TEMPERAMENT, PARENTAL ANXIETY AND THEIR ROLE IN THE DEVELOPMENT
OF CHILD ANXIOUS PSYCHOPATHOLOGY

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ABSTRACT

Much emphasis has been placed on parental contribution in the development and progression of psychopathology. Although it has been established that children of parents with anxiety disorders are at greater risk for developing anxious psychopathology themselves (e.g., Dadds & Roth, 2001; Rosenbaum, Biederman, Hirschfield, Bolduc, & Chaloff, 1991), the specific developmental path is not yet completely understood (Kendall & Ollendick, 2004). This cross-sectional study examined how child temperament (e.g., negative affectivity - NA) and parental anxiety symptom expression were related to the development of anxiety in a clinically referred sample of 570 children and adolescents. Structural equation modeling provides evidence that both child NA and parental anxiety were associated with child anxiety. Secondly, the relationship between parent and child anxiety symptoms was significant even when controlling for the influence of child NA. In other words, there was an association between child and parent anxiety that was not accounted for by the temperamental dimension of NA. Finally, in younger children (ages 7 to 11), parent anxiety’s influence on child anxiety was weaker than in older children (ages 12 to 19), suggesting that, as children grow older, the influence of parental anxious behavior grows stronger.
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Introduction: Childhood Anxiety

Anxiety is among the most common disorders of childhood and adolescence, with lifetime prevalence rates between 6 and 18%, depending on age, gender, and diagnostic category (Anderson, Williams, McGee, & Silva, 1987; Costello, Egger, & Angold, 2005; Ihle & Esser, 2002; Kashani et al., 1987; Roth, Dadds, & Weems, 2004). Some anxiety can be normal and adaptive, such as fleeing from a genuinely dangerous situation. Yet, anxiety can also be extremely debilitating if anxious feelings are evoked by a subjectively scary but objectively benign situation or object. Diagnosis of an anxiety disorder can be warranted for a child whose anxiety is excessive in frequency and/or duration, and that anxiety impairs her functioning at home, in school and/or with peers (Ollendick, Shortt, & Sander, 2005). Anxiety disorders developed in childhood are likely to continue into adulthood, and are often positively correlated with other non-anxiety behavior disorders (Albano, Chorpita, & Barlow, 2003). Understanding the development of excessive anxiety in children and adolescents is critical to developing preventative strategies.

Anxiety Runs in Families

Although investigators have identified several risk factors associated with anxiety, prior research has focused on parental contributions in the development and progression of anxiety disorders in children. This focus has been motivated by the observation that children of parents with anxiety disorders are at greater risk for developing anxious psychopathology themselves, when compared to children of parents without anxiety disorders (Dadds & Roth, 2001; McClure, Brennan, Hammen, & Le Brocque, 2001; Rosenbaum, Biederman, Hirschfield, Bolduc, & Chaloff, 1991). In particular, a recent
study by Cooper, Fearn, Willets, Seabrook, and Parkinson (2006) found a strong association between maternal and child anxiety.

Even though prior research has failed to identify a single direct pathway from parental variables to childhood anxiety, two major causal paths are often theorized to account for this transmission: inherited temperament and environment (Dadds & Roth, 2001; Lonigan & Phillips, 2001; Vasey & Dadds, 2001). This study offers a model for how child temperament and parental environment fit into the development and maintenance of child anxiety. More specifically, it investigates whether a clear association exists between parental anxiety and the emergence of childhood anxiety, above and beyond inherited temperament.

Risk Factors

Genetics

Many twin studies have found a heritable risk for developing anxiety disorders (Ollendick et al., 2005). One’s genes are thought to account for approximately one third of the variance when measuring anxiety, as indicated by both twin and adoption studies (Albano et al., 2003; Eley & Gregory, 2004). Still, genetic studies of childhood anxiety also demonstrate that environmental factors play a large role in the development of these disorders (Eley, 2001; Eley & Gregory, 2004). The influence of shared environment accounts for 20% of the variance in the development of childhood anxiety, and nonshared environment accounts for the remaining 50% (Eley & Gregory, 2004).

Shared environment refers to experiences common to all siblings, and nonshared environment refers to effects which makes family members different from one another. The influence of shared environment on the development of anxiety is quite unique, as it
rarely has a significant impact on other behavioral disorders in children and adults (Eley & Gregory, 2004). In her 2001 review, Eley suggests that maternal psychopathology is the most obvious factor in a shared environment that contributes to anxiety. Nevertheless, it is difficult to isolate the part of maternal anxiety that influences a child through shared environment versus the influence of shared genes.

Genetics appear to contribute a general vulnerability for anxiety and depression, but not for specific anxiety disorders (Albano et al., 2003; Ollendick et al., 2005). For example, Eley and Stevenson (1999) found that genetic variance accounted for most of the correlation between anxiety and depression, but that environmental measures were responsible for discriminating between anxiety and depression. This study reinforced Eley’s findings in 1997 that shared genes explained a great portion of the variance in 490 twin pairs with anxious and depressive symptoms (ages 8-16). Kendler, Neale, Kessler, Heath and Eaves (1992), in their study of monozygotic twins, also found that genetic predisposition supplies a general risk factor for both anxiety and depression, but not for specific anxious or depressive disorders. Furthermore, Kendler et al. found that stressful events in childhood, in addition to a genetic disposition, place individuals at greater risk for the development of mood and anxiety disorders in adulthood. Finally, while Andrews, Stewart, Morris-Yates, and others (1990) found no evidence for the inheritance of specific anxiety disorders in their large twin study, they concluded that a general inclination toward neurosis was heritable.

Altogether, these genetic studies suggest there is room for environment to supplement genetics in the emergence of anxiety disorders. They also imply that
environmental influences, unlike anxiety-prone genes, may be disorder-specific in some cases (Chorpita & Barlow, 1998).

Temperament

Research on temperament may help clarify what portion of a child's anxiety is inherited and what remainder is caused by environmental influences. Inherited temperament represents a theoretical means of transmitting parents' inclination for anxiety to his or her child. It is defined as a set of personality traits, including emotionality and behavioral style, which are inherited and usually appear during the first years of life. Temperament is commonly seen as an enduring, stable quality across development (e.g., Lonigan & Phillips, 2001; Thomas & Chess, 1977). For instance, retrospective studies have shown that a difficult temperament in infancy is associated with mood difficulties later in life (Dadds & Roth, 2001). Gray (1982), and Watson and Clark (1984) agree that a general risk factor involving sensitivity to negative stimuli is associated with harmful emotional and behavioral outputs. Their separate theories of inherited temperament are discussed below.

Gray's behavioral inhibition model of anxiety. According to Gray (1982), the brain has an aversive motivational operating system called the "behavioral inhibition system" (BIS). This physiological mechanism is sensitive to signals for punishment, signals for nonreward, and novelty. When confronted with anxiety-relevant cues, the BIS controls the experience of anxiety by inhibiting behavior that may lead to negative or painful outcomes. Thus, BIS activation causes a decrease in movement toward goals. Gray argues that BIS functioning is responsible for feelings such as anxiety, fear, frustration and sadness in response to anxiety-relevant cues. The inhibition system is
characterized mainly by increased attention, vigilance, and processing of threat relevant information, and does not involve traditional conceptualizations of anxiety such as physiological arousal (e.g., fast heartbeat). In sum, people with high BIS sensitivity should be especially responsive to punishment cues, they should experience great anxiety in situations with cues of impending punishment, and they might be predisposed to anxiety or depressive disorders. Gray's model of personality parallels the tripartite model below. Whereas the BIS construct is similar to the negative affectivity construct in the tripartite theory, the questionnaires and data used in this study rely on the latter, newer model.

*The tripartite model.* Many researchers have studied anxious temperament via a large, general construct of temperamental emotional distress. In 1984, Watson and Clark labeled this construct "negative affectivity". Seven years later, they used a tripartite model of emotion to specify the association between Negative Affect (NA), Positive Affect (PA), and Physiological Hyperarousal (PH). Watson and Clark used these three variables to help explain the relationship between anxiety and depression, with NA being a broad factor related to depression and anxiety, (low) PA being a specific factor related to depression, and PH being a specific factor related to anxiety.

Negative Affectivity (NA) was initially proposed after observing the high comorbidity of anxiety and depressive disorders in clinical populations (Watson & Clark, 1984). NA is defined as a “stable, highly heritable general trait dimension with a multiplicity of aspects ranging from mood to behavior” (Clark, Watson, & Mineka, 1994, p.104). High NA is a composite of distress and disengaging that includes a variety of negative moods such as anger, contempt, disgust, guilt, fear, and nervousness, whereas
low NA signifies a state of calmness and serenity (Watson, Clark & Tellegen, 1988, p. 1063). An individual with high NA has the propensity to feel tired, distressed, worried and sad (Turner & Barrett, 2003).

Positive Affectivity (PA), the second factor, is associated with energy, enthusiasm, mental alertness, interest, joy, and determination. Low PA, on the other hand, is related to lethargy and tiredness (Watson, Clark & Carey, 1988). Whereas depression has been found to represent a mixed state of high NA and low PA, anxiety is characterized by high NA with no important relation to PA (Watson et al., 1988). In clinical samples, anxiety disorders are often found alone without comorbid depression, whereas depression is usually comorbid with anxiety (Albano et al., 2003; Cole, Peeke, Martin, Truglio, & Ceroczynski, 1998). This asymmetrical comorbidity suggests that anxiety may developmentally precede depression in an individual with high NA (Albano et al., 2003).

The last factor, Physiological Hyperarousal, (PH), relates to anxiety in that it represents autonomic nervous system alterations (such as increased heart rate) in response to anxiety-provoking situations (Lonigan, Phillips, & Hazen, 2004). However, recent research with adults and children has shown that PH is only related to some anxiety disorders (e.g., panic disorder) and not others (Brown, Chorpita, & Barlow, 1998a; Chorpita, 2002; Chorpita, Plummer, & Moffitt, 2000; Mineka, Watson, & Clark, 1998). Studies have also found that PH might not be temperamental in nature, and therefore not as useful when examining the development of anxiety (Lonigan & Phillips, 2001).
Adult negative affectivity and internalizing psychopathology. NA appears to distinguish anxiety and depression from other diagnoses, whereas PA helps distinguish anxious from depressed symptoms for purposes of treatment and research (Clark & Watson, 1991; Mineka, Watson, & Clark, 1998; Watson, Clark & Carey, 1988; Watson, Clark & Tellegen, 1988). If an individual has high NA, anxiety and/or depression may develop. However, if an individual has high NA and low PA, depression is more likely to manifest itself. As NA relates directly to anxiety and PA does not, this study will focus primarily on the former construct.

NA has been found to correlate with, and is thought to underlie, both depressed and anxious symptomatology in adults (Brown et al., 1998a; Lonigan, Carey & Finch, 1994; Watson, Clark, & Carey, 1988). Brown et al. (1998a), using a clinic-referred sample of 350 adults with anxiety and mood disorders, found that NA influenced anxiety and depressive disorders, and that low PA was significantly related to depression. Their evaluations of various structural models were consistent with the tripartite model (Brown et al., 1998a). Thus, the NA construct may be of great importance when studying the development and maintenance of emotional disorders in adults.

Child negative affectivity and internalizing psychopathology. The tripartite model has also been studied in child and adolescent populations, both clinical and non-clinical, and findings have been relatively consistent with studies on adults.

With regard to clinical populations, Lonigan, Carey, and Finch (1994) found that measures of low PA differentiated children with depression from children with anxiety in their study of 233 inpatient children with either an anxiety or a depressive disorder. That is, low PA scores were able to detect children with depression. These children reported
more problems related to loss of interest and low motivation, and they had more negative
views of themselves. In contrast, anxious children reported that they worry more about
the future, their well-being, and the reactions of others. The two groups did not vary in
terms of reporting being annoyed by things or feeling alone and isolated (Lonigan et al.,
1994). These findings suggest there is a general negative affectivity element in both
anxiety and depression disorders. Lonigan et al. (1994) concluded that these disorders in
children have unique features which can be detected by using self-report instruments. In
particular, the positive affectivity construct may differentiate between anxiety and
depression in children.

Joiner, Catanzaro, and Laurent (1996) also found support for the tripartite model in
children. They conducted an exploratory factor analysis of 116 child psychiatric
inpatients and found that three factors emerged (depression, anxiety, and negative affect)
which were consistent with the three factor model of negative emotions. Chorpita,
Albano and Barlow (1998) uncovered further evidence for the tripartite model when
investigating latent factors associated with anxiety and depression in a sample of 216
children and adolescents with anxiety and mood disorders. Chorpita et al. (1998) used
multiple informants (child and parent measures) to gather information for their
exploratory analysis of the structure of negative emotions. They concluded that a three
factor solution (i.e., tripartite model) was the best fitting model of symptoms of anxiety
and depression in a clinical sample of children.

Several community studies have revealed similar results. For example, Lonigan,
Hooe, David, and Kistner (1999) examined the relationship of PA and NA constructs to
anxiety and depression in a school sample of 365 children and adolescents. They
discovered that child measures of NA and PA had results consistent with adult findings. Specifically, NA was strongly related to measures of anxiety symptoms and depression symptoms. PA, especially in the adolescent sample, was more strongly related to the measure of depressive symptoms than to the measure of anxiety symptoms.

Furthermore, in a study of 270 students (4th to 11th graders) over the course of seven months, Lonigan, Phillips and Hooe (2003) established that high NA predicted higher future anxiety and low PA predicted higher future depression. These authors also found that NA and PA were moderately consistent over time, supporting the view that these constructs are due to temperamental and not situational factors. As such, Lonigan et al.’s (2003) findings reinforce the theory that NA and PA have a role in the development of anxiety and depression in children.

When investigating these issues in a large school sample of 1,578 children and adolescents, Chorpita (2002) concluded that the tripartite model was maintained across gender and age groups. Specifically, he found that NA correlated positively with anxiety and depression, and that depression was negatively related to PA. The PH construct was associated with panic only, and not with the other anxiety disorders, a finding consistent with Brown et al.’s (1998a) study of adults. Moreover, while Generalized Anxiety Disorder (GAD) was not significantly related to PH in Chorpita’s (2002) study of children, the two variables were negatively associated in Brown and colleagues’ (1998a) study of adults. Although not statistically significant, Chorpita (2002) also observed a negative association between GAD and PH that grew stronger with age.

In sum, many studies of children and adolescents have found that NA is a general risk factor underlying both anxiety and affective disorders in this population (Chorpita,
Daleiden, Moffitt, Yim, & Umemoto, 2000; Lonigan & Phillips, 2001; Ruschena, Prior, Sanson, & Smart, 2005). As a general risk factor, NA is more than just a state of anxiety and/or depression; it is conceptualized as a trait and is stable across time (Lonigan, Hooe, David & Kistner, 1999; Lonigan, Phillips & Hooe, 2003). Furthermore, prior research provides evidence that the symptoms of psychological disorders are different from temperament. When the common variance between symptoms of disorders and temperament is removed from psychological measures, there continues to be a significant relationship between the two variables (e.g., Lemery, Essex, & Smider, 2002; Lengua, West, & Sandler, 1998).

Summary of the Negative Affectivity Construct

Prior research on NA in both adults and children has arrived at the same conclusion: NA is related to depressive and anxious symptomatology and persists over time. Because NA is thought to be stable and heritable and is found in both children and adults, parents with high NA are at greater risk of having anxiety and depression difficulties, and are potentially at greater risk of having children with high NA. Studying NA is vital to understanding the trajectory of anxiety. Few studies have investigated this hypothesized link between parent and child NA.

Child’s Environment and Parental Responses

Genetics and temperament are likely important, distal risk factors in the development of anxiety disorders. Nevertheless, although researchers have concluded that some aspect of temperament is associated with later development of anxiety disorders, not all children with an anxious temperament develop these problems (Hudson & Rapee, 2004). There must be other—environmental—factors that predict the
occurrence of anxiety disorders in at-risk children. In fact, the evolution of anxiety may largely be the result of environment (Eley, 2001).

Many environmental variables, such as peer relationships, traumatic social events and other conditioning experiences might be responsible for the persistence of anxiety difficulties in children. Researchers who have studied the maintenance of a general vulnerability for anxiety stress the role of the early environment, particularly the influence of parents (McClure et al., 2001). Consequently, the following review of environmental contributors to child anxiety focuses on parental responses.

*Unintentional reinforcement.* One manner in which environmental factors may affect anxiety is via unintentional reinforcement. Children with high NA may respond to anxiety-provoking situations in a manner that reinforces their symptomatology (Vasey & Dadds, 2001). These children may cope with stressful stimuli by avoiding them. This evasion decreases uncomfortable anxiety, and thereby increases the likelihood one will avoid similar situations in the future. Moreover, such behavior could decrease their opportunities to habituate to and master important developmental contexts. Avoiding a negative consequence is reinforced with a positive outcome, a process called negative reinforcement in operant conditioning terms.

Anxious children’s avoidance responses persist even in the absence of the original aversive stimulus. These avoidant behaviors also contradict established helpful responses in anxious situations – the main cognitive behavioral treatment for anxiety is exposure to the feared stimulus. Short-term decisions to avoid have long-term consequences. By avoiding the anxiety-provoking situation, children with high NA reduce their chances of
a corrective experience (e.g., Kendall, 1992). Accordingly, reducing immediate avoidance in children is imperative to long-term anxiety-reduction (Kendall, 1992).

Avoidance behaviors and excessive neediness in anxious children also increase the likelihood that parents, peers, and teachers will initially shelter them, and then ultimately reject such behavior. These adults and peers may end up pushing these children towards autonomy and interaction with challenging stimuli too quickly (Dadds & Roth, 2001). According to the unintentional reinforcement theory, forcefully or hastily goading an anxious child toward independence will lead to, and later increase, the child’s stress and fear, which leads to more and more avoidance (Fox & Calkins, 1993). Instead, these parents should systematically desensitize their child toward challenging situations. Otherwise, the poor parental behaviors mentioned earlier will make the child even more likely to avoid in the future, and the negative cycle will continue to repeat itself (Dadds & Roth, 2001).

Overprotection. Inhibited or avoidant behaviors by the child might also foster more “helping” behaviors from parents, sometimes called overprotection. Overprotection can be defined as “the degree to which parents limit and constrain the behavior of the child, particularly in threatening or novel environments” (Chorpita, 2002, p. 126). These constraints can encourage maladaptive behaviors in children. Retrospective questionnaire studies indicate that parental over-involvement is indicative of a child’s anxiety (Hudson, Flannery-Schroeder, & Kendall, 2004). Children’s perceptions of over-control and anxious childrearing by parents have been found to be correlated with increases in anxious symptomatology (Muris & Merckelbach, 1998). Data from observational studies suggest that parents of anxious children are more likely to facilitate avoidance (Dadds,
Barrett, Rapee, & Ryan, 1996), which is the opposite of a helpful response when children encounter challenging situations (Vasey & Dadds, 2001).

For example, Hudson and Rapee (2001) gave children a moderately stressful puzzle task, and then assessed mothers' behaviors during performance of the task. Mothers of anxiety disordered children behaved differently than those of non-anxious children. In particular, the anxious children's mothers gave more unsolicited help and were more intrusive during the task. Findings from this study strengthen the theory that parents of children with anxiety disorders often attempt to prevent their children from feeling distressed, and therefore do not allow them to solve problems on their own. Because anxious parents limit their child's exposure to potentially distressing or threatening situations, it is believed that children with internalizing disorders have little experience successfully coping with such situations. As a result, these children are even less likely to submit themselves to the avoided situation, and will probably miss out on important developmental contexts and experiences (Vasey & Dadds, 2001).

Modeling. Many observational studies have found that modeling or conditioning methods in family exchanges serve to heighten a child's expressed anxiety. For example, in a study by Barrett, Rapee, Dadds and Ryan (1996b), 233 nonclinic children (ages 7 to 14) were asked to generate explanations in response to an ambiguous description of a hypothetical situation (e.g., moving toward a group of laughing peers). Parents were invited to complete the same procedure, after which both parents and children were gathered together to discuss and develop a mutual solution. Following conversations with their parents, children with anxiety disorders interpreted more threat than the control group and the oppositional-children group. They were also more likely to change from a
pro-social response to a more anxious interpretation. These findings suggest that parents of anxious children often model maladaptive anxiety, and that in turn, their children report more anxiety than they initially indicated.

These findings have been replicated by Shortt, Barrett, Dadds, and Fox (2001a), and Chorpita, Albano, and Barlow (1996). These investigators also found that anxious children (ages 6-14, and 9-13, respectively) are more likely to evaluate ambiguous situations as threatening as compared to nonclinic controls. In addition, while the former study provided evidence that maternal distress was associated with the child’s heightened avoidance after the family conversation, the latter study found that parental levels of anxiety influence their child’s anxiety.

Dadds et al. (1996) used the ambiguous situation task with pre and post-situation discussions to show that parents of anxious children often promote avoidant behaviors. He compared interactions between 66 anxious children (ages 7-14) and their mothers with 18 non-anxious children and their mothers. Anxious children reported more avoidant coping responses after conversations with their parents. Also, parents of anxious children were more likely to agree with and support anxious interpretations and avoidant strategies suggested by their children as compared to parents of non-anxious children.

A more recent observational study of play situations (Turner, Beidel, Roberson-Nay & Tuervo, 2003) compared the behavior of clinically anxious mothers to non-clinically anxious mothers. Turner et al. found that anxious mothers reported feeling higher levels of distress when observing their children playing. However, the two groups of mothers did not differ in measures of anxious affect or the number of anxious statements directed at their children. There are several ways to interpret these findings. It
is possible that mothers behave differently in an observational situation versus a non-experimental, real-life parent-child interaction. It could also be that anxious mothers communicate their internal anxiety to children in subtle ways that are not detected by the observational method used in this study.

For instance, Gerull and Rapee (2002) found that young children learn anxious behaviors by using their mothers as a reference for emotional cues. These authors examined whether a mother’s fearful response to threatening stimuli (rubber spider and rubber snake) would affect her infant’s behavior (15 to 20 months old). Initially, the mother was instructed to respond to the stimuli in front of her child with either a fearful face and tone of voice, or a happy face and tone. After a few minutes delay, the infant was again presented with the stimuli, but this time his or her mother’s face was neutral. Children were more likely to avoid the object if it was initially presented with a fearful expression from their mothers. If an infant’s stimulus-response can be altered so easily by parental responses, it seems plausible that even more dramatic effects might occur for children who are already temperamentally inhibited. The amount of parental anxiety may also affect a child’s response to the modeling of such behavior. The more anxious the parent, the stronger the parent’s fearful or ambiguous behavior, and the more this behavior can shape their child’s conduct (Hudson & Rapee, 2004).

A recent study of 3-4 year-olds found that maternal anxiety and temperamental inhibition were independently associated with child anxiety (Shamir-Essakow, Ungerer, & Rapee, 2005). In addition, the highest levels of anxiety were found in children who were behaviorally inhibited, insecurely attached, and had a mother who was also anxious. However, the measure Shamir-Essakow et al. used to evaluate maternal anxiety (i.e., the
State-Trait Anxiety Inventory) assesses several symptoms which are not anxiety specific. It can best be conceptualized as a measure of general negative affectivity/distress rather than of pure anxiety (Kennedy, Schwab, Morris, & Beldia, 2001). Shamir-Essakow et al.’s results can therefore only be interpreted as supporting a link between child anxiety and maternal negative affectivity more generally, as opposed to maternal anxiety specifically. It remains unclear whether the link between maternal anxiety and child anxiety is due to genetic and/or environmental influences (i.e., parenting).

Attachment and social learning theories. The exacerbation of internalizing symptoms by children and parents can be conceptualized with attachment and social learning theories (Dadds & Roth, 2001). Bowlby (1982) viewed an infant’s relationship with its primary caregiver as crucial for later development. The foundation of an infant’s attachment is the mother’s (or primary caregiver’s) responsiveness to the child’s needs and signals. An infant whose mother is responsive will learn to expect an accessible, caring mother and tend to feel secure in the infant-mother relationship. As the child grows, he or she learns to use the mother as a secure base, and will feel confident exploring the world around him or her. In contrast, a child who does not experience a secure relationship with the mother will tend to be anxious about maintaining closeness to the mother and will not have a secure base to extend outward from (Fuendling, 1998).

According to the attachment model, child anxiety problems are influenced by unsuitable parenting, or a child’s difficult temperament that leads to difficulty parenting (and in turn, the application of inappropriate parenting styles) (Warren, Huston, Egeland, & Sroufe, 1997). For instance, maternal anxiety has been found to contribute to a child’s insecure attachment (Manassis, Bradley, Goldberg, Hood, & Swinson, 1994). Manassis et
al. (1994) found that 80% of preschool children who had mothers diagnosed with an anxiety disorder were insecurely attached. Warren et al. (1997) found that the interaction of early insecure attachment and temperament might be predictive of later anxiety problems. Conversely, a secure attachment style may serve to buffer anxiety (Manassis, Hudson, Webb, & Albano, 2004). For example, Pincus and Barlow (2002) found that an intervention designed to improve the attachment between parent and child in separation-anxious children resulted in most children no longer meeting diagnostic criteria for separation anxiety disorder.

In contrast to the attachment model, social learning theory (SLT) places much less emphasis on temperament, and much more weight on the parental processes that establish and maintain internalizing behaviors (i.e., rewards and consequences). In SLT, the potential for the occurrence of a behavior is considered a function of the expectancy that the behavior will lead to particular reinforcements, and the attractiveness of these reinforcements for an individual in a given situation (Rotter, 1971). Attachment theory predicts that parental reassurance of a child’s anxious behavior would make it easier for the child to self-soothe, leading to fewer anxious behaviors over time. On the contrary, SLT predicts that parental soothing of this behavior would reinforce such conduct, and that punishing the maladaptive behaviors should eventually eliminate them (Dadds & Roth, 2001).

Parent treatment. If parental behaviors exacerbate child anxiety, it follows that reducing parental anxiety-producing behaviors should lower child anxiety. Several studies have demonstrated that involving the family in the treatment of a child’s anxiety has beneficial outcomes and enhances the efficacy of individual cognitive behavioral
therapy (CBT) (e.g., King et al., 1998; Shortt, Barrett, & Fox, 2001; Silverman, Kurtines, Ginsburg, Weems, Lumpkin, & Carmichael, 1999; Waters, Barrett, & March, 2001). For instance, Zastowny, Kirshenbaum, and Meng (1986) studied mothers who underwent treatment for their own anxiety, in combination with assisting their child’s treatment of anxiety. Specifically, these mothers helped their children with the correct use of anxiety reducing skills, monitored practice of the skills, and reminded their children of when to use the skills. Post-treatment, the children’s reduction in fear responses to the intended situation mirrored their mothers’ decreased fear reactions in the same situation.

Additionally, Creswell, Schniering, and Rapee (2005) found that a family-based intervention reduced both maternal and child threat interpretation.

Barrett et al. (1996a) also found that parent training plus child treatment was more effective than child CBT alone in the treatment of anxious children, but only for younger participants (ages 7 to 10). They suggested older children (ages 11 to 14) might want to assert more autonomy and thus prefer to solve problems on their own. Parent training in the Barrett et al. study consisted of three features: (1) how to reward brave behaviors, (2) how to deal with their own anxieties, and (3) brief training in communication skills.

Moreover, in their comparison of family and child-only treatments in families with and without anxious parents, Cobham, Dadds, and Spence (1998) reported that anxious children with an anxious parent benefited the most from combined intervention. The outcome variable in this study was child post-treatment diagnosis. Bögels and Boers (2002) also found that their “family cognitive-behavior therapy” was effective for children and adolescents with serious anxiety disorders. Finally, and more recently, Wood, Piacentini, Southam-Gerow, Chu, and Sigman (2006) discovered that family CBT
(as opposed to individual CBT) was associated with greater improvement on independent evaluators’ ratings of parent reports of child anxiety (but not on child self-reports).

Summary of Parental Responses

Collectively, prior research provides strong evidence that parents can maintain their child’s internalizing disorders via modeling, overprotection, unintentional reinforcement and encouragement of child maladaptive behaviors. These parental responses are often intensified if a parent is anxious or stressed. High parental anxiety is thought to hamper their own coping skills and produce specific parenting behaviors that increase their child’s anxiety. This, in turn, raises their child’s vulnerability to developing an anxiety disorder (Ginsburg et al., 2004).

Interaction of Temperament and Environment

Parental responses can act in concert with coercive parent-child cycles and temperament to escalate childhood psychopathology (Dadds & Roth, 2001). Initially, a child with an anxious temperament who is confronted with challenging situations will seek closeness from his or her parents, and may be rewarded with attention. Over time, this child will learn that his or her behavior is sometimes rewarded with attention, and so will continue acting “difficult” until noticed. Parents who provide attention will learn that the child stops acting out when he or she is recognized (Dadds & Roth, 2001).

Eventually, parents might try to manage this anxious temperament style through two different behaviors. Parents could attempt to protect their child from novel situations by not allowing him or her to enter such situations (i.e., overprotection). Or, a child’s excessive neediness might motivate parents to behave in a rejecting manner and encourage autonomy and interaction with novel stimuli in challenging situations (Dadds
Either strongly pushing a child toward independence or overprotecting him or her will lead to, and later increase, the child’s stress and fear, which leads to more attention-seeking (Fox & Calkins, 1993). As explained above, parents are likely to respond to such behaviors with initial attention, thereby repeating the cycle (Dadds & Roth, 2001).

This pattern can be likened to Patterson’s (1982) coercive model of family conflict, which holds that parental responses to a child’s behaviors serve to escalate the behaviors, which in turn exaggerate parental behaviors. Cycles of reward and punishment for negative child behaviors repeat, and an intermittent schedule of reinforcement is created that probably strengthens the child’s insecurity and psychopathology (Dadds & Roth, 2001).

In a study by Moore, Whaley, and Sigman (2004), mothers of anxious children, regardless of their own anxiety, were less warm and gave less autonomy to their children. This contradicts popular theories that parental anxiety (manifested as overprotective behavior) results in child anxiety. Instead, Moore et al.’s finding supports the reverse hypothesis: some anxious children may shape their parents’ behavior to be more protective. In one of the few longitudinal studies on this topic, Rubin, Nelson, Hastings, and Asendorpf (1999) found that child anxiety predated parental overprotection.

The most revealing examination of interactions between child temperament and parental anxiety would be a longitudinal study that clarifies the direction of effects; specifically, a study which tests the mutual prediction of temperament and parental anxiety over time. This has not been done yet. Moreover, longitudinal research to date has focused on parental behavior, not parental anxiety. These studies have linked parental
behavior, such as over-involvement, to anxiety disorders in children with moderate consistency (Hudson & Rapee, 2004).

For instance, Lengua and Kovacs (2005) examined child temperament (ages 8 to 11) and parenting (i.e., acceptance, involvement, inconsistent discipline) over time, and found that child irritability predicted more inconsistent discipline by the parent. Nevertheless, the parenting variables studied here were more related to externalizing rather than internalizing problem behaviors. Future studies need to elucidate not only the direction of effects between temperament and parenting behaviors, but also the specific components of temperament and parenting (such as parental anxiety) that correlate the two variables.

**Summary of Risk Factors**

Genetics, temperament, and environmental factors all appear to contribute to one’s vulnerability for developing anxiety disorders. Many environmental factors, including parental behavior, serve to influence a child’s temperament. Lonigan and Phillips (2001) encourage investigation of the interaction between temperamental (i.e., NA) and environmental factors since they believe these two variables have a large influence on the expression of symptoms, even in children with high temperamental risk. More research is needed to tease out the familial transmission of anxiety via temperament from environmental causes of child psychopathology, such as anxious parental behaviors (Dadds & Roth, 2001).

Researchers have proposed several developmental pathways that lead to anxiety disorders. The principle of *equifinality* states that any outcome may result from numerous and varied pathways. A single pathway to a disorder would constitute a rarity (Toth &
The second principle of multifinality states that different outcomes can be the result of the same starting point. For instance, any single risk factor associated with the development of an anxiety disorder (e.g. high NA) is likely to result in several outcomes, and not just be specific to anxiety disorders. Thus, it is important to understand the characteristics within and outside a person that can worsen or improve early differences, and continue or interrupt early adjustment and development. The developmental psychopathology perspective focuses on the origins and course of anxiety disorders, its antecedents and consequences, and its variations and expressions within development (Ollendick, Shortt, & Sander, 2005; Rutter, 1985; Toth & Cicchetti, 1999).

Age Differences

Age differences are important to consider when developing an etiological model of child psychopathology (Vasey & Dadds, 2001). In fact, the literature's support of familial transmission of internalizing disorders via a biological-environmental interaction (Rosenbaum et al., 1991) necessitates the investigation of age differences. This will help parse out the contribution of temperament from the effects of environmental factors such as attachment and modeling. It is possible that a child’s temperament, as measured by NA, will relate to child anxious symptoms fairly strongly in earlier ages. In contrast, parental anxious behaviors are hypothesized to relate less to child anxiety in early childhood. As the child grows older, NA may have the same or slightly less of an impact on child anxiety, whereas parent anxiety symptoms would have a stronger relation to child symptoms. This connection might be stronger because the child has been submitted to an environment where he or she has learned these anxious behaviors; either by modeling the parents, or by reinforcement of the behaviors via parental responses.
Current Study

Although researchers have determined that anxiety runs in families, no study to date has estimated the degree to which child anxiety is associated with parental behavior versus child temperament. Some difficulties of this task are methodological. Most research conducted to assess parental responses to anxiety has been retrospective, requiring individuals with anxiety to recall early experiences with their parents (Chorpita, 2002). There are many flaws with this approach—recall bias being the most obvious (Chorpita & Barlow, 1998). Also, despite the fact that observational studies have documented parents acting in many ways to increase their child’s anxiety, prior research has only examined the specific context of one particular task. A final problem with these retrospective and observational studies is that many have not used clinical samples to investigate parental contributions to child anxiety (e.g., Barrett et al., 1996; Chorpita, Brown, & Barlow, 1998b).

To provide a clearer picture of the progression of childhood anxiety, this cross-sectional study examined how child temperament and parental anxiety symptom expression are related to the development of anxiety in a clinic-referred population of children and adolescents. Child temperament was measured by negative affectivity (NA). Prior investigations have already found that high NA in adults is related to high anxiety in adults (Brown, Chorpita, & Barlow, 1998a). The current study attempted to replicate these findings. In addition, it was predicted that children of parents with high NA would have high NA. As high NA is theoretically linked to the development of anxiety, it followed, then, that parents with anxiety would also have anxious children.
A model that posited a direct influence of parent NA on child NA, with two pathways influencing child anxiety (child NA and parental anxiety), was predicted to have the best explanation for the data. The specific hypotheses of the study were as follows:

1. Two main models were tested: the hypothesized model and the theoretical alternative model. The former suggests that there are two main paths predicting child anxiety – parental anxiety and child NA (see Figure 2). The first path, showing that child anxiety is predicted by a significant path from parental anxiety, would reveal a parental behavioral contribution to child anxiety.

1.1. It was hypothesized that statistically significant completely standardized path coefficients ($p < .05$) would be observed from parent anxiety to child anxiety, from child NA to child anxiety, from parent NA to parent anxiety, and from parent NA to child NA.

1.2. To investigate whether parent NA directly influences child anxiety, the hypothesized model includes a third path from parent NA to child anxiety. It was predicted that the path coefficient from parent NA to child anxiety would not be significant (see Figure 2).

1.3. In addition to the evaluation of path coefficients, the hypothesized model was predicted to be a good fit according to a series of fit indices.

2. The second model, a theoretical alternative (see Figure 3), would be contrasted with the hypothesized model. The alternative model suggests that children influence their parents' anxiety. In the alternative model, child anxiety is predicted by child NA, and
child anxiety and NA in turn predict parental and child anxiety. It was predicted that
the alternative model would be an inferior fit compared to the hypothesized model.

2.1. As the alternative and hypothesized models are not nested, a traditional test of \( \chi^2 \)
difference can not be used to test the differences in fit between them. Instead, it
was decided that the difference between the two models would be evaluated by a
set of fit indices (described in the Method section below).

2.2. It was hypothesized that statistically significant completely standardized path
coefficients \( (p < .05) \) would be observed from parent NA to child NA, from
parent NA to parent anxiety, from child NA to child anxiety, and from child
anxiety to parent anxiety. It is important to note that in this model, the path from
child to parent anxiety was hypothesized to be significant, as it represented the
same path as in the hypothesized model, only reversed (in the hypothesized
model, the directionality runs from parent to child anxiety).

2.3. It was also predicted that the path from child NA to parent anxiety in the
alternative model would not be statistically significant \( (p > .05; \) see Figure 3),
indicating that the direction of symptom influence is from parent to child (as the
hypothesized model predicts), and not from child to parent.

3. If the hypothesized model was supported, then a third model would test the effects of
age on the relationship between parent anxiety and child anxiety, and between child
NA and child anxiety (see Figure 4). The third model purported that the effect sizes of
parental anxiety’s influence on child anxiety would be significantly higher in children
12 and older as opposed to children 11 and younger, and that the effect of child NA
on child anxiety would be slightly weaker in the older group as compared to the younger group.

3.1. The two age groups would be evaluated using a multi-sample analysis (e.g., Jöreskog & Sörbom, 1993). The first step of this analysis involves testing whether the hypothesized model fit the data from both children and adolescents equally well, as indicated by a non-significant ($p > .05$) $\chi^2$ value.

3.2. In addition, this analysis was predicted to have good fit as evaluated by a series of fit statistics. A non-significant $\chi^2$ value signifies equivalent structure and is a prerequisite for further testing of equality across groups. Also, in this first step, it was predicted that the path coefficient from parent to child anxiety would be higher for the older age group as compared to the younger children.

3.3. If, in step 1, the data fit equally well for both groups and the path coefficient from parent to child anxiety was higher for the older group as compared to the younger group, a second solution would be tested. The second step of this multi-sample analysis involves testing the equivalence of the two influences (parent anxiety and child NA) on child anxiety across age groups. Here, the paths from parent to child anxiety and from child NA to child anxiety would be constrained to be equivalent across the two age groups. This permits a test of whether the parameter estimates are significantly different in younger children versus older children. Because the models in steps 1 and 2 are nested, a traditional nested $\chi^2$ test can be employed to test the difference between the two steps. It was hypothesized that this $\chi^2$ test will be significant ($p < .05$), indicating that the tested parameters are not equivalent for the different age groups. This would
confirm the hypotheses that parent anxiety influences child anxiety more in the older group compared to the younger group, and that child NA influences child anxiety less in children 12 and older as opposed to 11 and younger.

This study attempted to establish whether parental anxiety has a significant and unique association with child anxiety, even when accounting for child temperament. It also sought to understand the role of a child’s age in this process. If it is determined that parental anxiety plays substantial a role in the emergence of child anxiety, above and beyond parental influence on child temperament, the next step would be to explore the exact mechanism by which this transmission takes place. For example, future studies could scrutinize this relationship by testing specific parenting behaviors like overprotection.

Method

Participants

Participants consisted of 570 families (i.e., one biological mother and one child/adolescent) obtained from 1,000 consecutive referrals for diagnostic mental health assessments to the Child and Adolescent Stress and Anxiety Program (CASAP) at the University of Hawai‘i’s Center for Cognitive Behavior Therapy (CCBT). CASAP provides comprehensive mental health assessment and treatment services to children and adolescents with a range of emotional and behavioral problems, and specializes in childhood anxiety disorders. Referral sources for CASAP include parents, and officials at the Hawai‘i State Department of Education (DOE) and Department of Health (DOH). Families were excluded if either the child or the parent was unable to participate in the interview or unable to complete a substantial portion of the questionnaires. There were
relatively few cases where fathers attended the mental health assessment ($n = 50$). As such, only families where the biological mother was present were selected for analysis.

A sample size of 88 would be adequate for the present analyses, based on an alpha level of .05 and a power equal to .80 with a moderate effect size (.3) (Cohen, 1992). However, as model evaluation involves identifying the latent factor structure of the dependent variables, there are additional considerations for sample size. Guadagnoli and Velicer (1988) offer guidelines for minimum sample size required to obtain a stable sample covariance matrix for input into the analyses. Based on 13 indicators, 4 latent variables, and predicted indicator factor loadings of .6 or higher for the present model, the recommended minimum sample size was estimated to be approximately 100 participants.

Child and adolescent participants were between the ages of 7 and 19 (only one 19 year-old), with a mean of 12.8 years (standard deviation = 2.97). The sample consisted of 380 males (67%) and 190 females (33%). Parental marital status was reported as follows: married, 48.2%; divorced, 20.5%; separated, 7.2%; widowed, 2.0%; and single, 14.4%. Marital status was not reported for 7.5% of the mothers. Median household income was $40,000, although income data was not reported for 36.2% of cases.

The ethnic composition of the sample, as reported by parents or guardians, was as follows: multiethnic, 42.5% ($n = 242$); Caucasian, 14.9% ($n = 85$); Japanese, 7.7% ($n = 44$); Hawaiian, 6.0% ($n = 34$); Filipino, 5.3% ($n = 30$); Chinese, 2.1% ($n = 12$); African American, 0.9% ($n = 5$); Samoan, 1.8% ($n = 10$); Hispanic, 1.2% ($n = 7$); Korean, 1.1% ($n = 6$); Tongan, 0.5% ($n = 3$); Guamanian, 0.2% ($n = 1$); Southeast Asian, 0.2% ($n = 1$), Portuguese, 0.2% ($n = 1$); Native American, 0.2% ($n = 1$), and other, 6.8% ($n = 39$).
Eight percent ($n = 48$) of the sample did not report their ethnicity. All children reported speaking English at school.

Although diagnostic information was not used in the present study, diagnoses of participants were determined using a semi-structured diagnostic clinical interview for parents and children, the Anxiety Disorders Interview Schedule for Children-IV, Child and Parent Versions (ADIS-IV-C/P, Silverman & Albano, 1996).

The data in this study was largely archival. To ensure that it was entered properly, 100 case files were randomly selected for re-entry and subsequent comparison to the original database. A priori, an error rate of over 5% was considered unacceptable. The random data re-entry revealed that all measures used in this study had an acceptable error rate (e.g., below 5%), obviating further data re-entry.

Model Indicators

The measurement model is presented in Table 1 and includes indicators and corresponding latent variable names. What follows is a description of the model indicators (a copy of all measures used is provided in the appendices).

Positive and Negative Affect Schedule for Children (PANAS-C; Joiner, Catanzaro, & Laurent, 1996). The PANAS-C is a downward extension of the adult PANAS (Watson, Clark, & Tellegen, 1988). The PANAS-C is a 27-item self-report measure, consisting of two scales, Positive Affect (PA) and Negative Affect (NA). The former scale contains 12 items and the latter contains 15 items. All items consist of mood adjectives that are rated by the respondent in terms of the frequency with which each emotion has been experienced during the past week. Respondents are asked to rate each emotion adjective on a five-point Likert scale, ranging from “very slightly or not at all” to
"extremely." Sample items are “sad, frightened, excited, and calm.” Both the PA and NA scales of the PANAS-C have demonstrated good levels of internal consistency (.84 and .80, respectively; Joiner et al., 1996). This scale has also demonstrated sound construct validity and discriminant validity (Laurent et al., 1999; Chorpita & Daleiden, 2002). In addition, Chorpita and Daleiden (2002) found that the PANAS-C NA scale was able to discriminate anxious and depressive disorders from all other disorders, and the PA scale was able to further discriminate depressive from non-depressive disorders. In the present study, the NA scale was split evenly into three model indicators (5 items each) for the latent variable child NA (see Table 1).

**Depression-Anxiety-Stress Scales 21 (DASS 21; Lovibond & Lovibond, 1995).**

The DASS-21 is a shorter version of the original DASS scale. It consists of three scales that evaluate symptoms of depression, anxiety and stress in adults. These scales better discriminate between anxiety and depression than past self-report measures, and they have demonstrated good reliability and discriminant validity (Lovibond & Lovibond, 1995). When the DASS-21 is administered, individuals are asked to rate how much each statement applied to them within the last week. All three scales are rated from 0, “did not apply to me at all,” to 3 “applied to me very much, or most of the time.” Sample items are: “I found it hard to wind down” and “I felt down-hearted and blue.”

Anthony, Bieling, Cox, Enns and Swinson (1998) demonstrated internal consistency to be .94 for the depression scale, .87 for the anxiety scale, and .91 for the stress scale. Similarly, Brown, Chorpita, Korotitsch and Barlow (1997) demonstrated internal consistency to be .96, .89, and .93 for the same scales, respectively. This latter
group of researchers also conducted test-retest analyses within a two-week time setting, which revealed strong correlations ($r = .71 -.81$).

The DASS-21 scale has demonstrated high concurrent validity ($r = .84$; Brown et al., 1997), and Anthony et al. (1998) found that the DASS-21 showed both convergent and discriminant validity. Moreover, Lovibond (1998) conducted a retest of the DASS with 882 participants, three to eight years after the initial administration, and found that reports were moderately stable over time ($r = .3$ to .5).

Finally, Brown et al. (1997) found the DASS-Stress scale was strongly associated with NA, whereas the Anxiety Scale appeared to measure anxiety symptoms more specifically. Brown et al. concluded that the Stress scale may be a more accurate index of overall severity of negative emotion than traditional scales, which measure specific emotions like anxiety and depression. Thus, in the present study, the DASS-Stress scale was used as a measure of parent NA, whereas the DASS-Anxiety Scale was used as a measure of parent anxiety. The items from the DASS Stress and Anxiety subscales were split into three indicators each in the present study (3 Stress subscale indicators were used for parent NA, and 3 Anxiety subscale indicators were used for parent anxiety; see Table 1).

Revised Child Anxiety and Depression Scale (RCADS). The RCADS (Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000) is a 47-item adaptation of the Spence Children’s Anxiety Scale (SCAS; Spence, 1998) designed to correspond more closely to DSM-IV anxiety disorders, while also incorporating a subscale for major depression. On this measure, children are asked to rate the extent to which each item is true on a scale of
0 to 3, corresponding to anchors of “never,” “sometimes,” “often,” and “always.” Sample items include “I worry about things” and “I feel worthless.”

The RCADS consists of six subscales: Panic Disorder, Obsessions/Compulsions, Social Phobia, Separation Anxiety, Generalized Anxiety, and Major Depression. The RCADS has demonstrated good structural factorial validity, internal consistency, one-week test-retest reliability, and convergent and discriminant validity for children and adolescents ages seven and older in both clinical and non-clinical samples (Chorpita et al., 2000a; Chorpita, Moffitt, & Gray, 2005). As such, it is a reliable and valid measure of internal distress. Also, when compared to traditional measures of internal distress, the RCADS generally shows greater relation to specific diagnoses (Chorpita et al., 2005). In the current study, the items from the RCADS anxiety subscales, but not the major depression subscale, were divided into two model indicators (see Table 1).

**Dimensional Ratings of Clinical Severity (DRs).** Dimensional Ratings of Clinical Severity (DRs) are clinician-derived ratings based on the interference rating scale developed previously for the ADIS-C/P (Silverman & Nelles, 1988). Ratings of 0 to 8, with larger numbers indicating greater degrees of distress and impairment, are provided for each diagnostic area on the basis of information obtained during each child and parent interview. More specifically, the assessing clinician employs ratings of “0” to indicate the absence of any symptoms associated with a given syndrome, “2” to suggest the presence of a few or slight features of the syndrome, “4” to indicate the presence of several or definite symptomatic features, “6” to reflect the presence of many or marked symptomatic features, and “8” to indicate the presence of very many or severe features of the disorder. A numerical rating is assigned for each symptom area regardless of whether
diagnostic criteria for that area were met by the individual’s symptom endorsement. In a previous investigation of 36 clinic-referred children, DRs were found to demonstrate good to excellent interrater reliability for both child and parent ratings (ranging from $r = .63$ for the Oppositional dimension to $r = 1.0$ for the Agoraphobia dimension; Gray, Francis, & Chorpita, 2001).

Preliminary support for the convergent validity of DRs was also indicated. Specifically, significant correlations were observed between the DRs and corresponding child and parent report measures (Gray et al., 2001), including the Revised Child Anxiety and Depression Scale (RCADS; Chorpita, Yim et al., 2001), the Revised Children’s Manifest Anxiety Scale (RCMAS; Reynolds and Richmond, 1978), the Children’s Depression Inventory (CDI; Kovacs, 1980/1981), and the Child Behavior Checklist (CBCL; Achenbach, 1991).

In the present investigation, after the administration of the ADIS-IV, the same intake assessor who conducted the interview assigned a DR to the child for each diagnostic area using information obtained in separate child and parent interviews (diagnostic areas: separation anxiety, social anxiety, specific fear/phobia, generalized anxiety, panic, agoraphobia, obsessions/compulsions, and posttraumatic stress). Thus, each child was assigned two different ratings for each diagnostic area, one based on the child report alone, and one based on the parent report alone. Each DR subscale score (e.g., social anxiety) could range from 0 to 8, whereas the total DR anxiety score for the separate child and parent scales could range from 0 to 64.
Procedure

Following initial referrals, graduate student clinicians telephoned the child or adolescent’s legal guardian to conduct a short screening interview. For children with previous diagnoses of Autism or Mental Retardation, prospective assessments were not recommended and more appropriate referrals were made instead. Interviews were scheduled for the remaining children and their parents or legal guardians. Before the interview, informed consent to participate in a semi-structured diagnostic interview and for the use of materials for subsequent research purposes was obtained from the parent. Informed assent was obtained from the child for the same intents.

Next, a mental health evaluation of the child or adolescent was conducted by trained graduate students attending UH Manoa. ADIS-IV-C and ADIS-IV-P interviews were administered consecutively. During the child interview, the parent filled out both participant and self-report questionnaires. Similarly, the child or adolescent filled out several self-report questionnaires during the parent interview. The RCADS and the PANAS-C were administered to children and the DASS-21 was administered to parents as part of the standard battery of assessment measures.

After the initial assessment, a child’s teachers, previous or current mental health providers, and other important informants (e.g., another relative living with the child part-time, probation officers, etc.) were contacted for concise, unstructured telephone interviews.

Analyses

Factor analytical strategy. Because structural equation modeling (SEM) can be used to accommodate measurement error (Jaccard & Wan, 1996), it was chosen as the
most appropriate method to evaluate the models described in the hypotheses section. The observed sample variance-covariance matrix of the dependent measures was analyzed using LISREL 8.72, a linear structural relations computer program (Jöreskog & Sörbom, 1993).

To allow for proper model identification, at least two indicators per latent variable are needed to prevent underidentification. As such, the items on the questionnaires used (RCADS, PANAS-C and DASS-21) were split two- or three-ways (e.g., the RCADS was split into 2 indicators with 18 and 19 items each; see Table 1). Jaccard and Wan (1996) recommend using three indicators per latent variable, but note that split-half approaches are well-recognized and often used in psychometrics for reliability estimation. Multiple indicators can estimate regression coefficients between the true latent variables while accounting for measurement error (Jaccard & Wan, 1996).

Fit indices. Because different fit indices are sensitive to unique artifactual influences (e.g., the chi square fit index increases with sample size), multiple fit indices were used to provide a more conservative and reliable evaluation of the models. These indices included the traditional Goodness of Fit Index (GFI; Jöreskog & Sörbom, 1993) and the Standardized Root Mean Square Residual (RMR; Jöreskog & Sörbom, 1993), which test the absolute fit of the model; the Incremental Fit Index (IFI; Bentler, 1990), which tests the fit of the model relative to a fully orthogonal “independence” model; the Root Mean Square Error of Approximation (RMSEA; Steiger, 1990), which evaluates the model relative to degrees of freedom; and Akaike’s Information Criterion (AIC; Akaike, 1987), which is the model $\chi^2$ plus 2 times the number of parameters.
Conventionally, scores of .90 and above on the GFI and the IFI represent good model fit, as do scores below .05 on the RMR. Cudeck and Browne (1992) suggest that RMSEA values at or below 0.08 represent acceptable fit, and that values at or below 0.05 represent good fit. An inferential test for close fit (defined as RMSEA < .05) was also employed in the current study, with a nonsignificant result ($p > .05$) suggesting good model fit (Cfit; Cudek & Browne, 1992). Lastly, the AIC was included because of its utility for comparatively evaluating non-nested models. Lower AIC values represent better model fit.

Modification indices. Modification indices (Sörbom, 1989) are focused tests that examine fit more closely to determine potential sources of poor fit (see above). These were employed to examine items for models that did not fit sufficiently well. For each fixed parameter in a model, a modification index is an estimate of the decrease in the $\chi^2$ badness-of-fit measure if the parameter was untied. A lower $\chi^2$ value indicates improved model fit. When sound theoretical arguments supported a modification of the model, the revised alternative was comparatively tested against the original model for significance of fit. If new indicators were created in order to improve overall model fit, the fit statistics for these models were evaluated for their enhancement over prior models.

Results

Internal Consistency of Indicators

Internal consistency coefficients (i.e., Cronbach’s coefficient alpha) were calculated for all model indicators as well as related scales. Also, since two-way and three-way split model indicators were developed for all scales, the new sub-scales were examined for their reliability. The coefficients for all relevant measures are presented in
Table 2. Most measures and indicators demonstrated adequate reliability, except for the DSTR2 indicator. Cronbach’s alpha is only one of many gauges of reliability, and may not be the best estimate of reliability for measures with few items. Using a larger number of items produces higher Cronbach’s alpha values and vice versa (Streiner & Norman, 2003). As some of the indicators in this study were comprised of only two to three items (see Table 1), correlations between indicators were deemed a more appropriate estimate of reliability than Cronbach’s alpha.

Correlations of Indicators

Scales that were split two- or three-ways were examined for their reliability by observing the correlations between the indicators. Thus, correlations between the 3 split-versions of the PANAS-C, the DASS-21 Stress and Anxiety scales, respectively, and the 2-split versions of the RCADS were examined. These indicators were deemed reliable if they correlated .55 or higher with each other. The correlations between these two- or three-way split indicators were good, ranging from .61 to .92 (correlations of selected indicators for the tested models can be found in Table 4). As discussed above, Cronbach’s alpha yielded somewhat low reliability for one of the indicators derived from the DASS-21 (DSTR2), but according to correlations between indicators, all three DASS-21 indicators were significantly highly correlated with one another (.61 to .71; see Table 4). Consequently, indicators derived from the DASS-21 were deemed reliable and were included in subsequent analyses.

Missing data. Although complete data were available for most measures, missing data were observed in a few self-report measures for a small portion of the sample. These omissions did not appear to be systematically related to any characteristics of the
participants or the measures themselves. Specifically, 10% of all items across all cases and measures were missing. Also, less than 10% of items on individual measures were missing, except for a few items on the PANAS-C. Thirteen percent of the cases had item 15 on the PANAS-C missing, and approximately 12% of the cases had items 21-23, 25, 14 and 19 of the PANAS-C missing. All cases had at least two measures filled out. Seventy-one of the cases had two measures filled out (12.5%), 314 of the cases had three measures filled out (55.1%), and 185 of the cases had all four measures filled out (32.5%).

Nevertheless, since approximately 10% of the data were missing, analyses using the method of multiple imputation (MI) were performed to correct for these omissions. Rubin (1987) notes that MI permits the use of any “complete data” method on incomplete data sets. More importantly, MI is a robust method under varying assumptions about the missing data mechanism (e.g., if the data are either missing completely at random or missing at random; See Brown 2006), and where the amount of missing data is significant (Brown 2006; Rubin, 1987).

According to Brown (2006), simple imputation procedures such as mean or regression imputation are not advised because they produce underestimates of variances and overestimates of correlations among the variables that contain imputed data. MI corrects these problems by introducing random variation into the imputed data. In MI, the missing values for each case are calculated on the basis on the observed values for other cases, but random noise is included in order to maintain the appropriate degree of unevenness in the imputed data. This process has to be performed multiple times;
otherwise, the resulting standard errors would be too small (Brown, 2006). MI procedures as described by Rubin and Brown are available in LISREL (e.g., version 8.72).

Using a sample of 570 participants, all covariance matrices in the present study were created by following the three basic steps of the MI process (Brown, 2006). First, five imputed data sets were generated with the multiple imputation command in LISREL. Second, the five data sets were analyzed with standard analytic procedures using LISREL. Third, results from the five analyses were combined by LISREL into a single set of parameter estimates, standard errors, and test statistics. Parameter estimates were created by simply averaging the estimates across the five analyses. For the detailed formula of the standard error calculation, see either Allison (2003) or Rubin (1987).

**Non-normal data.** Prior to conducting analyses, data was routinely screened (using PRELIS 2.72) to identify problems such as non-normality. The proposed set of indicators (see Table 3) was found to be multivariately positively skewed ($z = 9.58, p < .01$) and had a significant multivariate kurtosis ($z = 7.45, p < .01$). The joint evaluation of non-normality in terms of both skewness and kurtosis yielded a chi-square of 147.27 (significant). Upon inspection of individual indicators, all had fairly normal skewness and kurtosis (Z-scores ranging from 0-1, which were non-significant), except for those indicators which were derived from the DASS-21. The reason for the high non-normality of the DASS-21 scores may be that the DASS-21 has a Likert-type response format that asks people about psychological problems and it measures the upper end of anxiety and depression difficulties. For many of the children and adolescents who are referred to the Stress and Anxiety Clinic, their mothers do not have severe stress or anxiety. When most
mothers respond “0” to the items on the DASS-21, this produces skewed data. See Table 3 for skewness and kurtosis of the proposed set of indicators.

Adjusted statistics were needed in order to correct for non-normality and safely proceed with the structural equation modeling. Brown (2006) states that weighted least squares (WLS) is an appropriate method to use with non-normal data, provided that one has a large enough sample size. The WLS fit function is weighted by variances/covariances and kurtosis to adjust for violations in multivariate normality (Brown, 2006). According to the formula provided by Brown (2006; see equation 3.14, p. 68), WLS requires that the sample size exceeds $p(p+1)/2 + p$ (i.e., the number of indicators*(the number of indicators + 1) /2 + the number of indicators). The result for this study was 13(13+1)/2 + 13 = 104. The sample size in the current study exceeded this figure, making WLS a good choice for estimator (overall $N = 570$, and $n = 235$ and 335, for the younger and older samples, respectively). WLS requires both covariance and asymptotic matrices be used as inputs for the analyses, as opposed to the traditional Maximum Likelihood Method (MLM), which only requires input of the former matrix. When an asymptotic matrix has been inputted into the analyses, standard errors are considered robust to non-normality (Brown, 2006). The covariance and asymptotic matrices for the analyses were generated using PRELIS, and all statistics reported from this point forward are based on WLS analysis.

Model Testing

Model evaluation relied on contrasting a hypothesized model with a theoretical alternative. The latter model proposed a different conceptual relationship among the
latent variables and was evaluated for quality of fit relative to the hypothesized model (see hypotheses above).

**Measurement model.** Before evaluating any of the structural models, an initial measurement model was tested that allowed all latent variables to be freely intercorrelated (see Figure 1). Also, correlated error was modeled a priori between the two RCADS indicators, and between mother and child dimensional ratings (DRs). This measurement model showed adequate model fit (see Table 5, Measurement Model). Following this first test, the modification indices were 57.12 for parent DRs and parent NA, and 36.90 for parent DRs and parent anxiety. Because parent DRs were intended to be used as a measure of child anxiety, and no theoretical rationale exists to justify the observed correlated errors, neither of these suggested modifications was adopted (i.e., untying the parameters).

Additionally, when taking a closer look at the correlations between both parent and child DRs and the two RCADS anxiety indicators, child DRs and the RCADS indicators had a correlation of .57 and .59, whereas these correlations for parent DRs were only .23 and .26 (see Table 4). It was decided, therefore, that parent DRs would be discarded as an indicator. Child DRs (in addition to the two RCADS indicators) was retained as a measure of child anxiety in subsequent models. With parent DRs removed, 12 indicators remained in the revised measurement model.

Re-estimation of the measurement model with parent DRs omitted yielded better individual fit statistics, and suggested good model fit overall (from $\chi^2 = 144.22$ and AIC = 203.41, to $\chi^2 = 65.41$ and AIC = 126.82; see Table 5 for more statistics). In the revised measurement model, each of the 12 indicators had strong and statistically significant
loadings \( (p < .01) \) on their respective latent factors. These 12 indicators were used in consequent models. The superior fit of the revised measurement model suggests that poor indicator selection (i.e., parent DRs) weakened the fit of the initial model.

*Hypothesized model.* The hypothesized structural equation model was tested next (see Figure 2). This model included the same error theory that was used in the measurement model (i.e., correlated error among certain indicators), but with parent DRs removed, it was no longer possible to model measurement error between parent and child DRs. The main hypothesis tested by this model was that parental anxiety would influence child anxiety above and beyond the contribution of child NA. Also, it was theorized that parent NA would be related to child NA, and that parent NA would not directly influence child anxiety, but rather indirectly via parent anxiety and child NA.

Fit statistics for the hypothesized model appear in Table 5 and suggest good model fit. Consistent with earlier predictions, the path from parent negative affect to child anxiety did not add anything to the model (completely standardized non-significant path coefficient: -.13; see Figure 5).

*Revised hypothesized model.* As a direct path from parent NA to child anxiety was not supported, the model was re-tested without it (see Figure 6). This revised hypothesized model yielded estimates similar to those in the original hypothesized model (see Table 5, revised hypothesized model). With the exception of the \( \chi^2 \) test—a conservative test for perfect model fit that is known to penalize larger sample sizes (e.g., Gerbing & Anderson, 1993)—these statistics uniformly suggested good model fit (see Table 5). In the revised model, statistically significant completely standardized path coefficients were observed from parent anxiety to child anxiety (.10), from child NA to
child anxiety (.83), from parent NA to parent anxiety (.86), and from parent NA to child NA (.13). Hence, it appeared that parent anxiety, above and beyond child NA, contributed to the variance in child anxiety.

*Theoretical alternative model.* The revised hypothesized model was compared against an alternative (see Figure 3) in which child anxiety and NA were modeled to affect parent anxiety. If all paths were significant in the alternative model, it would lead one to suspect that children influence their parents’ anxiety instead of parents influencing their children’s anxiety, or that the relationship is bi-directional. In the alternative model, the path from child to parent anxiety was hypothesized to be significant, as it represented the same path as in the hypothesized model, only reversed (in the hypothesized model, the directionality runs from parent to child anxiety). However, if the path from child NA to parent anxiety in the alternative model was not significant, it would suggest that parents influence children’s anxiety, and not the other way around. In addition to evaluating the significance of these paths, the proposed model was hypothesized to provide a statistically superior fit of the observed data according the fit indices described above.

In the alternative model, statistically significant completely standardized path coefficients were observed from child anxiety to parent anxiety (.22), from child NA to child anxiety (.84), from parent NA to parent anxiety (.87), and from parent NA to child NA (.15; See Figure 7). As both the revised hypothesized model and the alternative model were not nested, a traditional nested $\chi^2$ test could not be employed. Instead, the AIC statistic was used because of its utility in comparatively evaluating non-nested models. The AIC values for the two models were identical (126.70). Fit statistics also

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indicated that the alternative model showed comparable fit to the proposed model (see Table 5). Nevertheless, consistent with prediction, the alternative model had a statistically significant and negative completely standardized coefficient (-.23) from child NA to parent anxiety, signifying that child NA does not influence parent anxiety (see Figure 7). Given that the alternative model did not add anything to the original model, and it had a statistically significant negative path, the revised hypothesized model was kept for later analyses.

_Age._ Next, the sample was divided into two groups: children ages 7-11 \( (n = 225) \), and adolescents ages 12-19 \( (n = 335) \). This was done in order to test the applicability of the overall model from previous analyses (i.e., the revised hypothesized model) to various age groups. Although a different dividing point (e.g., median) would have provided groups of similar size, the 12 year-old cutoff was based on cognitive and developmental theories of self-reported child anxiety (e.g., see Chorpita, Albano, & Barlow, 1996), and the change in anxiety disorders that tends to occur around age 12 (e.g., the prevalence of specific phobias and separation anxieties in children versus social anxieties in adolescents; Gullone, 2000; Ollendick, King & Muris 2002).

The two age groups were evaluated in a multi-sample analysis (e.g., Jöreskog & Sörbom, 1993) that first entailed testing whether the hypothesized solution would fit the data from both children and adolescents equally well. Simultaneous multi-sample analysis of the child and adolescent groups yielded a GFI of .99, an RMSEA of .027, and an IFI of .97, all of which suggest that an equivalent structure across age groups fits the data very well (see Table 5). For this solution, \( \chi^2 = 116.80, df = 96, p = .07 \). This evidence of equivalent structure is a prerequisite for further testing of equality across groups. The
second step of this multi-sample analysis involved testing the equivalence of the two influences (parent anxiety and child NA) on child anxiety across age groups.

Here, a second solution was estimated that constrained the parameters from parent to child anxiety and from child NA to child anxiety to be equivalent across the two age-groups. This permitted a test of whether the two parameter estimates were significantly different in younger children compared to older children (as hypothesized). There was reason to believe this hypothesis would be substantiated, because in step 1 (see above), the completely standardized path coefficient from parent to child anxiety in the younger age group (.06) was not statistically significant from 0, whereas in the older group this path coefficient (.10) was statistically significant. Both age groups had significant completely standardized path coefficients in step 1 for the influence of child NA on child anxiety (.86 and .81, respectively).

In step 2, these two parameters (parent to child anxiety and child NA to child anxiety) were fixed in the Beta matrices to require the same values for both older and younger children. The extent to which such manipulations degrade model fit in relation to the previous solution is thought to signify differences in the parameter values across groups (Jöreskog & Sörbom, 1993). The second solution led to an overall Chi-square of 127.90, $df = 98, p = .023$. Because the models in steps 1 and 2 were nested, a traditional nested $\chi^2$ test could be employed. This $\chi^2$ test of the difference between the two steps was significant ($\chi^2_{diff} = 11.10, df = 2, p = .004$), indicating that the tested parameters were not equivalent for older and younger groups. In other words, the two models are not equally fitting.
Thus, the two groups' path coefficients differ when holding constant the effects of parent anxiety and child NA on child anxiety. One can conclude that not only does the covariance structure fit when the path coefficients are allowed to differ across the two groups, but the fit worsens when those path coefficient values are constrained to be the same across these groups. One can claim that the two age groups have unequal path coefficients (e.g., they are statistically different from each other).

Discussion

In this study, the best fitting model suggested that parent anxiety contributed to child anxiety symptoms, above and beyond the influence of a child's anxious temperament. Specifically, in all of the structural equation models that used the entire sample ($N=570$), the path from parent to child anxiety was significant, even when accounting for child NA (temperament). These results are consistent with previous findings that implicate the role of early experience in the development of anxiety (e.g., Lonigan & Phillips, 2001; McClure et. al., 2001). Interestingly, in the hypothesized model, parent NA did not directly influence child anxiety (i.e., it had a non-significant path). Instead, it influenced child anxiety indirectly via parental anxiety or child NA. That is, in this sample, parental NA alone was insufficient to produce child anxiety. Both parental anxious behaviors and child NA mediated this effect. The superior fit of the revised hypothesized model over the alternative model (as indicated by the latter’s negative and non-significant path from child NA to parent anxiety) also provides support for the former model.

Furthermore, parent anxiety appeared to influence child anxiety significantly more in the older group (ages 12-19) compared to the younger group (ages 7 to 11).
Conversely, child NA influenced child anxiety significantly more in the younger sample. In the 7 to 11-year old sample, parent anxiety was not even a significant predictor of child anxiety. This may suggest that the older children had more time to learn anxious behaviors from their parents, and so anxiety transmission via parents’ anxiety was stronger. With regard to a possible diminishing influence of child NA on child anxiety in the older group, it seems logical that, as parents’ anxiety becomes more influential in producing child anxiety, the influence of child NA on child anxiety may get somewhat “drowned out.” Alternatively, it may also mean that if anxiety persists into adolescence, parents start to become more anxious.

The observed differences between children and adolescents may also be explained by the commonly held view that adolescence is a period of transition (e.g., Cicchetti & Toth, 1996). For example, anxiety disorders tend to shift from specific phobias and separation anxiety in childhood towards more social, academic and health-related anxieties in adolescence (e.g., Bauer, 1976; Strauss & Last, 1993; Vasey & Dadds, 2001). This shift in anxiety symptoms towards more social anxieties may be due to the fact that adolescents are more socially involved than children, and that they undergo more environmental changes (i.e., puberty, dating, new schools, peer influences, etc.; Chavira & Stein, 1995). Accordingly, adolescents are more inclined to adapt behaviors from, and be influenced by, other people in their environment. Parents, who usually comprise a large part of an adolescent’s social interactions, may have a powerful influence on their child’s anxiety during this time.
Limitations

One significant limitation of this study, and of much related research, is the imprecise measurement of constructs. Self-report data were used for all the constructs (e.g., the DASS-21 and PANAS-C) except child anxiety, and so an assessment of method variance was not viable for the parent anxiety, child NA, or parent NA constructs. It was largely impossible to disentangle method variance from the variance unique to the constructs of interest (Cole, Truglio, & Pecke, 1997). Also, the operational definitions of some of the constructs (e.g., parent anxiety, parent and child NA) were constrained by the items on the DASS-21 and the PANAS-C.

For example, even though the DASS-21 stress scale (used in this study to measure parent NA) correlates highly with NA in other studies (e.g., Brown et al., 1997), it may not be the best measure of parent NA. The items on the DASS-21 stress scale appear to be measuring parental tension and worry. The items on the DASS-21 anxiety scale, however, are more representative of anxious behavior. Hence, the stress scale may measure the non-observable part of anxiety, whereas the anxiety scale measures observable anxiety. It is therefore possible that this study’s findings of parent anxiety (and not parent NA) being directly related to child anxiety are due to children being attuned to their parents’ observable anxiety, and not noticing or picking up on their parents’ stress (tension and worry).

Conversely, parents may be influenced by their child’s anxiety, and not NA, because parents may not notice their child’s stress when it constitutes more of an internal experience. This might help to explain the finding of a significant path from child to parent anxiety, and a non-significant path from child NA to parent anxiety. Thus, the idea...
that children influence their parents' anxiety cannot be ruled out from this study's findings. The measures used here are by no means definitive measures of anxiety and NA. It will be necessary to replicate the current study's findings in future studies that use more robust measures and take into account method variance.

Response bias, such as a socially desirable response style, is also a concern when relying mainly on self-report measures. As people with high levels of anxiety may be more worried about pleasing others, a socially desirable response style might be correlated with measures of anxiety. In one study with a community sample, Dadds, Perrin and Yule (1998) found that children's social desirability scores were predictive of their level of anxiety. In order to correct for this contribution to method variance, measures of social desirability should be considered for inclusion in future models that use self-report measures. Another problem with self-report data in this field is that adolescents might be more accurate than younger children in self-reporting their internal states (e.g., Edelbrock, Costello, Dulcan, Kalas, & Conover, 1985).

To better offset response bias in youth, future studies may need to include additional informants such as observers' ratings or peer nominations. This new data could allow the identification of models that can control for all method variance simultaneously. Still, an answer to the choice of mono- versus multi-informants is not obvious. Many have argued (e.g., Campbell & Rapee, 1996; Carver & Scheier, 1996) that anxiety is a wholly internal state and that individual reports of these states are preferable. Another difficulty is that observers' reports (e.g., peer, parent, etc.) would be dependent on the type of situations they observed, the degree of emotional articulation in the child, and the internal state evaluated. Consequently, the level of agreement between children
and observers (i.e., multi-informants) may underestimate children's anxiety states (e.g., see Edelbrock et al., 1985; Silverman & Eisen, 1992). Underestimation was found in the current study, as clinician ratings of parent reports (parent DRs) correlated highly with parent symptoms, but did not correlate well with the latent construct of child anxiety in the measurement model.

A related limitation of this study is the fact that biological mothers' reports were the only indicator of parental influence on child anxiety. Even though the effect of mothers' anxiety on child anxiety was significant, it was small (.10) compared to the influence of child NA (.83). A mother is only one environmental influence on a child's life. Including fathers (or other significant caregivers) in structural modeling may yield a greater effect of parent influence on child anxiety. Also, mothers' and fathers' psychopathology may influence children's internalizing behaviors differently depending on age. Connell and Goodman (2002) found that throughout childhood, mothers' behaviors have more influence than fathers' (r = .19 and r = .09, respectively). The opposite was true for adolescents in their study. In this older group, anxious behaviors were influenced more by paternal rather than maternal factors (r = .23 and r = .13, respectively). Bailey's (1994) finding that fathers are more involved in the care of adolescents than for younger children helps to explain this paradox.

Had the current study included fathers, a larger difference in parental contributions between the two age groups may have surfaced. Unfortunately, a relatively small number of fathers were present for the assessment in the current study, necessitating their omission from the analyses. Future cross-sectional studies would be more revealing if they investigated both paternal and maternal influences on child.
anxiety, as well as other important influences in a child’s environment (e.g., peers, siblings, and teachers).

Of course, even with multi-informant analyses, many complex causal effects cannot be tested with a cross-sectional model. Although the path between child NA and parent anxiety was non-significant in this study, these findings do not eliminate the possibility that child anxiety influences parent anxiety, and not the reverse. Ultimately, future research will need to corroborate the direction of these effects, because the assumption of causality in structural equation modeling is difficult and often relies on extant theories. For example, a model that posits child anxiety leading to child NA and child NA leading to parent NA is mathematically similar to the present model, but not supported by current etiological theory. Structural equation modeling simply investigates covariances in the measured variables. Etiological models of child anxiety propose a bidirectional relationship between child and parent anxiety (Bögels & Brechman-Toussaint, 2005; Dadds, 2002).

In order to better understand the directionality of childhood anxiety and NA, we must explore the present study’s findings in clinical as well as non-clinical samples. Aside from replicating these findings in more cross-sectional studies that possess a variety of samples with different measures (see above), longitudinal studies with clinical parent samples (e.g., those that only target parental anxiety in treatment) can establish whether reducing parent anxiety subsequently lowers child anxiety. Alternatively, longitudinal treatment studies that focus solely on child anxiety could see if the effects of this treatment include lower levels of parent anxiety. It would be interesting to document
whether stronger effects are found in longitudinal studies with clinical samples (e.g., those with diagnosed anxiety disorders) versus non-clinical samples.

Longitudinal modeling with non-clinical samples may also help to clarify developmental processes, identify other influential variables, and discover the point at which emerging vulnerabilities start to assume the structure implicit in models of clinical anxiety. Although logistically and ethically problematic, an even more authoritative investigation might involve a twin study, where one twin was adopted away and raised by non-anxious parents, and the other twin was raised by the original anxious parents.

As mentioned in the introduction, even if cross-sectional and longitudinal (or adoption) studies replicate the present one and establish with more certainty that parental anxiety contributes to child anxiety, we still will not know the exact parent mechanisms (e.g., overprotection, modeling, etc.) that create child anxiety. As the parental anxiety contribution to child anxiety is substantiated by additional studies, more specific models will need to investigate these vectors.

Finally, future research on early models of childhood anxiety will benefit from more diverse samples in terms of demographic factors like ethnicity and age. The ethnic diversity in this study, while extensive, mainly consisted of different Asian populations in Hawai‘i. Studies conducted on mainland America and elsewhere in the world may yield different results. Secondly, like most other research on childhood anxiety, this study did not investigate anxiety in early childhood (i.e., 0-6 year-olds; Cartwright-Hatton, 2006). In a recent longitudinal study of 112 children from age 2 to age 4, the authors found that early internalizing difficulties were predicted by parents’ anxiety/depression, over-involved parenting, and low “warm-engaged parenting” (Bayer, Sanson, & Hemphill,
The findings of the current study would be strengthened if parent anxiety was found to be less influential for 0-6 year olds than for older children and adolescents.

**Conclusion**

When compared with observational or retrospective research, this study provides a different perspective on the transmission of anxiety from parents to children. Important findings include the excellent fit of the revised hypothesized model, and the significant, albeit small, role of parent anxiety on child anxiety, above and beyond the contribution of child temperament. In the multiple-group solution analyses, parent anxiety appeared to be more influential in older children relative to younger children. These outcomes are consistent with the study’s hypotheses. In addition, the multiple-group solution represents a cross-validation of the revised hypothesized model because it found that the structure of the model held up across groups. If these findings are corroborated in the much-needed future studies mentioned above, we can not only advance the theoretical knowledge of the development of childhood anxiety, but also guide intervention and prevention efforts for these disorders.

Currently, the overall remission rate for children whose anxiety disorders are treated with cognitive-behavioral therapy is 56% (Cartwright-Hatton, Roberts, Chitsabesan, Fothergill, & Harrington, 2004). This bleak remission rate underscores the need for new therapeutic strategies. Researchers have already found that child anxiety decreases when parent anxiety is targeted for treatment (e.g., Cobham, Dadds, & Spence, 1998). Furthermore, collective evidence from studies reviewed in the introduction suggests that including anxious parents in the treatment of childhood anxiety may be wise, regardless of whether the effects of parent and child anxiety problems are bi-
directional. Continual study of the parental contributions to child anxiety will only help to facilitate newer, more effective treatment programs.
APPENDIX A: TABLES

Table 1. *Model Indicators and Their Abbreviations*

<table>
<thead>
<tr>
<th>Latent Variable</th>
<th>Indicator</th>
<th>Abbreviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child Negative Affect</td>
<td>PANAS-C NA, 5 items</td>
<td>PANAS1</td>
</tr>
<tr>
<td></td>
<td>PANAS-C NA, 5 items</td>
<td>PANAS2</td>
</tr>
<tr>
<td></td>
<td>PANAS-C NA, 5 items</td>
<td>PANAS3</td>
</tr>
<tr>
<td>Parental Anxiety</td>
<td>DASS-21 Anxiety subscale, 3 items</td>
<td>DANX1</td>
</tr>
<tr>
<td></td>
<td>DASS-21 Anxiety subscale, 3 items</td>
<td>DANX2</td>
</tr>
<tr>
<td></td>
<td>DASS-21 Anxiety subscale, 2 items</td>
<td>DANX3</td>
</tr>
<tr>
<td>Parental Negative Affect</td>
<td>DASS-21 Stress subscale, 3 items</td>
<td>DSTR1</td>
</tr>
<tr>
<td></td>
<td>DASS-21 Stress subscale, 3 items</td>
<td>DSTR2</td>
</tr>
<tr>
<td></td>
<td>DASS-21 Stress subscale, 2 items</td>
<td>DSTR3</td>
</tr>
<tr>
<td>Child Anxiety</td>
<td>RCADS Anxiety Scales, 18 items</td>
<td>RTOT1</td>
</tr>
<tr>
<td></td>
<td>RCADS Anxiety Scales, 19 items</td>
<td>RTOT2</td>
</tr>
<tr>
<td></td>
<td>Dimensional Ratings Child</td>
<td>Child DRs</td>
</tr>
<tr>
<td></td>
<td>Dimensional Ratings Parent</td>
<td>Parent DRs</td>
</tr>
</tbody>
</table>

*Note.* PANAS-C NA = Positive and Negative Affect Schedule for Children, Negative Affect Scale; DASS 21 = Depression-Anxiety-Stress Scales, 21 item version; RCADS = Revised Child Anxiety and Depression Scale (only the Anxiety scale was used in this study). The DASS-21 Stress and Anxiety Scales and the PANAS-C NA were both divided into three indicators by selecting every third item (9 indicators total). Also, the RCADS Anxiety scale was divided into two indicators by selecting every other item.
Table 2. Means, Standard Deviations and Internal Consistency (Cronbach’s Alpha) of All Indicators and Relevant Scales for the Total Sample (N=570)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Overall</th>
<th>N</th>
<th>Mean(SD)</th>
<th>( \alpha )</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS-C NA TOTAL</td>
<td>570</td>
<td>30.7(12.0)</td>
<td>.91</td>
<td></td>
</tr>
<tr>
<td>PANAS1</td>
<td>570</td>
<td>10.5(4.5)</td>
<td>.79</td>
<td></td>
</tr>
<tr>
<td>PANAS2</td>
<td>570</td>
<td>10.1(4.6)</td>
<td>.81</td>
<td></td>
</tr>
<tr>
<td>PANAS3</td>
<td>570</td>
<td>10.1(4.0)</td>
<td>.72</td>
<td></td>
</tr>
<tr>
<td>RCADS ANX TOTAL</td>
<td>570</td>
<td>25.4(18.0)</td>
<td>.96</td>
<td></td>
</tr>
<tr>
<td>RTOT1</td>
<td>570</td>
<td>12.5(9.2)</td>
<td>.90</td>
<td></td>
</tr>
<tr>
<td>RTOT2</td>
<td>570</td>
<td>12.9(9.2)</td>
<td>.90</td>
<td></td>
</tr>
<tr>
<td>Child DRs</td>
<td>570</td>
<td>8.8(7.1)</td>
<td>.74</td>
<td></td>
</tr>
<tr>
<td>Parent DRs</td>
<td>570</td>
<td>8.2(6.3)</td>
<td>.68</td>
<td></td>
</tr>
<tr>
<td>DSTR TOTAL</td>
<td>570</td>
<td>4.4(4.5)</td>
<td>.82</td>
<td></td>
</tr>
<tr>
<td>DSTR1</td>
<td>570</td>
<td>1.9(1.9)</td>
<td>.73</td>
<td></td>
</tr>
<tr>
<td>DSTR2</td>
<td>570</td>
<td>1.6(2.0)</td>
<td>.46</td>
<td></td>
</tr>
<tr>
<td>DSTR3</td>
<td>570</td>
<td>.96(1.3)</td>
<td>.64</td>
<td></td>
</tr>
<tr>
<td>DANX TOTAL</td>
<td>570</td>
<td>2.2(3.5)</td>
<td>.88</td>
<td></td>
</tr>
<tr>
<td>DANX1</td>
<td>570</td>
<td>1.0(1.6)</td>
<td>.69</td>
<td></td>
</tr>
<tr>
<td>DANX2</td>
<td>570</td>
<td>.61(1.2)</td>
<td>.70</td>
<td></td>
</tr>
<tr>
<td>DANX3</td>
<td>570</td>
<td>.57(1.1)</td>
<td>.64</td>
<td></td>
</tr>
</tbody>
</table>

Note. PANAS-C NA = Positive and Negative Affect Schedule for Children, Negative Affect Scale; RCADS ANX = Revised Child Anxiety and Depression Scales, Anxiety scale only; DSTR = Depression-Anxiety-Stress Scales, 21 item version, Stress scale only; DANX = Depression-Anxiety-Stress Scales, 21 item version, Anxiety scale only. The PANAS-C NA scale was divided into every third items to create three indicators (PANAS1-3). The DASS-21 Stress and Anxiety Scales were also divided into three indicators by selecting every third item (DSTRESS1-3 and DANX1-3, respectively). Also, the RCADS anxiety scale was divided into two indicators by selecting every other item (RTOT1-2).
Table 3. Kurtosis and Skewness for the Proposed Set of Indicators

<table>
<thead>
<tr>
<th>Indicator Abbreviation</th>
<th>Kurtosis</th>
<th>Skewness</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS1</td>
<td>-2.15</td>
<td>1.30</td>
</tr>
<tr>
<td>PANAS2</td>
<td>-2.84</td>
<td>1.78</td>
</tr>
<tr>
<td>PANAS3</td>
<td>-1.94</td>
<td>1.18</td>
</tr>
<tr>
<td>DANX1</td>
<td>-2.10</td>
<td>7.09</td>
</tr>
<tr>
<td>DANX2</td>
<td>0.87</td>
<td>9.36</td>
</tr>
<tr>
<td>DANX3</td>
<td>1.29</td>
<td>9.61</td>
</tr>
<tr>
<td>DSTR1</td>
<td>-3.80</td>
<td>3.20</td>
</tr>
<tr>
<td>DSTR2</td>
<td>-3.16</td>
<td>3.00</td>
</tr>
<tr>
<td>DSTR3</td>
<td>-3.65</td>
<td>5.86</td>
</tr>
<tr>
<td>RTOT1</td>
<td>-0.57</td>
<td>0.32</td>
</tr>
<tr>
<td>RTOT2</td>
<td>-0.81</td>
<td>0.45</td>
</tr>
<tr>
<td>Child DRs</td>
<td>-1.39</td>
<td>0.81</td>
</tr>
<tr>
<td>Parent DRs</td>
<td>-1.31</td>
<td>0.77</td>
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</tbody>
</table>
Table 4. Zero-order Pearson Correlation of Selected Indicators for Full Sample (N=570)

<table>
<thead>
<tr>
<th></th>
<th>DSTR 1</th>
<th>DSTR 2</th>
<th>DSTR 3</th>
<th>DANX 1</th>
<th>DANX 2</th>
<th>DANX 3</th>
<th>Child DRs</th>
<th>RTOT 1</th>
<th>RTOT 2</th>
<th>PANAS 1</th>
<th>PANAS 2</th>
<th>PANAS 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSTR 1</td>
<td>0.64(*)</td>
<td>2</td>
<td></td>
<td>0.61(*)</td>
<td>0.57(*)</td>
<td>0.62(*)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>DSTR 2</td>
<td></td>
<td>0.71(*)</td>
<td>0.61(*)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DSTR 3</td>
<td></td>
<td></td>
<td>0.61(*)</td>
<td>0.57(*)</td>
<td>0.62(*)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>DANX 1</td>
<td></td>
<td></td>
<td></td>
<td>0.58(*)</td>
<td>0.54(*)</td>
<td>0.59(*)</td>
<td>0.72(*)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DANX 2</td>
<td></td>
<td></td>
<td></td>
<td>0.58(*)</td>
<td>0.52(*)</td>
<td>0.64(*)</td>
<td>0.72(*)</td>
<td>0.78(*)</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>DANX 3</td>
<td></td>
<td></td>
<td></td>
<td>0.58(*)</td>
<td>0.52(*)</td>
<td>0.64(*)</td>
<td>0.72(*)</td>
<td>0.78(*)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Child DRs</td>
<td>0.10(*)</td>
<td>0.10(*)</td>
<td>0.12(*)</td>
<td>0.08</td>
<td>0.12(*)</td>
<td>0.10(*)</td>
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</tr>
<tr>
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<td>0.12(*)</td>
<td>0.10(*)</td>
<td>0.11(*)</td>
<td>0.12(*)</td>
<td>0.10(*)</td>
<td>0.11(*)</td>
<td>0.57(*)</td>
<td></td>
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</tr>
<tr>
<td>RTOT 2</td>
<td>0.10(*)</td>
<td>0.11(*)</td>
<td>0.13(*)</td>
<td>0.13(*)</td>
<td>0.12(*)</td>
<td>0.12(*)</td>
<td>0.59(*)</td>
<td>0.92(*)</td>
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<tr>
<td>PANAS 1</td>
<td>0.10(*)</td>
<td>0.10(*)</td>
<td>0.07</td>
<td>0.03</td>
<td>0.06</td>
<td>0.04</td>
<td>0.51(*)</td>
<td>0.64(*)</td>
<td>0.66(*)</td>
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<td>PANAS 2</td>
<td>0.11(*)</td>
<td>0.11(*)</td>
<td>0.10(*)</td>
<td>0.04</td>
<td>0.06</td>
<td>0.04</td>
<td>0.53(*)</td>
<td>0.67(*)</td>
<td>0.68(*)</td>
<td>0.80(*)</td>
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<td>0.13(*)</td>
<td>0.13(*)</td>
<td>0.11(*)</td>
<td>0.09(*)</td>
<td>0.45(*)</td>
<td>0.62(*)</td>
<td>0.63(*)</td>
<td>0.79(*)</td>
<td>0.76(*)</td>
<td></td>
<td></td>
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<tr>
<td>Parent DRs</td>
<td>0.28(*)</td>
<td>0.26(*)</td>
<td>0.28(*)</td>
<td>0.19(*)</td>
<td>0.19(*)</td>
<td>0.22(*)</td>
<td>0.62(*)</td>
<td>0.23(*)</td>
<td>0.26(*)</td>
<td>0.27(*)</td>
<td>0.29(*)</td>
<td>0.22(*)</td>
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</tbody>
</table>

Note. * Correlation is significant at the .05 level or lower (2-tailed).
Table 5. Fit Statistics for the Structural Models

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$ (df)</th>
<th>$p$</th>
<th>AIC</th>
<th>RMSEA</th>
<th>Cfit</th>
<th>GFI</th>
<th>IFI</th>
<th>RMR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial Measurement Model</td>
<td>159.4 (57)</td>
<td>.00</td>
<td>218.5</td>
<td>.05</td>
<td>.27</td>
<td>.96</td>
<td>.99</td>
<td>.63</td>
</tr>
<tr>
<td>Revised Measurement Model</td>
<td>80.63 (43)</td>
<td>.00</td>
<td>149.8</td>
<td>.04</td>
<td>.92</td>
<td>.98</td>
<td>.99</td>
<td>.02</td>
</tr>
<tr>
<td>Revised Hypothesized Model</td>
<td>64.82 (47)</td>
<td>.04</td>
<td>126.8</td>
<td>.00</td>
<td>1.00</td>
<td>.98</td>
<td>1.0</td>
<td>.02</td>
</tr>
<tr>
<td>Revised Hypothesized Model with Age, Step 1</td>
<td>66.70 (48)</td>
<td>.04</td>
<td>126.7</td>
<td>.00</td>
<td>1.00</td>
<td>.98</td>
<td>1.0</td>
<td>.02</td>
</tr>
<tr>
<td>Revised Hypothesized Model with Age, Step 2</td>
<td>116.8 (96)</td>
<td>.07</td>
<td>237.0</td>
<td>.03</td>
<td>.99</td>
<td>.99</td>
<td>.97</td>
<td>.12</td>
</tr>
<tr>
<td>Revised Hypothesized Model with Age, Step 2</td>
<td>127.9 (98)</td>
<td>.02</td>
<td>231.5</td>
<td>.27</td>
<td>.99</td>
<td>.99</td>
<td>.97</td>
<td>.12</td>
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</tbody>
</table>

Note. AIC = Akaike’s Information Criterion; RMSEA = root mean square error of approximation; Cfit = test for close fit (RMSEA < .05); GFI = goodness of fit index; IFI = incremental fit index; RMR = standardized root mean square residual; Hypothesized Model = Model with parent anxiety, parent NA and child NA predicting child anxiety; Revised Hypothesized Model = Hypothesized Model without the path from parent NA to child anxiety; Alternative Model = Model proposing the direction of the anxiety transference is from child to parent; Revised Hypothesized Model with Age, Step 1 = the applicability of the model across ages 7-12 and 12-19; Revised Hypothesized Model with Age, Step 2 = test of whether the relations between parent anxiety and child anxiety were significantly different in younger children as compared to older children. The Revised Hypothesized Model (in bold) was retained as best fitting model for additional analyses.
Figure 1. Model 1: Measurement Model.
Figure 2. Model 2: Hypothesized Model. Note. NS = hypothesized non-significant path; * = hypothesized significant path.
Figure 3. Model 3: Alternative Model. Note. NS = hypothesized non-significant path; * = hypothesized significant path.
Figure 4. Model 4: Model of interaction effect of age.
Figure 5. Model 5: Hypothesized Model with completely standardized estimates. Note. NS = non-significant path; * = significant path (p < .05).
Figure 6. Model 6: Revised Hypothesized Model with completely standardized estimates. Note. NS = non-significant path; * = significant path (p < .05).
Figure 7. Model 7: Alternative Model with completely standardized estimates. *Note. NS = non-significant path; * = significant path (p < .05).
Figure 8. Model 8: Revised Hypothesized Model for the younger age group (7-11) with completely standardized estimates. Note. NS = non-significant path; * = significant path (p < .05).
Figure 9. Model 9: Revised Hypothesized Model for the older age group (12-19) with completely standardized estimates. Note. NS = non-significant path; * = significant path (p < .05).
Figure 10. Model 10: Model of interaction effect of age for full sample. Note. NS = non-significant path; * = significant path ($p < .05$).
PANAS-C

This scale has a number of words that describe different feelings and emotions. Read each item and then circle the best answer next to that word. Indicate to what extent you have felt this way during the past few weeks. There are no right or wrong answers.

<table>
<thead>
<tr>
<th></th>
<th>Very slightly or not at all</th>
<th>A little</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
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<tbody>
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<td>1.</td>
<td>Interested</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2.</td>
<td>Sad</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3.</td>
<td>Frightened</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4.</td>
<td>Excited</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5.</td>
<td>Ashamed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6.</td>
<td>Upset</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7.</td>
<td>Happy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8.</td>
<td>Strong</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9.</td>
<td>Nervous</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10.</td>
<td>Guilty</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<tr>
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<td>Energetic</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<td>12.</td>
<td>Scared</td>
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<td>2</td>
<td>3</td>
<td>4</td>
</tr>
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<td>Calm</td>
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<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14.</td>
<td>Miserable</td>
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<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15.</td>
<td>Jittery</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16.</td>
<td>Cheerful</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17.</td>
<td>Active</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18.</td>
<td>Proud</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19.</td>
<td>Afraid</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20.</td>
<td>Joyful</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>21.</td>
<td>Lonely</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>22.</td>
<td>Mad</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>23.</td>
<td>Disgusted</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>24.</td>
<td>Delighted</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<tr>
<td>25.</td>
<td>Blue</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>26.</td>
<td>Gloomy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>27.</td>
<td>Lively</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<tr>
<td></td>
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</tr>
<tr>
<td>1.</td>
<td>I worry about things</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>2.</td>
<td>I feel sad or empty</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>3.</td>
<td>When I have a problem, I get a funny feeling in my stomach</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>4.</td>
<td>I worry when I think I have done poorly at something</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>5.</td>
<td>I would feel afraid of being on my own at home</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>6.</td>
<td>Nothing is much fun anymore</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>7.</td>
<td>I feel scared when I have to take a test</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>8.</td>
<td>I feel worried when I think someone is angry with me.</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>9.</td>
<td>I worry about being away from my parents</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>10.</td>
<td>I get bothered by bad or silly thoughts or pictures in my mind</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>11.</td>
<td>I have trouble sleeping</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
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<td>12.</td>
<td>I worry that I will do badly at my school work</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
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<tr>
<td>13.</td>
<td>I worry that something awful will happen to someone in my family</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>14.</td>
<td>I suddenly feel as if I can’t breathe when there is no reason for this</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
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<tr>
<td>15.</td>
<td>I have problems with my appetite</td>
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<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>16.</td>
<td>I have to keep checking that I have Done things right (like the switch is off, or the door is locked)</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
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<td>17.</td>
<td>I feel scared if I have to sleep on my own</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
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<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
<td></td>
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<tr>
<td>18.</td>
<td>I have trouble going to school in the mornings because I feel nervous or afraid</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>19.</td>
<td>I have no energy for things</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>20.</td>
<td>I worry I might look foolish</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>21.</td>
<td>I am tired a lot</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>22.</td>
<td>I worry that bad things will happen to me</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>23.</td>
<td>I can’t seem to get bad or silly thoughts out of my head</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>24.</td>
<td>When I have a problem, my heart beats really fast</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>25.</td>
<td>I cannot think clearly</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>26.</td>
<td>I suddenly start to tremble or shake when there is no reason for this</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>27.</td>
<td>I worry that something bad will happen to me</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>28.</td>
<td>When I have a problem, I feel shaky</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>29.</td>
<td>I feel worthless</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
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<td>30.</td>
<td>I worry about making mistakes</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>31.</td>
<td>I have to think of special thoughts (like numbers or words) to stop bad things from happening</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>32.</td>
<td>I worry what other people think of me</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>33.</td>
<td>I am afraid of being in crowded places (like shopping centers, the movies, buses, busy playgrounds)</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td>34.</td>
<td>All of a sudden I feel really scared for no reason at all</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
</tr>
<tr>
<td></td>
<td>Question</td>
<td>Never</td>
<td>Sometimes</td>
<td>Often</td>
<td>Always</td>
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<tr>
<td>35.</td>
<td>I worry about what is going to happen</td>
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<tr>
<td>36.</td>
<td>I suddenly become dizzy or faint when there is no reason for this</td>
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<tr>
<td>37.</td>
<td>I think about death</td>
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<tr>
<td>38.</td>
<td>I feel afraid if I have to talk in front of my class</td>
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<tr>
<td>39.</td>
<td>My heart suddenly starts to beat too quickly for no reason</td>
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<tr>
<td>40.</td>
<td>I feel like I don't want to move</td>
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<tr>
<td>41.</td>
<td>I worry that I suddenly get a scared feeling when there is nothing to be afraid of</td>
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<tr>
<td>42.</td>
<td>I have to do something over and over again (like washing my hands, cleaning or putting things in a certain order)</td>
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<tr>
<td>43.</td>
<td>I feel afraid that I will make a fool of myself in front of people</td>
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<tr>
<td>44.</td>
<td>I have to do something in just the right way to stop bad things from happening</td>
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<tr>
<td>45.</td>
<td>I worry when I go to bed at night</td>
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<tr>
<td>46.</td>
<td>I would feel scared if I had to stay away from home overnight</td>
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<tr>
<td>47.</td>
<td>I feel restless</td>
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</tbody>
</table>
Please read each statement and circle a number 0, 1, 2 or 3 that indicates how much the statement applied to you over the past week. There are no right or wrong answers. Do not spend too much time on any statement.

*The rating scale is as follows:*

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1. I found it hard to wind down
2. I was aware of dryness of my mouth
3. I couldn't seem to experience any positive feeling at all
4. I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)
5. I found it difficult to work up the initiative to do things
6. I tended to over-react to situations
7. I experienced trembling (e.g., in the hands)
8. I felt that I was using a lot of nervous energy
9. I was worried about situations in which I might panic and make a fool of myself
10. I felt that I had nothing to look forward to
11. I found myself getting agitated
12. I found it difficult to relax
13. I felt down-hearted and blue
14. I was intolerant of anything that kept me from getting on with what I was doing
15. I felt I was close to panic
16. I was unable to become enthusiastic about anything
17. I felt I wasn't worth much as a person
18. I felt that I was rather touchy
19. I was aware of the action of my heart in the absence of physical exertion (e.g. sense of heart rate increase, heart missing a beat)
20. I felt scared without any good reason
21. I felt that life was meaningless
Based on the child and parent portion of the assessments, please assign a dimensional rating (DR) from 0 to 8 for the features of each disorder, whether or not criteria are met for that disorder. In order to make the measures sensitive, be sure to assign a zero (0) rating only when there is no trace or feature of that disorder. If multiple disorders or areas are present within one rating scale (e.g., multiple specific phobias), please indicate this by assigning an appropriately increased DR for that scale.

For the consensus version, please assign a DR from 0 to 8 for the features of each disorder, whether or not criteria are met for that disorder based on the collective information obtained during the course of the evaluation, teacher interviews, and your judgment about the relative validity of the various informants.

<table>
<thead>
<tr>
<th>Child Version</th>
<th>Parent Version</th>
<th>Consensus Version</th>
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</thead>
<tbody>
<tr>
<td>Separation Anxiety</td>
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<tr>
<td>Generalized Anxiety</td>
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<tr>
<td>Social Anxiety</td>
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<tr>
<td>Obsessions/Compulsions</td>
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<tr>
<td>Panic</td>
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<tr>
<td>Agoraphobia</td>
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<tr>
<td>Posttraumatic Stress</td>
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<tr>
<td>Specific Fear/Phobia</td>
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<tr>
<td>Depression/Dysthmic Disorder</td>
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<tr>
<td>Oppositional</td>
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<tr>
<td>Delinquent</td>
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<tr>
<td>Hyperactive</td>
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<tr>
<td>Inattentive</td>
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</tr>
</tbody>
</table>
REFERENCES


children: Effects on distress before, during, and after hospitalization for surgery.

*Health Psychology, 5, 231-247.*