A Longitudinal Examination of Family Factors in Childhood Anxiety: The Role of Parental Anxiety and Child Emotion Dysregulation

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ABSTRACT

Theoretical models specify that anxiety aggregates in families. Research confirmed maternal anxiety as a predictor of childhood anxiety; however, very little evidence exists in support of paternal anxiety's role in child anxiety as well as about potentially reciprocal relationships between parental and child anxiety. The parent-child anxiety transmission mechanisms are also not fully understood; the majority of previous research focuses on the child's acquisition of anxiety symptoms from a parent via cognitive processes. Recent integrative theoretical models propose that child emotion regulation processes might be involved in parent-child anxiety transmission. The current dissertation aimed to address these gaps in literature. Both studies utilized data from over 800 mothers, 400 fathers, and their children drawn from the longitudinal NICHD Study of Early Childcare and Youth Development. Measures of maternal anxiety, paternal anxiety, child anxiety, and child emotion dysregulation were collected over a nine-year period when children were between the ages of 6 and 15 years. Study I provided evidence of significant indirect effects from parental anxiety to child anxiety through child emotion dysregulation for both mother-child and the father-child relationships. Child emotion dysregulation was non-significant in the father-child path of a family model, despite significant direct effects. The results provide evidence for child emotion dysregulation as an underlying process of parent-child anxiety transmission. Study II provided evidence of significant bidirectional predictive links of maternal anxiety and child anxiety across ages 6, 8, 10, and 15 years tested in a mother-child cross-lagged path model. Significant predictive paths from paternal anxiety to child anxiety were found from ages 6 to 8 and a significant predictive path from child anxiety to paternal anxiety was found from age 10 to age 15 in a father-child cross-lagged model. Additional tests of family models confirmed that there were unique effects of both maternal and paternal anxiety on child anxiety over time. The results show the long-term impact of both maternal anxiety and paternal anxiety on child anxiety as well as child anxiety's reciprocal effects on parental anxiety. Both studies demonstrate the importance of both mothers and fathers in childhood anxiety etiology.

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GENERAL AUDIENCE ABSTRACT

Research has shown that anxiety might run in families, and that parental and child anxieties might reinforce each other. In research, most attention is paid to mothers and how their anxiety influences child anxiety, including how children learn their anxious experiencing from their mothers. Very little research has been dedicated to studying fathers and how their anxiety might impact their children. The current two studies of this dissertation wanted to better understand how maternal and paternal anxiety shape child anxiety over the years. Study I tested whether the way how children regulate their emotions is influenced by parental anxiety and whether it contributes to their own anxiety. The evidence shows both mother and father anxiety influence children's regulation of their emotions, and in turn, this contributes to child anxiety. Therefore, how children control their emotions impacts how parental anxiety shapes child anxiety. Study II tested whether parental anxiety influences child anxiety consistently over time as children become adolescents. Study II also tested whether child anxiety contributes to parental anxiety over the years. Results showed that maternal and child anxiety consistently foretold each other when children were at 8, 10, and 15 years old. The results for fathers were more complicated and showed that father anxiety likely influences child anxiety when children are younger, while child anxiety influences paternal anxiety when children are older, during their teenage years. Both studies highlighted showed the importance of both mother and father anxiety to better understand child anxiety.

Dedication

To my parents, whose relentless support of my education made this degree possible. Your love for learning is forever embedded in me.

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Chapter 1: Introduction

Childhood Anxiety Prevalence

Anxiety is one of the most common mental health disorders in children. The Anxiety and Depression Association of America (ADAA, 2020) estimates that around 25% of children experience an anxiety disorder. The prevalence of childhood anxiety, however, varies depending on the data source. The lowest prevalence of 7% for children between 3 and 17 years of age has been reported by The Centers for Disease Control and Prevention (CDC, 2020) based on the study sampling over 300,000 households (Ghandour et al., 2019). In contrast, based on a nationally representative sample of 10,123 adolescents aged 13 to 18 years (Merikangas et al., 2010), The National Institute of Mental Health (NIMH, 2017) reports the highest prevalence of almost 32%.

These discrepancies in the prevalence estimates of child and adolescent anxiety are partially related to age, with higher prevalence of anxiety being in adolescent samples (Merikangas et al., 2010). The discrepancies are also related to different operationalizations of anxiety in prevalence studies. For example, the CDC-reported study by Ghandour et al. (2019) was designed to produce national estimates of mental health disorders. In this study, caregivers were asked whether they were ever told by a healthcare provider that their child had anxiety. This is potentially problematic since anxiety, as an internalizing disorder, does not necessarily have an outward disturbing manifestation and, unless there is a severe impact on normal functioning, it might not raise enough attention for a parent to seek a healthcare provider's opinion. Since no other formal evaluation of anxiety was part of the Ghandour et al. (2019) study, anxiety rates could have been underrepresented. In comparison, an NIMH-reported study by Merikangas et al. (2010) utilized an interview-based diagnostic tool to determine the anxiety

diagnosis in an adolescent sample, which likely increased the accuracy of prevalence estimates. Over 30% reported by the NIMH is therefore a more accurate estimate of anxiety prevalence in adolescence.

Additionally, changes in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) might have an impact on tracking anxiety prevalence over time. Diagnostic tools designed to distinguish between anxiety types, such as The Anxiety Disorders Interview Schedule for *DSM-IV* (ADIS-IV, Silverman & Albano, 1996), are based on the *DSM* and, therefore, are dependent upon changes introduced in new editions of the manual. To illustrate, significant changes to conceptualizations of anxiety types occurred with the publication of *DSM-5* (American Psychiatric Association, 2013), namely, the inclusions of separation anxiety disorder and selective mutism disorder and the exclusions of obsessive-compulsive disorder, posttraumatic stress disorder, and acute stress disorder from the family of the anxiety disorders (American Psychiatric Association, 2013). Researchers conducting prevalence studies using DSM diagnoses need to account for such changes. Bittner et al. (2007), for example, included the diagnosis of overanxious disorder from *DSM-III-R* in addition to anxiety types from *DSM-IV* (American Psychiatric Association, 1994), due to its unique features, despite its omission from the more recent *DSM-5* manual (American Psychiatric Association, 2013). Such changes in the conceptualization of anxiety types might make it harder to track the prevalence of anxiety disorders over time.

It is important to note that childhood anxiety disorders are often accompanied by other mental health disorders. High comorbidity has been found with for example depression, conduct disorder, and attention deficit/hyperactive disorder (Bittner et al., 2007). In fact, rates of comorbidity are estimated to be approximately 50% for other internalizing disorders like

depression (Beidel & Alfano, 2011). In comparison, anxiety disorders have a lower likelihood of co-occurrence with externalizing disorders; for example, only 11% of twelve-year-olds had a dual diagnosis of an anxiety disorder and an externalizing disorder in a study using a community sample (Yoo et al., 2009).

Definition and Diagnostic Criteria of Childhood Anxiety

Experiences of high anxiety and excessive fear that interfere with everyday functioning and result in behavioral disturbances are core characteristics of anxiety disorders, as defined in *DSM-5* (American Psychiatric Association, 2013). Fear is an emotional response to a known and immediate threat that is accompanied by a physiological arousal and autonomic activation (i.e., fight-or-flight response) or autonomic inhibition (i.e., freeze response; American Psychiatric Association, 2013). Anxiety is an emotional and cognitive response to an unknown threat that is future-oriented and generalized. Both fear and anxiety serve a survival function of alerting to danger (Steimer, 2002). Further labeled under the umbrella term *anxiety*, the *DSM-5* (American Psychiatric Association, 2013) recognizes 12 distinct, although highly comorbid, types of anxiety, each with mutual symptoms of heightened anxiety, fear, and avoidant behavior. The diagnostic criteria for each anxiety disorder differ based on the situations or objects that trigger the anxiety response, which needs to exceed the range of developmentally appropriate anxious presentation and persist for at least 6 months.

During childhood and adolescence, a certain degree of anxiety is considered functional and even beneficial (Owens et al., 2012). In addition to anxiety's protective function, anxiety responses, such as increased vigilance and heightened arousal, play an important role in attention and motivation, and contribute to optimal performance, for example at school or in sports (Lupien et al., 2007). Anxiety's negative impact on functioning is therefore an important

diagnostic criterion for anxiety disorders among children and adolescents (American Psychiatric Association, 2013).

Further, anxiety that is normative at one developmental stage, such as separation anxiety during toddlerhood, could be considered pathological during other developmental stages, such as separation anxiety in adolescence (McClure & Pine, 2006). The *DSM-5* (American Psychiatric Association, 2013) reflects this developmental perspective by organizing the anxiety types by typical age at onset of the diagnosis: separation anxiety (onset in preschool), selective mutism (onset under age 5), specific phobia (between the ages 7 and 11), social anxiety (age 13), panic disorder (between ages 20 and 24), agoraphobia (between ages 25 and 29), generalized anxiety disorder (age 30). However, this organization does not reflect the fact that many anxiety disorders in childhood occur before the typical onset age (Merikangas et al., 2010). For instance, 25% of children between the ages of 8 and 15 years were found to be diagnosed with a generalized anxiety disorder (Bushnell et al., 2019). Additionally, several studies focused on pediatric anxiety disorders note that anxiety disorders typically emerge around age 6, earlier than any other developmental psychopathology (Merikangas et al., 2010). Finally, different types of anxiety disorders in the child and adolescent population are also highly likely to co-occur with each other and, despite high anxious symptomatology and interference with functioning, their anxious presentation might not fall within diagnostic criteria of a specific anxiety disorder. For example, in a sample of 198,450 children between the ages of 3 and 17 years, 55% presented with an unspecified anxiety disorder, a percentage higher than any other anxiety type (Bushnell et al., 2019).

Anxiety is conceptualized as a tripartite model whereas its physiological, cognitive, and behavioral components are engaged in a negative cycle that maintains or increases anxiety

(Beidel & Alfano, 2011). Physiological responses to anxiety (e.g., increased heart rate, rapid breathing) that have frequency and intensity beyond normative fears reinforce a subjective distress conceptualized as a negative cognition (e.g., difficulty maintaining attention, *what if* thoughts with negative scenarios, and repetitive and dominant thoughts about the object of anxiety). The subjective distress/negative cognition is reinforced by a subsequent negative behavioral response that typically presents in a form of avoidant behavior (e.g., avoidance of anxiety-triggering situations) but can also result in an array of dysfunctional behaviors (e.g., difficulty sleeping and/or eating, difficulty starting or completing a task). Concluding the negative cycle, avoidant behavior further reinforces the physiological responses (Beidel & Alfano, 2011).

Anxiety symptoms in children and adolescents manifest more commonly as persistent, excessive, and recurring worries and fears with (e.g., separation anxiety, social anxiety) or without (e.g., generalized anxiety) a specific focus, difficulty controlling anxious thoughts, feelings of apprehension and dread, somatic complaints (e.g., headaches, stomachaches, nausea), heightened physiological arousal (e.g., sweating, shaking, heart palpitations), mood changes (e.g., irritability), and sleep disturbances (American Psychiatric Association, 2013). Linked to anxiety are internalizing symptoms that separate the physiological, cognitive, and behavioral components into anxious/depressed, somatic complaints, and withdrawn behavior domains (Achenbach, 1991). Somatic complaints refer to physiological discomfort/pain that is attributed to emotional distress and withdrawn behavior is characterized by avoidance of triggers that induce anxious response. While somatic complaints and withdrawn behavior were identified as separate clusters of internalizing disorders (Achenbach, 1991), they most commonly manifest within anxiety disorders (Beidel & Alfano, 2011).

The Impact of Childhood Anxiety

Childhood anxiety has been found to have both an immediate and a long-term impact on a child's health and functioning, especially in social and academic domains (ADAA, 2020). Headaches, stomachaches, nausea, vomiting, fatigue, and muscle tensions and muscle pains are common anxiety-related health complaints in children (American Psychiatric Association, 2013). Sleep problems, such as restless sleep, difficulty falling asleep, and nightmares are also more common for children with anxiety (Shanahan et al., 2014). Because of their interference with child's learning processes, such as attention and memory, anxiety symptoms have direct negative impact on academic success and school performance (Langley et al., 2004). Anxious adolescents are likely to engage in defiant behavior and school avoidance (Garland, 2001). Anxiety also has a negative impact on children's and adolescents' social competence and relational skills, as it limits children's ability to successfully handle social situations and form and maintain friendships (Scharfstein et al., 2011).

The long-term negative impact of childhood anxiety on health outcomes has also been documented among adults. It was found that the majority of adults diagnosed with an anxiety disorder experienced the onset of clinically significant anxiety symptoms during childhood (Beidel & Alfano, 2011; Shanahan et al., 2015). The NIMH (2017) estimates that approximately 30% of adults have an anxiety disorder. Additionally, young adults who had been diagnosed with generalized anxiety as children were found to have poor functioning across health (e.g., having a serious or chronic illness), financial (e.g., being impoverished or having a difficulty maintaining employment), and interpersonal (e.g., lower quality of relationships and higher social isolation) domains (Copeland et al., 2014). Similarly, individuals experiencing an anxiety disorder in

adolescence had significantly higher risk of depression, substance abuse, and poor school outcomes at the age of 21 (Woodward & Fergusson, 2001).

High rates of childhood anxiety disorders and the severity of symptoms and their impact on children's functioning throughout their lives demonstrate the need to understand the determinants of anxiety in childhood. Further, given the long-term consequences of childhood anxiety on adult well-being, understanding the determinants of childhood anxiety and the processes that underlie the developmental pathways from childhood into adolescence and adulthood are crucial in identifying ways to alter the course of anxiety trajectories, and therefore, to reduce the rates of anxiety in adulthood. This dissertation will focus on identifying the determinants of anxious developmental pathways as well as on understanding the mechanisms that underlie these pathways as children transition from childhood into adolescence.

Determinants of Childhood Anxiety

Parental influences have been at the center of efforts to understand childhood anxiety etiology (e.g., Chorpita & Barlow, 1998; Ginsburg & Schlossberg, 2002; Manassis & Bradley, 1994). Childhood anxiety is believed to be a result of a child's generalized biological vulnerabilities, such as inherited genetic predispositions, and the child's generalized psychological vulnerabilities to anxiety that originate in family environment (Barlow, 2000). Parents therefore influence a child's anxiety through heredity as well as through family environment, and specifically, parental factors (McLeod et al., 2011). Additionally, the child's biological vulnerabilities might also interact with a specific psychological vulnerability, such as if there is a particularly significant and/or traumatic event that happened to a child (Barlow, 2000).

It is not fully understood whether genetic risk needs to be present for the development of clinically significant anxiety in the presence of severe and persistent adverse environmental factors (such as harsh parenting; McLeod et al., 2011); however, genetic factors are believed to account for approximately 30% of the variance in child anxiety through interactive and additive effects (Eley & Gregory, 2004). In addition to genetic predispositions, recent research has provided evidence that there exists an epigenetic link in the transmission of anxiety disorders from mothers to their children. These studies tested prenatal risks in animal models and found that severe stress during the prenatal period altered the offsprings' ability to adequately regulate their anxiety responses (Keating, 2017). Genetic and epigenetic links therefore provide a partial understanding of childhood anxiety etiology.

When genetic and temperamental vulnerabilities, such as behavioral inhibition, are present, childhood anxiety disorders are believed to develop as a result of multiple risk exposure over time (Vasey et al., 2014). There is a consensus among anxiety researchers that factors related to the family environment, specifically parental influences, significantly contribute to the development and maintenance of anxiety symptoms in childhood (Ginsburg et al., 2004; Ranøyen et al., 2015). For example, parenting dimensions of rejection and overcontrol have been commonly studied as parental risk factors. A meta-analysis of 47 studies found parental overcontrol to have a slightly larger effect on child anxiety than parental rejection (d=.52 compared to d=.41); additionally, low autonomy-granting as a subdimension of overcontrol, was found to have an even larger effect on child anxiety (d=.93; McLeod et al., 2007). Another subdimension of overcontrol, intrusive control, operationalized as taking over a task that a child can complete themselves, has been studied and confirmed in efforts to bring greater specificity to understanding the parenting-child anxiety link (McLeod et al., 2011). However, while parenting

practices have been clearly shown to play an important role in the child anxiety etiology, the same meta-analysis also found that parenting accounted for only 4% of the total variance in childhood anxiety (McLeod et al., 2007).

Thus, other parental factors besides direct parenting practices need to be considered. Several theoretical models of family factors in childhood anxiety highlight the importance of parental anxiety in child anxiety etiology (Ginsburg & Schlossberg, 2002). Maternal anxiety, specifically, has been confirmed by empirical studies to predict child anxiety in both cross-sectional and longitudinal samples (Costa & Weems, 2005; Lebowitz et al., 2016). As mentioned, the parent-child anxiety link is partially explained by genetics (Gregory & Eley, 2007). However, the mechanisms of anxiety transmission from a parent to a child beyond heredity are not yet fully understood (Lebowitz et al., 2016).

Mechanisms of Parent-Child Anxiety Transmission

To date, only one mechanism of parent-child transmission has been identified in the empirical literature, namely cognitive distortions (Lebowitz et al., 2016). The transmission of cognitive distortions from a parent to a child has been found to underlie several parent-child anxiety pathways, such as parental modeling (i.e., parents behaving and parenting in ways consistent with anxious presentation), child social referencing (i.e., children using parental cues as guides for their own behavior), and parenting practices (i.e., such as above-mentioned parental control; Lebowitz et al., 2016). Parents with anxiety were found to have negative cognitive distortions in a form of an interpretation bias that lead to ambiguous situations being interpreted as threatening (Gallagher & Cartwright-Hatton, 2008). A child's interactions with an anxious parent might encourage the child to develop similar cognitive distortions and see problems in a catastrophic manner (McLeod et al., 2011). Child anxiety is then reinforced through parent-child

interactions as desirable, since children look up to their parents' behavior for social referencing – for cues to interpret situations. It is presumed that children develop schemas that might generalize all unknown situations or persons as dangerous (Egliston & Rapee, 2007). The threat bias that is characteristic for anxious parents might also influence the parents' parenting behaviors and lead to limiting of the child's autonomy in order to protect the child. Therefore, interpretation bias associated with parental anxiety is learned by a child through modeling, but it is also directly communicated by a parent through overcontrolling parenting behaviors.

To summarize, children learn to interpret the world in a distorted way from their parents via exposure to multiple parental influences, such as parent-child communication and direct parenting practices (Lebowitz et al., 2016). According to the tripartite model of anxiety, cognitive distortions reinforce physiological responses to anxiety and result in avoidant behavior (Beidel & Alfano, 2011). Physiological responses to anxiety, however, are also accompanied by negative emotional states, such as feelings of irritability, dread, and apprehension, that can result in avoidant behavior and reinforcement of anxiety (Cisler et al., 2010). Therefore, emotion regulation has been recently proposed as a mechanism of parent-child anxiety transmission (Cisler et al., 2010; Nolte et al., 2011).

A recent Interpersonal Stress Regulation Model (Nolte et al., 2011) postulated that ineffective child emotion regulation strategies lead to the development of childhood anxiety and to the reinforcement of child anxious symptomatology over time. According to this model, a child's ineffective emotion regulation strategies are a result of the anxious parents' failure to adequately co-regulate their child's reactivity during stressful experiences (Nolte et al., 2011). To my knowledge, Nolte et al. (2011) theoretical model has not been empirically tested, and empirical studies examining the role of child emotion regulation in anxiety transmission remain

scarce. Study I of this dissertation therefore proposes to study whether child emotion dysregulation can explain the parent-child anxiety link. A confirmation of the importance of emotion regulation processes, in addition to the already known cognitive processes, in the role of parent-child anxiety transmission, would significantly contribute to our understanding of anxiety in families and provide a new target for anxiety family interventions with a goal to reduce anxiety transmission from a parent to a child.

Parental Anxiety Influences in Child Anxiety

Studies examining intergenerational transmission of anxiety from parents to children have mainly built upon theoretical models of childhood etiology that do not distinguish between maternal and paternal anxiety influences (e.g., Ginsburg & Schlossberg, 2002). In these theoretical models, maternal anxiety and paternal anxiety are perceived as interchangeable. While maternal anxiety as a risk factor for childhood anxiety has been substantiated by significant body of empirical evidence (e.g., Drake & Kearney, 2008), the results from paternal anxiety studies are mixed and more research is needed to empirically support the role of paternal anxiety in child anxiety etiology. A theoretical model proposed by Bögels and Phares (2008) highlights the need of separating maternal and paternal anxiety influences on child anxiety because of potential differential impact of mothers and fathers on child's outcomes across different developmental stages (Cabrera et al., 2011, McKinney & Renk, 2008). Thus, in the currently proposed studies, the effects of maternal and paternal anxiety on child anxiety will be differentiated.

Further, there are two important aspects of childhood anxiety etiology that were postulated by the theoretical models, specifically models by Ginsburg and Schlossberg (2002) and by Bögels and Phares (2008), that are rarely examined by empirical studies: the

developmental pathways of childhood anxiety and the reciprocal relations of the parent and the child factors. Parental and child anxiety factors influence childhood anxiety onset and maintenance within the context of child development. As outlined in the *DSM-5* (American Psychiatric Association, 2013), anxious presentations differ based on the age of the child. Furthermore, children are continuously exposed to parental influences and the anxiety models informed by developmental literature (e.g., Bögels & Phares, 2008) suggest that maternal and paternal influences change depending on the age of the child. In order to understand the developmental pathways of anxiety from childhood to adulthood, it is crucial to identify how maternal and paternal factors contribute to the development and maintenance of anxiety symptoms over time. To date, no research studies have tested the potential changes in parental anxiety over time and their effects on the developmental changes in child anxiety as children transition from childhood into adolescence.

Research that would take into account the reciprocity of parental and child anxiety also remains scarce, despite strong theoretical support. Theoretical models of childhood anxiety stress the reciprocity of parental factors and child factors in the development and maintenance of child anxiety symptoms over time (Ginsburg & Schlossberg, 2002; Bögels & Phares, 2008). The models suggest that it is the interaction between factors that ultimately leads to child anxiety. For instance, a temperamentally shy child might have a tendency to withdraw from a caregiver, which in turn might increase the caregiver's anxiety and alter parenting strategies used to parent the child.

To address the above-mentioned gaps in the literature, Study II of this dissertation utilizes a developmental lens and proposes to examine the reciprocal influences of parental and child anxiety over time as children transition from childhood into adolescence. This study will expand

the empirical evidence for the role of parental factors in childhood anxiety by testing theoretical aspects of childhood anxiety models that have been rarely studied. More importantly though, an understanding of developmental trajectories of childhood anxiety and its predictors will also contribute to our understanding of how developmental trajectories of anxiety can be altered and therefore prevented from continuation into adulthood.

Chapter 2: Literature Review

There is a consensus among anxiety researchers that anxiety aggregates in families (Ginsburg et al., 2004). In support of this idea are theoretical models of childhood anxiety etiology (e.g., Chorpita & Barlow, 1998; Ginsburg & Schlossberg, 2002) that emphasized the role of parental anxiety in the development and maintenance of childhood anxiety symptoms. Maternal anxiety, in particular, was found to predict child anxiety by a large body of research (Vasey et al., 2014). Several pathways were also proposed that described how parental anxiety transmitted to children (Lebowitz et al., 2016). Empirically, the most commonly tested have been processes involving the child's acquisition of cognitive interpretation bias that was found to be characteristic of anxious parents (Gallagher & Cartwright-Hatton, 2008). Recently, a theoretical model proposed by Nolte et al. (2011) suggested that in addition to cognitive bias, child emotion regulation might be a potential underlying process in parent-child anxiety transmission. Only a handful of studies considered maternal emotion regulation as a method of transmission, despite the theoretical support for this link (Kerns et al., 2017). Virtually no studies examined child emotion regulation processes as a mechanism of transmission. Study I therefore builds on the model proposed by Nolte and colleagues (2011) and examines the role of child emotion regulation in mother-child and father-child anxiety transmission.

As mentioned, theoretical models of childhood anxiety etiology have hypothesized the role of parental anxiety in child anxiety (Ginsburg & Schlossberg, 2002). However, while maternal anxiety was found to be a predictor of child anxiety in both cross-sectional and longitudinal studies (Lebowitz et al., 2016), very few studies examined the effects of paternal anxiety on child anxiety (Bögels & Phares, 2008). Differentiating between the effects of mothers on child anxiety and of fathers on child anxiety was proposed in a model by Bögels and Phares

(2008). The model suggested that mothers and fathers complete distinct developmental tasks with their children, and thus, their impact on child anxiety needs to be differentiated. For example, maternal anxiety might prevent a mother from providing a child with adequate comfort while paternal anxiety might prevent the father from engaging the child in age-appropriate play (Bögels & Phares, 2008). The deficient experiences in mother-related and father-related developmental tasks might contribute to child anxiety differently. However, very little empirical attention has been paid to the role of paternal anxiety in child anxiety. The evidence for paternal anxiety's effects on childhood anxiety remains mixed at best, perhaps due to the small number of studies examining paternal influences on child anxiety (Connell & Goodman, 2002). Therefore, both Study I and Study II test the effects of maternal and paternal anxiety on child anxiety separately.

Further, there are two other aspects stressed by theoretical models of childhood anxiety etiology (Bögels & Phares, 2008; Ginsburg & Schlossberg, 2002) that received only marginal research attention: developmental perspective on parental and child factors and the reciprocal relationships between parental and child factors. The purpose of a developmental lens is twofold: 1) to understand the impact of parental anxiety on child anxiety trajectories and 2) to distinguish the potentially differential magnitude of effects of maternal and paternal anxiety on child anxiety at different developmental stages. Despite theoretical suggestions that parental influences might differ over time as children transition into adolescence, no research studies have tested the potential changes in parental anxiety over time and their effects on the developmental changes in child anxiety (Bögels & Phares, 2008).

Finally, theoretical models theorized that parental anxiety influences child anxiety and child anxiety influences parental anxiety at any given time (Ginsburg & Schlossberg, 2002).

Empirical studies that examined the role of parental anxiety were predominantly one-directional and focused on the effects of parents on children. Therefore, little is known about the nature of the reciprocal influence and its potential to maintain or increase both parental and child anxiety over time. To address the gaps in literature regarding potential differential roles of mothers and fathers, and regarding the developmental perspective and reciprocal relationships, Study II examines the effects of maternal anxiety changes and the effects of paternal anxiety changes on child anxiety changes as well as the effects of child anxiety changes on maternal and paternal anxiety changes over time as children transition from childhood into adolescence.

This chapter gives an overview of literature relevant to both studies. The first part of this literature review discusses mechanisms of parent-child anxiety transmission and offers theoretical and empirical support for child emotion regulation as a potential mechanism of anxiety symptoms from a parent to a child.

The second part provides theoretical and empirical overview of parental anxiety research and addresses three core concepts that expand current literature on the role of parental anxiety in childhood anxiety etiology: differential role of mothers and fathers, developmental lens, and reciprocity of parental and child anxiety.

Parent-Child Anxiety and Mechanisms of Transmission

The mechanism of anxiety transmission from a parent to a child has been partially explained by heredity (Gregory & Eley, 2007). A child-inherited temperamental vulnerability, specifically in a form of behavioral inhibition, has been examined as a proxy of transmission and a mediator of genetic risk (Eley & Gregory, 2004). Child behavioral inhibition, which is defined as a temperamental predisposition to deficient competence at regulating emotional reactions (Drake & Ginsburg, 2012), has been found to significantly predict child internalizing problems

(Vasey et al., 2014). Heredity and behavioral inhibition, however, do not fully account for variance in child anxiety, since a significant relationship remained between maternal anxiety and child anxiety even after controlling for behavioral inhibition (Hudson et al., 2011). Behavioral inhibition thus explains the link between parental anxiety and child anxiety through interactive and/or additive effects with other mother-, father- and child-related factors being significant as well (Hudson et al., 2011).

In the literature, three key parent-child processes were identified as pathways of intergenerational transmission of anxiety: 1) parental modeling, 2) direct parenting practices, and 3) parent-child communication (Fisak & Grills-Taquechel, 2007). Parental cognitive distortions were found to underlie these pathways, identifying cognitive distortions as a mechanism of parent-child anxiety transmission (Lebowitz at al., 2016). Parents with anxiety were found to have negative cognitive distortions that are typical for anxiety disorders (American Psychological Association, 2013). Specifically, these cognitive distortions in a form of an interpretation bias that impacts perception of situations and filters ambiguous situations as threatening (Gallagher & Cartwright-Hatton, 2008). All three processes explain how children acquire these cognitive distortions.

First, parental modeling as a vehicle of intergenerational transmission is grounded in Bandura's social learning theory (1997). It suggests that children model parental anxious behaviors either through direct observation and replication or through vicarious learning of a parent's tendencies to communicate anxious thoughts and feelings and show avoidant behavior (Drake & Ginsburg, 2012). Since children look to their parents for social cues about how to interpret and behave in unfamiliar situations, parents who respond in an anxious manner model to their children that these situations should be perceived as dangerous. As a result, children

develop similar cognitive distortions as their parents, including a tendency to interpret ambiguous cues as threatening and a tendency to perceive problems in a catastrophic manner (McLeod et al., 2011). It is presumed that such developed cognitive schemas then generalize for children to all unknown situations or persons (Egliston & Rapee, 2007). Furthermore, parental interpretation bias prompts parents to see the world as threatening, and therefore parents are likely to reinforce their children's learned cautious responses as desirable. As such, anxious parents then indirectly reinforce anxiety in their children (McLeod et al., 2011).

Second, acquisition of anxiety symptoms has been attributed to direct parental influences, namely parenting practices. Particularly, parental over-control (i.e., high parental decision-making and overprotection and low autonomy-granting), as well as its subdimension intrusive control (i.e., parental interfering with tasks a child can handle), were found to be strongly and positively associated with anxiety in children (Ballash et al., 2006; McLeod et al., 2011). It is suggested that parents engage in over-control with the intent to protect their child from threatening situations (Hudson & Rapee, 2004). Due to their cognitive distortions, however, anxious parents are more likely to perceive threats where there are none, and their limiting of their child's autonomy has a negative impact on the child's sense of self-efficacy and sense of control (McLeod et al., 2011). Therefore, through overcontrol, parents fail to support their child's autonomy in exploring and learning, and thus parents interfere with the child's normal processes of fear habituation and mastery gaining (Creswell et al., 2011). As a result, children do not gain necessary self-regulatory abilities to handle fear and anxiety, and are likely to utilize strategies, such as avoidance, that further reinforce anxiety.

Finally, cognitive distortions are also transmitted from parent to child as a part of information transfer in parent-child communication. That is, anxious parents who see the world

as threatening will not only model fear responses and promote avoidant behavior, they will also openly communicate and give instructions to the child in line with their anxious thinking and biased perceptions which will lead to the child's internalizing of the fear responses and contribute to the child's anxiety (Fisak & Grills-Taquechel, 2007; Vasey et al., 2014).

As mentioned, all three potential pathways emphasize the role of cognition in both parental and child anxiety, more specifically, cognitive distortion by parents in a form of interpretation bias (Hudson & Rapee, 2004) that is acquired by their children. This is somewhat surprising, since anxiety is classified as emotional disorder (Lewis & Rudolph, 2014), although anxious thoughts have received much more attention both empirically and clinically than anxious feelings (cf. Cartwright-Hatton et al., 2004). Anxious thoughts are represented by worrying thoughts of intrusive nature that revolve around the object of fears, for example, failing or judgment from others. Anxious feelings manifest in emotional experiencing of anxiety, for example feeling afraid, panicked, agitated, restless, irritated, sad, angry (Cisler et al., 2010). According to the tripartite model of anxiety, anxious thoughts reinforce subjective distress/emotional experiencing which leads to avoidant behavior that further reinforces anxious thoughts and subjective distress (Beidel & Alfano, 2011). Emotional experiencing is therefore as critical to understanding anxiety as its cognitive component. Recently, an integrative model of childhood anxiety proposed by Nolte et al. (2011) suggested that emotions in anxiety, and more specifically how emotions are regulated, might play an important role in the parent-child anxiety transmission. This, however, remains to be empirically proven, and therefore Study I examines child emotion regulation as a mechanism for parent-child anxiety transmission.

Interpersonal Stress Regulation Model

The Interpersonal Stress Regulation Model (Nolte at al., 2011) builds on modern attachment theory and emphasizes the role of emotion regulation in parent-child relationships (Schore & Schore, 2008). Emotion regulation can be defined as extrinsic and intrinsic processes that are responsible for monitoring, evaluating, and modifying of emotional reactions in order to engage in goal-oriented behavior (Thompson, 1994). Emotion regulation constitutes of complex controlling mechanisms that, if maladaptive, significantly contribute to child psychopathology, including the anxiety symptomatology (McClure & Pine, 2006).

The Interpersonal Stress Regulation Model (Nolte at al., 2011) offers an integrative perspective that puts parent-child interaction at the heart of intergenerational anxiety transmission. Specifically, the way a parent responds to the child's bids for attention and the way the child approaches the parent to communicate their needs play a key role in the development of anxiety disorders and in the mechanism of anxiety transmission from a parent to a child. Drawing from the attachment theory formulated by Bowlby (1988) and recently expanded by Schore and Schore (2008), a child turns to a parent for comfort in a time of distress, and the child communicates the need for comfort verbally and/or non-verbally via bids for the parent's attention. If a parent responds to the child's bids successfully, the child's distress is soothed and an adequate emotion co-regulation between a parent and a child occurs (Mikulincer et al., 2003). However, an anxious parent is likely to respond to the child's bids in an inconsistent, unpredictable, and anxious manner, which leads to exacerbation of the child's distress (Schore & Schore, 2008). Since the child's distress is not soothed, but heightened, the child employs hyperactivating strategies in an effort to relieve distress; thus, the child increases their vigilance for comfort cues from their parent and increases the bids for attention (Nolte et al., 2011). The

Interpersonal Stress Regulation Model (Nolte et al., 2011) posits that if a child's experienced stress as a result of unsuccessful co-regulation within early caregiving environment is persistent, it has a direct impact on a child's physiological responses and causes functional changes in the child's brain (Nolte et al., 2011). Specifically, persistent stress leads to over-functioning of the hypothalamic–pituitary–adrenal (HPA) axis that becomes overactive and dysregulated. If stressors in the environment persist, the HPA-axis becomes chronically hyperactivated, predisposing a child to hypervigilant responses and biased-toward-negative interpretations of ambiguous or unknown stimuli which leads to anxiety (Taylor et al., 2011).

An anxious parent's unsuccessful co-regulation therefore results in the child's heightened and prolonged exposure to stress; however, it also prevents the child from learning adequate emotion regulation strategies via parent co-regulation. As mentioned before, in early childhood, children learn to regulate through co-regulation with their primary caregivers, therefore, emotion regulation skills are of interpersonal nature (Schore & Schore, 2008). In other words, parents who have not developed adequate emotion regulation skills themselves, cannot effectively co-regulate their children's emotions. Since children acquire their emotion regulation skills through socialization and co-regulation in early childhood (Eisenberg et al., 1998), the ineffective emotion regulation skills are passed down from parents to their children.

Adequate emotion regulation skills are crucial in child's successful regulation of stress (Ragen et al., 2016). Chronic hyperactivation is therefore a consequence of inadequate co-regulation provided by the caregivers that is further reinforced by stressful caregiving environment. Prevalent and consistent experience of inadequate co-regulation of stressors leads to anxiety as a predominant mode of stress regulation. The acquisition of adequate stress regulation skills – emotion regulation skills included - through the effective co-regulation

strategies within the secure attachment experiences has, on the other hand, the ability to serve as a protective factor in a face of other anxiety determinants (Nolte et al., 2011).

The Role of Emotion Dysregulation in Anxiety Transmission

The interpersonal stress regulation model (Nolte et al., 2011) postulated that children learn their regulatory strategies from their parents and that the child's learned inability to regulate their stress leads to anxiety. The link between parental anxiety and parental emotion regulation proposed by this model has empirical support in the literature. In particular, two experimental studies showed the links between maternal anxiety and child anxiety and the mother's dysregulated emotional response. In an experimental study by Kerns et al. (2017), 45 anxious mothers were asked to listen to a recording of a child in distress and indicate at what point they would comfort the child. The mothers, whose children were between the ages of 3 and 8, also completed anxiety assessments by reporting on their own and their child's anxiety symptoms. Maternal emotional dysregulation, in the form of higher negative affect and ineffective use of multiple emotion regulation strategies during the child's distress, were found to be associated with greater child anxiety. Since the indirect effect of maternal anxiety on child anxiety through maternal emotion regulation was confirmed, the authors suggested that helping anxious mothers to acquire effective emotion regulation skills might be useful in the treatment of child anxiety (Kerns et al., 2017).

Another experimental study compared maternal responses of 44 clinically anxious and 44 non-anxious mothers (Creswell et al., 2013). While observing interactions of clinically anxious children and clinically anxious mothers during a challenging task, anxious mothers reported experiencing more negative emotions and higher levels of anxiety when observing the child's struggles (Creswell et al., 2013). The higher levels of maternal negative emotions suggest that

anxious mothers have difficulty tolerating their children's negative emotions; therefore, the authors believed that strategies geared towards parental acceptance of negative emotions might be a successful supplement to current cognitive-behavioral therapeutic approaches to child anxiety treatment (Creswell et al., 2013).

Evidence from these studies shows that anxious mothers struggle with emotion regulation processes and, as a result, often respond to their anxious children in a manner that has tendency to reinforce the child's anxiety, rather than soothe it. As such, it appears that maternal emotion regulation is relevant to child's emotion regulation, and subsequently, to child's anxiety through ineffective regulation. In a rare study investigating the role of emotion dysregulation in intergenerational transmission of anxiety, parental emotion dysregulation was found to mediate the link between parental anxiety and child internalizing problems in a sample of 89 Chinese parents and their school-aged children (Han et al., 2016). The authors suggested that emotion regulation strategies might be partially genetically transmitted as well as acquired in the process of socialization. Interestingly, this study analyzed separate mediation models for internalizing problems and for different types of anxious symptomatology (e.g., separation anxiety, generalized anxiety, school anxiety, and social anxiety) and found that the mediation models were only supported for internalizing symptoms and separation anxiety. The authors speculated that perhaps a developmental lens needs to be considered; school-aged children's generalized, social, and school anxiety might be more peer-oriented whereas separation anxiety is more parent-oriented (Han et al., 2016).

In the empirical literature, maternal emotion regulation is often connected to the concept of family accommodation through the parent's difficulty to tolerate the anxious child's distress that leads to reinforcement of the child's anxiety. In a sample of 48 mothers of children between

3 and 8 years of age, Kerns et al. (2017) found that maternal anxiety positively predicted child anxiety via ineffective maternal emotion regulation and greater family accommodation pathways. Family accommodation represents all behaviors that family members, and parents, in particular, engage in order to help the child avoid anxious distress (Norman et al., 2015). Research focusing on neural networks showed that mothers responded to a child's distress with greater cortisol reactivity and hyperactivation of amygdala and hypothalamus, therefore experiencing greater distress themselves. Attempts to accommodate the child and ease the child's distress thus might be attempts to help with the mothers' own distress, that they struggle to soothe due to their poor emotion regulation skills (Norman et al., 2015).

Interestingly, most empirical research has focused on the role of parental emotion regulation in the parent-child anxiety link. However, child emotion regulation has also been robustly found in the literature to predict child anxiety symptoms (Loevaas et al., 2018) and maladaptive emotion regulation strategies have been found to be present for children across different anxiety diagnoses (Keil et al., 2017). In their chapter discussing social anxiety and emotion regulation, McClure and Pine (2006) make the point that it is not the presence of anxiety and fear, but rather a deficiency in regulatory processes involved in handling the anxiety and fear reactions that are critical in anxiety becoming a disordered experience. One of very few studies that examined parental anxiety and child emotion regulation utilized an infant sample (Kaitz et al., 2010). Mother-child interactions and infant emotion dysregulation were recorded for 34 anxious mother-child dyads and 59 non-anxious mother-child dyads. Infants of anxious mothers were found to show more inhibited and flattened affect, which, as the authors hypothesized, could be a way of handling the mother's anxious presentation (Kaitz et al., 2010).

A theoretical link between parental anxiety, child emotion dysregulation, and child anxiety has been clearly formulated (Nolte et al., 2011), and there is empirical evidence that maternal anxiety is related to maternal emotion regulation and child anxiety (Creswell et al., 2013) and child emotion regulation is related to child anxiety (Keil et al., 2017). However, no studies so far tested the hypothesis that child emotion dysregulation is an important underlying process in transmission of parent-child anxiety. Study I therefore builds on Nolte et al.'s (2011) model and proposes to examine the role of child emotion dysregulation in the anxiety transmission from both the mothers and the fathers to their children.

The Role of Parental Anxiety in Childhood Anxiety Etiology

Parental anxiety as a determining factor of child anxiety symptoms development during childhood was proposed by several theoretical models (Ginsburg et al., 2004). Two of these models – a family model postulated by Ginsburg and Schlossberg (2002) and a model postulated by Bögels and Phares (2008) also discussed factors that played a role in how parental anxiety impacted child anxiety. Specifically, Ginsburg and Schlossberg's (2002) model highlighted the reciprocity of parental and child anxiety in a context of the child's development. The importance of developmental lens was also stressed by the Bögels and Phares' (2008) model, that, furthermore, proposed to distinguish maternal and paternal anxiety and their differential impact in the course of child development. Despite a large body of literature studying parental anxiety and child anxiety, neither one of these aspects have received much empirical attention. In this section, I will review the theoretical models and related empirical literature followed by a discussion about the importance of studying the differential impact of mothers and fathers while considering the developmental lens and consideration and the reciprocity of a parent-child anxiety relationship.

Ginsburg and Schlossberg's (2002) model delineated several child factors and parent factors as determinants of childhood anxiety and stressed reciprocal relationships between these factors. Inspired by an older model by Manassis and Bradley (1994) that integrated temperamental and attachment perspectives on childhood anxiety, the family model considered temperament and attachment to be the stepping stones for other family factors. For a child, early behavior inhibition and insecure attachment to a parent were hypothesized to lead to a higher sympathetic arousal that lead to social and behavioral withdrawal and subsequent peer rejection. Such negative experiences with parents and peers were hypothesized to result in acquisition of cognitive distortions and maladaptive coping strategies that eventually resulted in child anxiety. For a parent, parental behavioral inhibition as a temperamental vulnerability and insecure attachment history were hypothesized as resulting in parental anxiety that lead to anxiety-enhancing parental behaviors. Anxiety-enhancing parental behaviors, for example, anxious modeling, overcontrolled parenting, and accommodation of child's anxiety behaviors, then reinforced and enhanced child anxiety, and therefore interfered with a child's adaptive cognitive, emotional, and social development (Ginsburg & Schlossberg, 2002).

In addition to the child and the parent factors being in reciprocal relationships with each other, both child and parent factors were hypothesized to interact with several contextual factors, such as marital discord, demographic factors, environmental stressors, and social support. Child and parent factors, their reciprocal relationships and their interactions with contextual factors were hypothesized to be impacted by child's developmental trajectory that was defined in this model as developmental events and cognitive maturation (Ginsburg & Schlossberg, 2002).

Despite its comprehensive attempt to capture all relevant family factors, the discussed family model (Ginsburg & Schlossberg, 2002) did not consider potential differential roles of

mothers and fathers in childhood anxiety etiology. This was remedied by a model proposed by Bögels and Phares (2008) that focused exclusively on distinguishing the mother and the father role in childhood anxiety etiology. Based on empirical data about differing child-rearing practices, Bögels and Phares' (2008) model postulated that a father's contribution to the child's anxiety differs from the mother's as the child progresses through the developmental stages. Specifically, the authors suggested that parents' experienced anxiety differentially prevents them from appropriately engaging with their child in important developmental tasks. Since developmental tasks vary for mothers and fathers during the course of the child's development, the influence of maternal anxiety and of paternal anxiety on child anxiety manifests differently (Bögels & Phares, 2008). For example, during infancy and early preschool years (0-4 years), paternal anxiety might prevent fathers from engaging in tasks related to developmentally appropriate challenge and play by encouraging less risks or having a flattened affect when playing with a child. Maternal anxiety might, on the other hand, prevent mothers from providing appropriate care. During adolescence, paternal anxiety might prevent the father from helping their child transition into the outside world while it might hinder the mother from providing enough autonomy for their child (Bögels & Phares, 2008).

The model proposed by Bögels and Phares (2008) has several drawbacks, the main one being that it is not offer any specificity with regard to pathways of parent-child anxiety transmission. It describes that a child's anxiety is impacted by preventing the parent from engaging in developmental tasks, however, it does not explain how the child's anxiety is affected. It does not discuss whose parental anxiety might potentially have a larger impact and at what developmental stage or whether both parents contribute to the child's anxiety the same, just through different mechanisms. The authors themselves suggested their model was preliminary

and needed empirical investigation (Bögels & Phares, 2008). Nevertheless, this model is noteworthy since it is the only family factors model that clarifies the potential differences between the mother and the father role, and it does so using a developmental perspective. Informed by this model, Study II of this dissertation will examine the effects of maternal anxiety and the effects of paternal anxiety on child anxiety separately.

Both discussed models of childhood anxiety identified parental anxiety as an important factor in the development and maintenance of child anxiety (Bögels & Phares, 2008; Ginsburg & Schlossberg, 2002). Supporting the idea that anxiety aggregates in families and that it is transmitted intergenerationally, substantial empirical evidence showed maternal anxiety to be a significant predictor of child anxiety (Costa & Weems, 2005; Drake & Kearney, 2008; Vasey et al., 2014). Mothers of anxious children were diagnosed with an anxiety disorder almost three times more likely than mothers of non-anxious children (Cooper et al., 2006). When examining the role of maternal anxiety in childhood anxiety etiology, multiple studies clearly confirmed maternal anxiety as a predictor of child anxiety, regardless of their cross-sectional or longitudinal nature and the age of the children sampled (Burstein et al., 2010; Creswell et al., 2013). For example, in a cross-sectional study of over 800 adolescents, anxiety disorder of mothers as well as anxiety/depression dual diagnosis were found to predict adolescent anxiety (McClure et al., 2001). This study found no relationship between a depression only diagnosis of mothers and adolescent anxiety, suggesting that the effects of maternal anxiety on child anxiety are unique and not interchangeable with any other internalizing disorder (McClure et al., 2001). In another cross-sectional study, significant associations between maternal anxiety and child anxiety were found for 88 children aged 6 to 17 (Costa & Weems, 2005).

Several longitudinal studies analyzed the effects of maternal anxiety more broadly by examining the impact of maternal internalizing symptoms of anxiety and depression on child outcomes. For example, a study of 921 Norwegian families found that maternal anxiety and depression predicted child externalizing problems, such as temper tantrums and lying, that began to manifest when the children were only 18 months old (Nilsen et al., 2013). This study did not examine child anxiety outcomes; however, early maternal psychopathology was found to predict later adolescent depression through a pathway of earlier exhibited externalizing behaviors. Similarly, trajectories of maternal psychological distress operationalized as maternal anxiety and depression were tracked in another Norwegian sample of 400 participants from childhood into adulthood. Consistently high maternal distress levels during childhood and adolescence predicted child anxiety and depression outcomes in young adulthood (Nilsen et al., 2016).

Such finding confirms the necessity to look at the effects of maternal anxiety on child outcomes developmentally. Results from these studies suggest that maternal anxiety has a potential to predict child anxiety past childhood and into young adulthood; however, more research is needed to understand the potential differential effects of maternal and child anxiety at different time points during child development. Study II will therefore examine the longitudinal effects of maternal anxiety on child anxiety at four time points between the ages of 6 and 15.

The effect of paternal anxiety on child anxiety has received much less research attention compared to maternal anxiety. Research focused on paternal factors in child anxiety etiology has only started to get more attention in last five years, comparatively late to maternal factors that have been studied for the past twenty tears (cf. Ginsburg et al., 2004). There are only a handful of studies that solely examined the role of paternal anxiety in child anxiety etiology; the majority of studies also examined the role of mothers, although the two influences were tested separately.

This was the case in an Australian study of over 800 adolescents whose mothers were diagnosed with depression (McClure et al., 2001). The results showed that maternal, but not paternal, anxiety was found to be a significant predictor of adolescent anxiety. Authors of this study found it surprising that there were no effects found for fathers and argued that perhaps gender bias prevented fathers from accurately reporting their anxiety symptoms (McClure et al., 2001).

Similar results were found by a meta-analysis that separately examined associations between maternal and paternal psychopathology and child internalizing behaviors and found small effects for both mothers and fathers, with the effects of mothers being slightly stronger (Connell & Goodman, 2002). However, after teasing out the effects of parental mental health disorders, such as depression, bipolar disorder, schizophrenia, and substance abuse, maternal anxiety was found to have a large effect on child internalizing behaviors while paternal anxiety showed no effect (Connell & Goodman, 2002). The authors of this meta-analysis argued that these findings were potentially a result of higher prevalence of anxiety disorders in females, and therefore, children were more likely to be exposed to more significant anxiety symptoms from their mothers than from their fathers.

Weak or no effects of paternal anxiety on child anxiety found in empirical literature are somewhat surprising since there is no theoretical basis discussed in the anxiety literature that would suggest anxious mothers are substantially more influential than anxious fathers. Possibly, the low number of empirical studies focusing on paternal anxiety could be contributing to the inconclusiveness of findings. To illustrate, there were only eight studies included in the Connell and Goodman (2002) meta-analysis that examined paternal anxiety compared to 24 studies that examined maternal anxiety. Even though this meta-analysis is of an older date, a recent meta-analysis focused on parenting practices demonstrates that focus on father influences has not

increased much between 2002 and 2016. Out of 31 studies included, 28 effect sizes were reported for mothers and 12 for fathers, in samples consisting of 5,728 mothers and children and 1,019 fathers and children, respectively. No studies included in this meta-analysis examined fathers alone (Möller et al., 2016).

In a rare examination that focused on paternal anxiety only, there was no direct effect of paternal anxiety on child anxiety, when controlling for parenting behaviors, specifically for encouragement of independence and overprotection (Gibler et al., 2018). The effects were tested using an actor-partner interdependence model in a sample of 94 fathers and their children. However, this study tested the effects of paternal anxiety for 12- to 30-month-olds and, and anxiety was therefore operationalized as the infants' inhibited temperament. Even though the authors argued that inhibited temperament is a proxy for anxiety at such young age (Gibler et al., 2018), inhibited temperament without other symptoms of anxiety and an impact on functioning, for example in a form of inconsolable crying, may simply refers to child's shy nature and not anxiety.

Commonly, empirical studies do not differentiate between the maternal and the paternal effects on child anxiety. Such mixed mother-father studies consider the maternal and paternal influences to be interchangeable; however, they might be underestimating or overestimating the parental effects at any given developmental stage of a child. Parental studies that do not distinguish the parental roles often have child samples with a wide age range, which further raises questions about the specificity of parent-child anxiety relationships at different developmental stages. To illustrate, parental anxiety was found to significantly predict child anxiety and depression in a sample of 97 parents and their children aged 6 to 14 years (Burstein et al., 2010). A similar study considered parental internalizing problems (i.e., anxiety,

depression, phobia, obsessive-compulsive behaviors) and found them to significantly predict child anxiety in a community sample of 157 7- to 18-year-olds (Drake & Kearney, 2008).

While such studies demonstrate the importance of parental anxiety in childhood anxiety etiology, they contribute little to the understanding of the unique effects of maternal and paternal anxiety on child anxiety at different developmental stages. Rather, studies like these imply that parental anxiety impacts child anxiety equally regardless of whether the child is in preschool age or in adolescence; although, it is important to note that these studies were cross-sectional and did not examine the predictive effects of parental anxiety over time. The role of parental anxiety in child anxiety etiology, however, cannot be fully understood without teasing out unique parental as well as developmental effects. Any potential implications of the research results would be hard to make, for example, for family anxiety treatment, if there is not a clear understanding of how and when mothers' and fathers' anxiety influences children's anxiety at different developmental stages.

The Differential Roles of Maternal and Paternal Anxiety

Drawing upon the Bögels and Phares (2008) model that highlighted the importance of studying the effects of maternal anxiety and of paternal anxiety on child anxiety separately, Study II will test the extent to which maternal anxiety and paternal anxiety are equally relevant or whether they each uniquely contribute to child anxiety. As outlined in the model proposed by Bögels and Phares (2008), a possible explanation of maternal and paternal anxiety's differential impact might lie in the unique roles that mothers and fathers play in a child's life. For example, a recent longitudinal study of 480 adolescents that examined the unique roles of mothers and fathers in adolescent emotion regulation showed that maternal support and paternal behavioral control were associated with adolescent emotion regulation (Van Lissa et al., 2019). The authors

suggested that their results reflected a notion that during adolescence, mother-child relationships were of supportive nature and father-child relationship were of activation nature (Paquette, 2004). There are currently no similar studies of parental and child anxiety; however, since adequate emotion regulation skills are relevant for anxiety development (McClure & Pine, 2006), similar influence of unique mother and father roles could be expected.

Another explanation is that since direct maternal practices were found to be more influential in younger children than older children (McLeod et al., 2007), perhaps, paternal influence becomes stronger for children as they transition into middle childhood and adolescence. Developmentally, children form closer bonds with their primary caregivers during early childhood, which are usually mothers (Lewis & Lamb, 2003). As children mature and transition into middle childhood, fathers become important role models for social referencing, and therefore, the impact of paternal anxious modeling might be larger (McLeod et al., 2007). In support of this developmental perspective is a recent study that showed maternal over-control (i.e., excessive parental regulation of child's thoughts, feelings and behaviors) played a unique role in anxiety among elementary-school children and paternal over-control played a unique role in anxiety of adolescents (Verhoeven et al., 2012). This study compared parenting practices of 179 parents with their elementary-school aged children and 127 parents with their adolescent children who were recruited from Dutch schools. The authors argued that their findings showed fathers to be of a higher importance to children in adolescence. During adolescence, children's developmental task is to seek autonomy and turn to the outside world; the authors argue that fathers represent this outside world. If fathers are more controlling, they send a signal to their adolescent children that the world is not a safe place, which in turn reinforces adolescents' anxiety symptoms (Verhoeven et al., 2012).

Such findings illustrate that maternal and paternal roles need to be clearly distinguished and, given that maternal and paternal tasks and their impact on child anxiety vary in the course of child development, this distinction needs to be made within the context of child development.

The Need for Developmental Lens

Developmental aspects of child anxiety are therefore critical to consider in order to understand the intersections of parental and child anxiety. A developmental perspective is also needed to establish potential continuity or discontinuity between earlier and later child symptomatology and factors that potentially predict this continuity over time, such as parental anxiety. Sameroff (2004) suggests that in order for any disorder to maintain its continuity over time, the experiences that contribute to the disorder need to continue at the same level or increase. Increases in the disordered experience then lead to child's poorer regulatory skills and lowered level of adaptive functioning (Sameroff, 2004). It is possible that exposure to parental anxiety over time will maintain, if not increase, a child's anxious symptomatology. However, the current empirical literature studying the effects of parental anxiety on the developmental pathways of child anxiety seems to be missing. Studying the effects of parental anxiety on child anxiety over time is crucial in order to understand how child anxiety maintains and/or increases during childhood and into adolescence. Such an understanding will inform family intervention efforts focused on altering developmental pathways of child anxiety and on preventing them from continuing into adulthood. To consider the developmental perspective and to determine the continuity or discontinuity of child anxiety over time, Study II will examine potential developmental changes in child anxiety as they are predicted by changes in maternal anxiety and changes in paternal anxiety over time.

The Reciprocity of Parent-Child Anxiety Relationship

Finally, the vast majority of parental anxiety studies examine a one-directional influence from a parent to a child, even though theoretical models highlight the transactional nature between family factors and child anxiety. For example, Manassis and Bradley (1994) described how a child's temperament influences parental responses to child's needs, and therefore, the parent-child relationship. Similarly, Ginsburg and Schlossberg's (2002) model stressed reciprocal relationships between parent-related anxiety factors and the child-related anxiety factors.

Research that takes bidirectional effects of parental anxiety on child anxiety and vice versa into the consideration remains scarce. In one of the few exceptions, in a study of 88 mothers and their children aged between 7 and 12 years, anxious and non-anxious mothers were observed during a challenge task activity that their children were completing. Maternal reactivity in a form of raised anxiety to a display of an anxious child's behaviors - observed by maternal anxious facial expressions, body movements, and speech as well as maternal displays of overprotective and intrusive behavior during the task - demonstrated that there are bidirectional effects of parent-child anxiety (Creswell et al., 2013). Specifically, anxious mothers anticipated having difficulty with their reactivity if their child struggled with a task; once their child expressed anxiety during the task, the anxious mothers were more likely to rate their emotions during task negatively. The findings of this experimental study clearly showed that mothers were impacted by their children and children were impacted by their mothers (Creswell et al., 2013).

The importance of a reciprocity has been supported in clinical research examining the effectiveness of anxiety treatment. Children who received combined cognitive behavioral therapy and parental anxiety management treatment were more likely to maintain anxiety

symptoms below clinical significance at three-year follow-up than children who received child-focused treatment only (Cobham et al., 2010). The higher effectiveness of joint parent-child treatment clearly demonstrates that parental anxiety and child anxiety are intertwined.

Given the theoretical background and some empirical evidence that suggests that individual family members' anxieties reinforce each other, which further supports the notion that anxiety aggregates in families (Ranøyen et al., 2015), it is necessary to consider the bidirectional effects when studying the impact of parental anxiety on child anxiety. However, since current empirical evidence is scarce, Study II proposes to examine the potential differential effect of maternal and paternal anxiety on child anxiety and the reciprocal effects of child anxiety on maternal and paternal anxiety over time.

Study Purpose

Study I: Child Emotion Dysregulation as a Mediator of Parent-Child Anxiety

The purpose of the proposed longitudinal study is to determine whether child emotion dysregulation can be considered a mediator in the intergenerational transmission of anxiety in families. Although child emotion dysregulation has been found to predict child anxiety by several studies (Keil et al., 2017), the role of child emotion dysregulation in parent-child transmission has never been examined. Instead, studies examining the mechanisms of transmission largely focused on the role of cognition, specifically, on a child's acquisition of cognitive distortions from a parent (McLeod et al., 2011). However, since there is evidence that children learn their emotion regulation strategies from their parents through co-regulation in attachment relationships (Nolte et al., 2011), it is likely that children will acquire poor regulatory skills that contribute to their anxiety from their parents. Therefore, child emotion dysregulation appears to be an important underlying process of intergenerational anxiety transmission, in

addition to cognitive distortions. Currently, no empirical studies have tested child emotion dysregulation as a mediator of parent-child anxiety link over time.

Identification of intergenerational anxiety transmission mechanisms in families allows for a better understanding of how anxiety aggregates in families and how children's exposure to parental anxiety results in children acquiring anxiety symptoms themselves. Such understanding will expand options for anxiety treatment in families by offering new targets for effective treatment. For example, if child emotion regulation is confirmed to play a significant role, interventions targeting child emotion regulation strategies might prove to be successful in the treatment of child anxiety.

This study further offers greater specificity in understanding maternal and paternal anxiety and their roles in intergenerational anxiety transmission. Currently, there are also no studies that would differentiate between the role of a mother and the role of a father when it comes to the parent-child anxiety link. Theoretical models suggest that mothers and fathers differ in how their anxiety impacts the anxiety of their child (Bögels & Phares, 2008) over time. It might be of a particular importance to identify distinct impact of a maternal anxiety in comparison to a paternal anxiety when it comes to anxiety transmission, since making the assumption that both parents contribute equally might result in misinformed, and therefore, ineffective treatment strategies.

This study will examine child emotion dysregulation as a potential mediator of the relationship between parent anxiety and child anxiety over time. Separate models for maternal and paternal anxiety will be tested to determine whether differences exist in the transmission mechanism for mothers and fathers. In a third and final model, both maternal and paternal pathways will be tested to determine unique, partially redundant, or fully redundant effects. By

testing the emotion dysregulation as a potential mechanism of transmission of anxiety from a parent to a child, this study will significantly contribute to the anxiety literature by expanding on the current knowledge of what is considered to be the vehicle of child anxiety symptoms acquisition in the parental context. Further, given that the effects will be tested longitudinally and separately for mothers and for fathers, this study will significantly expand the current understanding of specificity of mother-child and father-child anxiety transmission.

Research questions/hypotheses.

R1.1: Does maternal anxiety at Age 6 positively predict child anxiety at Age 15 (Figure 1)?

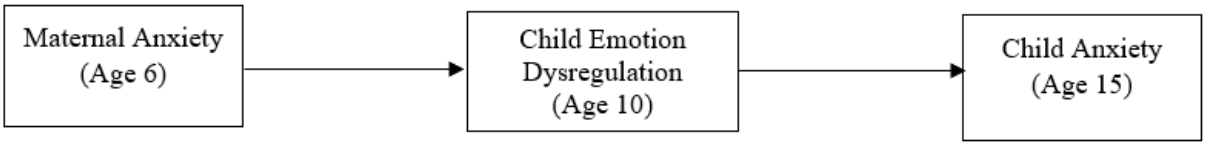
R.1.2: Does child emotion dysregulation at Age 10 mediate the relationship between maternal anxiety at Age 6 and child anxiety at Age 15?

H1.1: Maternal anxiety at Age 6 is expected to positively predict child anxiety at Age 15.

H1.2: Child emotion dysregulation at Age 10 is expected to mediate the relationship between maternal anxiety at Age 6 and child anxiety at Age 15.

Figure 1

Child Emotion Dysregulation Mediates the Relationship Between Maternal Anxiety and Child Anxiety over Time



R2.1: Does paternal anxiety at Age 6 positively predict child anxiety at Age 15 (Figure 2)?

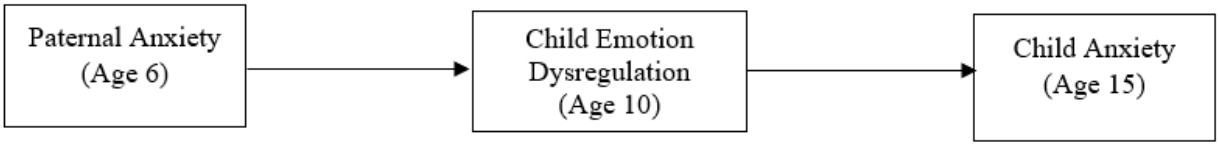
R.2.2: Does child emotion dysregulation at Age 10 mediate the relationship between paternal anxiety at Age 6 and child anxiety at Age 15?

H2.1: Paternal anxiety at Age 6 is expected to positively predict child anxiety at Age 15.

H2.2: Child emotion dysregulation at Age 10 is expected to mediate the relationship between paternal anxiety at Age 6 and child anxiety at Age 15.

Figure 2

Child Emotion Dysregulation Mediates the Relationship Between Paternal Anxiety and Child Anxiety over Time



R3.1: Does paternal anxiety at Age 6 positively predict child anxiety at Age 15 above and beyond the effects by maternal anxiety (Figure 3)?

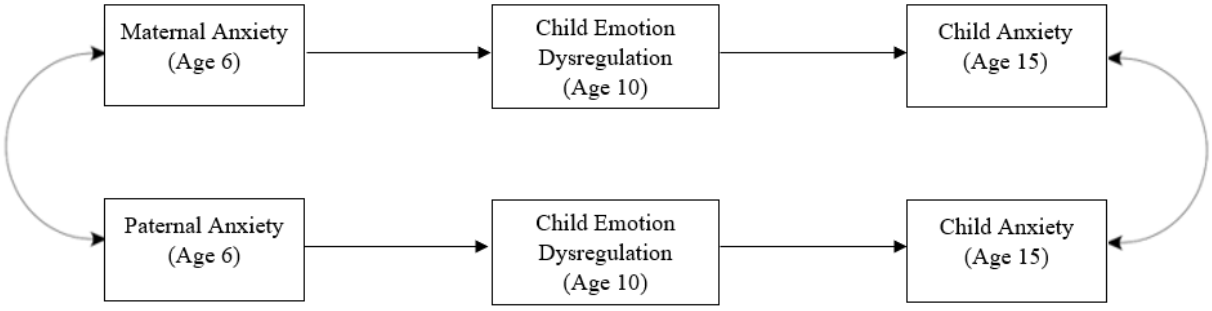
R3.2: Does child emotion regulation at Age 10 mediate the relationship between paternal anxiety at Age 6 and child anxiety at Age 15 above and beyond the effects by maternal anxiety?

H3.1: Paternal anxiety at Age 6 is expected to positively predict child anxiety at Age 15 above and beyond the effects by maternal anxiety.

H3.2: Child emotion regulation at Age 10 is expected to mediate the relationship between paternal anxiety at Age 6 and child anxiety at Age 15 above and beyond the effects by maternal anxiety.

Figure 3

Child Emotion Dysregulation Mediates the Relationship Between Maternal Anxiety and Child Anxiety and Paternal Anxiety and Child Anxiety over Time



Study II: Bidirectional Effects of Parent-Child Anxiety from Early Childhood into Adolescence

Theoretical models and parental anxiety literature reveal three important aspects of parent-child anxiety that are overlooked in current empirical literature, namely differential impact of maternal and paternal anxiety on child anxiety, developmental lens, and reciprocal relationships of parent-child anxiety. Study II proposes to examine these three aspects by testing the bidirectional longitudinal effects of maternal and paternal anxiety and child anxiety from early childhood into adolescence.

Finding a study that examined maternal and paternal effects on child anxiety separately is rare. A significant body of literature has examined maternal anxiety and its impact on child anxiety at different ages and across populations (e.g, Costa & Weems, 2005), and there is strong evidence in support of maternal anxiety as a predictor of child anxiety (Vasey et al., 2014). Research into paternal anxiety, however, has been scarce and its results mixed (Bögels & Phares, 2008). Drawing upon the theoretical models that distinguish mother and father roles and their unique impact on child anxiety at different stages of child development (Bögels & Phares, 2008), the proposed study attempts to address this gap in the literature by testing the effects of maternal anxiety on child anxiety and of paternal anxiety on child anxiety both separately and together. Maternal and paternal effects on child anxiety will be tested simultaneously to determine whether there exists unique, partially redundant, or fully redundant paternal effects, above and beyond maternal effects.

Further, theoretical models suggest that it is necessary to consider developmental perspective when examining differential impact of mothers and fathers since the impact of maternal and paternal anxiety might vary in how and how much it interferes with a child's

cognitive, emotional, and social development at different development stages (Bögels & Phares, 2008) and therefore contribute to child anxiety in unique ways. Thus, Study II will examine how changes in maternal and paternal anxiety impact child anxiety at four time points that capture a child's developmental transition from early childhood (Age 6) into adolescence (Age 15).

Finally, theoretical models (e.g., Ginsburg & Schlossberg, 2002) as well as the results from experimental studies suggest that, while maternal anxiety predicts child anxiety, anxious children's behavior also increases maternal reactivity and, subsequently, maternal anxiety (Creswell et al., 2013). However, considerations of the bidirectional effects of maternal anxiety on child anxiety over time have been scarce. Additionally, virtually no studies have examined potential bidirectional effects of paternal anxiety and child anxiety. This study therefore examines the effects of maternal and paternal anxiety on child anxiety as well as child anxiety on maternal and paternal anxiety.

Understanding developmental changes in child anxiety and its predictors over time will contribute to current knowledge about parent-child anxiety transmission and about the continuous trajectories of child anxiety into adulthood. Such understanding will also identify the targets of intervention in order to alter child anxiety trajectories and potentially prevent them from continuing into adulthood.

Research questions/hypotheses.

R1.1: Do maternal anxiety and changes in maternal anxiety predict developmental changes in child anxiety from Age 6 until Age 15 (Figure 4)?

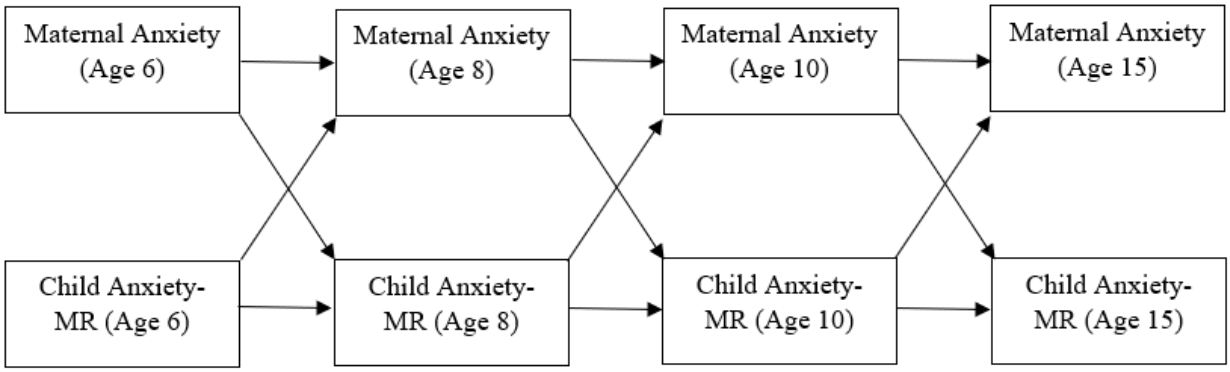
R1.2: Do child anxiety and developmental changes in child anxiety predict changes in maternal anxiety from Age 6 until Age 15?

H1.1: Maternal anxiety and changes in maternal anxiety are expected to positively predict the developmental changes in child anxiety at Age 8, Age 10, and Age 15.

H1.2: Child anxiety and developmental changes in child anxiety are expected to positively predict changes in maternal anxiety at Age 8, Age 10, and Age 15.

Figure 4

Cross-lagged Model of Maternal Anxiety and Child Anxiety from Age 6 until Age 15



Note. MR= Mother report.

R2.1: Do paternal anxiety and changes in paternal anxiety predict developmental changes in child anxiety from Age 6 until Age 15 (Figure 5)?

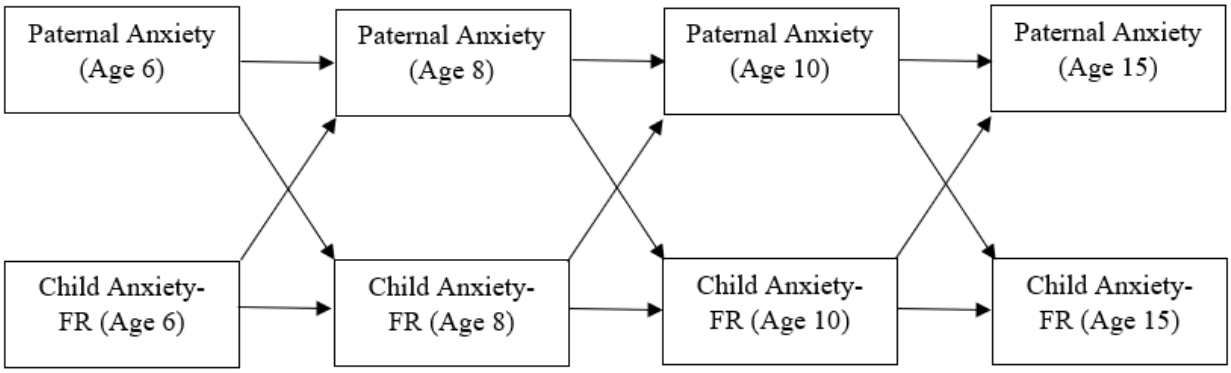
R2.2: Do child anxiety and developmental changes in child anxiety predict changes in paternal anxiety from Age 6 until Age 15?

H2.1: Paternal anxiety and changes in paternal anxiety are expected to positively predict the developmental changes in child anxiety at Age 8, Age 10, and Age 15.

H2.2: Child anxiety and developmental changes in child anxiety are expected to positively predict changes in paternal anxiety at Age 8, Age 10, and Age 15.

Figure 5

Cross-lagged Model of Paternal Anxiety and Child Anxiety from Age 6 until Age 15



Note. FR= Father report.

R3.1: Does maternal anxiety and changes in maternal anxiety in predict developmental changes in child anxiety from Age 6 until Age 15 above and beyond the effects by paternal anxiety (Figure 6)?

R3.2: Does paternal anxiety and changes in paternal anxiety predict developmental changes in child anxiety from Age 6 until Age 15 above and beyond the effects by maternal anxiety?

R3.3: Does child anxiety and changes in predict changes in maternal anxiety from Age 6 until Age 15 above and beyond the effects by paternal anxiety?

R3.4: Does child anxiety and changes in child anxiety predict changes in paternal anxiety from Age 6 until Age 15 above and beyond the effects by maternal anxiety?

H3.1: It is expected that maternal anxiety and changes in maternal anxiety positively predict the developmental changes in child anxiety over time above and beyond the effects by paternal anxiety.

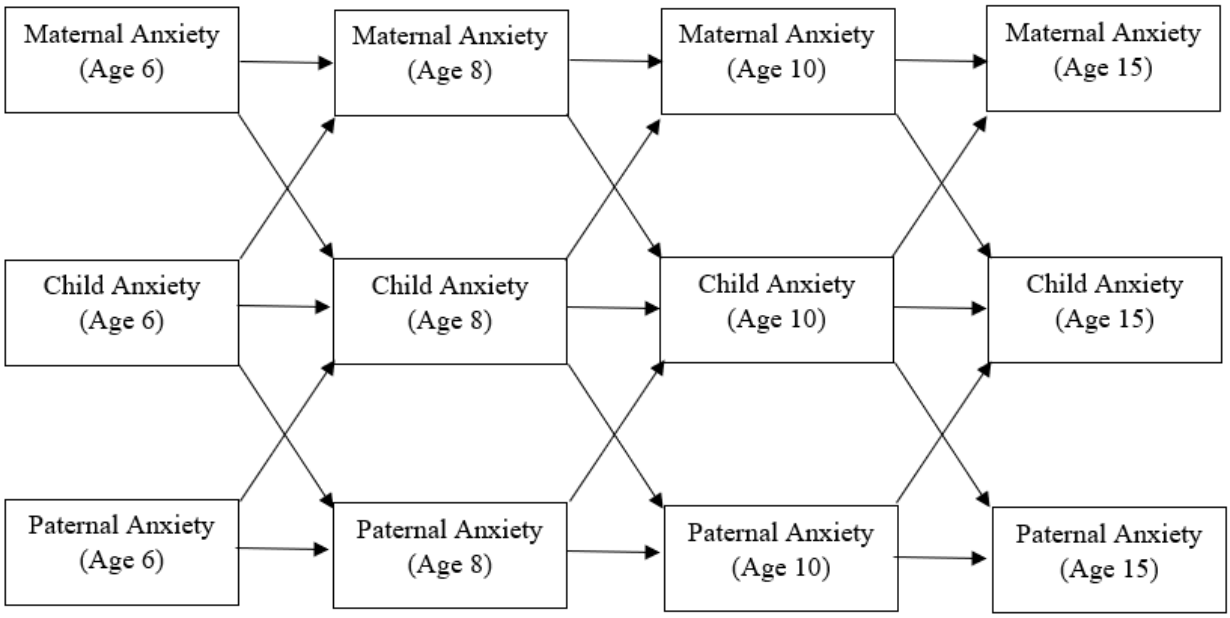
H3.2: It is expected that paternal anxiety and changes in paternal anxiety positively predict the developmental changes in child anxiety over time above and beyond the effects by maternal anxiety.

H3.3: It is expected that child anxiety and changes in child anxiety predict changes in maternal anxiety over time above and beyond the effects by paternal anxiety.

H3.4: It is expected that child anxiety and changes in child anxiety predict changes in paternal anxiety over time above and beyond the effects by maternal anxiety.

Figure 6

Cross-lagged Model of Maternal Anxiety, Paternal Anxiety, and Child Anxiety from Age 6 until Age 15



Chapter 3: Methods

Participants

Participants are families from the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development (NICHD SECCYD) dataset (*Eunice Kennedy Shriver* National Institute of Child Health and Human Development, National Institute of Health [NIH], Department of Health and Human Services [DHHS], 2007). The NICHD Study is a longitudinal project initiated to examine how differences in childcare experiences relate to children's developmental outcomes. Data collection began in 1991 and concluded in 2007 and was completed at 17 timepoints in total. The study enrolled a diverse sample of children and families at ten locations across the United States (NICHD Study of Early Child Care and Youth Development: Phase I, 1991-1995). Participants were recruited at designated hospitals and families with healthy and full-term babies were recruited. Conditionally random sampling was used to select 1,364 participants from a pool of over 6,000 families. This sampling method enabled the researchers to only include families with a particular work-life balance (60% of mothers who planned to work or study full-time, 20% of mothers who planned to work or study part-time, and 20% of mothers who stayed at home) and who reflected economic, educational, and ethnic diversity. Exclusion criteria included: (a) mothers younger than 18 years of age at the time of the child's birth, (b) families who did not anticipate remaining in the catchment area for at least 3 years, (c) children with obvious disabilities at birth or who remained in the hospital more than 7 days postpartum, and (d) mothers who were not sufficiently conversant in English (US Department of Health and Human Services, NICHD Study of Early Child Care and Youth Development: Phase I, 1991-1995).

Procedure

All assessments were focused on a focal child that was followed from birth until the age of 15. NICHD study was conducted at multiple sites, including hospitals where data were collected shortly after the focal child was born, the research laboratories of participating Universities, the families' homes, and at the childcare/school sites of focal children. Data were collected using direct observations, interviewers, questionnaires, and direct testing. Interviews were completed in person or over the phone. Parents of focal children also completed self-report questionnaires reporting on their own functioning as well as functioning of their child. Children completed self-report measures for the first time when they were 15 years old. More information about the specific data collection methods used for each timepoint can be found in NICHD Study of Early Child Care and Youth Development Historical Overview document (US Department of Health and Human Services, *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, 2020).

Study I: Child Emotion Dysregulation as a Mediator of Parent-Child Anxiety

Study Sample

Study I utilized data from three timepoints of the NICHD study: Phase II (when a child was at Age 6, conducted in 1998), Phase III (at Age 10, conducted in 2002), and Phase IV (at Age 15, conducted in 2007). For the purposes of this study, the study sample was limited to mother and father respondents only; all other caregiver types (e.g., grandparents, siblings) were removed from the sample prior to analysis.

The final sample included 811 mothers and 437 fathers; the 437 fathers and corresponding mothers comprised a family sample. In the mother sample, the mean age of the children at Age 6 was 6.4 (*SD*=0.5, ranging from 6 to 8) and there was almost an equal ratio of

boys to girls (48.5% to 51.5%, respectively). The mean age of mothers was 34.97 years (*SD*=5.47, ranging from 24 to 52) and they were predominantly White (86%, *n = 694*), followed by Black/African American (10%, *n = 82*), and Other (e.g., Asian and Pacific Islander, American Indian, Eskimo and others; 4%, *n = 35*). Children in the mother sample were also mainly White (83%, *n = 675*), then Black/African American (10%, *n = 81*); Other race was reported for 7% (*n = 55*) of the children.

In the father sample, children's mean age at Age 6 was 6.5 (*SD*=0.5, ranging from 6 to 8) with the ratio of boys to girls being 49.5% to 50.5%, respectively. The fathers' mean age was 38.3 years (*SD*=5.71, ranging from 23 to 54). The majority of fathers (93%, *n = 410*) reported being White, followed by Black/African American (4%, *n = 18*), and Other (3%, *n = 12*). Children in the father sample were mostly White (92%, *n = 404*), then Black/African American (4%, *n = 57*); Other race (4%, *n = 16*). The mean annual household income reported by mothers was between $60,000 and $70,000 (*M*=11.69, *SD*=4.50).

Measures

Demographic variables. A demographic questionnaire was completed by mothers; mother's age, mother's race, child's race, father's age, father's race, and family income were included in the study. Mothers reported their age and the father's age in years when the focal child was born. To calculate the mean parental age at Age 6, 6 years were added to the mother's and to the father's reported age. Mothers also reported their race, the father's race, and the child's race as follows: *1= American Indian, Eskimo, and Aleut, 2 = Asian and Pacific Islander, 3 = Black/African American, 4= White, and 5 = Other*. Race variable was recorded as *1= White, 0 = Other*. Family income was reported by mothers at Age 10; there were 27 income categories reported ranging from *1= less than $5,000* to *27 = more than $1,000,000*.

Maternal anxiety and paternal anxiety. State–Trait Anxiety Inventory (STAI, Spielberger et al., 1983) is a widely used self-report measure that assesses the presence and severity of anxiety symptoms in adult populations. Two subscales of the measure evaluate current state of anxiety (S-Anxiety) and general inclination toward anxiety traits (T-Anxiety). Each subscale consists of 20 items and the minimum total score for each subscale is 20 points and the maximum is 80 points. STAI has good psychometric properties with internal consistency alpha coefficients ranging from 0.86 to 0.95 across populations of different ages (Spielberger et al., 1983).Validity of the measure was also widely demonstrated across different populations, including for example anxiety assessment of mothers during pregnancy (Gunning et al., 2010). Content validity was confirmed by comparing STAI to symptoms of generalized anxiety disorder defined in *DSM-IV* (Okun et al., 1996).

Only the S-Anxiety subscale was administered to participants in the NICHD study and two changes were made to the subscale. First, the number of items was reduced from 20 to 10, and second, the wording of the questions was adjusted slightly; instead of reporting on their current state, mothers and fathers were asked to report how they felt in the past week. The list of 10 selected items can be found in Appendix A.

The mother and father respondents completed the shortened version of the S-Anxiety subscale when children were at Age 6 (Phase II). The responses ranged from *1=not at all* to *4=very much*. Example items included “I am worried about possible misfortune.” “I feel nervous.” and “I am jittery.” Four items were reverse-coded, and the total scores were calculated by summing of the item scores for each parent. Higher total score indicated higher anxiety symptomatology. Despite the item reduction from 20 to 10 items, the internal consistency of the measure in the current sample was good for mother (α =.87) and father (α =.86) respondents.

Child anxiety. In the NICHD study, Child Behavior Checklist (CBCL; Achenbach, 1991), Parent Report for children aged 4 to 18 was used by parents to rate their child's behavioral and emotional problems. The CBCL (Achenbach, 1991) is a well-established measure that consists of 113 items describing child problem behaviors; parents rate their children on a 3-point Likert Scale (*0=not true*, *2=very true or often true*). Items are summed into two main subscales: externalizing and internalizing behaviors. Internalizing behavior subscale is comprised of three domains: 1) Social Withdrawal domain that contains items related to avoidance of social interactions, 2) Somatic Complaints domain that contains items related to somatic discomfort, such as headaches and itching, and 3) Anxiety/Depression domain that contains items related to symptoms of anxiety and depression. CBCL has strong psychometric properties. Internal consistencies of the whole measure as well as its subscales and domains range between α =.80 and α =.97 (Achenbach & Rescola, 2001). Concurrent validity was demonstrated with *DSM*-described symptomatology (Ebesutani et al., 2010).

For the purposes of the proposed study, child anxiety at Age 6 (Phase II) and Age 15 (Phase IV) was assessed with the CBCL-Anxiety Problems (CBCL-AP; Achenbach et al., 2003) scale, a 6-item measure used by parents to rate their child's anxiety symptoms. Achenbach and colleagues (2003) developed CBCL-AP scale by selecting six items from the Child Behavior Checklist (CBCL; Achenbach, 1991) Anxiety/Depression subscale that matched the DSM-described anxious symptomatology. The list of the six items can be found in Appendix A. The 6-item scale was developed based on expert selection to reflect the *DSM-IV* diagnostic criteria. Example items include: "[child] clings to adults or too dependent" and "[child is] nervous, high-strung, or tense." Total scores were calculated by summing of all six items. The total scores were calculated separately for mother-reported child anxiety and for father-reported child anxiety in

each time point. In the family model, total scores of mother-reported and father-reported child anxiety were summed and then averaged. Higher score indicated higher anxiety symptomatology. Reported internal consistency for CBCL-AP measure was .82 (Achenbach et al., 2003). In the current sample, the internal consistency alpha coefficients were acceptable; mother-reported child anxiety: α =.60 and α =.70; father-reported child anxiety: α =.63 and α =.72 for Ages 6 and 15, respectively.

Child emotion dysregulation. Mothers and fathers evaluated their child's ability to deal with strong emotions using the Parent Report of Children's Reaction Scale (PRCR) when the child was 10 years old (Age 10, Phase III). The PRCR scale was modified from a 40-item Affective Intensity Measure – Long Form (AIM; Larsen, 1984), which measures the extent of emotional reactivity to life events and is considered an affect regulation measure (Larsen, 2009; Zelkowitz & Cole, 2016). The adapted PRCR scale consisted of 10 items and mothers and fathers rated their children on a 5-point Likert scale (*1=never, 5=always*). The list of all 10 items can be found in Appendix A. Example items include "When angry, it is easy for my child to still be rational and not overreact." and "My child is calm and not easily aroused." Five items were reverse-coded. Total scores for mother-reported child emotion dysregulation and for father-reported child emotion dysregulation were calculated by adding the scale item scores. Higher score indicates higher emotional reactivity, and therefore higher emotion dysregulation. In the study sample, the internal consistently alpha coefficients were α =.80 for mothers and α =.72 for fathers.

Statistical Approach

Missing data. Statistical software Amos (v25.0, Arbuckle, 2017) does not allow bootstrapping procedures on data with missing values. Cases with missing values of included

study variables in at least one timepoint were therefore removed. There were 181 (18%) cases removed from a mother sample and 219 (33%) cases removed from a father sample. Inspection of removed data revealed that majority (90% in the mother sample and 65% in the father sample) of cases with missing data only completed assessments for one of three timepoints used in this study. When comparing the data with missing values to the data without, there were no statistically significant differences on the main study variables in the mother-child sample: maternal anxiety [$F(1, 894)= 1.87$, $p= 0.17$], child emotion dysregulation [$F(1, 990)= 1.66$, $p= 0.20$], and child anxiety [$F(1, 884)= .34$, $p= 0.59$]. However, when comparing the data on demographic variables, there were statistically significant differences on all demographic variables in the mother-child sample: maternal age [$F(1, 990)= 16$, $p= 0.00$]; family income [$F(1, 942)= 13.33$, $p= 0.00$]; child race $\chi^2(1, N= 992)= 14.47$, $p= .00$; mother race $\chi^2(1, N= 992)= 12.39$, $p= .00$; and child sex $\chi^2(1, N= 992)= 3.18$, $p= .05$.

On the other hand, there were statistically significant differences across two main study variables, but no statistically significant differences on demographic variables in the father-child sample: paternal anxiety [$F(1, 655)= 6.25$, $p= 0.01$]; child emotion dysregulation [$F(1, 513)= 7.98$, $p= 0.01$]; child anxiety [$F(1, 473)= 2.24$, $p= 0.16$]; father race $\chi^2(1, N= 655)= .57$, $p= .45$; child race $\chi^2(1, N= 656)= .52$, $p= .47$; and child sex $\chi^2(1, N= 656)= 1.28$, $p= .26$.

Data analysis. Path analysis with observed variables was conducted in statistical software Amos (v25.0, Arbuckle, 2017). Paths were set to be freely estimated and the error terms of endogenous variables were correlated at each timepoint. Demographic variables that were significantly correlated ($p\leq .5$) with the dependent variable (child anxiety at Age 15) were controlled for in each model. Standardized path coefficients were reported. To determine the significance of the mediating effect, four linear regressions were conducted in SPSS (v25.0)

following the steps according to Baron and Kenny (1986). Sobel test calculator was utilized to confirm the mediation (Preacher & Leonardelli, 2020, http://quantpsy.org/sobel/sobel.htm). The statistical significance of the indirect effect in the path analysis was also determined by performing a bootstrapping procedure in Amos (v25.0, Arbuckle, 2017), allowing for 5,000 resamples and setting the confidence interval at 95%. Bias correction was used due to the positive skewness of dependent variable. According to Hayes (2013), since a bootstrapping procedure relies on random sampling with replacement, it is the best suited method to test the statistical significance of the indirect effect as a method relying on random sampling with replacement.

Study II: Bidirectional Effects of Parent-Child Anxiety from Early Childhood into Adolescence

Study Sample

Four timepoints from three phases of data collection were used in this study: Age 6 (Phase II) conducted in 1998, Age 8 (Phase III) conducted in 2000, Age 10 (Phase III) conducted in 2002, and Age 15 (Phase IV) conducted in 2007. For the purposes of this study, the study sample was limited to mother and father respondents only, all other caregiver types (e.g., grandparents, siblings) were removed from the sample prior to analysis.

The resulting sample included 1006 mothers and 678 were fathers. The family sample was comprised of 678 mothers and fathers. In the mother sample, the mean age of children at the first timepoint was 6.4 (*SD*=0.5, ranging from 6 to 8 years of age) and there was almost equal ratio of boys to girls (49.8% to 50.2%, respectively). The mean age of mothers at Age 6 was 34.7 years (*SD*=5.5, ranging from 30 to 58). Mothers were predominantly White (85%, *n = 858*), followed by Black/African American (10%, *n = 103*), and Other race, e.g., Asian and Pacific

Islander, American Indian, Eskimo, and others (5%, *n = 45*). Children in the mother sample were also mainly White (83%, *n = 835*), then Black/African American (10%, *n = 105*) and Other race was reported for 7% (*n = 66)* of the children.

In the father sample, the mean age of children at Age 6 was 6.44 (*SD*=0.5; ranging from 6 to 8 years of age; male to female ratio: 51% to 49%). The mean age of fathers was 37.29 (*SD*=8.3, ranging from 29 to 60). The majority of fathers (92%, *n = 618*) reported being White, followed by Black/African American (5%, *n = 33*), and Other race, e.g., Asian and Pacific Islander, American Indian, Eskimo, and others (3%, *n = 19*). Race composition of children in the father sample was White (92%, *n = 614*), Black/African American (4.5%, *n = 30*), and Other race (3.5%, *n = 26*). Mean annual household income reported by mothers was between $60,000 and $70,000 (*M*=11.69, *SD*=4.50).

Measures

Demographic variables. A demographic questionnaire was completed by mothers; mother's age, mother's race, child's race, father's age, father's race, and family income were included in the study. Mothers reported their age and the father's age in years when the focal child was born. To calculate the mean parental age at Age 6, 6 years were added to the mother's and to the father's reported age. Mothers also reported their race, the father's race, and the child's race as follows: *1= American Indian, Eskimo, and Aleut, 2 = Asian and Pacific Islander, 3 = Black/African American, 4= White, and 5 = Other*. Race variable was recorded as *1= White, 0 = Other*. Family income was reported by mothers at Age 15; there were 27 income categories reported ranging from *1= less than $5,000* to *27 = more than $1,000,000*.

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severity of anxiety symptoms in adult populations. Two subscales of the measure evaluate current state of anxiety (S-Anxiety) and general inclination toward anxiety traits (T-Anxiety). Each subscale consists of 20 items and the minimum total score for each subscale is 20 points and the maximum is 80 points. STAI has good psychometric properties with internal consistency alpha coefficients ranging from 0.86 to 0.95 across populations of different ages (Spielberger et al., 1983).Validity of the measure was also widely demonstrated across different populations, including for example anxiety assessment of mothers during pregnancy (Gunning et al., 2010). Content validity was confirmed by comparing STAI to symptoms of generalized anxiety disorder defined in *DSM-IV* (Okun et al., 1996).

Only S-Anxiety subscale was administered to participants in the NICHD study and two changes were made to the subscale. First, the number of items was reduced from 20 to 10 and second, the wording of the questions was adjusted slightly; instead of reporting on their current state, mothers and fathers were asked to report how they felt in the past week. The list of 10 selected items can be found in Appendix A.

The mother and father respondents completed the shortened version of the S-Anxiety subscale when children were at Age 6 (Phase II), Age 8, Age 10 (Phase III), and Age 15 (Phase IV). The responses ranged from *1=not at all* to *4=very much*. Example items included “I am worried about possible misfortune.” “I feel nervous.” and “I am jittery.” Four items were reverse-coded, and the total scores were calculated by summing of the item scores for each parent. Higher total score indicated higher anxiety symptomatology. Despite the item reduction, the internal consistency of the measure in the current sample was good for both mother and father respondents and across all time points (mothers: α=.87, α=.87, α=.87, and α=.90; fathers: α=.86, α=.86, α=.87, and α=.87 for Ages 6, 8, 10, and 15, respectively).

Child anxiety. In the NICHD study, the Child Behavior Checklist (CBCL; Achenbach, 1991), Parent Report for children aged 4 to 18 was used by parents to rate their child's behavioral and emotional problems. The CBCL (Achenbach, 1991) is a well-established measure that consists of 113 items describing child problem behaviors; parents rate their children on a 3-point Likert Scale (*0=not true*, *2=very true or often true*). Items are summed into two main subscales: externalizing and internalizing behaviors. The internalizing behavior subscale is comprised of three domains: 1) Social Withdrawal domain that contains items related to avoidance of social interactions, 2) Somatic Complaints domain that contains items related to somatic discomfort, such as headaches and itching, and 3) Anxiety/Depression domain that contains items related to symptoms of anxiety and depression. CBCL has strong psychometric properties. Internal consistencies of the whole measure as well as its subscales and domains range between α =.80 and α =.97 (Achenbach & Rescola, 2001). Concurrent validity was demonstrated with *DSM*-described symptomatology (Ebesutani et al., 2010).

For the purposes of the proposed study, child anxiety at Age 6 (Phase II), Age 8, Age 10 (Phase III), and Age 15 (Phase IV) was assessed with the CBCL-Anxiety Problems (CBCL-AP; Achenbach et al., 2003) scale, a 6-item measure used by parents to rate their child's anxiety symptoms. Achenbach and colleagues (2003) developed CBCL-AP scale by selecting six items from the Child Behavior Checklist (CBCL; Achenbach, 1991) Anxiety/Depression subscale that matched the DSM-described anxious symptomatology. The list of the six items can be found in Appendix A. The 6-item scale was developed based on expert selection to reflect the *DSM-IV* diagnostic criteria. Example items include: "[child] clings to adults or too dependent" and "[child is] nervous, high-strung, or tense." Total scores were calculated by summing of all six items. The total scores were calculated separately for mother-reported child anxiety and for father-reported

child anxiety in each time point. Higher score indicated higher anxiety symptomatology. Reported internal consistency for CBCL-AP measure was .82 (Achenbach et al., 2003). In the current sample, internal consistency alpha coefficients were acceptable; mothers: α=.60, α=.66, α=.67, and α=.67; fathers: α=.65, α=.64, α=.66, and α=.72 for ages 6, 8, 10, and 15 respectively.

Statistical Approach

Missing data. Similar rates of missing data were observed across the four timepoints in both mother-child and father child dyads. For mother-child variables, 0%, 10%, 12%, and 19% of maternal anxiety cases and 0%, 9%, 11%, and 18% of child anxiety-MR were missing on at least one item across Ages 6, 8, 10, and 15. For the father-child variables, 0%, 21%, 29%, and 39% of paternal anxiety cases and 0%, 20%, 28%, and 39% of child anxiety-FR were missing at least one item across Ages 6, 8, 10, and 15. Data with missing values were handled by the full information maximum likelihood estimation (FIML), which is considered the most current method to handle missing data (Wilson et al., 2014). FIML allows to retain the sample size, and it estimates standard errors accurately (Schlomer et al., 2010). Compared to multiple imputation (MI) method, FIML does not calculate missing values; rather, it produces reliable estimates in place of missing data and therefore, it is considered superior (Allison, 2009). Since FIML estimates the parameters of interest from the data, it is the recommended approach for data with over 10% of missingness (Little et al., 2014).

Data analysis. Data were analyzed in Amos (v25.0, Arbuckle, 2017) using cross-lagged path analysis (Kearney, 2017) with observed variables to determine the bidirectional effects of parental anxiety and child anxiety over time and to examine potential developmental changes of parental anxiety and child anxiety as the child transitioned from childhood into adolescence. Cross-lagged path analysis allows to examine the stability (i.e., the degree to which variable

values remain the same over time) and the bidirectional effects of a parent variable predicting child variable measured at later time point and child variable predicting parent variable measured at a later time point (Kearney, 2017). Three cross-lagged models modeling maternal anxiety and child anxiety (Figure 11), paternal anxiety and child anxiety (Figure 12), and both maternal and paternal anxiety and child anxiety (Figure 13) at Age 6, 8, 10, and 15 were tested. The error terms for endogenous variables were permitted to be correlated within a given construct. Demographic variables that were significantly correlated ($p \leq .5$) with the dependent variables were controlled for in each model. Model fit was evaluated using the chi-square test, the Comparative Fit Index (CFI), and Root Mean Square Error of Approximation (RMSEA; Hu & Bentler, 1999) to determine whether the models fit the data. CFI and RMSEA was used as alternative fit indices since the chi-square test is known to be sensitive to sample size.

Chapter 4: Results

Study I: Child Emotion Dysregulation as a Mediator of Parent-Child Anxiety

Preliminary Analysis

Table 1 includes means, standard deviations, and indicators of normality for all study variables. After inspection of the descriptive statistics associated with the study variables, there appeared to be no violations of normality, with the exception of child anxiety at Age 15 (both mother and father report) being significantly skewed to the left. A square-root transformation was used on the child anxiety variable to address the moderately positive skewness of the data (Howell, 2007; Tabachnick & Fidell, 2007). After transformation, child anxiety-mother report (skewness = .94, kurtosis = - .20) and child anxiety-father report (skewness = 1.00, kurtosis = -.27) appeared to be normally distributed. All further analysis used transformed child anxiety variables.

Table 1 also includes correlations between the main study variables. The main study variables were also correlated with the demographic variables, namely child race, child sex, mother age, mother race, father age, father race, and family income to determine which demographic variables should be entered in the models as covariates. For mothers, all main study variables were correlated; however, only one covariate, namely child's sex (r_{pb} = .17, $p \leq .01$) was significantly positively correlated with mother-reported (MR) child anxiety at Age 15. Similarly, for fathers, all study variables were significantly positively correlated, and only child's sex (r_{pb} = .12, $p \leq .05$) was significantly correlated with father-reported (FR) child anxiety at Age 15.

Table 1

Descriptive Information and Correlations Between Main Study Variables

Variables	*M*	*SD*	Range	Variance	Skewness	Kurtosis	1	2	3	4	5
1. Maternal Anxiety (Age 6)	17.40	5.19	10-39	26.99	.88	.98	-				
2. Paternal Anxiety (Age 6)	16.51	4.77	10-36	22.78	.90	.95	.20**	-			
3. Child Emotion Dysregulation-MR (Age 10)	33.47	5.82	15-49	33.85	-.09	-.04	.15**	.12*	-		
4. Child Emotion Dysregulation-FR (Age 10)	32.10	4.89	18-46	23.99	.32	.12	.13**	.13**	.40**	-	
5. Child Anxiety-MR (Age 15)	.78	1.30	0-9	1.7	2.51	8.30	.28**	.11*	.30**	.17**	-
6. Child Anxiety-FR (Age 15)	.83	1.40	0-8	2.25	2.11	4.46	.09	.24**	.24**	.28**	.41**

Note. MR= mother report, FR= father report; **$p \leq .01$, *$p \leq .05$

Mother-Child Model

The mother-child mediation model was tested using Amos (v25.0; Arbuckle, 2017) in a sample of 811 mothers and children. Controlling for mother race (β= .10, $p \leq .01$) and child sex (β= .11, $p \leq .01$) that both positively predicted the dependent variable, a significant positive direct effect was found from maternal anxiety at Age 6 to child anxiety-MR at Age 15 (β= .20, $p \leq .01$) There was also a significant positive direct effect from maternal anxiety to child emotion dysregulation at Age 10 (β= .18, $p \leq .01$) and from child emotion dysregulation to child anxiety at Age 15 (β= .23, $p \leq .01$). The size of the standardized indirect effect from maternal anxiety at Age 6 to child anxiety at Age 15 through child emotion dysregulation at Age 10 was β= .04, 95% CI [.022; .063].

To determine whether the standardized indirect effect was significant and, therefore, whether child emotion dysregulation mediated the relationship between maternal anxiety and child anxiety, two tests of mediation were conducted. First, using the Baron and Kenny (1986) approach to determine mediation, four linear regressions were conducted: 1) regressing child anxiety on maternal anxiety [$F(1, 809) = 52.30$, $p \leq .01$], 2) regressing child anxiety on child emotion dysregulation [$F(1, 809) = 64.73$, $p \leq .01$], 3) regressing child emotion dysregulation on maternal anxiety [$F(1, 809) = 26.24$, $p \leq .01$] and 4) regressing child anxiety on both maternal anxiety and child emotion dysregulation [$F(2, 808) = 52.31$, $p \leq .01$]. Since independent variables positively and significantly predicted the dependent variables in all four regression equations, the unstandardized regression coefficient for maternal anxiety predicting child anxiety (B = .033) was compared to unstandardized regression coefficient for maternal anxiety predicting child anxiety (B = .028) when child emotion dysregulation was included in the model. The reduction of the coefficient size indicated that there was partial mediation. The significance

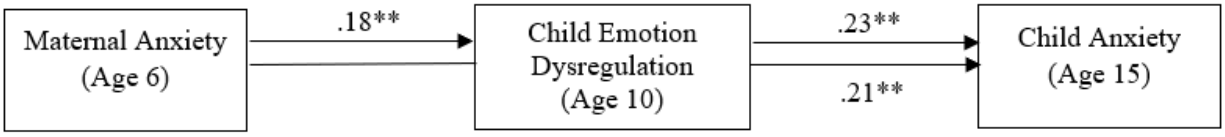
of the reduction was additionally confirmed by the Sobel test (p = .00). Unstandardized regression coefficients and standard errors used to calculate the Sobel test statistic are included in Table 2.

A bootstrapping procedure was used as a second test of mediation (Preacher & Hayes, 2004). Due to the positive skewness of the dependent variable, bias-corrected bootstrapping was performed using 5,000 resamples and requesting 95% confidence intervals. The bootstrapped standardized indirect effect was β = .04, CI [.022; .063], indicating that the indirect effect was significant, and therefore child emotion dysregulation was found to positively mediate the longitudinal relationship between maternal anxiety and child anxiety (Figure 7). The final model accounted for 14% of the explained variance in child anxiety at Age 15. Model fit indices provided evidence of good fit [$\chi^2(5) = 9.23$, $p \geq .05$; CFI= .97; RMSEA= .03].

In support of our hypothesis, both mediation tests showed that child emotion dysregulation at Age 10 partially mediated the relationship between maternal anxiety at Age 6 and child anxiety-MR at Age 15.

Figure 7

Standardized Regression Coefficients for the Relationship between Maternal Anxiety and Child Anxiety as Mediated by Child Emotion Dysregulation



**$p \leq .01$

An additional mother-child model was tested that took the bidirectional relationship of maternal and child anxiety into consideration. In the additional model, child anxiety at Age 6 was entered, as well as controlling for maternal anxiety at Age 6 and correlated demographic

variable of child sex (β= .18, $p \leq .01$). The standardized regression coefficients in this model were identical to the previous model and statistically significant: a positive direct effect from maternal anxiety at Age 6 to child emotion dysregulation at Age 10 (β= .18, $p \leq .01$) and from child emotion dysregulation at Age 10 to child anxiety at Age 15 (β= .23, $p \leq .01$) in addition to a positive direct effect from maternal anxiety at Age 6 to child anxiety-MR at Age 15 (β= .22, $p \leq .01$). The significance of indirect effect of maternal anxiety at Age 6 on child anxiety at Age 15 through child emotion dysregulation at Age 10 was confirmed using the bootstrapping procedure, β= .04, CI[.022; .062]. This model also explained 13% in child anxiety variance. However, model fit this model was poor [$\chi^2(9) = 114.83$, $p \leq .05$; CFI= .66; RMSEA= .12), suggesting that the data did not support this model. Controlling for the bidirectionality of mother-child anxiety therefore did not alter the results from the original model and did not weaken the effect of maternal anxiety on child anxiety via child emotion dysregulation.

Father-Child Model

It was hypothesized that child emotion dysregulation would also mediate the effect from paternal anxiety at Age 6 to child anxiety-FR at Age 15. The father-child mediation model was tested in a sample of 437 fathers and children. Child sex was added in the model as a covariate, and it positively predicted child anxiety-FR at Age 6 (β= .09, $p \leq .05$). The direct effect from paternal anxiety to child anxiety was positive and statistically significant (β= .20, $p \leq .01$). There were also positive and statistically significant direct effects found from paternal anxiety at Age 6 to child emotion dysregulation at Age 10 (β= .13, $p \leq .01$) and from child emotion dysregulation at Age 10 to child anxiety at Age 15 (β= .22, $p \leq .01$; Figure 8). The size of the standardized indirect effect was β= .03, 95% CI [.008; .056].

The significance of the standardized indirect effect was tested following Baron and Kenny's (1986) method to determine mediation. The regression analyses, 1) regressing child anxiety on paternal anxiety [$F(1, 438) = 25.18$, $p \leq .01$], 2) child anxiety on child emotion dysregulation [$F(1, 438) = 28.73$, , $p \leq .01$], 3) child emotion dysregulation on paternal anxiety [$F(1, 438) = 7.62$, $p \leq .01$], and 4) child anxiety on both maternal anxiety and child emotion dysregulation [$F(2, 437) = 24.97$, $p \leq .01$] were all positive and statistically significant. Therefore, the unstandardized regression coefficient for paternal anxiety predicting child anxiety (B = .04) was compared to unstandardized regression coefficient for paternal anxiety predicting child anxiety (B = .03), when child emotion dysregulation was included in the model. Since there was a reduction in a coefficient size, it can be concluded that child emotion dysregulation partially mediated the relationship between paternal and child anxiety. The significance of the reduction was confirmed by the Sobel test ($p \leq .05$). Unstandardized regression coefficients and standard errors used to compute the Sobel test statistic can be found in Table 2.

A bias-corrected bootstrapping procedure with 5,000 resamples and 95% confidence intervals was also performed to test the significance of the indirect effect. The standardized indirect effect was found to be significant (β= .03, CI [.008; .056]). This model accounted for 11% of the explained variance in child anxiety. Model fit indices provided evidence of good model fit [$\chi^2(2) = 2.63$, $p \geq .05$; CFI= .99; RMSEA= .03]. The mediation analysis therefore confirmed the hypothesis that child emotion dysregulation at Age 10 partially mediated the relationship between paternal anxiety at Age 6 and child anxiety-FR at age 15.

Table 2

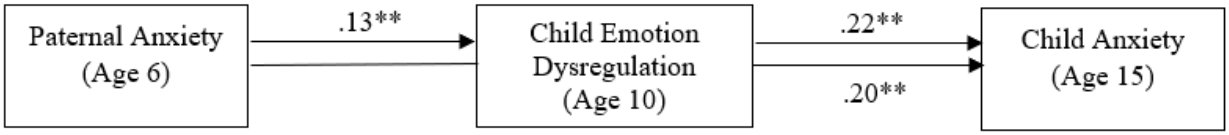
Unstandardized Regression Coefficients and Standard Errors

Mother-Child Regression	Unstandardized B (SE)	Sobel Test Statistic (SE)
Child Emotion Dysregulation on Maternal Anxiety	.20 (.04)	4.16 (.001)**
Child Anxiety on Child Emotion Dysregulation (and Maternal Anxiety)	.03 (.004)	
Father-Child Regression		
Child Emotion Dysregulation on Paternal Anxiety	.14 (.05)	2.34 (.002)*
Child Anxiety on Child Emotion Dysregulation (and Paternal Anxiety)	.03 (.007)	

Note. $^{**}p \leq .01$, $^{*}p \leq .05$

Figure 8

Standardized Regression Coefficients for the Relationship between Paternal Anxiety and Child Anxiety as Mediated by Child Emotion Dysregulation



$^{**}p \leq .01$

An additional model that considered the bidirectional nature of the father-child anxiety was also tested for fathers and their children. Child anxiety at Age 6 was included in the model as a control variable for paternal anxiety at Age 6. The standardized regression coefficients in this model were identical to the previous model. Controlling for child sex (β= .10, $p \leq .05$), there were positive significant direct effects from paternal anxiety at Age 6 to child anxiety at Age 15 (β= .20, $p \leq .01$), paternal anxiety at Age 6 to child emotion dysregulation at Age 10 (β= .13, $p \leq .01$), and from child emotion dysregulation at Age 10 to child anxiety at Age 15 (β= .21, $p \leq .01$). The direct effect from paternal anxiety at Age 6 to child anxiety-FR at Age 15 was also

significant (β= .20, $p \leq .01$). The significance of the indirect effect of maternal anxiety on child anxiety was confirmed using the bootstrapping method, β= .03, CI[.006; .063]. This model explained 11% in child anxiety variance. As with the additional mother model, model fit was poor [$\chi^2(5) = 54.97.94$, $p \leq .05$; CFI= .57; RMSEA= .15) and adding child anxiety at Age 6 as a control variable did not alter the results of the original father-child model.

Family Model

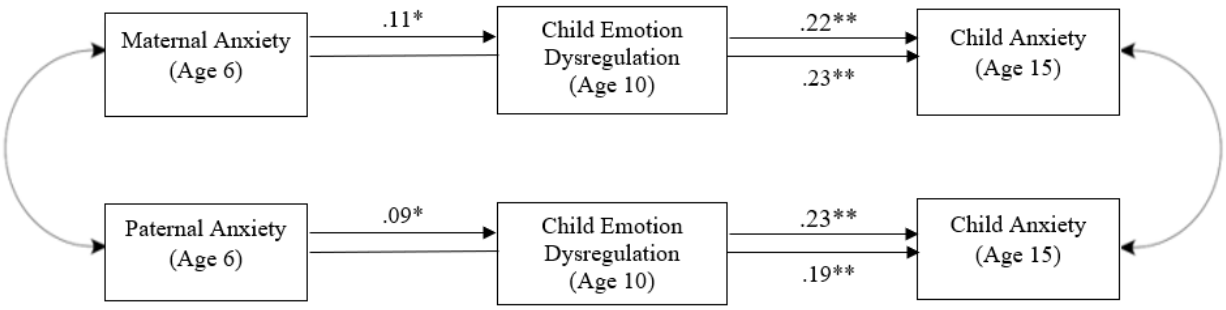
Family model was tested in a sample of 437 families. To determine whether child emotion dysregulation-FR at Age 10 mediated the relationship between paternal anxiety at the Age of 6 and child anxiety-FR at Age 15 above and beyond the effects by maternal anxiety, mediation effects were tested in one family model while controlling for child sex (β= .14, $p \leq .01$) that positively predicted child anxiety-MR at Age 15. Mother race variable that was positively correlated with child anxiety-MR at Age 15 was not significant in the model (β= .02, $p \geq .05$), nor was child sex predicting child anxiety-FR at Age 15 (β= .09, $p \geq .05$), despite it being positively correlated with child anxiety-FR at Age 15. In the mother path, there were positive significant direct effects from maternal anxiety at Age 6 to child anxiety-MR at Age 15 (β= .23, $p \leq .01$), from maternal anxiety at Age 6 to child emotion dysregulation at Age 10 (β= .11, $p \leq .01$), and from child emotion dysregulation at Age 10 to child anxiety-MR at Age 15 (β= .22, $p \leq .01$; Figure 9). In the father path, there were positive significant direct effects from paternal anxiety at Age 6 to child anxiety-FR at Age 15 (β= .19, $p \leq .01$), from paternal anxiety at Age 6 to child emotion dysregulation at Age 10 (β= .09, $p \leq .05$), and from child emotion dysregulation at Age 10 to child anxiety-FR at Age 15 (β= .23, $p \leq .01$). The significance of the mediating effect β= .024, CI [.004; .050] was confirmed by the bootstrapping procedure for the mother path; however, the mediating effect was not significant for the father path, β= .021, CI [-.002;

.501]. Chi-square goodness of fit for this model was not significant, [$\chi^2(16) = 26.10$, $p \geq .05$], and other fit indices indicated a good fit (CFI= .96; RMSEA= .04).

In the family model, child emotion dysregulation did not mediate the relationship between paternal anxiety and child anxiety-FR, therefore our hypothesis was not supported.

Figure 9

Standardized Regression Coefficients for the Mother-Child Anxiety Relationship and Father-Child Anxiety Relationship as Mediated by Child Emotion Dysregulation



*$p \leq .05$, **$p \leq .01$

Study II: Bidirectional Effects of Parent-Child Anxiety from Early Childhood into Adolescence

Preliminary Analysis

Table 3 includes means, standard deviations, and indicators of normality for all study variables. After inspection of study variables' descriptive information, there appeared to be no violations of normality for maternal anxiety and paternal anxiety variables. Child anxiety variables reported by both mothers and fathers were all skewed left. To address the moderate positive skewness and non-normality of child anxiety data distribution, Howell (2007) suggested a square-root data transformation. After transforming the data in SPSS (v25.0), the variables appeared to be normally distributed (Table 3). All further analysis used transformed child anxiety variables.

Bivariate correlations between main study variables can be found in Table 4. Correlations were also conducted between the demographic variables, namely child sex, child race, mother age, mother race, father age, father race, and family income, and the main study variables to determine which demographic variables should be entered in the models as covariates. Two demographic variables were negatively correlated with maternal anxiety at Age 15, namely mother age (r_{pb} = -.13, $p \leq .01$) and family income (r = -.18, $p \leq .01$). Mother race (r_{pb} = .1, $p \leq .01$) and child sex (r_{pb} = .12, $p \leq .01$) were positively correlated with child anxiety-mother report (MR) at Age 15. Family income was negatively correlated with paternal anxiety at Age 15 (r = -.14, $p \leq .01$) and child sex was positively correlated with child anxiety-father report (FR) at Age 15 (r_{pb} = .11, $p \leq .01$).

Table 3

Descriptive Information for All Study Variables

Variables	*M*	*SD*	Range	Variance	Skewness	Kurtosis
Maternal Anxiety (Age 6)	17.47	5.20	10-39	27.06	.84	.84
Maternal Anxiety (Age 8)	17.44	5.10	10-38	25.97	.81	.63
Maternal Anxiety (Age 10)	17.74	5.73	10-40	29.45	.78	.69
Maternal Anxiety (Age 15)	17.83	5.73	10-40	32.85	.95	.98
Child Anxiety-Mother Report (Age 6)	1.23	1.44	0-9	2.09	1.66 *(.22)*	3.72 *(-1.02)*
Child Anxiety-Mother Report (Age 8)	1.24	1.55	0-12	2.40	1.77 *(.35)*	4.40 *(- .98)*
Child Anxiety-Mother Report (Age 10)	1.22	1.60	0-12	2.53	1.97 *(.43)*	5.74 *(- .89)*
Child Anxiety-Mother Report (Age 15)	.80	1.33	0-9	1.75	2.5 *(.93)*	8.20 *(- .23)*
Paternal Anxiety (Age 6)	16.78	4.88	10-36	23.91	.88	.76
Paternal Anxiety (Age 8)	16.89	4.97	10-39	24.74	.83	.95
Paternal Anxiety (Age 10)	16.60	5.08	10-37	25.32	.96	1.04
Paternal Anxiety (Age 15)	16.31	4.76	10-36	23.02	.94	.85
Child Anxiety-Father Report (Age 6)	1.43	1.57	0-11	2.43	1.47 *(.09)*	3.28 *(-1.10)*
Child Anxiety-Father Report (Age 8)	1.24	1.52	0-9	2.15	1.50 *(.35)*	2.4 *(-1.08)*
Child Anxiety-Father Report (Age 10)	1.09	1.55	0-8	2.07	1.93 *(.65)*	4.88 *(-.90)*
Child Anxiety-Father Report (Age 15)	.82	1.37	0-8	1.93	2.13 *(.99)*	4.55 *(-.26)*
Child Anxiety-Averaged (Age 6)	1.29	1.19	0-7	1.43	1.21 *(-.17)*	1.79 *(-.70)*
Child Anxiety-Averaged (Age 8)	1.13	1.20	0-6.5	1.44	1.45 *(.09)*	2.20 *(-.82)*
Child Anxiety-Averaged (Age 10)	1.11	1.34	0-9.5	1.78	1.90 *(.29)*	5.40 *(-.78)*
Child Anxiety-Averaged (Age 15)	.77	1.14	0-6.5	1.30	2.28 *(.70)*	5.90 *(-.33)*

Note. Skewness and kurtosis of child anxiety variables after square-root transformations are italicized.

Table 4

Correlations Between the Main Study Variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. Maternal Anxiety (Age 6)	-																		
2. Maternal Anxiety (Age 8)	.50*	-																	
3. Maternal Anxiety (Age 10)	.43*	.50*	-																
4. Maternal Anxiety (Age 15)	.45*	.40*	.42*	-															
5. Child Anxiety-MR (Age 6)	.26*	.22*	.20*	.14*	-														
6. Child Anxiety-MR (Age 8)	.22*	.29*	.20*	.14*	.55*	-													
7. Child Anxiety-MR (Age 10)	.23*	.27*	.26*	.19*	.52*	.63*	-												
8. Child Anxiety-MR (Age 15)	.23*	.28*	.25*	.23*	.40*	.51*	.54*	-											
9. Paternal Anxiety (Age 6)	.20*	.14*	.07	.14*	.11*	.10	.15*	.07	-										
10. Paternal Anxiety (Age 8)	.17*	.17*	.16*	.14*	.06	.05	.11	.07	.53*	-									
11. Paternal Anxiety (Age 10)	.08	.12*	.12*	.13*	.07	.08	.14*	.10	.50*	.49*	-								
12. Paternal Anxiety (Age 15)	.18*	.20*	.16*	.24*	.13*	.10	.18*	.15*	.42*	.47*	.44*	-							
13. Child Anxiety-FR (Age 6)	.10	.13*	.13*	.07	.34*	.30*	.31*	.20*	.20*	.13*	.15*	.14*	-						
14. Child Anxiety-FR (Age 8)	.10	.15*	.15*	.08	.32*	.41*	.37*	.32*	.21*	.22*	.17*	.23*	.51*	-					
15. Child Anxiety-FR (Age 10)	.10	.19*	.10	.07	.25*	.36*	.46*	.32*	.28*	.27*	.28*	.28*	.45*	.56*	-				
16. Child Anxiety-FR (Age 15)	.08	.11	.08	.06	.16*	.27*	.32*	.40*	.22*	.21*	.19*	.26*	.34*	.47*	.50*	-			
17. Child Anxiety-AV (Age 6)	.20*	.20*	.19*	.11*	.77*	.50*	.50*	.36*	.18*	.12*	.14*	.17*	.83*	.50*	.42*	.33*	-		
18. Child Anxiety-AV (Age 8)	.19*	.27*	.20*	.12	.51*	.81*	.58*	.48*	.18*	.17*	.16*	.21*	.49*	.83*	.55*	.44*	.61*	-	
19. Child Anxiety-AV (Age 10)	.23*	.29*	.23*	.14*	.46*	.58*	.87*	.51*	.23*	.22*	.24*	.27*	.43*	.52*	.80*	.47*	.54*	.66*	-
20. Child Anxiety-AV (Age 15)	.19*	.22*	.17*	.16*	.33*	.45*	.53*	.81*	.20*	.16*	.19*	.24*	.32*	.45*	.49*	.83*	.41*	.54*	.60*

Note. MR=mother report, FR=father report, AV=averaged mother and father report; *$p \leq .01$

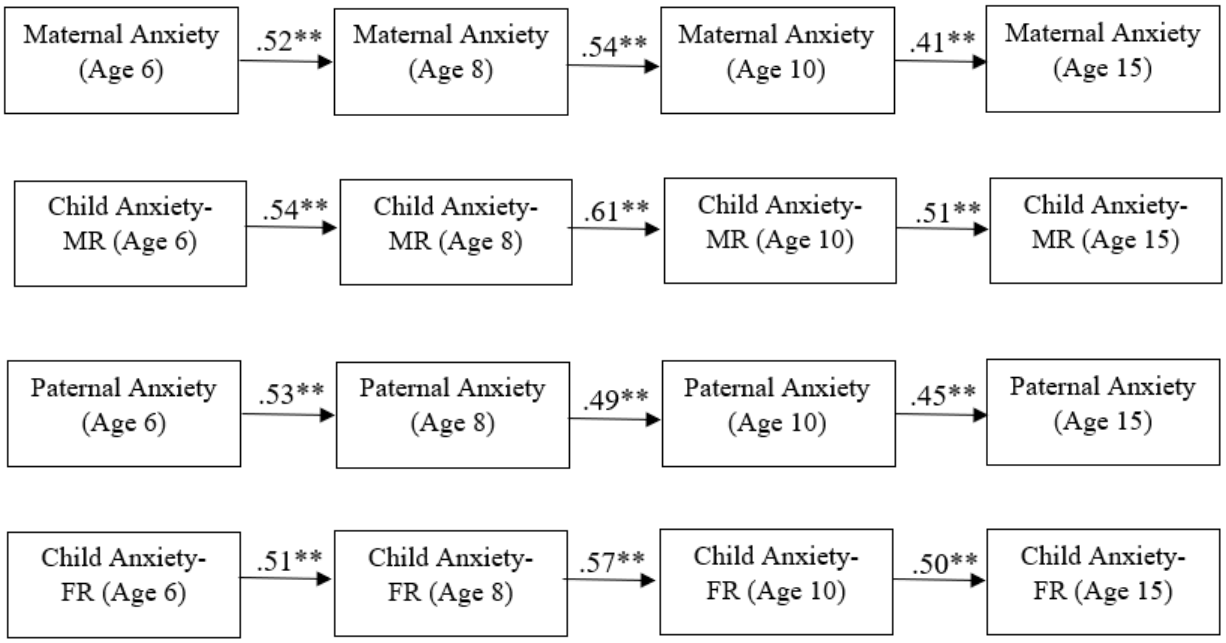
Mother-Child Model

A cross-lagged path model using Amos (v25.0; Arbuckle, 2017) was tested to examine the hypothesized bidirectional relationships between maternal anxiety and child anxiety-MR over the course of 9 years. First, the stability paths of both maternal anxiety and child anxiety-MR were specified and separately tested. All autoregressive paths were significant (Figure 10). Second, cross-lagged effects were specified and covariates were added to the model; mother age was not significant, but family income ($\beta = -.09$, $p \leq .01$), predicted maternal anxiety at Age 15 and mother race ($\beta = .11$, $p \leq .01$), and child sex ($\beta = .09$, $p \leq .01$), predicted for child anxiety-MR at Age 15. The path analysis showed that all bidirectional effects of maternal and child anxiety-MR were significant over time (Figure 11). Maternal anxiety and child anxiety-MR from Age 6 to Age 10 explained 19% and 31% of variance in maternal anxiety and child anxiety-MR at Age 15, respectively. The chi-square goodness of fit for this model was significant, [$\chi^2(36) = 504.99$, $p \leq .05$], and alternative fit indices provided evidence of poor model fit (CFI= .80; RMSEA= .11). Model fit was deemed to be poor due to CFI and RMSEA not meeting the cut-off criteria (CFI ≥.90, RMSEA < 0.08; Kline, 2005).

As hypothesized, the cross-lagged model showed that maternal anxiety and changes in maternal anxiety positively predicted the changes in child anxiety at Ages 8, 10, and 15. Child anxiety and developmental changes in child anxiety were also found to positively predict changes in maternal anxiety at Ages 8, 10, and 15.

Figure 10

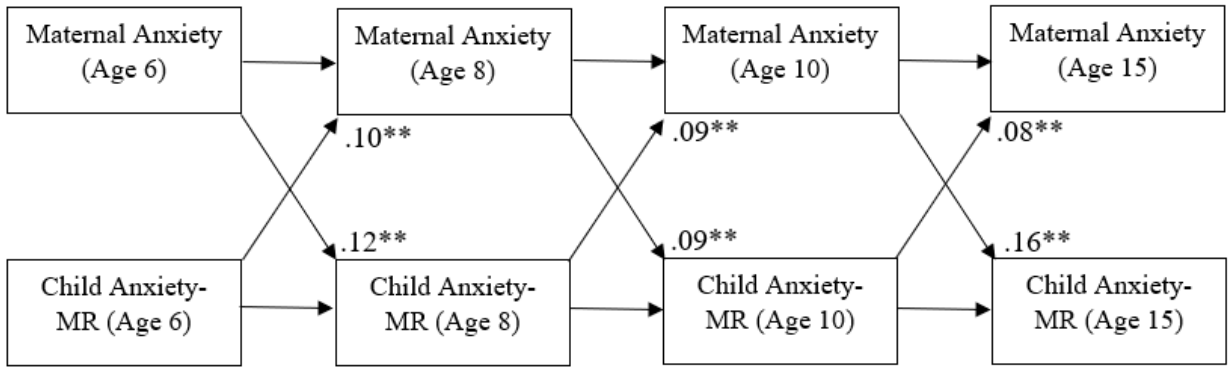
Standardized Regression Coefficients for Stability Paths in the Mother-Child Model (Maternal Anxiety and Child Anxiety-MR) and in the Father-Child Model (Paternal Anxiety and Child Anxiety -FR)



Note. MR = mother report, FR = father report; $^{**}p \leq .01$

Figure 11

Standardized Regression Coefficients for Autoregressive Paths between Maternal Anxiety and Child Anxiety-MR over the Course of 9 Years



Note. MR = mother report; $^{**}p \leq .01$

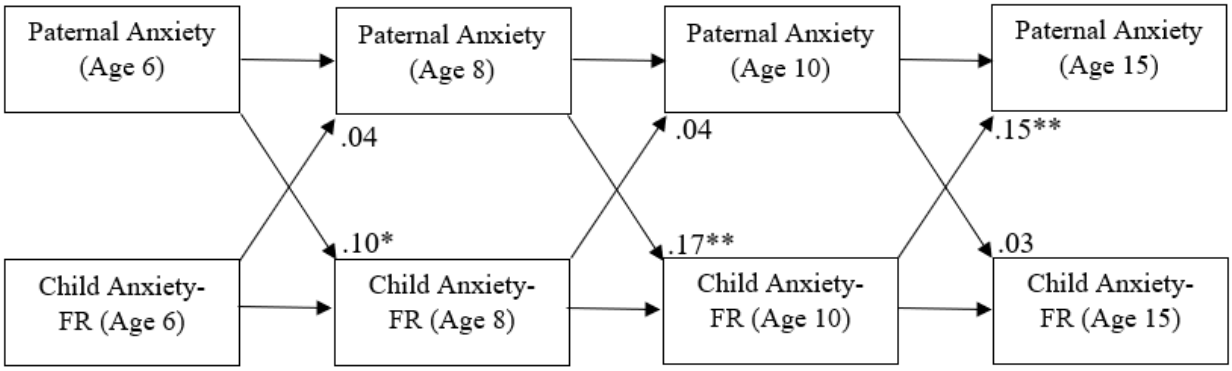
Father-Child Model

A cross-lagged path model was also tested to determine the role of paternal anxiety in child anxiety-FR over time. The stability of paternal anxiety and child anxiety-FR over the course of 9 years was also confirmed in the father-child model (Figure 10). Including family income as a covariate predicting paternal anxiety at Age 15 (β= -.08, $p \leq .05$) and child sex as a covariate predicting child anxiety-FR at Age 15 (β= .12, $p \leq .01$), the cross-lagged model testing the bidirectional relationships of paternal anxiety and child anxiety-FR over a period of 9 years showed that three predictive paths were significant: paternal anxiety at Age 6 predicting child anxiety-FR at Age 8 (β= .10, $p \leq .01$), paternal anxiety at Age 8 predicting child anxiety-FR at Age 10 (β= .17, $p \leq .01$), and child anxiety at Age 10 predicting paternal anxiety at Age 15 (β= .15 $p \leq .01$; Figure 12). Paternal anxiety and child anxiety from Age 6 to Age 10 explained 23% in paternal anxiety at Age 15 and 25% in child anxiety-FR at Age 15. The chi-square goodness of fit for this model was significant, [$\chi^2(28) = 242.62$, $p \leq .05$], and alternative fit provided evidence of poor fit (CFI= .82; RMSEA= .11).

The cross-lagged analysis showed that our second hypothesis was only partially supported. Paternal anxiety and changes in paternal anxiety are positively predicted the developmental changes in child anxiety measured at Ages 8 and 10, but not at Age 15. On the contrary, child anxiety and developmental changes in child anxiety did not predict changes in paternal anxiety at Ages 8 and 10, but they did positively predict changes in paternal anxiety at Age 15.

Figure 12

Standardized Regression Coefficients for Autoregressive Paths between Paternal Anxiety and Child Anxiety-FR over the Course of 9 Years



Note. FR = father report; *$p \leq .05$, **$p \leq .01$

Family Model

Finally, a third model was tested utilizing the family sample of 678 mothers and fathers to determine whether the effects of maternal anxiety and of paternal anxiety on child anxiety are unique and predictive when the other parent's anxiety is included in the model. In order the test this hypothesis, one measure of child anxiety was constructed by averaging mother-reported child anxiety and father-reported child anxiety. However, when the averaged child anxiety measure was included in the family model, the path analysis yielded unexpected and inconsistent estimates; for example, significant paths from the individual models became nonsignificant and the nonsignificant paths from the individual models became significant. To test the accuracy of these results, two more family models were tested, one with mother-reported child anxiety and one with father-reported child anxiety. When mother-reported child anxiety was tested, the significance of the standardized regression coefficients did not differ from the previously reported mother-child model; however, multiple father-child paths were not significant, such as paternal anxiety at Age 6 predicting child anxiety-MR at Age 8 ($\beta = .01$, $p \geq .05$) and paternal anxiety at Age 8 predicting child anxiety-MR at Age 10 ($\beta = .05$, $p \geq .05$). Paternal anxiety at Age

10 did not predict child anxiety-MR at Age 15, which was consistent with the individual father-child model, however, the direction of the prediction was negative (β= - .01, $p \geq .05$)

When father-reported child anxiety was tested, the significance of the standardized regression coefficients did not differ from the previously reported father-child model, while several mother-child paths were not significant, such as maternal anxiety at Age 6 predicting child anxiety-FR at Age 8 (β= .02, $p \geq .05$) and maternal anxiety at Age 10 predicting child anxiety-MR at Age 15 (β= .01, $p \geq .05$).

Given these inconsistencies of the results, it appeared that there was a discrepancy between mother-reported anxiety and father-reported anxiety, despite significant correlations (see Table 4 for correlations at each timepoint) between the two constructs.

Multivariate regression was conducted next to test whether mother-reported child anxiety at Age 6, 8, and 10 predicted father-reported child anxiety at Age 15 and whether father-reported child anxiety at Age 6, 8, and 10 predicted mother-reported child anxiety at Age 15. It was found that father-reported child anxiety did not significantly predict mother-reported child anxiety at Age 15 [$F(6, 434) = 39.20$, $p \leq .01$], and only mother-reported child anxiety at Age 6 significantly predicted father-reported child anxiety at Age 15 [$F(6, 405) = 33.12$, $p \leq .01$] (see Table 5), a further indication that mother-reported and father-reported child anxiety might be distinct constructs.

Table 5

Regression Coefficients for Mother-Reported and Father-Reported Child Anxiety

	Unstandardized B (SE)	Standardized β	t	*p*
Child Anxiety-Mother Report (Age 15)				
Child Anxiety-MR (Age 6)	.09 (.05)	.09	1.91	.06
Child Anxiety-MR (Age 8)	.22 (.05)	.24	4.51	.00
Child Anxiety-MR (Age 10)	.28 (.05)	.32	5.96	.00
Child Anxiety-FR (Age 6)	-.04 (.04)	-.04	-.83	.41
Child Anxiety-FR (Age 8)	.08 (.05)	.09	1.74	.08
Child Anxiety-FR (Age 10)	.01 (.05)	.01	.21	.84
Child Anxiety-Father Report (Age 15)				
Child Anxiety-MR (Age 6)	-.10 (.05)	-.10	-1.95	.05
Child Anxiety-MR (Age 8)	.03 (.06)	.03	.46	.65
Child Anxiety-MR (Age 10)	.10 (.05)	.10	1.82	.07
Child Anxiety-FR (Age 6)	.14 (.05)	.14	2.92	.00
Child Anxiety-FR (Age 8)	.25 (.05)	.25	4.62	.00
Child Anxiety-FR (Age 10)	.27 (.05)	.27	4.97	.00

Note. MR= mother report, FR= father report

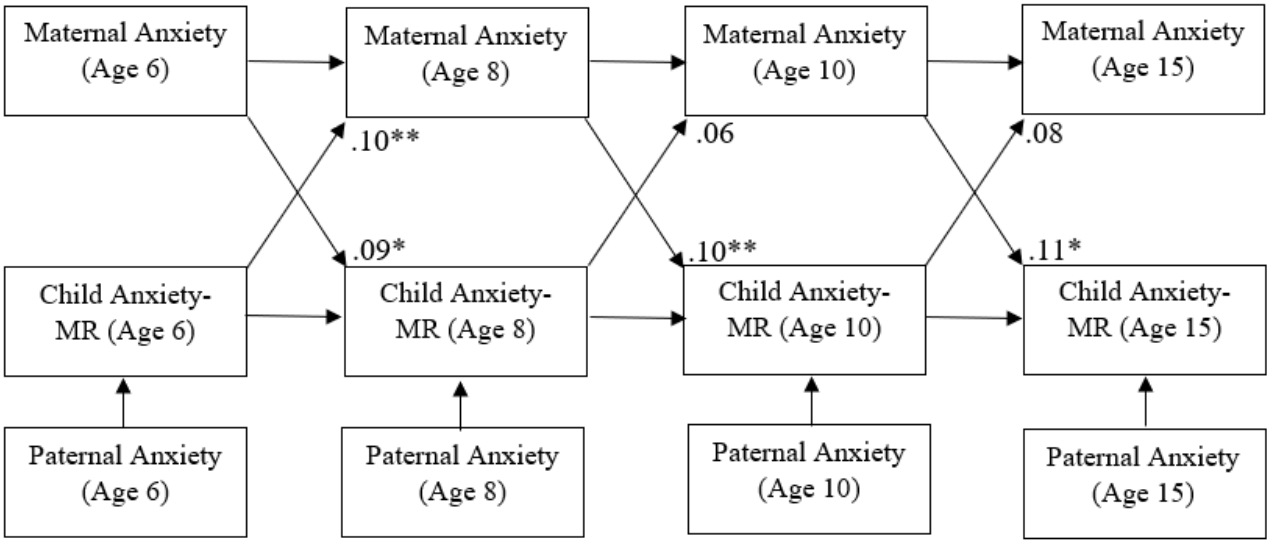
The original family model was therefore modified and, in order to test for unique parental effects, paternal anxiety was entered into a mother-child model with mother-reported child anxiety as a control variable and maternal anxiety was entered into a father-child model as a control variable. Further, family income predicting maternal anxiety at Age 15 (β= -.11, $p \leq .05$) and child sex predicting child anxiety-MR at Age 15 (β= .12, $p \leq .01$) were added as covariates. After controlling for paternal anxiety at each time point, all cross-lagged paths between maternal anxiety and mother-reported child anxiety remained positive and significant, with the exception of child anxiety at Age 8 predicting maternal anxiety at Age 10 and child anxiety at Age 10 predicting maternal anxiety at Age 15 (Figure 13). Maternal anxiety and child anxiety explained

30% of the variance in child anxiety at Age 15 and 18% of the variance in maternal anxiety at Age 15. The chi-square goodness of fit for this model was significant, [$\chi^2(64) = 479.53$, $p \leq .05$], and other fit indices were poor (CFI= .80; RMSEA= .10).

In partial support of our hypothesis, the results show that maternal anxiety and changes in maternal anxiety at each timepoint predict developmental changes in child anxiety beyond the effects of paternal anxiety. On the contrary, changes in child anxiety at Age 8 and Age 10 were not found to predict changes in maternal anxiety at Age 10 and Age 15 when paternal anxiety was included in the model.

Figure 13

Standardized Regression Coefficients for Autoregressive Paths between Maternal Anxiety and Child Anxiety-MR over the Course of 9 Years, Controlling for Paternal Anxiety



Note. MR = mother report; *$p \leq .05$, **$p \leq .01$

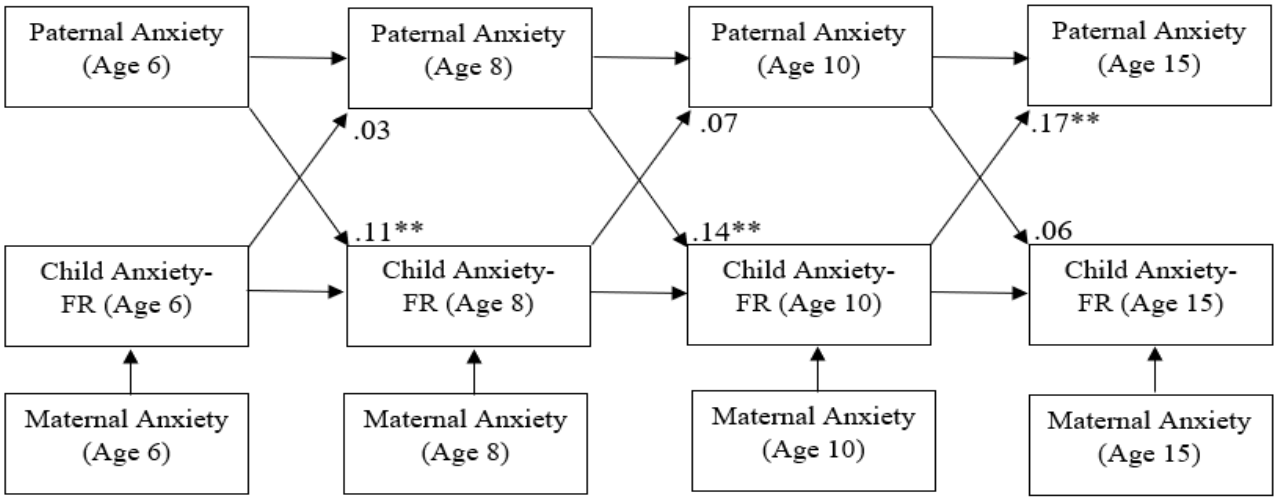
After controlling for maternal anxiety at each timepoint, three cross-lagged paths between paternal anxiety and father-reported child anxiety were significant; paternal anxiety at Age 6 predicting child anxiety-FR at Age 8, paternal anxiety at Age 8 predicting child anxiety-FR at Age 10, and child anxiety-FR at Age 10 predicting paternal anxiety at Age 15 (Figure 14).

Paternal anxiety and child anxiety at previous timepoints explained approximately 24% and 26% in paternal anxiety and in child anxiety, respectively. The chi-square goodness of fit for this model was significant, [$\chi^2(64) = 435.42$, $p \leq .05$], and alternative fit indices were poor (CFI= .80; RMSEA= .09).

The results of the cross-lagged analysis only partially supported our hypothesis that paternal anxiety and its changes would predict child anxiety and its changes over time and vice versa above and beyond the effects of maternal anxiety. Compared to the results of the father-child model, including maternal anxiety in the model did not alter the significance or non-significance of the effects.

Figure 14

Standardized Regression Coefficients for Autoregressive Paths between Paternal Anxiety and Child Anxiety-FR over the Course of 9 Years, Controlling for Maternal Anxiety



Note. FR = father report; *$p \leq .05$, **$p \leq .01$

Chapter 5: Discussion

This dissertation focused on examining the role of parental anxiety in the etiology of childhood anxiety. The purpose of Study I was to determine whether child emotion dysregulation could serve as an underlying mechanism of anxiety transmission from parents to children. The purpose of Study II was to understand the reciprocal relationships between parental and child anxiety over the course of nine years as children transitioned from early childhood into adolescence. Both studies tested the effects of maternal and paternal anxiety separately as well as together to better understand the role of fathers in childhood anxiety etiology research.

Study I: Child Emotion Dysregulation as a Mediator

The Study I yielded two main findings: 1) As hypothesized, parental anxiety longitudinally predicted child anxiety across all models; and 2) As hypothesized, child emotion dysregulation partially mediated the relationship between mother-child anxiety and father-child anxiety in separate models. Contrary to our hypothesis, child emotion dysregulation was not found to be a significant mediator in a father-child path of the family model.

The evidence of parental anxiety's influence during early childhood on child's adolescent anxiety provided by this study contributes to the wealth of research proving the relevance of parental anxiety in childhood anxiety etiology (Vasey et al., 2014). The long-term impact of early parental anxiety on child anxiety outcomes in adolescence demonstrated by the results further deepens the need to understand the processes through which early parental psychopathology continues to shape a child's level of functioning over time. Our study found that child emotion dysregulation partially mediated the parent-child anxiety link, therefore providing an insight into processes that underlie the pathways of parent-child transmission.

Child emotion dysregulation has been commonly researched for its association with child anxiety (Loevaas et al., 2018; Schneider et al., 2018); however, assumptions about the directions of child emotion dysregulation-child anxiety relationship have been difficult to conclude from predominantly cross-sectional research. In our study, child emotion dysregulation proved to be a developmental antecedent to child anxiety as well as a consequence of child's exposure to parental anxiety. Interestingly, when processes of emotion regulation were previously examined in parent-child anxiety transmission research, it was parental emotion regulation that was found to be associated which child anxiety (Kerns et al., 2017). Our study is therefore the first one to test and confirm child emotion dysregulation as opposed to parental emotion dysregulation as a mechanism of anxiety transmission between parents and their children. Given previous research findings, it is probable that both parental and child emotion regulation processes underlie the mechanism of transmission.

Nolte et al.'s (2011) Interpersonal stress regulation model based on attachment theory (Bowlby, 1988) provides a theoretical explanation of how child emotion dysregulation carries the impact of parental anxiety into the child's life and anxious experiencing. According to this model, parental anxiety prevents parents from successfully co-regulating their children's distress which results in child's development of poor regulatory capacities, such as high emotional reactivity. Children struggling with emotion regulation were found to have reduced capacity to regulate their affective states, including their anxious feelings (Young et al. 2019). Our study therefore also provides a partial validation for Nolte et al.'s (2011) model.

Alternatively, or perhaps in a complimentary process, child's emotion regulation skills could also be a result of socialization that anxious parents engage in (Eisenberg et al., 1998). Anxious parents are likely to teach emotion regulation skills that contribute to child's heightened

anxiety, since parents themselves utilize regulatory skills that are insufficient in regulating their own anxiety. The child then acquires regulatory skills via direct and indirect socialization methods that are likely inadequate in keeping the child's anxiety within functional levels.

Mechanisms of parent-child anxiety transmission were previously researched solely on a cognitive level. Specifically, the child's acquisition of anxious cognitive distortions from their parents was identified as underlying process involved in transmission (Lebowitz at al., 2016). Our results significantly expand this understanding by bringing attention to and confirming that emotional processes are involved in transmission. Our results that found partial mediation indicate that other mechanisms beyond child emotion dysregulation contribute to transmission. It is likely that cognitive and emotional aspects of transmission processes interact, are additive, or perhaps determining of each other. Possibly, emotion dysregulation skills are learned by children within the same context of socializing parent-child interactions as cognitive distortions. For example, it was specified that anxious parents perceive neutral situations as threatening and they directly and indirectly communicate this information to their child, teaching the child the same cognitive distortions (McLeod et al., 2011). It could be that parental cognitive distortions are accompanied by parental anxious regulation, reinforcing the cognitive message by emotional experiencing. It is also possible that parental communication of cognitive distortions leads directly to child's emotion dysregulation, which either goes unnoticed by a parent preoccupied with their own anxious experiencing or it is potentially reinforced by an anxious parent as a desirable and accurate response to parental anxious communication.

The interplay of child and parental cognitive distortions and emotion regulation in intergenerational transmission would also be influenced by what facets of emotion regulation are measured. Emotion regulation is a multifaceted construct that involves complex regulatory

strategies (Thompson, 1994) and thus has varying operational definition across child emotion regulation studies. Study I operationalized child emotion dysregulation as a child's high emotional reactivity to life events (Larsen, 1984), which seemed particularly fitting given that anxiety manifests in heightened emotional experiencing (American Psychiatric Association, 2013; Gregory et al., 2019). It is, however, important to keep in mind that this operationalization likely captures only one aspect of the child's emotional regulatory capacities and, therefore, more research is needed to understand emotion regulation as a mechanism of parent-child anxiety transmission in depth as well as in its interconnection with cognitive distortions.

Study II: Bidirectional Effects of Parent-Child Anxiety

Study II examined bidirectional effects for parent-child anxiety over time. The bidirectional effects were confirmed for both parents, although contrary to our hypothesis, paternal anxiety effects were less consistent over time than maternal anxiety effects. Maternal anxiety was found to predict child anxiety across all time points between the ages 6 and 15. Maternal anxiety is a well-documented predictor of child anxiety (Costa & Weems, 2005), and our results echo other longitudinal studies that focused on early maternal psychopathology and distress and later adolescent outcomes more broadly, finding maternal risk factors to predict internalizing and externalizing symptoms in Australian and Norwegian samples (Betts et al., 2014; Nilsen et al., 2013). Our results are, however, noteworthy for studying maternal anxiety and child anxiety as a specific outcome of maternal anxiety in a US sample and for capturing the positive predictive effects that remained significant across four time points. This is the first study to show continuous and consistent impact of maternal anxiety on child anxiety over a nine-year period that spanned across different developmental stages, beginning in early childhood and ending in adolescence. There are many developmental milestones and transitions that a child

experiences during this time span (Mundy et al., 2013); it is therefore intriguing that maternal anxiety's influence on child anxiety remained unfaltering over time and with child's changing developmental needs. Assumptions can be made about potentially different underlying processes playing a role in mother-child anxiety transmission at various stages of development. For example, younger children's anxiety could be a result of direct parenting practices, such as overcontrol, while adolescent anxiety could be a result of internalized inadequate regulatory skills or lowered self-competence (Affrunti & Ginsburg, 2012). Nevertheless, the effects of maternal anxiety observed across developmental and temporal events point towards the robustness of anxiety transmission between mothers and children.

Further, Study II found that there was reciprocity between maternal anxiety and child anxiety. There is evidence of reciprocity in many aspects of parent-child relationships, such as parenting and temperament (Kiff et al., 2011); however, reciprocal anxiety relationships in families are often assumed, but rarely studied. One recent longitudinal study of adoptive parents and their children found child anxiety predicting maternal – but not paternal – anxiety from age 7 to age 8 (Ahmadzadeh et al., 2019). Both mother-child and father-child models in this study provided evidence for reciprocity of parent-child anxiety relationships, albeit with differences between the parents. While child anxiety positively predicted paternal anxiety only at one time point, child anxiety was found to positively predict maternal anxiety across all four time points. Maternal and child anxiety were therefore found to be closely intertwined and interconnected in their own trajectories.

The implications of child anxiety's reciprocal influence in maternal anxiety might alter our understanding of parenting practices that anxious parents utilize with their children. Parenting practices of overcontrol and low autonomy granting were found to be used by anxious

parents and result in child anxiety (McLeod et al., 2007). What if these parenting practices, however, were responses to children experiencing anxiety, rather that parental agency choices? For example, if an anxious child is cautious about going somewhere or playing with someone, a parent might be more inclined to provide more detailed guidance with closer monitoring, and a parent might be more likely to overstep child's boundaries in an effort to help the child and elevate child's anxiety. This dynamic is explained as family accommodations that families of anxious children make (Norman et al., 2015). High accommodation, high overcontrol and low autonomy further reinforce, not elevate child anxiety, leading family into a vicious cycle.

Another important finding that emerged from Study II's analysis was the stability of family members' anxiety. Maternal anxiety, paternal anxiety, and child anxiety were each found to be stable over time by all models. The developmental trajectories of child anxiety are not likely to alter themselves as a function of time or maturing. Several studies confirmed the stability of anxiety pathways during the course child development (Mesman et al., 2001; Nilsen et al., 2013), but the stability of maternal and paternal anxiety pathways is a novel finding. The continuity of anxiety trajectories of each parent and their child, in addition to the reciprocity of parent-child anxiety relationships provide further support for the notion that anxiety aggregates in families (Ginsburg & Schlossberg, 2012). This study shows that parental and child anxiety are interconnected throughout a substantial period of child's development and, therefore, child anxiety needs to be understood in relations to parental anxiety and within family relationships.

The Role of Paternal Anxiety in Child Anxiety Etiology

Both Study I and Study II tested the effects of maternal and paternal anxiety separately as well as together to better understand the role of fathers in childhood anxiety etiology. Paternal anxiety has been seldomly investigated in child anxiety research, and the studies that did

examine paternal anxiety as a predictor of child anxiety often found it non-significant, as evidenced by the Connell and Goodman's (2002) meta-analysis that found paternal anxiety having no effect on child internalizing symptoms overall (Connell & Goodman, 2002).

Based on the difference in mother-reported and father-reported child anxiety constructs that emerged in this study, it can be speculated that non-significance of potential previous studies' results could have been attributed to measurement, since it is common practice to obtain mother reports when assessing constructs in children, and child anxiety is no different. Discrepancies in mother-reported and father-reported child anxiety have been reported before (Jansen et al., 2017); it was nevertheless surprising that the discrepancies were severe enough to change the strengths of predictive effects. Similar results were found by Ahmadzadeh et al. (2019), whose study found paternal anxiety predicting child anxiety but only when father-reported child anxiety was included in their model. It appears that not only parental anxiety influences parental perceptions of their child's anxiety, but each parent also perceives their child's anxiety in their own way. Our findings raise a question whether the lack of evidence for paternal anxiety has been due to studies only obtaining maternal reports of child anxiety. Potential inclusion of mother-reported child anxiety in examination of paternal anxiety influences could have resulted in biased estimates and understated importance of paternal anxiety in child anxiety. Our study, therefore, reiterates the importance of including both parents' assessments of child anxiety in analyses, especially if the examination of paternal anxiety is of interest.

Nevertheless, there is very little literature supporting the role of fathers in child anxiety etiology, despite theoretical assumptions held by multiple models (Bögels & Phares, 2008; Ginsburg & Schlossberg, 2002). The current studies both show that paternal anxiety is

significantly important for child's development of anxiety, evidence that shifts our understanding of parental roles in childhood anxiety. The focus of child anxiety studies on maternal factors including maternal mental health seems to have communicated that the role of fathers is marginal or unimportant. The results of our studies indicate the opposite, suggesting that parental anxiety is integral to the development and maintenance of child anxiety over time.

Study I found that both maternal and paternal anxiety experienced by parents in the child's preschool years positively predicted a child's anxiety in adolescence. The effect from paternal anxiety to child anxiety also remained significant when maternal and paternal influences were tested together in a family model, and therefore, it can be concluded that paternal anxiety is a unique predictor of child anxiety above and beyond maternal anxiety. The results suggest that there are long-term consequences of parental anxiety that extend into the child's adolescent years and that the exposure to parental anxiety at a younger age predisposes children to experience anxiety symptoms nine years later.

Study I also found that child emotion dysregulation mediated both mother-child and father-child anxiety links in separate models. Paternal anxiety was found to have an influence on child anxiety through the same mechanism of child emotion dysregulation as maternal anxiety. To our knowledge, no empirical studies have previously examined the role of paternal anxiety in child emotion regulation processes and the evidence of fathers' involvement in child emotion regulation in general is mixed (Bariola et al., 2012). Results of Study I clearly demonstrated that child emotion dysregulation in middle childhood was predicted by early childhood paternal anxiety. Interestingly, the mediation test, however, did not find a significant indirect effect from paternal anxiety to child anxiety in the family model when maternal anxiety was included in the model. The result of the mediation test is somewhat surprising since all direct paths in the model

were statistically significant, including the direct path from paternal anxiety to father-reported child emotion dysregulation and the direct path from father-reported child emotion dysregulation to child anxiety. The mediation effect was also significant in the father-child model. The non-significance of the mediation effect in the family model would suggest that there is no unique effect of child emotion dysregulation mediating paternal anxiety and child anxiety; however, given the significance of the direct paths, it is also likely that the non-significant statistical test occurred due to redundancy of maternal anxiety and paternal anxiety effects.

It is, however, unclear, whether equal importance of paternal anxiety means that paternal anxiety manifests in child anxiety and processes involved in father-child anxiety transmission the same way as maternal anxiety. Based on the emotion socialization literature that differentiates between maternal and paternal parenting practices and its influences (Cheung et al., 2018), it is possible that paternal anxiety manifests in different parenting practices than maternal anxiety. It is very likely that mothers will have a different way of shaping child's emotion regulation than fathers. Mothers might more likely influence their child's regulation via nurturing, while fathers possibly more through play. While our results show that parental anxiety results in child emotion dysregulation and child anxiety regardless of how it happens, understanding of specificity in parent-child transmission is needed in order to identify the factors that can buffer, alter, or interrupt the transmission.

Similar questions about specificity of paternal anxiety influence arose from results of Study II. The bidirectional effects of paternal anxiety and child anxiety were not found to be consistent across time points, unlike for maternal anxiety. Rather, paternal anxiety at ages 6 and 8 positively predicted child anxiety at ages 8 and 10 while there were no reciprocal effects from child anxiety; interestingly, the effects flipped at the age of 10, when child anxiety was found to

predict paternal anxiety at age 15 but there was no reciprocal relationship from paternal anxiety at age 10 to child anxiety at age 15. It appears that paternal anxiety has greater consequences in earlier childhood and middle childhood than in adolescence. This is a surprising finding since literature suggests fathers take on a more important role in their children's upbringing during adolescence (McLeod et al., 2007). Perhaps there are developmental processes in play that are related to adolescence as a developmental period and that are protective from paternal anxiety influences. In their theorizing paper of father-child relationship during child development, Paquette (2004) argued that father-child relationship is of an activating nature and that the role of fathers is to connect children to the world. Since adolescence is a period of self-discovery, renegotiation of a parent-child relationship, and of greater orientation toward peers (Collins & Steinberg, 2006), the turn toward the world occurs through adolescent's push for autonomy and independence as opposed to paternal involvement, thus potentially acting as a buffer from paternal influences including paternal anxiety.

It is, however, also likely that paternal involvement and influence are important in adolescence, but the mechanism through which fathers influence their child's anxiety is no longer relevant in adolescence. For example, paternal anxiety might transmit to child anxiety as a more of a result of direct parenting. As children enter adolescence and spend less time under direct parental monitoring (Metzger et al., 2016), fathers might have less opportunities or be less engaged in direct parental practices of low autonomy granting and overcontrol that have been found to be associated with child anxiety (McLeod et al., 2011). This is in contrast with previous research that found paternal overcontrol playing a unique role in adolescence in a sample of Dutch children (Verhoeven et al., 2012). On the other hand, a parenting-child anxiety meta-analysis revealed paternal parenting to be as important, if not more, as maternal parenting in a

sample of children under 5 years of age (Möller et al., 2016), which would be more in line with our findings.

The possibility of parent-specific mechanism of transmission emerges further when comparing the strengths of associations. Paternal anxiety had the strongest association with child anxiety from age 8 to age 10. Maternal anxiety, on the other hand, had the strongest association with child anxiety from age 10 to age 15 - when the effect of paternal anxiety was found to be nonsignificant. These differences suggest that there might be unique processes involved through which each parent's anxiety manifests in child anxiety, including differential parenting or differential modeling of anxious behavior. It is also important to note that even though there was no direct effect found from paternal anxiety at age 10 to child anxiety at age 15, in Study I, paternal anxiety at age 6 was found to positively predict child anxiety at age 15. Thus, it could be assumed that the exposure to paternal anxiety in younger childhood has a greater impact on child's anxiety symptoms than exposure in middle childhood, and the transmission of anxiety from paternal anxiety experienced at younger age to child anxiety in adolescence might occur through internalized processes, such as child emotion dysregulation studied in Study I, rather than through direct influences.

Future Directions

Findings of both studies of this dissertation contribute to our current knowledge about parental and child anxiety as well as inspire further research. Study I identified child emotion dysregulation as a mechanism of parent-child anxiety transmission that focused on emotional, rather than cognitive processes as vehicles of the transmission. Future research is needed to understand the interplay between the cognitive and emotional processes, the direction of their relationship and possible interactive or additive effects. Future research is also needed to

understand the mechanism of transmission in greater detail. This study found that parental anxiety predicted child emotion dysregulation; however, it is not clear whether parental emotion dysregulation processes, as suggested by theoretical explanations, play a role in this link or not.

Previous research connected parental anxiety, particularly maternal anxiety, with maternal emotion regulation and since child emotion regulation is assumed to be learned from parental regulation, it is likely that parental regulatory processes are involved in parental anxiety-child emotion regulation link. Research investigating both parent and child emotion regulation underlying parent-child anxiety link is therefore needed.

To add a greater specificity and a more thorough understanding of parent-child anxiety link, different types of emotion regulation need be examined. As mentioned, there are many facets of emotion regulation beyond the child's ability to respond to strong emotions, which was this study's conceptualization of emotion regulation. The significant findings of this study encourage further research into other facets of child emotion dysregulation as well in order to understand what aspects of emotion regulatory capacities are potentially more relevant in development and maintenance of childhood anxiety as well as in its transmission between family members. Greater understanding of different facets of emotion regulation would therefore allow for more informed research of the interplay between cognitive and emotional processes, as mentioned previously. It would also allow for identification of emotion regulation strategies that have a potential to alter developmental trajectories of anxiety and therefore inform therapeutic interventions.

Study II provided evidence for reciprocal mother-child and father-child anxiety relationships over a nine-year period. Maternal and paternal anxiety's influence differed at the age of 10, when maternal anxiety predicted child anxiety at age 15, but paternal anxiety did not.

More research is needed to understand whether a father's influence in early and middle childhood is unique from maternal influence or whether it could be considered interchangeable. Future research needs to focus on identifying the underlying processes of anxiety transmission with greater specificity to determine whether there are any differences in how anxiety transmits from each parent and across developmental periods. For example, is the vehicle of mother-child transmission the same from early childhood to middle childhood than it is from middle childhood to adolescence? Is the vehicle of transmission from paternal anxiety in early childhood to child anxiety in middle childhood the same as it is for mother-child transmission?

Finally, more research is needed to understand what underlies these father-child anxiety effects and whether the direct impact of paternal anxiety possibly ceases in adolescence, as suggested by the results. Identifying specific mechanisms of transmission would therefore help to determine factors that have a potential to buffer the effect of paternal anxiety on child anxiety in adolescence.

Implications for Practice

Findings of both studies directly inform child anxiety treatment and have the potential to translate into concrete changes in how childhood anxiety is approached in clinical treatment. In summary, the studies yielded three main findings: 1) Anxiety aggregates in families, as evidenced by child and parental anxiety reinforcing each other developmentally over time; 2) Paternal anxiety is an important contributor to child anxiety, as evidenced by significant effects of paternal anxiety on child anxiety; and 3) Child emotion dysregulation is an underlying process of intergenerational transmission of anxiety, as evidenced by significant mediating effects.

Study findings show that parental anxiety has a strong longitudinal impact on child anxiety. Mother-child anxiety trajectories were found to be closely intertwined and predictive of

each other into child's adolescent years, while paternal anxiety impacted child anxiety directly until the age of 10 and indirectly via child emotion dysregulation until the age of 15. These results strongly suggest that parental inclusion in child anxiety treatment would be beneficial. It appears that inclusion of both parents in treatment for children younger than 10 years old would be necessary to successfully address child's anxiety symptoms and inclusion of mothers would be important for children during adolescence.

Parental influences in child anxiety development and maintenance therefore should not be overlooked by clinical practice, yet it seems to happen. The recommended treatment for childhood anxiety continues to be individual cognitive-behavioral therapy and medication (CBT; ADAA, 2020). The recommendation of CBT is certainly justified, since it has received a significant amount of attention in research that supported its effectiveness. For example, participation in a 12-week combination therapy of CBT and medication was found to reduce child anxiety symptoms for up to 68% of children (Ginsburg et al., 2011). However, a six-year follow up showed that almost half of the children relapsed (Ginsburg et al., 2014), while only about 20% remained in stable remission.

Individual CBT treatment is therefore an effective approach for child anxiety treatment; however, as Ginsburg et al.'s (2014) follow-up study illustrated, it appears CBT's effectiveness has limitations and it does not remain stable over time. This is not surprising given the findings in the current two studies which showed that child anxiety was predicted by parental anxiety. A child who receives individual treatment returns to the family unit where they are exposed to the same anxious socialization that is provided by their anxious parents. Without treatment support, the child can easily revert and relapse under the influence of parental anxiety. In other words, parental anxiety that contributed to child's anxiety symptoms development in the first place

would continue to reinforce a child's anxiety symptoms over time, as was shown by the cross-lagged models in Study II.

In order to provide an alternative to CBT that would include parental involvement, Lebowitz et al. (2020) conducted a comparison trial with children randomly assigned to individual CBT or parent-based treatment aimed at reducing parental accommodation of child anxiety. Family accommodation is an interpersonal feature of child anxiety that involves parental behaviors aimed at alleviating or avoiding the child's anxious distress. Family accommodation behaviors typically include parental participation in a symptom-driven behavior, such as parents changing their schedules or family routines, speaking on behalf of the child, and facilitating avoidance of school or social functions (Norman et al., 2015). At the end of the trial, the authors concluded that the CBT approach and the parent-based approach were comparably effective. Significantly greater reduction of anxiety accommodation occurred only in the parent-based program (Lebowitz et al., 2020).

The concept of family accommodation shows the susceptibility of parents to the child's anxiety and the interconnectedness of parental and child anxious distress. Parents who are likely to accommodate their child's anxiety are also likely to struggle with regulating their own anxiety, particularly around the child's anxious presentation. Family accommodation therefore shows how parental and child anxiety reinforce each other, which was also demonstrated by findings of Study II.

Interestingly, Lebowitz et al.'s (2020) parent-based treatment that included interventions for parents only was similarly successful in reduction of child anxiety symptoms as individual CBT administered to children only. These results suggest that change on either parental or child level is sufficient for the child's anxiety symptom reduction. It is possible that child's lowered

anxiety as a result of less parental accommodation also lowered parental anxiety in turn. Results from the current study would support interventions aimed at parental change; however, given the longitudinal reciprocity of maternal and child anxiety revealed by Study II findings, it is unclear whether long-term remission of child anxiety symptoms could be maintained by parent intervention only and whether reduction of child anxiety as a consequence of less parental accommodation would be potent enough to have a reciprocal impact on parental anxiety. In order to effectively target child anxiety and maintain anxiety symptom reduction over time, current study findings indicate that both parents, but especially mothers, would need to be involved in treatment that would be aimed at addressing both parental and child anxiety symptomatology.

Alternatively, a mechanism of parent-child anxiety transmission could be targeted in treatment in order to disrupt the child's acquisition of anxious symptoms from parents. Study I furthered our understanding of how parent-child anxiety transmission by identifying child emotion dysregulation as one of its mechanisms. Identification of child emotion dysregulation's role in child anxiety maintenance opens new possibilities for effective anxiety treatment. CBT is a treatment that is predominantly focused on targeting anxious thoughts and anxious behaviors. CBT's focus on anxious thoughts was also mirrored by research that primarily examined cognitive distortions as mechanisms of parent-child anxiety transmission. Study results provide some evidence indicating that targeting child emotion dysregulation could have potentially equally positive effects. Moreover, including parents in treatment focused on emotion regulation might be easier, since the idea that children learn their emotion regulation strategies from their parents is commonly accepted. A treatment that would help parents with child's coregulation would also inherently include helping parents with their regulatory strategies.

A family therapy approach to child anxiety treatment would therefore require active participation and involvement of parents during and between therapy sessions. An important component of targeting child emotion dysregulation during therapeutic treatment is supporting the parents of an anxious child to help them stay regulated when their child becomes dysregulated and shows anxious distress. Findings of Study II appear to be consistent with what happens in a therapy room when a child becomes anxious during a session and shares their anxious experiencing with their parent. Anxious parents will likely have a tendency to mirror a child's anxious dysregulation by becoming distressed themselves and, subsequently, will respond to the child in an anxious manner that would further fuel the child's anxiety. A therapist can support parents by helping them to recognize and become aware of their own anxious dysregulated response as well as of the impact that their anxious reactivity has on their interactions with their child that further reinforce child anxiety. Parents can learn multiple emotion regulation strategies during therapy treatment that would help them remain calm and regulated when faced with their child's anxiety. In turn, anxious children will be able to safely share their anxious distress without worrying about the impact on their parent. A calm and regulated parent will also model a different response to their anxious child that is unfamiliar to the child at first, but that the child will perceive as calming and safe if the parental regulated response remains consistent. As a result, an anxious child will feel more understood in their interactions with their parent and will also learn more appropriate and regulated responses to perceived distressing events that would reduce the child's anxiety, as opposed to exacerbate it.

Limitations

Limitations shared by both studies are related to sample and measurement. First, both studies utilized samples that were fairly homogenic with majority of participants being White

and middle-class. Second, both studies used parental reports as a child anxiety measure rather than objective measures of anxiety gained through observation or child reports. Given the discrepancy in mother-reported and father-reported child anxiety, it would be interesting to see whether including objective child anxiety ratings or self-reports would change the results in this study.

In Study I, the software (Amos, v25.0; Arbuckle, 2017) used to conduct the mediation tests required data with no missing values to compute confidence intervals, and therefore list-wise deletion was employed to obtain such a sample. List-wise deletion is not a preferred way to treat missing values due to its reduction of sample size that might result in a loss of statistical power and in potential sample bias. Comparison analysis revealed that cases with and without missing values differed on some demographic variables in the mother-child sample. Perhaps attrition associated with longitudinal data collection contributed to these differences. Despite the differences on demographic variables, however, there were no differences on main study variables, and therefore it can be assumed that the differences on demographic variables did not affect study findings. Comparison analysis in father-child sample found statistically significant differences on two main study variables, specifically paternal anxiety and child emotion dysregulation, but no statistically significant differences in child anxiety and demographic variables. There was no loss of statistical power due to a large sample size in either sample; however, given the number of missing cases, there is no way of knowing whether deleting these cases had an impact on final study results.

Further, the largest number of mother and father participants were included in Study I and Study II. This resulted in different sample sizes for the mother-child sample and for the father-child sample. Given that all fathers in the father-child sample had a matching mother in

the mother-child sample, but a large number of mothers in the mother-child sample did not have a matching father, it is possible that there are important differences between the mothers included in the mother-child models and the mothers included in the family models that could have impacted study findings. Without understanding and measuring these differences and addressing them, it is not possible to know whether study findings were impacted. Alternatively, a listwise deletion approach could have been used that would have reduced the mother-child sample to include only mothers with matching fathers. However, by doing so, it is likely that the mother-child sample in both studies would have been prone to sample bias as that is one of the limitations of listwise deletion.

Finally, emotion regulation is a complex concept that is often operationalized differently in empirical studies. In this study, child emotion dysregulation was operationalized as the extent of emotional reactivity to life events (Larsen, 1984). Individuals with anxiety have been shown to react faster to events and with higher intensity of emotions (Cisler et al., 2010), and therefore Larsen's (1984) operationalization of emotion dysregulation seems well-suited. Additionally, a recent study focused on child emotion regulation and adolescent internalizing symptoms utilized the same measure of emotion dysregulation (Gregory et al., 2019). However, given that emotion regulation is a multifaceted construct, ideally different aspects of emotion regulation would be measured.

In Study II, several models' fit was under the cut-off score of what is a considered a good fitting model. While model fit is an important indicator in analysis with latent constructs, it appears that it could be less of a concern when observed variables are included in the model. Multiple factors, such as model complexity that increases with the use of longitudinal data, impact the value of model fit indices and contribute to the challenge of obtaining adequate model

fit. It is likely that the complexity of the cross-lagged longitudinal model negatively impacted the overall model fit in our analyses. Data analysis was executed without any error messages and provided the regression coefficients of interest, despite the model overall providing some evidence of inadequate fit to the data. Since the units of analysis in path models are based on observed variables, there needs to be a balance between obtaining coefficients of effects versus achieving excellent fit. Hu and Bentler (1998) noted that it is important to focus on adequacy and interpretability of parameter estimates in the determination of whether a model is valid and whether a model provides new information about studied phenomenon, in this case about the development of anxiety in children.

Poor model fit might also be related to model misspecification as well as sensitivity to small local misspecifications (Hu & Bentler, 1999), which was observed in Study 1 where the initial model had an excellent fit and adding control variables provided evidence of a very poor fit. Finally, Marsh et al. (2004) argued that an overreliance on model fit cut-off values can lead to rejecting acceptable models and thus type I error, and therefore they advocated for acceptable-misspecified models if there is adequacy and interpretability of parameter estimates, which was achieved in this study.

Conclusion

The two studies of this dissertation focused on expanding our understanding of the role of parental anxiety in child anxiety etiology. The main contributions of the studies to the current literature include: 1) a confirmation that fathers and their anxiety significantly and longitudinally impacted child anxiety - a notion that was theorized but lacked empirical evidence; 2) the identification of an additional underlying mechanism of parent-child transmission, namely child emotion dysregulation, which contributes to the acquisition of anxiety symptoms via cognitive

processes, thus expanding potential targets for anxiety family treatment; and 3) evidence for both longitudinal and reciprocal relationships between parental and child anxiety, providing additional support to the notion that anxiety aggregates in families, thus further highlighting the impact of parental anxiety that persists over time and across developmental stages.

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Appendices

Appendix A

Table A

Measures

Variable	Measure	Items	Score Range
Maternal and Paternal Anxiety	State-Trait Anxiety Scale (modified from Spielberger et al., 1983)	1. I felt calm. 2. I was tense. 3. I felt at ease. 4. I was worrying over possible misfortune. 5. I felt nervous. 6. I was jittery. 7. I was relaxed. 8. I was worried. 9. I felt steady. 10. I felt frightened.	*1=not at all - 4=very much*
Child Anxiety	Child Behavior Checklist – Anxiety Problems (Achenbach et al., 2003)	1. Clings to adults or too dependent 2. Fears certain animals, situations, or places, other than school 3. Fears going to school 4. Nervous, high-strung, or tense 5. Too fearful or anxious 6. Worries	*0=not true - 2=very true or often true*
Child Emotion Regulation	Parent Report of Children's Reaction Scale (adapted from Larsen, 1984)	1. When my child feels an emotion, either positive or negative, my child feels it strongly. 2. After finishing a difficult task, my child feels delighted or elated 3. My child responds very emotionally to stories, movies and events. 4. My child is calm and not easily aroused. 5. When angry, it is easy for my child to still be rational and not overreact. 6. When happy, my child is contented and calm rather than exhilarated and excited. 7. When my child experiences anxiety, the anxiety is normally very strong. 8. Even when happy, sad, or upset, my child does not get highly emotional. 9. When happy, my child is bursting with joy. 10. My child is slow to become angry, nervous or upset.	*1=never - 5=always*

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MEMORANDUM

DATE: September 26, 2018

TO: Megan Leigh Dolbin-MacNab, Katarina Krizova, Christine Kaestle, Carolyn Shivers

FROM: Virginia Tech Institutional Review Board (FWA00000572, expires January 29, 2021)

PROTOCOL TITLE: Family Factors in Childhood Anxiety

IRB NUMBER: **18-790**

Effective September 25, 2018, the Virginia Tech Institution Review Board (IRB) approved the New Application request for the above-mentioned research protocol.

This approval provides permission to begin the human subject activities outlined in the IRB-approved protocol and supporting documents.

Plans to deviate from the approved protocol and/or supporting documents must be submitted to the IRB as an amendment request and approved by the IRB prior to the implementation of any changes, regardless of how minor, except where necessary to eliminate apparent immediate hazards to the subjects. Report within 5 business days to the IRB any injuries or other unanticipated or adverse events involving risks or harms to human research subjects or others.

All investigators (listed above) are required to comply with the researcher requirements outlined at:

http://www.irb.vt.edu/pages/responsibilities.htm

(Please review responsibilities before the commencement of your research.)

PROTOCOL INFORMATION:

Approved As:	**Exempt, under 45 CFR 46.101(b) category(ies) 4**
Protocol Approval Date:	**September 25, 2018**
Protocol Expiration Date:	**N/A**
Continuing Review Due Date*:	**N/A**

*Date a Continuing Review application is due to the IRB office if human subject activities covered under this protocol, including data analysis, are to continue beyond the Protocol Expiration Date.

FEDERALLY FUNDED RESEARCH REQUIREMENTS:

Per federal regulations, 45 CFR 46.103(f), the IRB is required to compare all federally funded grant proposals/work statements to the IRB protocol(s) which cover the human research activities included in the proposal / work statement before funds are released. Note that this requirement does not apply to Exempt and Interim IRB protocols, or grants for which VT is not the primary awardee.

The table on the following page indicates whether grant proposals are related to this IRB protocol, and which of the listed proposals, if any, have been compared to this IRB protocol, if required.

Date*	OSP Number	Sponsor	Grant Comparison Conducted?

* Date this proposal number was compared, assessed as not requiring comparison, or comparison information was revised.

If this IRB protocol is to cover any other grant proposals, please contact the IRB office (irbadmin@vt.edu) immediately.