issues might explain the apparent link between earlier anxiety symptoms and later depression. First, sub-threshold levels of symptoms are not commonly assessed or reported in studies of comorbidity, so elevations in risk may simply be caused by the high correlation between anxiety and depression. That is, children diagnosed with anxiety disorders may have concurrent depressive symptoms, and these co-occurring, sub-threshold symptoms may account for the apparent link between anxiety and subsequent depressive disorder in adolescence (Zahn-Waxler, Klimes-Dougan & Slatterty, 2000). Since comorbidity rates are likely to be underestimated when diagnostic data are solely used, research on this topic requires the integration of a dimensional approach to symptom assessment.

Second, it is important for studies that test hypotheses about the predictive validity of anxiety and depressive symptoms to ensure that measures employed have sufficient discriminant validity (Stark & Laurent, 2001). Although self-report measures of anxiety and depression are generally good at identifying anxious versus non-anxious children and depressed versus non-depressed youth, respectively, these instruments are not very sensitive to differences among types of internalizing disorders (Brady & Kendall, 1992; Seligman, Ollendick, Langley, & Baldacci, 2004). Relatedly, some of the most widely used, well-established measures of anxiety and depression symptom assessment in youth include overlapping or even identical items (Cummings et al., 2014; Stark & Laurent, 2001), making their usefulness in predicting one another over time questionable.

Most studies of sequential comorbidity have focused on anxiety as a predictor and depression as an outcome, rather than the reverse (e.g., Bittner et al., 2007). To our knowledge, only five longitudinal studies to date have tested the hypothesis that anxiety predicts future

depression in youth using balanced designs that control for prior symptoms in each domain (Cohen et al., 2014; Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Keenan, Feng, Hipwell, & Klostermann, 2009; Lavigne, Hopkins, Gouze, & Bryant, 2015; Snyder et al., 2009). This work has produced mixed results, with some studies reporting that anxiety predicts future depression, but not vice versa (Cole et al., 1998; Snyder et al., 2009), one study reporting that anxiety and depression are associated with higher levels of one another in subsequent years (Lavigne et al., 2015) and others reporting that anxiety and depression most strongly predict themselves over time (Cohen et al., 2014; Keenan et al., 2009; Snyder et al., 2009).

It is unclear whether these inconsistent findings depend on the discriminant validity of questionnaires employed in these studies or the raters involved. For example, Cole and colleagues (1998) and Lavigne and colleagues (2015) found cross-lagged associations between anxiety and depressive symptoms, but did not use questionnaires with discriminant validity. Synder and colleagues (2009) reported cross-lagged associations between anxiety and depression for teacher's (but not mother's) reports. Previous work has shown that informants show, at best, only modest levels of agreement regarding youth's internalizing problems (De Los Reyes & Kazdin, 2005; Duhig, Renk, Epstein, & Phares, 2000) and that integrating information from multiple informants can lead to different conclusions regarding the correlates or risk factors for these problems (De Los Reyes & Kazdin, 2005). Thus, more research is required to understand whether the patterns of co-occurrence depend on whether data is obtained from self, parent, or teacher ratings.

Another important (substantive) explanation for the high rate of comorbidity is that anxiety and depression are associated because they share common etiological influences (Kendler, Gardner, Gatz, & Pedersen, 2007; Mathew et al., 2011; Rice, van den Bree, & Thapar, 2004). Evidence supporting the shared etiology hypothesis includes twin and family studies showing that a large proportion of the overlap in anxiety and depression is due to common genetic disposition (Kendler et al., 2007; Middeldorp, Cath, Van Dyck, & Boomsma, 2005; Rice et al., 2004). Several researchers have suggested that the overlap between anxious and depressive symptoms may be rooted in biologically-based, temperamental differences in positive and negative emotionality (Watson, Gamez, & Simms, 2005). Positive emotionality refers to approach behaviors such as high-intensity pleasure, smiling and laughter, activity, and affiliation and maps closely onto the personality construct of extraversion. Negative emotionality comprises

dimensions of anger, frustration, emotional intensity, difficultness, and fear and is closely linked to the personality construct of neuroticism (Caspi & Shiner, 2006). Extensive evidence in the mood and anxiety literature shows that high levels of negative emotionality are a risk factor for the development of both mood and anxiety disorders (tripartite model; Clark & Watson 1991; Lonigan, Phillips, & Hooes, 2003; Steer, Clark, Kumar, & Beck, 2008). Low levels of positive emotionality are posited to distinguish depression from anxiety. However, recent research indicates that some anxiety disorders, including generalized anxiety disorder and social anxiety disorder, are also characterized by low levels of positive emotionality (Watson et al., 2005). Most research in this area has failed to employ a multivariate perspective of the temperament-psychopathology relation, thus little is known how the combination of positive and negative emotionality predict comorbid psychopathologies (Tackett, 2006), especially within a prospective longitudinal framework.

In investigating these three potential pathways, it is important to consider how other individual and environmental characteristics may promote or inhibit the expression of anxiety and depressive disorders. Although genetic influences are likely to play a significant role in the common etiology of anxiety and depression, a great deal of research also implicates parenting behaviors in the intergenerational transmission of depression and anxiety (McLeod, Weisz, & Wood, 2007; McLeod, Wood, & Weisz, 2007). The quality of parenting and the parent-child relationship are widely regarded by developmental psychopathologists as playing an important role in the development and maintenance of childhood anxiety and depression (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000). For example, attachment theory emphasizes that early caregivers help establish patterns of emotion regulation via sensitive responsiveness to the child's emotional cues (Sroufe, Carlson, Levy, & Egeland, 1999). Parenting behaviors that help children learn how to develop emotion regulation capacities (e.g., parental emotional availability, including high parental warmth and sensitivity, and low hostility) have been found to be associated with children's ability to regulate affect and manage stress (Yap et al., 2007). Both anxious and depressed mothers tend to show less warmth and sensitivity and more control and hostility toward their children (Goodman et al., 2011; McLeod, Weisz, & Wood, 2007; McLeod, Wood, & Weisz, 2007). Thus, the quality of parenting and parental history of internalizing disorders represent important risk factors which may influence the co-development or differential expression of anxiety and depression over time.

The Present Study

The present study compared three alternative hypotheses that may explain the high degree of co-occurrence between anxiety and depression in youth: 1) the experience of anxiety increases risk for later depression; 2) anxiety and depression are prospectively related due to overlap in measurement of symptoms; and 3) early anxiety and later depressive symptoms are associated because of shared etiological risk factors (e.g., temperament). To test these competing (but non-mutually exclusive) hypotheses, we compared the extent to which anxiety and depression symptoms predicted the occurrence of future symptoms, as assessed by both children and their mothers, across 3 waves of data collection (9 years), spanning from pre-adolescence to late adolescence among a community sample of children and families. We employed an autoregressive cross-lagged panel model to test these hypotheses, which allows for the examination of both the stability of symptoms and the predictive association between two variables over time, each controlling for effects at earlier time points (Selig & Little, 2011). We modeled the temporal relation between anxiety and depression using some of the most widely used children's self-report and parent-report measures of childhood anxiety and depression symptoms, as well as revised scoring methods for these scales, which show discriminant validity for differentiating between anxiety and depression. This allowed us to test the hypothesis that item overlap on the questionnaire measures may be responsible for the previously stated finding that early anxiety is related later depression. In addition, we were able to examine whether effects differed according to the child's or parent's perspective of symptoms. Finally, we included data from both boys and girls and tested whether gender moderated the anxiety-depression relation or other study effects.

To address these questions, the following hypotheses were proposed. First, item overlap on common self-report questionnaires were expected to account for the previously stated finding that early anxiety is related to later depression. Second, preschool temperament characterized by low positive emotionality and high negative emotionality was expected to predict the parallel development of anxiety and depressive symptoms and comorbid diagnoses of anxiety and depressive disorders in adolescence, above and beyond the effects of established predictors of internalizing problems, including family income, parental internalizing diagnoses and maternal emotional availability. It is relatively rare for a single study on this topic to examine the influence of parental diagnoses, family interaction patterns, and child temperament on the co-

development of anxiety and depression. Thus, we did not make any specific predictions about whether these other contextual factors would differentially promote or inhibit the development of anxiety and depression. Finally, we did not have specific predictions regarding whether study effects would vary according to child gender or source of the informant, given the inconsistent results reported in the literature (reviewed above).

Method

Participants

The participants in the current study are part of the Concordia Longitudinal Risk Project, a large, prospective, intergenerational community-based research project that examines the processes that are associated with positive versus negative social and health outcomes across the life course families from disadvantaged backgrounds (Schwartzman, Ledingham, & Serbin, 1985). The original longitudinal study began in 1976 with the screening of the school populations at 22 inner-city schools serving French-speaking children living in predominantly lower income neighborhoods of Montreal, Quebec. Many of the original participants who have been followed over time have become parents, and smaller subsets of these participants have been selected for follow-up studies, providing opportunities to examine long-term prediction of family functioning and the inter-generational transfer of health and psychosocial risk (Serbin, et al., 2011). The sub-samples used for each of these follow-up studies are representative of the original sample in terms of gender, family SES, neighborhood, and behavioral characteristics in childhood.

The Current Sample. A sub-sample of original participants in the Concordia Project who had preschool aged children during the first wave of the current study were identified and invited to participate in the present study. Children were included in the present analyses if they participated in at least 2 of 4 of the waves of data collection, resulting in a final sample of 143 (64 boys). Approximately 82% of invited families (eligible *N*=175) participated during the period of the current study (see Procedures, below). These 143 families did not differ from those who did not participate (*N*=32) or from the complete sample of families in the Concordia Project (*N*=550; including families with children above or below the target age for the present study) in terms of family income, maternal education, neighborhood disadvantage, rate of single parenthood, child behavior characteristics, or parenting (analyses of representativeness within sample; all p values >.10).

Procedures. When children were preschool aged (*M*=3.63 years, *SD*=1.56), a home visit was conducted. During this home visit, the child's mother, father, and an experimenter completed questionnaires assessing the child's temperament. At Time 2, 3, and 4 children were subsequently assessed for anxiety and depression on questionnaire-based measures, at approximately 3-year intervals (Time 2 of the present study: pre-adolescence: age 9-12, *M*=10.74 years, *SD*=0.77; Time 3: early adolescence: age 12-15; *M*=13.38 years, *SD*=0.84; Time 4: late adolescence: age 15-18; *M*=16.51 years, *SD*=0.88). Time 2 involved another home visit, where mother's emotional availability was observed and coded during dyadic parent-child interactions. At Time 3 and Time 4, youth who rated high on screening measures of anxiety and depression were administered diagnostic interviews. Families and children were compensated with a nominal honorarium. These procedures were approved by the university's Institutional Review Board.

Measures

Self-Report. We assessed youth's self-reported symptoms of depression and anxiety with a French language translation of the Children's Depression Inventory (CDI; Kovacs, 1984) and the Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1985) at Time 2 (age 10), Time 3 (age 13), and Time 4 (age 16). The CDI is a 27-item, self-report instrument that assesses depressed affect, somatic symptoms, depressive behaviour, low self-esteem, and anhedonia (Cronbach's α: Time 2=.83; Time 3=.83; Time 4=.64). Each item consists of three statements scored in order of increasing severity from 0 to 2. Children select one sentence from each group that best describes themselves for the previous 2 weeks. The CDI is one of the most widely used and reliable measures of childhood depression. The RCMAS is a 37-item, self-report measure that assesses anxiety symptoms in children and adolescents, including physiological symptoms, worry, over-sensitivity, social and concentration concerns, and social desirability (Cronbach's α: Time 2=.84; Time 3=.83; Time 4=.86). Each item consists of a statement that children rate as either being true or false of themselves ("yes" or "no"). The RCMAS has demonstrated good convergent and construct validity in child and adolescent populations (Reynolds & Richmond, 1985).

We also calculated scores for the CDI and RCMAS, using revised scoring methods that show good discriminant validity for anxiety and depression (Stark & Laurent, 2001). The revised scores were used to examine our second hypothesis, whether overlapping symptoms within

anxiety and depression scales inflate the correlation between these constructs and their predictive relations over time. Nine items composed the unique 9-item depression scale (items 1, 3, 5, 7, 8, 10, 14, 26, 27), which composed largely of items representing sad affect and a negative view of oneself, and seven items composed the abbreviated anxiety scale (items 2, 10, 14, 22, 25, 29, 37), which assessed worry and nervousness (See Table 1). Each revised scale demonstrated acceptable internal consistency across each time point (CDI-Revised: Cronbach's α: Time 2=.67; Time 3=.77; Time 4=.74; RCMAS-Revised: Cronbach's α: Time 2=.72; Time 3=.70; Time 4=.65).

Mother-Report. At each time point, mothers completed a French language translation of the Child Behavior Checklist (CBCL 4-18/parent form; Achenbach, 1991). Mothers rated how well the items reflected their child, from 0 ("not true") to 2 ("very true or often true"). Responses were scored to calculate the widely used Anxious/Depressed (items 14, 29, 30, 31, 32, 33, 35, 45, 50, 52, 71, 81, 91, 106, 108, 112) and Withdrawn/Depressed (items 5, 42, 65, 69, 75, 102, 103, 111) syndrome scales. In order to examine whether symptom overlap inflates the prospective relations between anxiety and depression, we also used revised scales of the CBCL, which have been developed based on factor analysis and expert consensus to specifically measure anxiety (items 29, 30, 31, 45, 46, 50, 112) and depressive symptoms (items 12, 14, 18, 33, 35, 54, 76, 77, 91, 100, 102, 103; see Table 1). These scales show better discriminant validity than the original Anxious/Depressed and Withdrawn/Depressed syndrome scales (Lengua, Sadowski, Friedrich, & Fisher, 2001). Internal consistency for the widely used syndrome scales (Anxious/Depressed: Cronbach's α: Time 2=.81; Time 3=.77; Time 4=.79; Withdrawn/Depressed: Cronbach's α: Time 2=.64; Time 3=.74.; Time 4=.69) and revised scales (Anxiety scale: Cronbach's α: Time 2=.76; Time 3=.67; Time 4=.77; Depression scale: Cronbach's α: Time 2=.77; Time 3=.73; Time 4=.57) at each time point was generally acceptable.

Diagnostic Outcomes. At Time 3 and 4, children who were rated at or above the borderline clinical range on screening measures for anxiety or depression (above the 94th percentile cut-off) were administered clinical interviews to assess for the presence of internalizing disorders. The Anxiety Disorders Interview Schedule for DSM–IV ADIS Child and Parent Version (ADIS–C/P; Silverman & Albano, 1996) and the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime version (K-SADS-PL; Kaufman, Birmaher, Brent, Rao, & Ryan, 2006) were administered by trained evaluators

(graduate level clinical psychology students) to determine DSM-IV diagnoses of anxiety and depressive disorders. Children and their mothers were interviewed via telephone. Information relevant to current and lifetime diagnoses of anxiety and depression were considered in the analyses, representing a cumulative risk for anxiety and depressive disorders from early childhood through adolescence. Scores ranged from 0 to 1: 0 (no symptoms of anxiety disorder or depression) or 1 (above threshold for diagnosis). Children who did not reach screening cut-offs were considered to be at low risk for an internalizing disorder and were coded as not having a DSM-IV internalizing disorder.

Temperament. Temperament was measured using the Buss and Plomin EAS Temperament Survey (EAS; Buss & Plomin, 1984). It is composed of 20 questions, which are rated on a five-point Likert scale. Questions are groups into three subscales that correspond to temperament dimensions of Emotionality, Activity, and Sociability. Mothers and fathers completed a French language translation of this questionnaire. Experimenters completed a parallel version of this questionnaire after observing the child in his or her home.

Mothers, fathers and experimenters' ratings of children's Emotionality on the EAS were transformed to z-scores and averaged to create a multi-informant scale representing **negative emotionality** (Buss, 1991). Similarly, mother's, father's and experimenter's ratings of children's Sociability and Activity on the EAS were transformed to z-scores and averaged to create a multi-informant scale representing **positive emotionality** (Buss, 1991). As reviewed above, positive emotionality is linked to the personality construct of extraversion and reflects approach behaviors such as pleasure, activity and affiliation. If only one parent rating was available, the scale was calculated using the experimenter's and the available parent's rating. Approximately 98% of mothers and 79% of fathers completed the questionnaires. Within the sample, internal consistency for the negative emotionality scale (Cronbach's α: Mothers=.74; Fathers=.81; Experimenter=.93) and positive emotionality scale (Cronbach's α: Mothers=.66; Fathers=.67; Experimenter=.73) was acceptable. Inter-rater agreement, indexed by the correlation between mothers', fathers', and experimenters' ratings on positive and negative emotionality scales was modest, with correlations ranging from .14 to .31. This level of inter-rater agreement is similar to what is reported in the literature (e.g., Seifer, Sameroff, Dickstein, Schiller, & Hayden, 2004) and highlights the need for a multi-informant approach. Positive and negative emotionality were modestly correlated (r=.16, p =.06) but were not differentially related to symptoms of anxiety or

depression at each time point. A temperament variable was created by subtracting the positive emotionality scale score from the negative emotionality scale score. Higher values on this temperament variable reflect high levels of negative emotionality and low levels of positive emotionality, which is hypothesized to represent shared risk for anxiety and depression (Watson et al., 2005).

Emotional Availability. At Time 2 (age 10), mothers and their children engaged in a 4-min Jenga task (a game whereby players take turns removing a block from a tower and balancing it on top). Dyadic interactions were videotaped and coded for maternal sensitivity, structuring, hostility, and child responsiveness and involvement on a 5- to 9-point scale using the Emotional Availability Scales Middle Childhood Version (Biringen, Robinson, & Emde, 1993). For the purposes of the current paper, we focused on the ratings of maternal behaviour (i.e., maternal sensitivity, structuring, and hostility). Note that, although the EA dimension is nonhostility, we inverted the scores and used the label of "hostility" (Stack et al., 2012). To assess inter-rater reliability, 25% of the sample were randomly selected and double-coded by trained coders who were blind to the study's hypotheses. Intraclass reliability coefficients revealed highly satisfactory levels for all EA Scales (*r*'s=.87–.97). These dimensions were used to create a factor representing maternal emotional availability. Principle Axis Factor (PAF) extracted one factor with an Eigenvalue of 2.13, which explained 71% of the variance. Sensitivity and structuring loaded highly and positively on the factor (with factor loadings of .94 and .82, respectively) while hostility loaded negatively on the factor (with a factor loading of -.51).

Parental Mental Health. A psychologist conducted the Structured Clinical Interview for DSM-IV (SCID-IV; First et al., 1995) with the parent who was the participant in the original study. Information relevant to the lifetime diagnoses of anxiety and depressive disorders were considered in the analyses. Scores ranged from 1 to 3: 1 (no symptoms of anxiety disorder or depression), 2 (some symptoms but below diagnostic threshold) or 3 (above threshold for diagnosis).

Child's Gender. Given that gender is often a predictor of anxiety and depression (Zahn-Waxler et al., 2008), as well as temperament (Else-Quest et al., 2006), gender was also included as a predictor in all models.

Child's Age. Because the age of children at each measurement point varied (e.g., children were between age 9 and 12 at Time 2) and because intervals between measurement

points were also somewhat variable (3 years was average, but there was variability due to availability and other practical issues such as summer holidays or family illness) child age was included as a time-varying covariate (i.e., age was controlled at each wave).

Family Income. Yearly family income was included as a control in all models.

Analytic Strategy

Analyses were conducted using an autoregressive cross-lagged (ARCL) panel model approach (Selig & Little, 2011), a model which allows for simultaneous examination of longitudinal influences of one construct on another, and vice versa, while also controlling for contemporaneous associations between constructs, and the stability of each construct over time. In all of the tested models, we allowed the residuals of anxiety and depression to covary within each time period. To enhance model parsimony, the magnitude of similar path coefficients were set to be equal over the three measurement points (Kline, 2005). The hypothesized model is presented in Figure 1. All models were estimated via Mplus Version 7 (Muthén & Muthén, 2012). Unlike traditional regression analysis, SEM allows for the estimation of multiple indirect effects. An effects decomposition of the indirect effects of temperament on the development of anxiety and depression was produced using the MODEL INDIRECT command of MPlus, such that we could determine the pathways through which temperament was linked to the development of anxiety and depression.

Missing Data. All available observations were included in the analysis, using the Full Information Maximum Likelihood (FIML) approach of Mplus, which is a robust estimation method when data are missing at random or completely at random (Enders & Bandalos, 2001). In the present study, the amount of missing data for the depression and anxiety variables of interest ranged between 11% and 45% (M=25.8%), although the majority (over 76%) of participants completed assessment measures on at least 3 of 4 waves of data collection. Little's missing completely at random test showed that data were missing completely at random $\chi 2$ (848)=912.54, p=.06, suggesting that the use of FIML for our analyses would not produce biased parameter estimates (Enders & Bandalos, 2001).

Testing Model Fit. Model fit was assessed by examining the chi-square statistic test, with good-fitting models yielding non-significant chi-square goodness-of-fit tests. Because the chi-square index is highly sensitive to sample size, we considered three other fit indexes, which are relatively independent of sample size, in conjunction with the chi-square statistic, including:

the comparative fit index (CFI); the root mean square error of approximation (RMSEA) and the standardized root mean square residual (SRMR). CFI values greater than .95 indicate good fit, and values between .90 and .95 indicate adequate fit. Generally, RMSEA valued less than .05 are considered to indicate good fit, values between .05 and .08 indicate adequate fit, and values larger than .10 indicate poor fit. SRMR values range from 0 to 1.0, with values less than .09 considered adequate (Mueller & Hancock, 2008).

Results

Descriptive Statistics. We first computed means, standard deviations and bivariate correlations for all measures used in the analysis (See Table 1). By the time they had reached adolescence (Time 3 and Time 4), approximately 17% of youth in this sample (8 boys, 12 girls) had experienced one or more anxiety or mood disorders, based on the K-SADS and ADIS-C diagnostic interviews. These rates are consistent with epidemiological studies that cite the cumulative prevalence of emotional disorders for youth aged 9 to 16 years at 15% (17% for girls; 13% for boys; Costello et al., 2003). In our sample, approximately 9% of participants had experienced a depressive disorder, most commonly Major Depressive Disorder, and 13% had experienced an anxiety disorder, most commonly Generalized Anxiety Disorder, Social Phobia and/or Specific Phobia. Of those who met criteria for a mood or anxiety disorder, 30% experienced comorbid anxiety and depressive disorders. Consistent with the literature (Costello et al., 2003), girls were more likely to develop a depressive disorder than boys (9 girls; 2 boys, $t(119)= -1.97$, $p< .05$). There was no statistically significant difference between rates of anxiety disorders in males versus females (9 girls; 7 boys, $t(119)= -0.08$, $p >.05$).

Bivariate correlations between self-report symptoms of anxiety and depression on the revised scales ranged from .20 to .36 at each time point versus a range of .40 to .64 for the total scale scores. Bivariate correlations between mother-rated anxiety and depression on the revised scale ranged from .37 to .57 at each time point versus .53 to .62 on the traditional syndrome scales. These data confirm better discriminant validity of anxiety and depression between the revised scales, as compared to the total scales or traditionally used syndrome scales.

Hypothesis #1: Anxiety Predicts Prospective Depression

We tested the hypothesized model presented in Figure 1 four times. First, we examined children's self-reported and mother-reported symptoms using the total scale scores of widely used questionnaire measures of childhood anxiety and depressive symptoms (children's self-

report: CDI and RCMAS total scales; mother-report: CBCL's Anxious/Depressed and Withdrawn/Depressed scales). Next, we tested the model using revised scoring methods for these questionnaires, which show discriminant validity for assessing anxiety and depression, as described above. Goodness-of-fit statistics revealed that all models showed good fit to the data (see Table 2). Parameter estimates for the four models appear in Table 3. The autoregressive beta weights (β) produced by each model were generally large and statistically significant, suggesting that individual differences between children were stable over time.

In the first set of models (total scale models), the cross-lagged paths were significant at the .05 level and were consistent with the hypothesis that early anxiety is related to later depression (but not vice versa). However, when revised scoring scales were used, which increase the discriminant validity of these measures, the relation between anxiety and later depression was reduced substantially and was no longer statistically significant. Instead, anxiety and depression were the strongest predictors of themselves over time. These findings suggest that overlapping items on anxiety and depression measures may be responsible for the previously stated finding that early anxiety is related to later depression.

Hypothesis #2: Shared Temperament Risk

To test our second hypothesis, that anxiety and depression co-occur due to shared temperamental risk, we included temperament as a time-invariant predictor and examined the indirect effect of T1 temperament on the development of anxiety and depression at T2, T3 and T4, using the revised scoring scales for both youth and mother reports. In these models, we controlled for the effects of yearly family income, parental history of internalizing disorders, and maternal emotional availability (assessed at T2). The results of these models are presented in Figures 2 and 3, respectively. Each model showed excellent fit to the data: youth self-report: $\chi 2$ (43)=43.075, p=.47, CFI=0.999, RMSEA=.003 CI_{95}(.000, .057), SRMR=.065; mother-report: $\chi 2$ (43)=50.473, p=.20, CFI=0.964, RMSEA=.035, CI_{95}(.000, .069), SRMR=.062. As predicted, preschool temperament characterized by high levels of negative emotionality and low levels of positive emotionality was statistically significantly related to both anxiety and depression assessed in pre-adolescence via youth and mother reported data. Low levels of maternal emotional availability predicted greater levels of youth and mother-reported depression, and female sex predicted greater levels of youth self-reported anxiety and depression. In the mother-report model, presence of parental internalizing disorder predicted greater depressive symptoms

in children. Age and family income did not predict anxiety and depression, above and beyond the effects of the other predictors in the model.

We tested for the presence of indirect effects through the MODEL INDIRECT command in Mplus. Analyses revealed that temperament was related to later anxiety and depression through the parallel prediction of anxiety and depression, rather than through cross-lagged links from anxiety to depression or depression to anxiety, in both youth self-report and mother-report models. That is, a statistically significant specific indirect effect of temperament → T2 anxiety → T3 anxiety → T4 anxiety emerged (children's self-report: β=.02, SE=.01, $p< .05$; mother-report: β=.06, SE=.03, $p<.05$), whereas pathways linking temperament and depression through anxiety were not statistically significant (ps > .25). Similarly, with regards to depression, analysis of the specific indirect effects revealed that temperament was most strongly related to T4 depression, directly through T2 and T3 depression (children's self-report: β=.03, SE=.02, $p<$ .05; mother-report: β=.08, SE=.03, $p< .01$). In other words, in both the self-report and mother report data, the effect of temperament on depression was *not* mediated through anxiety, but rather via the continuity in depressive scores across time. Similarly temperament predicted anxiety symptoms through continuity in anxiety scores over time, rather than by mediation through depressive symptoms.

Gender Differences

Preliminary model testing comparing the variance-covariance matrices of boys and girls showed that the baseline model (without predictors) for both youth self-report and mother-report data showed no significant interactions with gender. These models fit equally well for both boys and girls and the regression paths did not vary in magnitude across the groups. These data indicate the sequential relations between anxiety and depressive symptoms are similar for boys and girls.

To examine whether pathways to anxiety and depression were similar for boys and girls, interaction terms involving child gender and each of the predictor variables were simultaneously entered in the model. In predicting mother-rated and youth-rated depressive symptoms and mother-rated anxiety symptoms, interactions involving Parenting x Gender were statistically significant (mother-rated depression: b=-1.65, $p<.01$; youth-rated depression: b=-1.00, $p<.05$; mother-rated anxiety: b=-1.20, $p<.05$). We interpreted the observed interaction effects following suggestions by Aiken, West, and Reno (1991). The results revealed that poor emotional

availability (i.e., 1 SD below the mean) increased the odds of having higher T2 mother-reported anxiety and depressive symptoms, as well as T2 youth-rated depressive symptoms, but only for females. In contrast, among boys, poor emotional availability was not related to mother- or youth-reported anxiety or depressive symptoms. No other statistically significant interactions with gender emerged.

Diagnostic Data

In the final model, we examined whether gender, temperament, yearly family income, parental mental illness, and maternal emotional availability predicted children's anxiety and depression diagnostic outcomes using WLSMV for categorical outcomes in Mplus 7.0. Relatively little is known about how the temperament traits and family factors identified in the present study longitudinally predict diagnostic outcomes of depression and anxiety in youth, accounting for their high comorbidity. Of the 143 study participants, 121 had information relevant to diagnostic data and were included in the present analysis. Model coefficients and standard errors can be found in Table 4. Because Mplus uses the probit function for categorical outcomes (rather than the logit function), odds ratios are not provided. Fit indices suggested that the model fit the data well: $\chi 2(6)=4.160$, $p=.65$, CFI=1.000, RMSEA=.000, $CI_{95}(.000, .095)$, WRMR=.379. Results indicated that temperament characterized by high levels of negative emotionality and low levels of positive emotionality and parental history of an internalizing disorder significantly differentiated youth with a lifetime diagnosis of depression or anxiety from those who never developed an internalizing disorder. Although only marginally significant, girls were more likely to develop a depressive disorder than boys.

We also hypothesized that temperament characterized by low positive emotionality and high negative emotionality would increase risk for diagnostic comorbidity. To test this hypothesis, we conducted a one-way ANOVA testing for temperament differences among differing levels of comorbidity. Temperament differed significantly across the three levels of comorbidity (no disorder, 1 disorder [either an anxiety or depressive disorder], 2 disorders [comorbid anxiety and depressive disorders]), $F(2,120)=6.66$, $p< .01$. Follow-up comparisons of the three groups using the Bonferroni correction indicate that groups with psychopathology (2 disorder group: $M=1.44$, 95% CI [0.21, 2.67] and 1 disorder group: $M=0.74$, 95% CI [-0.08, 1.56]) had significantly higher temperament ratings than the group without a disorder (no disorder: $M=-0.15$, 95% CI [-0.42, 0.10]), p's $< .05$). Together, these findings indicate that

temperament is a powerful predictor of anxiety and depressive diagnoses. In particular, the *combination* of high negative emotionality and low positive emotionality predicted increasing rates of diagnostic comorbidity.

Discussion

It is well established that the concurrent comorbidity of anxiety and depression is substantial at both the diagnostic and symptom level in children and adolescents (Brady & Kendall, 1992; Costello et al., 2003). However, less clear is the extent and direction of sequential comorbidity; that is, when one disorder reliably precedes and predicts the other (Angold et al., 1999). This investigation is the first to use a longitudinal, community-based design to examine three possible explanations of the association between anxiety and depressive symptoms in youth: 1) anxiety increases the risk for later depression; 2) anxiety and depression are prospectively related due to overlap in measurement of symptoms and; 3) a common risk factor, temperament, explains the association between anxiety and depression.

Results of the auto-regressive cross-lagged panel analyses indicate that a causal model wherein anxiety increases risk for the development of depression is *not* the best explanation for the high rate of co-occurrence observed between anxiety and depression. Although anxiety symptoms predicted small increases in prospective depression when using total scores on common self-report questionnaires (RCMAS and CDI total scores for youth self-reports; CBCL Anxious/Depressed and Withdrawn/Depressed syndrome scales for mother reports), this effect was no longer statistically significant when revised scoring methods for these scales, showing good discriminant validity, were employed. Thus, item overlap on common questionnaire measures assessing anxiety and depression may overestimate the causal relationship between anxiety and future depression.

Consistent with more recent research (e.g., Cohen et al., 2014; Keenan et al., 2009), our results revealed that depression was the strongest predictor of future depression and anxiety was the strongest predictor of future anxiety. Both anxiety and depression showed a high degree of stability over time and little additional variance was explained by cross-domain symptom changes. Thus, past findings supporting the hypothesis that anxiety directly predicts future depression may be attributed to an overreliance on retrospective studies in which early depressive episodes or symptoms are underreported or sub-threshold symptoms are not accounted for (Merikangas et al., 2003; Moffitt et al., 2007).

The developmental psychopathology framework stresses that psychopathology is likely to result from multiple pathways, and equally that any one risk factor is likely to result in diverse outcomes, reflecting the concepts of equifinality and multifinality (Cicchetti & Toth, 2009). The latter has stimulated a recent surge in the search for common factors associated with the development of comorbid psychopathology (Nolen-Hoeksema, & Watkins, 2011). Findings from the present study found evidence for one such common factor: temperamental vulnerability characterized by high levels of negative emotionality and low levels of positive emotionality best explained the development of both anxiety and depression over time. Indirect effects analyses supported the hypothesis that temperament (as assessed in preschool by the child's mother, father, and as observed by an experimenter in a naturalistic setting) predicted the parallel development of anxiety and depression, rather than through cross-lagged links from anxiety to depression or depression to anxiety. Temperament was the most robust predictor of future anxiety and depression outcomes above and beyond other family and child factors which have been implicated in the development of internalizing problems, including parental history of anxiety and depression, maternal emotional availability, family income, and gender (Bayer, Sanson, & Hemphill, 2006; Goodman et al., 2011, Zahn-Waxler et al., 2008). These results were also robust across reporters, with both youth self-report and mother-report data showing similar patterns of association. To our knowledge, this is the first study to show that the combination of temperament traits of high negative emotionality and low positive emotionality, assessed at preschool age, are transdiagnostic risks for the co-development of anxiety and depression (as assessed at both the symptom and diagnostic level).

Gender Differences. Previous studies have documented marked gender differences in internalizing problems, with females showing greater levels of anxiety as early as preschool age and greater levels of depressive symptoms and clinical depression, beginning in early adolescence (Zahn-Waxler et al., 2008). Several prospective epidemiological studies of youth have reported that the female preponderance in anxiety may partly account for sex difference in rates of major depressive disorder that emerge in later adolescence (Breslau et al., 1995; Costello et al., 2003; Merikangas et al., 2003). In contrast to what has been reported in these previous studies, which have focused on diagnostic outcomes, we found no gender differences in the prospective associations between anxiety and depressive symptomatology.

Instead, we found gender differences in the pathways predicting the development of anxiety and depression. Specifically, the impact of maternal emotional availability on the development of internalizing symptomatology was moderated by gender, such that low maternal emotional availability was associated with increased internalizing symptomatology (especially depression) in girls but not boys. These findings are consistent with other research showing that girls tend to exhibit greater levels of depression and anxiety in response to family discord or interpersonal conflicts/stressors that boys (Zahn Waxler, Crick, Shirtcliff, & Woods, 2006). In general, adolescents with parents who are unresponsive, neglectful, coercive, harsh, or inconsistent have more difficulty developing secure attachment styles and adaptive emotion regulation skills (Yap et al., 2007). These adolescents are likely to have difficulties dealing with negative life events and stressors, which may increase their vulnerability to developing internalizing problems, especially within the adolescent years (Cyranowski, Frank, Young, & Shear, 2000). Among girls, who have the tendency to display strong affiliative styles and inter-dependent self-concepts, disrupted parental relationships may lead to especially heightened vulnerability to depression (Cyranowski et al., 2000). Current developmental models of psychopathology aim to identify the common risk factors which lead to multiple disorders, but have greater difficulty understanding why one individual with a particular risk factor develops one set of symptoms, while another with the same risk factor develops another set of symptoms (Nolen-Hoeksema & Watkins, 2011). Our results suggest that among females with a temperamental vulnerability, maternal emotional availability may play a particularly important role in the development of depressive symptomatology.

Limitations and Future Directions

The current study has several strengths, including the use of a prospective longitudinal design spanning multiple developmental periods, rigorous statistical methodology, a combined dimensional and diagnostic approach to assessment, and a non-clinical community sample. The study assessed early emerging temperament from a multi-informant perspective, including naturalistic observations and both symptom levels and diagnostic outcomes from two sources. In addition, the follow-up period of approximately 15 years provided an opportunity to prospectively document the onset and course of psychiatric conditions in youth.

Limitations of the present study should also be noted. Although repeated assessments enhance the power of our analyses, the sample size was relatively small, limiting our statistical

power to detect small predictive and interaction effects. Additionally, our diagnostic data were limited as we did not conduct clinical interviews with youth who rated low on screening measures of anxiety and depression and we did not interview youth at multiple time points. Consequently, we may have underestimated the prevalence of anxiety and depressive disorders within our sample. Relatedly, we examined summary categories of depressive and anxiety disorders, rather than specific diagnoses, as only a small number of participants met diagnostic criteria for an internalizing disorder at assessment. Thus, we were unable to examine how comorbidity may vary according to the specific anxiety or depressive diagnoses presented. Some resent research has suggested that trajectories of anxiety-depression comorbidity vary depending on the subtype of these disorders (see Cummings et al., 2014 for review). Future research should consider the heterogeneity among anxiety disorders when investigating the co-occurrence between anxiety and depression.

Finally, although the present study identified risk factors (i.e., temperament) that predicted the co-development of anxiety and depression, it did not focus on variables that predict their differentiation (although we did find preliminary evidence that parental emotional availability was more strongly related to depressive rather than anxiety symptoms, especially among girls). An interesting area of future research may be whether exposure to unique environmental experiences (e.g., peer and romantic relationships, grief/loss experiences, early life stress, traumatic events) explains why one disorder versus another develops within temperamentally vulnerable individuals. In addition, more research is needed to investigate how parenting and other environmental influences interact with child characteristics in a bi-directional process to produce internalizing syndromes (e.g., Serbin et al., 2015).

Conclusions and Implications for Intervention and Prevention

A traditional explanation for the high degree of comorbidity between anxiety and depression is that anxiety is causally related to the onset of depressive symptoms and that treating anxiety may be a means of preventing subsequent depression (Flannery-Schroeder, 2006; Garber, & Weersing, 2010). We did not find support for the hypothesis that anxiety predicts prospective depressive symptoms, when using measures that show discriminant validity for assessing anxiety and depression. These results suggest that treating anxiety disorders is not an effective method of preventing later depressive disorders. Instead, anxiety and depression most strongly predicted themselves over time, which may indicate the need for disorder-specific,

targeted interventions or alternatively, trans-diagnostic treatment approaches for anxiety and depression targeting their common risk factors.

Most notably, we found anxiety and depression are associated due to common temperament, characterized by high negative emotionality and low positive emotionality. This temperament profile was associated with large effects on anxiety and depression assessed 7 to 13 years later, as well with an increased risk of comorbid anxiety and depressive disorders. Our results suggest that treating emotionality in early childhood holds promise for reducing anxiety and mood disturbance, which becomes highly prevalent in the adolescent years. Increasing theoretical and empirical research has supported the idea that temperament can be used to guide prevention efforts and for identifying individuals at risk for the onset of mental illness (Rapee, 2002). Other potential modifiable risks include family factors such as parental expression of emotional availability, including warmth and sensitivity, appropriate structure, and non-hostility. These factors may interact with an emotionally liable temperament, especially among girls, to produce emotional disorders (Rapee, 2002). Additional research addressing the trans-diagnostic factors (e.g., positive and negative emotionality) of anxiety and depression, as well as the genetic or environmental variables that distinguish them, will be crucial to improving our understanding of their comorbidity and improving intervention efforts. Research investigating the etiology of comorbid psychopathology may lead to more successful treatment of youth with comorbid conditions, who are highly impaired, difficult to treat, and have traditionally received little attention in clinical research trials (Ollendick et al., 2008).

Table 1

Items in Abbreviated Symptom Scales.

Depression Items	Anxiety Items
Youth Self Report	
CDI 1. I am sad all the time	RCMAS 2. I'm nervous when things don't go right
CDI 3. I do everything wrong	RCMAS 10. I worry what my parents will say
CDI 5. I am bad all the time	RCMAS 14. I worry about what other people think
CDI 7. I hate myself	RCMAS 22. I worry about what's going to happen
CDI 8. All bad things are my fault	RCMAS 25. I have bad dreams
CDI 10. I feel like crying every day	RCMAS 29. I wake up scared some of the time
CDI 14. I look ugly	RCMAS 37. I worry something bad will happen
CDI 26. I never do what I am told	
CDI 27. I get into fights all the time	
Mother Report	
CBCL 12. Complains of loneliness	CBCL 29. Fears certain animals, situations, or places, other than school
CBCL 14. Cries a lot	CBCL 30. Fears going to school
CBCL 18. Deliberately harms self	CBCL 31. Fears he/she might think or do something bad
CBCL 33. Feels or complains that no one loves him/her	CBCL 45. Nervous, highstrung, or tense
CBCL 35. Feels worthless or inferior	CBCL 46. Nervous movements or twitching
CBCL 54. Overtired without good reason	CBCL 50. Too fearful or anxious
CBCL 76. Sleeps less than most kids	CBCL 112. Worries
CBCL 77. Sleeps more than most kids	
CBCL 91. Talks about killing self	
CBCL 100. Trouble sleeping	
CBCL 102. Underactive, or lacks energy	
CBCL 103. Unhappy, sad, or depressed	

Note: Some items are paraphrased because of space limitations. CDI = Children's Depression Inventory; RCMAS = Revised Children's Manifest Anxiety Scale; CBCL = Child Behavior Checklist 4/18.

Table 2. *Correlation Matrix, Means, and Standard Deviations*

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. Child sex (female)																			
2. Family income	-.09																		
3. Parent Anx/Dep	-.07	-.06																	
4. Emo Availability	.17	.11	-.12																
5. Temperament	.06	-.09	.09	.09															
6. Anx Disorder	.01	-.11	.21*	-.10	.23**														
7. Dep Disorder	.17^{t}	-.08	.30**	-.10	.31**	.39**													
8. T2 CDI-*R*	.18	-.06	.11	-.19^{t}	.21*	.03	.23**												
9. T3 CDI-*R*	-.02	-.06	.25*	.02	.05	.46**	.28**	.19^{t}											
10. T4 CDI-*R*	.15	-.04	.12	-.02	-.07	.52**	.55**	.19	.52**										
11. T2 RCMAS-*R*	.20^{t}	-.02	-.02	-.03	.29**	.10	.05	.36**	.18^{t}	-.01									
12. T3 RCMAS-*R*	.19^{t}	-.06	.21*	.00	.14	.16^{t}	.11	.02	.20*	.20	.26*								
13. T4 RCMAS-*R*	.36**	-.15	.00	.24^{t}	-.00	.19	.29**	.14	.11	.27*	.37**	.22^{t}							
14. T2 CBCL Dep-*R*	-.12	-.12	.37**	-.17	.28**	.45**	.33**	.12	.35**	.16	.30**	.21*	.02						
15. T3 CBCL Dep-*R*	-.05	-.17^{t}	.31**	-.12	.30**	.52**	.48**	.13	.51**	.41**	.15	.05	.10	.60**					
16. T4 CBCL Dep-*R*	.07	-.11	.23^{t}	-.02	.23*	.39**	.30**	.04	.19	.34**	.16	.10	.16	.51**	.53**				
17. T2 CBCL Anx-*R*	-.13	-.00	.26*	-.12	.17^{t}	.46**	.19^{t}	.00	.30**	.08	.26*	.13	-.02	.59**	.37**	.28*			
18. T3 CBCL Anx-*R*	.05	-.06	.16	-.05	.15	.37**	.18^{t}	.08	.06	-.01	.13	.15	.03	.31**	.51**	.29*	.50**		
19. T4 CBCL Anx-*R*	.19	-.21^{t}	.04	-.09	.07	.40**	.13	.11	.11	.12	.29*	.10	.10	.34**	.37**	.39**	.49**	.63**	
Mean (%)	(55%)	$53388	(35%)	0.00	0.03	(13%)	(9%)	11.01	10.82	10.84	2.75	2.81	3.17	2.62	2.03	1.60	2.14	1.87	1.55
SD	-	$35553	-	0.95	1.34	-	-	2.08	2.28	2.10	2.07	1.99	1.88	2.91	2.46	1.79	2.26	2.05	2.00

Note. Observed, rather than imputed, values are presented in this table. *R* indicates that the revised scales, using showing discriminant validity were used.

**p $\leq$.01, *p $\leq$ .05 ^{t} p $\leq$ .10. *N*=143.

Table 3. *Goodness-of-Fit Statistics for the Autoregressive Cross-Lagged Panel Models*

Model	X2 (*df*)	*p*	CFI	RMSEA (95%CI)	SRMR
Child self-report - Total scales	24.20 (*23*)	.39	0.991	.019 (.000, .072)	.084
Mother report - Total scales	14.89 (*23*)	.90	1.000	.000 (.000, .031)	.043
Child self-report - Revised scales[a]	21.11 (*23*)	.57	1.000	.000 (.000, .062)	.066
Mother report - Revised scales[a]	28.52 (*23*)	.20	0.971	.041 (.000, .084)	.063

Note. [a] Revised scales, showing discriminant validity. CFI = comparative fit index; RMSEA= root mean square error of approximation with 95% confidence interval; SRMR = standardized root mean square residual. *N*=143.

Table 4. *Parameter Estimates for the Autoregressive Cross-Lagged Panel Models*

	Child self-report Total scales		Mother report Total scales		Child self-report Revised scales[a]		Mother report Revised scales[a]	
	b (SE)	β	b (SE)	β	b (SE)	β	b (SE)	β
Autoregressive coefficients								
T2 Anx → T3 Anx	0.37** (0.09)	0.41**	0.46** (0.06)	0.53**	0.24* (0.09)	0.25*	0.50** (0.07)	0.53**
T3 Anx → T4 Anx	0.37** (0.09)	0.32**	0.46** (0.06)	0.55**	0.24* (0.09)	0.25*	0.50** (0.07)	0.54**
T2 Dep → T3 Dep	0.19* (0.08)	0.21*	0.40** (0.08)	0.40**	0.38** (0.08)	0.33**	0.43** (0.06)	0.53**
T3 Dep → T4 Dep	0.19* (0.08)	0.21*	0.40** (0.08)	0.46**	0.38** (0.08)	0.44**	0.43** (0.06)	0.55**
Cross-lagged coefficients								
T2 Anx → T3 Dep	0.24** (0.09)	0.22**	0.11* (0.05)	0.19*	0.11 (0.09)	0.09	0.06 (0.06)	0.03
T3 Anx → T4 Dep	0.24** (0.09)	0.23**	0.11* (0.05)	0.18*	0.11 (0.09)	0.11	0.06 (0.06)	0.04
T2 Dep → T3 Anx	-0.05 (0.08)	-0.06	0.19[t] (0.10)	0.13[t]	0.04 (0.08)	0.04	0.03 (0.07)	0.08
T3 Dep → T4 Anx	-0.05 (0.08)	-0.05	0.19[t] (0.10)	0.15[t]	0.04 (0.08)	0.05	0.03 (0.07)	0.07
Contemporous associations								
T2 Anx with T2 Dep	17.04** (4.04)	0.46**	5.76** (1.15)	0.56**	1.28** (0.41)	0.32**	3.73** (0.71)	0.58**
T3 Anx with T3 Dep	11.95** (3.25)	0.44**	3.06** (0.71)	0.51**	0.77[t] (0.45)	0.19[t]	1.59** (0.40)	0.46**
T4 Anx with T4 Dep	15.52** (3.50)	0.62**	1.45** (0.45)	0.38**	0.54 (0.36)	0.19	0.62* (0.27)	0.27*

Note. Child age and sex were included as time-varying covariates in all models. Unstandardized betas (bs) were constrained to be equal across all waves; standardized betas (βs) were not constrained. [a] Revised scales, showing discriminant validity. **p ≤ .01, *p ≤ .05 [t] p ≤ .10. *N*=143.

Table 5. *Results from the Diagnostic Outcomes Model*

	b (SE)	β
Lifetime Anxiety Disorder by Adolescence		
Gender	-.03 (.30)	-.02
Family Income	-.04 ^{t} (.02)	-.11 ^{t}
Parent Mental Illness	.30* (.15)	.25*
Emotional Availability	-.09 (.14)	-.08
Temperament	.28** (.11)	.36**
Model R^{2} = .22		
Lifetime Depressive Disorder by Adolescence		
Gender	.64 ^{t} (.39)	.26 ^{t}
Family Income	-.04 (.04)	-.09
Parent Mental Illness	.53** (.15)	.39**
Emotional Availability	-.13 (.18)	-.10
Temperament	.42** (.15)	.48**
Model R^{2} = .50		
Correlation Anxiety & Depression	.48** (.18)	.46**

Note. b = unstandardized coefficient; β = standardized coefficient. **p ≤ .01, *p ≤ .05 ^{t} p ≤ .10. *N*=121. $\chi 2(6)=4.160$, *p*=.65, CFI=1.000, RMSEA=.000, CI_{95}(.000, .095), WRMR=.379.

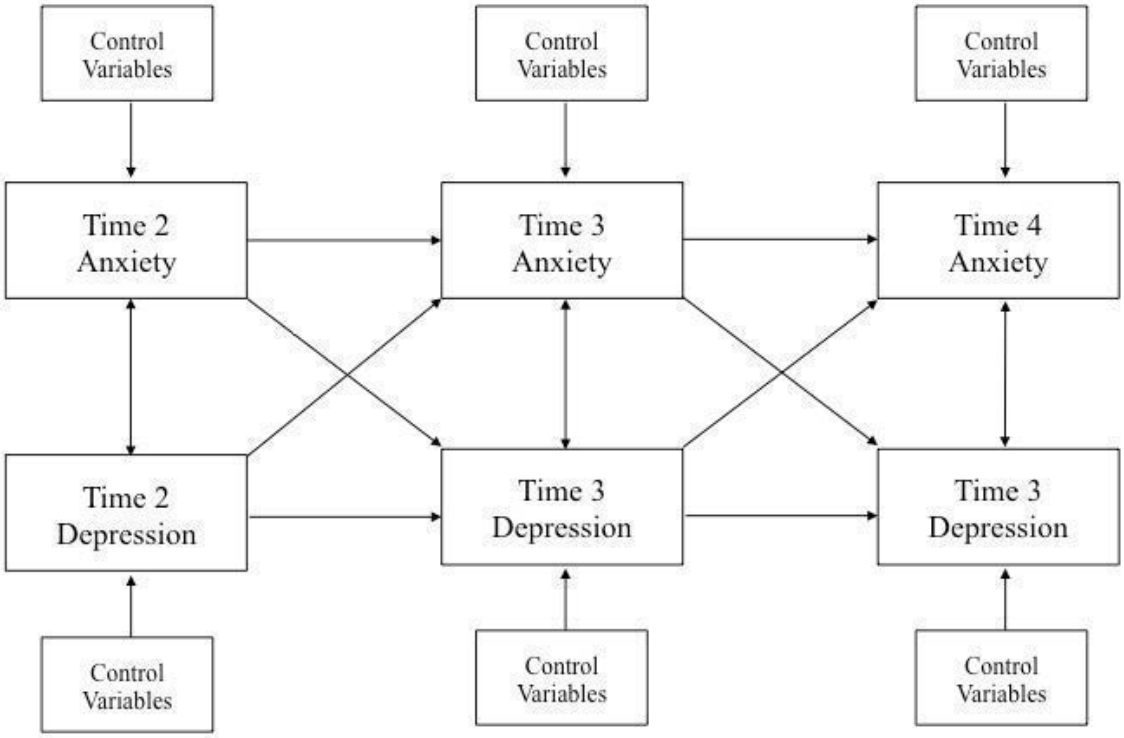

Figure 1. The conceptual model, testing for the reciprocal effects between youth's anxiety and depression symptoms.

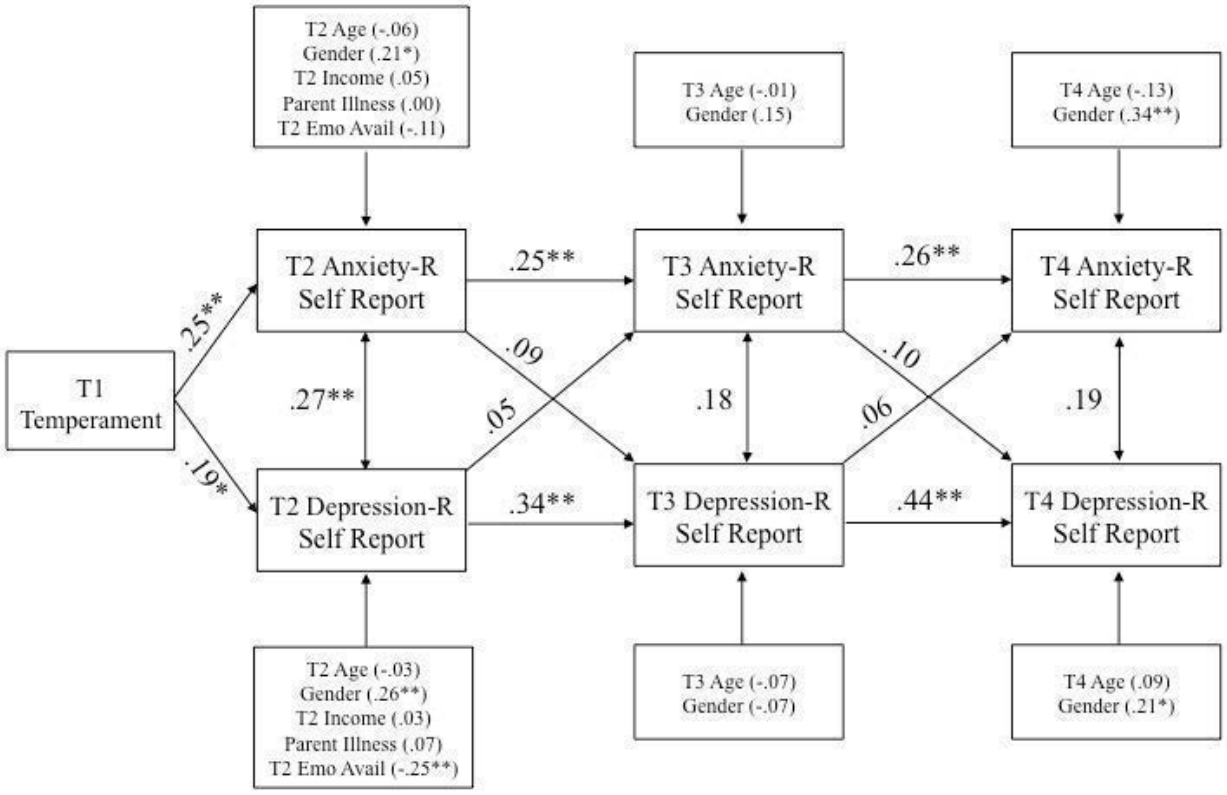

Figure 2. Standardized path coefficients for the reciprocal effects between child-reported anxiety and depression symptoms (Revised scales, showing discriminant validity), including predictors. $\chi 2$ (43)=43.075, p=.47, CFI=0.999, RMSEA=.003 CI_{95}(.000, .057). N=143.

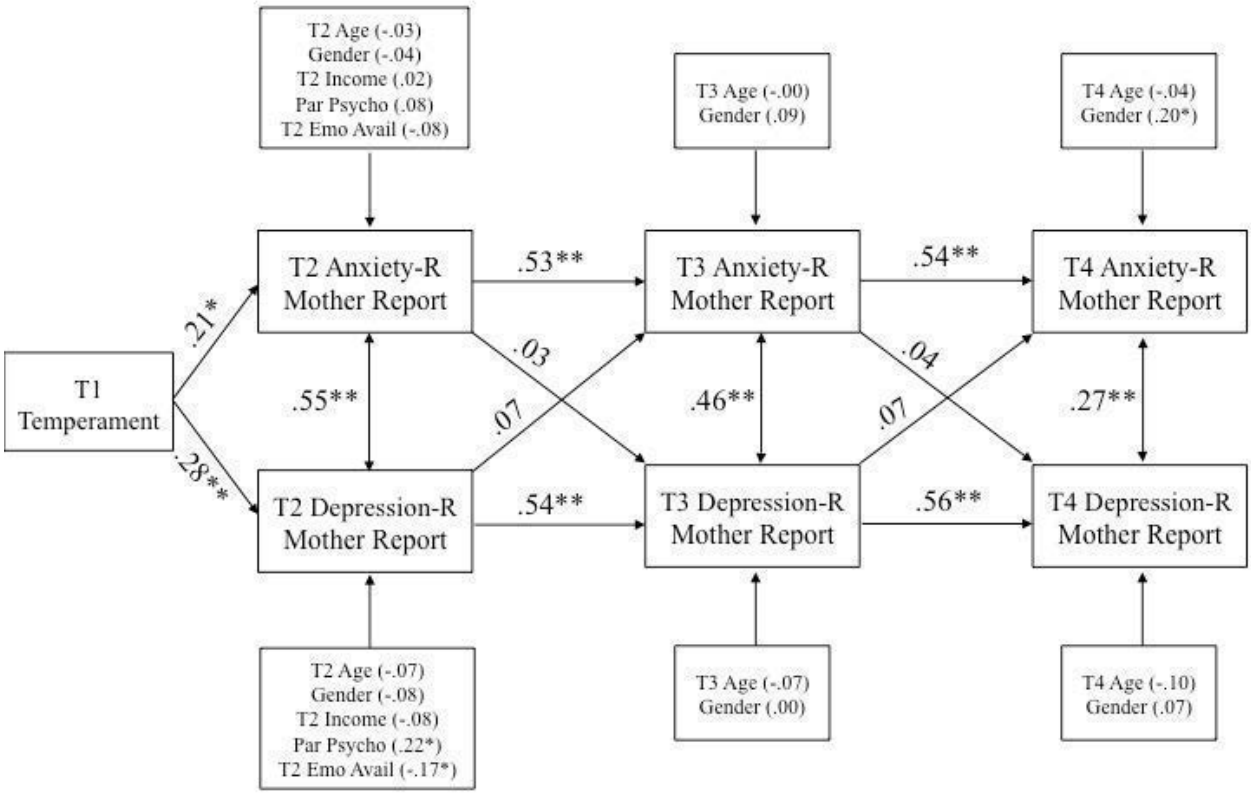

Figure 3. Standardized path coefficients for the reciprocal effects between mother-reported anxiety and depression symptoms (Revised scales, showing discriminant validity), including predictors. $\chi 2$ (43)=50.473, p=.20, CFI=0.964, RMSEA=.035, CI_{95}(.000, .069), SRMR=.062. N=143.

CHAPTER 5: GENERAL DISCUSSION

While significant progress has been made in the understanding, prevention, and treatment of specific psychological disorders, less progress has been made in the understanding of comorbid conditions, including their meaning, course, and causes. The overriding goal of the present dissertation was to provide a more comprehensive understanding of the mechanisms that underlie comorbidity in youth. The studies included in this dissertation explored several potential explanations for the co-occurrence of childhood psychopathology, both within and across diagnostic classes. Adopting a developmental psychopathology approach, the present studies modeled sequential patterns of internalizing and externalizing problems in a community-based sample of children assessed at ages 3, 7, 9, 13, and 16, and tested multiple theoretical pathways to co-occurrence.

The findings presented in the present studies make four distinct contributions to the comorbidity literature in youth by examining: (1) whether the experience of symptoms of one disorder was associated with the development of future symptoms of another disorder, controlling for baseline symptom levels in each domain; (2) whether common temperament risk factors predicted the co-development of internalizing and externalizing problems, and conversely, whether certain temperament dimensions predicted their differentiation; (3) whether other contextual variables, such as the quality of parenting, predicted children's mental health outcomes, separately or in combination with temperament factors (i.e., moderation); (4) whether parenting and other family factors interacted with child gender to differentiate trajectories of internalizing and externalizing problems in boys and girls. In addition, to testing the above-mentioned substantive explanations for comorbidity, Study 2 also tested whether methodological explanations contributed to the high rates of comorbidity observed for anxiety and depression. Finally, diagnostic outcomes were incorporated into the assessment procedures of Study 2, such that risk factors for co-occurring problems could be related to both symptom and diagnostic outcomes. Each of these contributions, as well as the strengths, limitation, future directions, and implications of the studies will be addressed below.

Although rates of concurrent comorbidity have been documented in many epidemiological and community-based studies, the longitudinal progression or sequential comorbidity of children's internalizing and externalizing problems has been rarely examined (Costello et al., 2003, Lilienfeld, 2003). By adopting a prospective, longitudinal approach that

followed children from age 3 to age 16, the studies included in the present dissertation were able to document the sequential progression of internalizing and externalizing problems across childhood and adolescence. Recent advances in statistical modeling procedures allowed us to simultaneously model symptom trajectories of both internalizing and externalizing disorders, such that bidirectional associations between symptom patterns could be examined, controlling for baseline symptoms in each domain. This represents an advance over previous research, which has previously limited analyses to examination of one disorder at a time or has failed to trace symptom fluctuations over time.

Preliminary results from Study 1 support the assertions made by many researchers that internalizing and externalizing problems appear to influence one another over time (Cummings et al., 2014; Wolff & Ollendick, 2006). Specifically, controlling for baseline symptoms in each domain, the experience of symptoms of internalizing problems appeared to protect against the development of future externalizing behaviour symptoms. Many researchers have hypothesized a causal relation between these symptom clusters, based on their high degree of co-occurrence when measured contemporaneously (Krueger & Markon, 2006). For example, Patterson and colleagues (1992) have proposed a causal link between externalizing and internalizing disorders such that the adverse consequences and life circumstances associated with externalizing disorders lead to social and academic problems that subsequently increase a child's vulnerability to depression ("failure" model: Capaldi, 1991). However, our results are more consistent with other research suggesting that internalizing problems are actually associated with lowered risk of later externalizing problems (Cunningham & Ollendick, 2010). Results from Study 1 may add clarity to this debate by demonstrating that internalizing and externalizing problems become increasingly differentiated across childhood and adolescence, as rates of co-occurrence decreased across the study period.

Study 2 investigated the longitudinal progression of symptoms within the class of internalizing symptomatology (i.e., anxiety and depression). Preliminary analyses revealed that anxiety predicted small increases in later depressive symptoms, but not vice versa, consistent with the pattern of anxiety preceding depression that has been established in numerous studies (Essau, 2003; Starr, Hammen, Connolly, Brennan, 2014). Causal model hypotheses have also been put forth to explain the longitudinal progression from anxiety to depression, whereby the social byproducts of anxiety, including peer rejection, problems in the parent-child relationship,

and isolation lead to the experience of other internalizing disorders, especially depression (Cummings et al. 2014; Zahn-Waxler et al., 2000). However, such mediating mechanisms are typically not explicitly tested in comorbidity research and the contribution of other methodological or substantive explanations for their longitudinal progression have not been systematically considered. To highlight this issue, our results indicated that symptom overlap among measures assessing anxiety and depressive symptoms explained a significant portion of this longitudinal progression. That is, when measures showing good discriminant validity were used, the association between earlier anxiety and later depression was no longer statistically significant. Unfortunately, as reviewed earlier in the thesis, many assessment instruments show poor specificity for discriminating anxiety from depression (Cummings et al. 2014), making it difficult to distinguish between artifactual and true sources of comorbidity. Thus, previous studies may have overestimated the degree to which anxiety and depression are causally related over time, by neglecting to consider the impact of methodological factors.

Another popular substantive explanation for the longitudinal progression between internalizing and externalizing problems is that common risk factors explain the development of symptoms in multiple diagnostic categories (Caron & Rutter, 1991; Krueger & Markon, 2006; Lilienfeld, 2003). Results from the present studies indicate that causal models, as described above, may not be the best explanation for the sequential progression of externalizing problems to internalizing problems or for anxiety to depression. Instead, results from the present studies indicate that the co-occurrence of these disorders was better explained by overlapping risk factors. Specifically, the temperamental trait of negative emotionality (i.e., difficulty regulating emotion) was identified as a common risk factor for both internalizing and externalizing problems, and for both anxiety and depression. In addition, low positive emotionality (i.e., the lack of experience of high-intensity pleasure) was identified as a common risk for both anxiety and depression. These temperament traits explained a great deal of the common variance across time, and the sequential relations between problems were no longer statistically significant when these temperament traits were taken into account.

These findings are consistent with previous work which has shown that negative emotionality is moderately positively correlated with both internalizing and externalizing disorders in children (Tackett, 2006; Tackett, Waldman, Van Hulle, & Lahey, 2011). Although negative emotionality has been linked to many forms of child psychopathology (Krueger &

Markon, 2006; Lilienfield, 2003), whether negative emotionality represents a pathway to comorbidity has not yet been tested empirically in a longitudinal framework. The present findings suggest that negative emotionality does represent one contributor to the covariation between these problems over time.

Additionally, the finding that the combination of high negative emotionality and low positive emotionality predicted the co-development of anxiety and depressive symptoms adds to the growing literature on Clark and Watson's tripartite model. The tripartite model was originally proposed to account for the high degree of symptom overlap and diagnostic comorbidity between anxiety and depression. This model posits that anxiety and depression share a common component of negative emotionality, but can be differentiated by low positive emotionality associated with depression and high physiological hyperarousal associated with anxiety (Clark & Watson, 1991). However, growing empirical evidence suggests that low positive emotionality is common to depression and many anxiety disorders, such as generalized anxiety, social phobia, and separation anxiety (Anderson & Hope, 2008; Chorpita, Plummer, & Moffitt, 2000). To date, most research on this topic has been conducted on adult samples and little is known how results extend to youth in community-based (i.e., non-clinical) samples. In addition, most studies have not been able to adequately assess whether the tripartite model is applicable across severity levels (i.e., symptom versus diagnostic outcomes), reporters, and longitudinal outcomes. The present results add to this literature by demonstrating that high negative emotionality and low positive emotionality contributed to the co-development of anxiety and depressive symptoms across time, with findings consistent across maternal and youth-self report measures. In addition, youth who had this combination of temperament traits were at heightened risk of experiencing comorbid anxiety and depression diagnoses in adolescence. These results are among the first to demonstrate that temperament traits assessed in early childhood operate as risk factors for co-occurring internalizing symptoms emerging later in development.

Current models of developmental psychopathology aim to identify the common risk factors which lead to multiple disorders, but have difficulty explaining why one individual with a particular risk factor develops one set of symptoms while another individual with the same risk factor develops another set of symptoms (Nolen-Hoeksema & Watkins, 2011). Thus, in addition to identifying pathways to comorbidity, another related goal of the present work was to illustrate how unique risk factors predict the differentiation of psychopathology over time. Results from

the current studies indicate that externalizing problems were predicted by earlier manifestations of activity (and associated impulsivity), while internalizing problems were uniquely predicted by social withdrawal. These findings are consistent with other theorists who view internalizing problems as over-controlled emotion and externalizing problems as under-controlled emotion (e.g., Eisenberg et al., 2001; 2004). Although similar findings have been documented in previous research (Campbell, Shaw, & Gilliom, 2000; Olson, Sameroff, Kerr, Lopez, & Wellam, 2005; Olson, Schilling & Bates, 1999; Rubin, Burgess & Coplan, 2002; Rubin et al., 2009), the vast majority of studies on childhood psychopathology simply identify the risk factors that are associated with the development of a specific psychological disorder. Few studies have been able to examine whether identified risk factors are specific to a particular disorder, by modeling outcomes for multiple disorders simultaneously, controlling for their overlap.

Moreover, the present results serve as an example of how risk factors may interrelate in a transactional manner. For example, parenting practices, including responsiveness and harsh discipline interacted with temperament traits to predict internalizing or externalizing problems. Results from these analyses showed that parenting effects were significant largely in their interaction with child temperament traits. For example, we found that among children rated high on social withdrawal, high levels of harsh discipline predicted higher internalizing problems. Similarly, among children rated highly on negative emotionality, low maternal responsiveness predicted greater levels of internalizing problems, while avoidance of harsh discipline protected children with lower levels of negative emotionality against externalizing problems. Together, these results indicate that the presence of positive parental supports may mitigate the relationship between temperament risk factors and subsequent psychopathology. However, among children who show difficult temperamental traits, negative parenting may place children at even greater risk for psychopathology. This may occur through a cycle in which parents and children influence one another's psychopathology (i.e., gene x environment interactions). These findings are an important addition to the literature, as relatively little is known about the mechanisms by which risk factors operate to produce psychopathology in childhood, and even less is known about the mechanisms by which risk factors operate to produce comorbid psychopathologies (Rutter, 1997).

Gender Differences

Previous studies have documented marked gender differences in psychopathology, with early-onset externalizing problems showing a marked male preponderance, while adolescent-onset internalizing disorders show a marked female preponderance (Achenbach, 1991; Zahn Waxler et al., 2008). Of relevance to understanding gender differences in developmental trajectories is whether the developmental progression of co-occurring internalizing and externalizing symptoms may differ for boys and girls. Some researchers have proposed a gender paradoxical effect wherein the risk for co-occurring problems would be greater among the sex that that was less frequently affected (Loeber & Keenan, 1994). For example, with respect to externalizing problems, girls, compared to boys, generally show a lower prevalence of externalizing problems. However, among girls who have developed a disruptive behavior disorder, the severity of the illness would be expected to be greater, increasing the risk for developing comorbid conditions (Loeber & Keenan, 1994; Wolff & Ollendick, 2006). In contrast, other researchers have suggested that due to statistical probability, the risk for comorbidity will be highest among the sex that is most commonly affected by the disorder. For example, because females are more likely to be affected by both anxiety and depressive disorders, they are at greatest likelihood of experiencing comorbid internalizing disorders (Breslau et al., 1995; Keenan et al., 2009). As such, the present studies tested whether there were gender differences in the pathways predicting the development of internalizing and externalizing problems, as well as anxiety and depression. We did not find evidence for gender differences in the progression of one disorder to another over time, although admittedly sample size limitations did not permit for a robust test of this hypothesis. Studies of co-occurring trajectories that span the full course of childhood and include both genders are scarce. However, some research suggests that rates of comorbidity may not be influenced by sex (Boylan, Vaillancourt, Boyle, & Szatmari, 2007), which may be in keeping with our findings of a lack of a sex difference in the sequential progression from one disorder to another.

Although we did not find evidence for gender differences in the progression of one disorder to another over time, we did find evidence of interactions between child gender and the identified risk factors. Specifically, in Study 1, high maternal responsiveness predicted lower internalizing and externalizing problems in girls, but higher levels of internalizing and externalizing problems in boys. In addition, Study 2 showed that the effects of maternal emotional availability were moderated by gender such that low maternal emotional availability

was associated with increased internalizing symptomatology (especially depression) in girls but not boys. These findings are consistent with other research showing that girls tend to exhibit greater levels of psychopathology in response to reduced maternal emotional supports that boys (Zahn Waxler et al., 2006). Understanding how comorbidity develops and how these effects may differ for boys and girls is an understudied area. More studies, allowing for better differentiation of findings by gender, such as the present studies, are needed.

Theoretical Contributions

Many pathways to comorbidity have been proposed by researchers (Krueger & Markon, 2006; Lilienfeld, 2003; Rutter & Caron, 1991), several of which were investigated in the present studies. However, pathways to comorbidity are not mutually exclusive. Instead, risk factors and risk processes are likely to be interrelated and serve to compound one another over time (Cicchetti & Rogosch, 1996; Rutter, & Sroufe, 2000). There is a paucity of research testing multiple pathways to comorbidity, which would shed light on the relative importance of various risk factors and processes. By incorporating a developmental psychopathology framework (Cicchetti & Toth, 2009), the present work attempted to capture a more complete picture of the various mechanisms by which risk factors may lead to the development of specific, or comorbid, disorders in childhood and adolescence. Findings from this thesis indicate that common risk factors most strongly accounted for the relationship between comorbid emotional and behavioral problems in youth. However, the common risks identified in these studies did not fully account for comorbidity. Instead, other processes, including the presence of one disorder placing a child at risk for the development of a future disorder, may also influence the development of comorbidity. Conversely, unique risk factors serve to account for the differentiation of disorders, either separately or in combination with other contextual risk factors. Further, the presence of overlapping symptom criteria may serve to inflate estimates of comorbidity, especially in the case of disorders within the same diagnostic class.

Based on the findings from the present studies, the following model is proposed to explain the development of comorbid internalizing and externalizing problems across childhood and adolescence (see Figure 1). First, common and unique risk factors may account for the initial onset and interaction between childhood psychological conditions, as well as their comorbidity. Due to common risk factors, internalizing and externalizing problems may be related such that one disorder increases the risk for a second disorder, both concurrently, and prospectively.

Overlap in symptomatology across disorders (represented by the area of overlap in Figure 1), may also increase the child's likelihood of meeting criteria for multiple psychopathologies. Overlapping symptomatology may represent both imprecision in diagnostic classification symptoms, but also true overlap in the expression of psychopathology across a range of psychological disorders. For example, a child with high levels of negative emotionality may be more likely to meet diagnostic criteria for multiple disorders because components of negative emotionality (i.e., irritability) are represented in the diagnostic criteria of many childhood psychological disorders. Thus, a child's development of internalizing or externalizing problems at Time 2 is a function of both his/her preexisting levels of internalizing and externalizing problems at Time 1, as well as the risk factors common to both disorders, and the degree of symptom overlap that is represented in the diagnostic criteria among disorders.

Next, unique risk factors may serve to differentiate the expression of psychopathology, such that certain disorders are more or less likely to co-occur, depending on the associated common and unique risk factors for each disorder. An example of this is that activity/impulsivity may represent a risk factor for externalizing problems, but be a protective factor against internalizing problems. Thus, the presence of high activity and impulsivity may decrease the likelihood of developing comorbid psychopathologies, either separately or in combination with other identified risk and protective factors (e.g., parenting). On the other hand, negative emotionality represents a risk factor for a wide range of psychopathologies. Negative parenting practices (i.e., harsh discipline, low responsiveness and warmth) may produce especially poor developmental outcomes in children with high levels of negative emotionality. Thus a child who presents with high levels of negative emotionality (especially in combination with negative parenting) is at heightened risk for experiencing comorbidity.

Overall, according to the model, common risk factors give rise to multiple disorders, while unique factors differentiate between these conditions. Although the present study did not find support for a direct (causal) link between disorders, the common risk factors identified in the present study did not fully explain the covariation in symptoms across time. Thus, a direct link between disorders may occur whereby the sequelae of one disorder may place an individual at increased risk for another disorder (Capaldi 1991; Cummings et al., 2014). Dashed lines are used to represent this theoretical pathway in Figure 1. Future research will be necessary to confirm the disorder sequelae (e.g., conflict in the parent-child relationship, social isolation, academic

difficulties, trouble with the law) that may increase risk for future disorders, controlling for baseline symptoms in each domain.

In sum, the results of the current studies and the proposed model incorporate common and unique factors related to the development of comorbidity over time. Both direct and indirect relationships between internalizing and externalizing problems have been reviewed and incorporated into this framework. Importantly, this model allows for several potential pathways for comorbidity, thus capturing the complexity of the developmental psychopathology approach to comorbidity.

Strengths and Limitations

The current research program demonstrated a number of strengths that increase its contribution to the research literature on the development of comorbidity in childhood. A major strength of the studies was their longitudinal design, which provided the opportunity to explore the temporal relations between symptom patterns over time and to examine whether common risk factors predicted their co-development. Additionally, recent advances in longitudinal data analytic methods provided us the opportunity to examination change in symptom patterns in multiple domains, simultaneously. This allowed us to detect previously unreported longitudinal associations between symptoms, as well as to examine whether symptom patterns at an earlier age played a part in trajectories of symptom patterns at later ages. These results extend the findings documented by previous studies, which have primarily utilized cross-sectional designs to assess comorbidity patterns at one time point.

Additionally, the assessment of temperament in early childhood, prior to the onset of serious emotional and behavioural problems, allowed us to begin to disentangle how risk factors influence the development of psychopathology over time. The wide range of predictor variables assessing aspects of the child, family, and environmental context is also unique to this literature. These variables were assessed using multiple means of data collection, including mother- and self-reported measures of behavioral and emotional symptoms, observational, mother- and father-rated coding of temperament, observation of parenting, and structured diagnostic interviews assessing both parent and child diagnoses. The inclusion of observationally-based measures allowed for a more in-depth examination of the constructs under investigation and lends itself to increased generalizability. This strategy also avoided the problem of shared rater variance (typically occurring when all measures are completed by a single rater), which is

common to this research area and is likely to inflate the associations between constructs. The strengths of this research design and the adoption of a developmental psychopathology approach allowed us to test multiple pathways to co-occurrence, which shed light on the relative importance of various risk factors and processes in producing comorbid child psychopathology.

Another strength of these studies is that they involved a community-based sample, rather than a clinical sample. Most other studies exploring patterns of comorbidity in youth have used clinical samples. Given that referral biases inherent to clinical samples alter patterns of comorbidity, the inclusion of a general-population sample afforded us the opportunity to explore how co-occurrence would appear in a sample more representative of the population at large.

There are a number of limitations of the current research that warrant discussion. Many of these limitations present areas for further investigation and ongoing study. First, these studies are correlational; thus, although the longitudinal associations suggest a direction of effects (e.g., such that temperament assessed early in childhood affects the development of co-occurring disorders throughout the late childhood and adolescent years), the direction of these effects cannot be determined conclusively. Because correlational designs can only suggest causality, an experimental design would be necessary to confirm that change in temperamental characteristics during the periods identified in the present study would affect the onset of psychopathology. Although temperament traits are often thought to underlie or predispose an individual to psychopathology ("vulnerability" model), the relationship is not necessarily unidirectional. Multiple models have been proposed to explain the relationship between temperament and psychopathology (Tackett, 2006). Other relevant pathways may be involved, including the possibility that the development of emotional or behavioral symptoms changes an individual's temperamental traits ("scar" model) or that dispositional traits may influence the course, severity, or presentation of a disorder ("exacerbation" model; Tackett, 2006). Finally, it could be that much of the taxonomic distinction between temperamental traits and psychological disorders is artificial and that many disorders are better conceptualized as extremes of temperament ("spectrum" model; Tackett, 2006).

Another limitation of the present design was the relatively small sample of families eligible for inclusion. Although finding large effects of temperament on the parallel development of children's internalizing and externalizing problems, bidirectional effects across symptom domains did not always reach levels of statistical significance. Review of the previous literature

suggests that when bidirectional effects were detected in previous studies, they were typically quite small in magnitude (e.g., Cohen et al., 2014; Gilliom & Shaw, 2004; Keenan et al., 2009; Lee & Bukowski, 2012). Even small bidirectional effects between symptom domains, which the present study did not have the power to detect, may have important implications for the development of children's psychopathology. Similarly, mediation effects, wherein the experience of one disorder places an individual at risk for another disorder due to the negative sequelae associated with the first disorder (e.g., social isolation/rejection, academic difficulties, conflict with the law) could not be examined in the present study. Although we found strongest evidence for a common risk factor model, hypotheses regarding mediation effects are commonly suggested in the comorbidity literature (Cummings et al., 2014; Wolff & Ollendick, 2006). Again, small mediation effects may have large influences on psychopathology as they accumulate and compound over time. Finally, the current studies did not have enough power to be able to divide the samples by sex and still detect significant results. Given that psychopathology is known to be a gender-differentiated phenomenon, future research utilizing larger samples should continue to consider how pathways to comorbidity may differ for boys and girls.

A significant drawback of the research presented in the present dissertation (and the corresponding proposed model) is its neglect of risk factors that might be unique to comorbid individuals. While the model allows for risk factors of internalizing and externalizing disorders to be compiled in an additive and synergistic manner, it does not account for factors which may be unique to children with comorbid disorders. For example, some risk factors not investigated in the present studies, such as neurocognitive deficits, early life stress, abuse/trauma, and genetic factors, may be unique to comorbid conditions (Cosgrove et al., 2011; Levy, 2010). Nevertheless, those studies that have examined risk factors in comorbid individuals seem to suggest the risk factors for comorbidity consist of a compilation of factors related to internalizing and externalizing disorders and that children with comorbid psychopathology experience the highest level of risk factors (Nottleman & Jensen, 1995; Oland & Shaw, 2005). Thus, separate risk factors, exclusive to comorbidity, may not exist.

Finally, the current research was exclusively focused on the development of psychopathology among a community-based study of lower-income youth living in Montreal,

Quebec Canada. Evaluation of these associations across countries and cultures will provide further insight into the etiology of developmental psychopathology.

Implications for Intervention and Prevention

Based on the results of the present studies and the proposed theoretical model of comorbidity, suggestions for future research and practice are warranted. In terms of clinical practice implications, these results highlight the differentiation between internalizing and externalizing problems, as well as between anxiety and depression. Although risk factors common to each problem were indicated, unique risk factors were also identified. These results suggest that the high level of comorbidity documented in the present studies in not simply a result of the disorders representing a different manifestation of the same underlying disorder.

Results of the present work suggest that a child's level of negative emotionality is intertwined with his or her risk for various disorders, including internalizing disorders like generalized anxiety and depression, as well as externalizing disorders like ADHD and oppositional defiant disorder. Relatedly, low positive emotionality was related to heightened risk for multiple expressions of internalizing disorders, including anxiety and depression. Understanding that negative emotionality and positive emotionality are contributors to the co-occurrence among these disorders may highlight avenues for early risk assessment, intervention, and perhaps prevention. Identifying early temperamental risk factors may help clinicians to identify children who are at risk for comorbidity and to develop more appropriate intervention and prevention efforts (Rapee, 2002). For example, as early differences in negative emotionality are identified, prevention programs may be designed to increase emotion regulation skills in these children, before the onset of one or more disorders. Future research should determine the extent to which temperament traits such as negative and positive emotionality in children are malleable, and if interventions aimed at reducing these difficult temperament traits decrease the risk for developing comorbid diagnoses (McClowry, Rodriguez & Koslowitz, 2008; Rapee, 2002).

Additionally, the significant parent x child temperament interactions found in the present studies indicate that parenting programs that aim to fostering children's adjustment may benefit from a more detailed assessment of temperament, in order to help parents to understand their children's behavioral tendencies and coping styles. It is possible that different parenting techniques are more effective for children with certain temperamental styles (McClowry et al.,

2008). Relatedly, the parent's understanding of goodness-of-fit between their child and their environmental context may lead to more effective parenting strategies that ultimately reduce the risk of psychopathology.

Finally, the high prevalence of comorbid internalizing and externalizing problems also highlights the need for interventions specific to comorbid populations. Previous research indicates that clinical interventions that are designed for specific disorders are often less efficacious when children have comorbid problems (Chase & Eyberg, 2008; Ollendick & King, 2004; Rohde, Clarke, Lewinsohn, Seeley, & Kaufman, 2001). Additionally, there is a need to account for treatment variables and risk factors specific to these comorbid individuals. More research is needed to identify effective treatment modalities for these comorbid individuals, who have historically been excluded from research and interventions studies.

Conclusions and Directions for Future Research

The two inter-related research studies presented in the present dissertation provide a comprehensive examination of the influence of temperament, parenting, and other family factors on the development of co-occurring symptom patterns across childhood and adolescence. In integrating multiple competing models of co-occurrence, the present studies demonstrate that the etiology of internalizing and externalizing problems is best understood when their co-development is considered, along with the risk factors that underlie symptom co-occurrence. The current results provide empirical support for the assertion that comorbidity is a real psychological phenomenon in need of further study, as well as pointing to possible avenues for improved assessment of comorbidity, classification of psychopathology, and prevention.

First, in terms of research, it is important for studies to use techniques that reduce artifactual comorbidity. As has been reviewed, various methodological techniques (e.g., use of clinical samples) may over inflate rates of comorbidity. To address some of these issues, it is recommended that studies make use of general population or community-based samples. Relatedly, although some researchers have attributed comorbidity to imprecisely drawn diagnostic boundaries and shared symptom structure (Krueger & Markon, 2006; Watson, 2005) the present results indicate that concurrent comorbidity is evident even when the specificity of diagnostic clusters of symptoms is improved. Thus, comorbidity seems to be a true phenomenon, rather than just a methodological artifact. However, researchers must continue to examine and rectify situations in which there is an overlap in symptoms across disorders, as well as to

determine if there are symptoms that accurately contribute to the differentiation of related disorders and to the identification of comorbidity.

The high degree of comorbidity identified in the present work also highlights the need to reconsider current categorical approaches to the classification of psychopathology. The current DSM taxonomy of psychopathology has become increasingly reliable, but has well documented weaknesses including (but not limited to) high comorbidity between seemingly disparate diagnostic categories, blurred boundaries between diagnostic categories, and high levels of heterogeneity within diagnostic categories, which leads to questions regarding its validity (Widiger & Samuel, 2005). The findings of the present studies, which illuminate the shared causal mechanisms that underlie the co-development of different disorders, are in line with recent movements to adopt a transdiagnostic approach to the study of psychopathology. The goal of the transdiagnostic approach is to develop empirically derived taxonomies based on shared features of mental disorders. The need for transdiagnostic approaches is reflected in the recent development of the Research Domain Criteria (RDoC) by the National Institute of Mental Health (Insel et al., 2010). In brief, the RDoC represents a strategic shift away from diagnostic categories to a new classification of psychopathology based on fundamental dimensions of neurobiological and behavioural functioning. Such an approach has the potential to pave the way toward an etiologically-based classification of psychopathology. A longitudinal, dimensional approach to symptom classification, as employed in the present studies, holds a number of advantages over more traditional categorical systems, including the potential to more effectively track the heterotypic continuity of psychiatric symptoms across development, which may help redefine boundaries between diagnostic categories.

This research highlights that in order to understand patterns of sequential comorbidity and the possibility that one may give rise to the other, a greater emphasis on longitudinal investigations is needed. While externalizing problems tend to precede internalizing problems and anxiety tends to precede depression in the majority of cases, these associations are reduced in magnitude when baseline symptom levels are accounted for. Longitudinal investigations of these disorders may clarify their developmental course as symptoms tend to wax and wane over time (Caron & Rutter, 1991; Wolff & Ollendick, 2006). This approach, which retains important information about symptom levels regardless of diagnostic threshold, may also be useful for

early detection, prediction of diagnostic course, and prevention of psychopathology (Franklin, Jamieson, Glenn, & Nock, 2015).

In addition, the results from the present thesis indicated that the co-occurrence between symptoms was best understood when the risk factors for each problem were taken into account. Longitudinal studies provide the opportunity to identify the antecedent risk factors of disorders as well as the mechanisms by which risk factors might explain the longitudinal progression from one disorder to another. It is both feasible and informative to examine the bidirectional influence of symptoms in multiple domains, as well as the predictors that serve to either explain their co-development or differentiation within a single statistical model. As statistical approaches incorporating multiple outcomes become more familiar, we anticipate that they will be increasingly incorporated into longitudinal research designs.

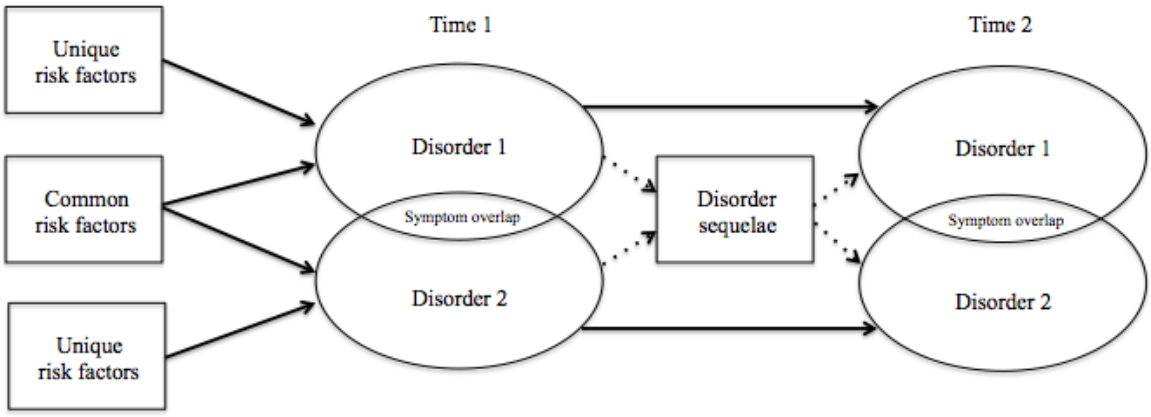

Figure 1. Model for the development of comorbid psychopathology in youth. According to this model, a child's development of a disorder at Time 2 is a function of both his/her preexisting levels of symptoms Time 1, as well as the risk factors common to both disorders and the degree of symptom overlap that is represented in the diagnostic criteria among disorders. Sequelae to a disorder (e.g., negative life events) may subsequently increase risk for a disorder at Time 2. Finally, unique risk factors serve to differentiate between conditions. Solid lines represent significant findings emerging from the present studies. Dashed lines represent theorized pathways requiring future research.

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