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ANXIETY SENSITIVITY, BEHAVIORAL INHIBITION, AND COGNITIVE BIASES AS

RISK FACTORS FOR ANXIETY: CUMULATIVE, INCREMENTAL, AND MEDIATED

INFLUENCES

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ABSTRACT

The present study aimed to advance understanding of the cumulative, incremental, and mediated influences of anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases on anxiety outcomes. Cumulative and multiple-risk factor models, as well as direct and indirect pathways linking anxiety sensitivity and behavioral inhibition with anxiety outcomes, were examined in 862 emerging adults. Exploratory cluster analyses were also performed in an effort to identify subgroups of participants with different constellations of risk. The cumulative and multiple-risk factor models significantly predicted anxiety outcomes, although the statistical prediction offered by the latter model was superior. Additionally, variability in each risk factor significantly predicted anxiety outcomes after controlling for the total number of risks, supporting the value of the content of risk to the prediction of anxiety outcomes. Structural equation modeling revealed that interpretive and judgment biases partially mediated pathways linking anxiety sensitivity and behavioral inhibition with anxiety outcomes. Anxiety sensitivity and behavioral inhibition were also directly linked with anxiety outcomes. Results were similar for males and females. Finally, cluster analyses revealed four clusters with varying combinations of risk and somewhat different levels of anxiety. Findings, implications for intervention, and limitations are discussed.

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DEDICATION

To my wife, Mariana, who gives meaning to everything that I do.

1. Introduction

The relation of interpretive and judgment biases to elevated anxiety and internalizing difficulties has been the subject of growing attention (Vasey & MacLeod, 2001; see Craske & Pontillo, 2001, and Weems & Watts, 2005, for excellent reviews). Research evidence suggests that men and women with anxiety commonly make threatening interpretations of, and misjudge their ability to cope with, ambiguous or neutral stimuli (Beck, Emery, & Greenberg, 1985; Mathews & MacLeod, 2002; McNally, 1996; Weems, Silverman, Rapee, & Pina, 2003). Research also suggests that interpretive and judgment biases may be causally related to elevated anxiety, thereby exacerbating risk for anxiety problems (e.g., Yiend & Mathews, 2002). However, two important areas on this topic have received relatively little study: (1) the contribution of interpretive and judgment biases relative to biologically-based anxiety precursors, such as anxiety sensitivity (Reiss & McNally, 1985; Stein, Jang, and Livesley, 1999; Taylor, Jang, Stewart, & Stein, 2008) and behavioral inhibition (Kagan, 2008), to the prediction of anxiety symptoms[1], and (2) the role of interpretive and judgment biases as potential mediators of the relation between anxiety sensitivity and behavioral inhibition, and elevated anxiety symptoms.

Important implications can stem from this research; on the one hand, findings may suggest that anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases predict anxiety *cumulatively*; to the extent that more of these risks are present, the higher the anxiety experienced by individuals, regardless of the content of risk (*cumulative risk* or *cumulative stress* hypothesis; Sameroff, Seifer, Baldwin, & Baldwin, 1993). On the other hand, this research may suggest that the content of risk is crucial to the process and that each of the risks *incrementally* predicts anxiety; such multiple risk-factor models (Deater-Deckard, Dodge,

Bates, & Pettit, 1998), where content of risk matters, allow for examination of different patterns of risk that may lead to anxiety. Finally, to the extent that the links between developmental precursors of anxiety—such as anxiety sensitivity and behavioral inhibition—and anxiety symptoms are mediated by interpretive and judgment biases, decisions about when or where to intervene may be facilitated. For example, findings may suggest early intervention targeting anxiety sensitivity and/or behavioral inhibition in order to prevent the future emergence of cognitive biases. Conversely, intervening at the level of cognition may prove more effective once cognitive biases are present, in part, due to their malleability relative to biologically-based risk factors (McNally & Foa, 1987).

With this in mind, the purpose of this study is to shed light on the aforementioned areas by testing three separate models: (a) a cumulative risk model examining whether anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases, cumulatively increase risk for anxiety symptoms; (b) a multiple risk-factor model that takes into account the content of risk to test whether each of these risks offer incremental contributions to the prediction of anxiety symptoms; and (c) a mediational model, to examine whether interpretive and judgment biases mediate the relation between anxiety sensitivity and behavioral inhibition, and anxiety symptoms (see Figure 1). In doing so, this study can begin to outline the often-assumed developmental progression from biologically-based anxiety precursors to distorted thinking to anxiety symptoms. This, in turn, can further our understanding of the development of anxiety problems and suggest potential avenues for interventions designed to preempt full-blown anxiety pathology.

1.1. Cumulative versus Incremental Risk for Anxiety

An individual rarely experiences a single stressor in isolation; instead, a constellation of interrelated stressors most often shape ontogenic development (Cicchetti, 2006). Although studies often show significant differences in outcomes based on a single risk factor, the proportion of variance explained by that single risk factor is typically small (Sameroff et al., 1993). For example, Deater-Deckard and colleagues (Deater-Deckard et al., 1998) found that child risk factors accounted for 4% of the variance in teacher reports of externalizing behavior, whereas a model that included various sociocultural, parenting, and peer risk factors in addition to child risk factors accounted for 36% of the variance. Similarly, Weems and colleagues (Weems, Costa, Watts, Taylor, & Cannon, 2007) found that anxiety sensitivity accounted for 11% of the variance in child-reported anxiety symptoms, whereas a model that included child demographic characteristics, and various cognitive and judgment biases in addition to anxiety sensitivity accounted for 50% of the variance. This suggests that cumulative risk models may more fully explain negative outcomes (Corapci, 2008; Evans & English, 2002; Sameroff, Gutman, & Peck, 2003). For example, strong empirical evidence suggests that it is not a single risk factor, but the number of risk factors that more accurately predicts psychiatric status (Rutter, 1979). Risk for psychiatric disorder in 10-year-olds increased from 2% in families with zero or one risk factors to 20% in families with four or more (Rutter, 1979). Similar results were found in other investigations examining a myriad of mental health and socio-emotional problems (e.g., Williams, Anderson, McGee, & Silva, 1990; Sameroff, Seifer, Zax, & Barocas, 1987). While significant progress has been made in the area of cumulative risk for negative outcomes, more research is needed to determine the combination of risks that may be important for specific psychological outcomes (see Koinis-Mitchell, 2008).

In the area of anxiety, consideration of both cumulative risk and multiple-risk factor models is rare (Weems & Silverman, 2008). While several studies have found support for the cumulative risk model with respect to externalizing problems (e.g., Biederman et al., 1995; Deater-Deckard et al., 1998) and other developmental outcomes (e.g., IQ; Sameroff et al., 1993; Liaw & Brooks-Gunn, 1994), the cumulative risk hypothesis has seldom been tested with regards to anxiety problems. In testing the first model, this study addresses this issue by positing a cumulative risk factor model where anxiety sensitivity and behavioral inhibition, as well as judgment and interpretive biases cumulatively increase risk for anxiety symptoms. That is, their sheer number confers risk in the form of generic stress—content of risk is irrelevant in this model.

Conversely, it is well-established that risk variables often covary in significant ways. With regard to anxiety, a plethora of evidence suggests that anxiety sensitivity (Silverman, Fleisig, Rabian, & Peterson, 1991) and a behaviorally inhibited temperament (Kagan, 2008) may lead to anxiety problems; similarly, research suggests that certain cognitive biases may place individuals at risk for anxiety (Weems et al., 2007). However, both research and theory have lagged behind in terms of efforts to conceptualize how these constructs covary and operate together. A test of the *incremental* prediction offered by each of these constructs can clarify whether and how these risks operate in combination with each other to predict anxiety problems. In testing the second model, this study incorporates content of risk as crucial to the development of anxiety and potentially identifies risk factors that incrementally explain anxiety problems, thereby quantifying the severity posed by different risks factors *in the context of* other risks.

Importantly, these models account for risk factors that theoretically differ in terms of developmental origins, an extension over available theorizing and one which more accurately

represents the complexity that characterizes the emergence of specific problems such as anxiety (Koinis-Mitchell, 2008; Weems & Silverman, 2008).

At the same time, while there is research evidence implicating behavioral inhibition and anxiety sensitivity in the early emergence of anxiety problems, little is known about their interrelation as well as the potential progression from these risk factors to cognitive risk factors emerging later in development—interpretive and judgment biases. Research suggests that children who are born with a biological predisposition for high reactivity and fearful responding to novel stimuli or situations (i.e., high behavioral inhibition) are at risk for developing anxiety problems (Biederman et al., 2001; Kagan, Reznick, & Snidman, 1988). Similarly, research indicates that the biologically-based fear of anxiety-related sensations (e.g., perspiration, stomach discomfort; i.e., anxiety sensitivity) serves as a risk factor for anxiety in both children (Weems, Hayward, Killen, & Taylor, 2002) and adults (Reiss, 1991). Exactly how these biologically-based factors lead to the development of anxiety problems, however, is not fully understood.

In testing the third model, this study sheds light on the issue of whether the relation of behavioral inhibition and anxiety sensitivity to anxiety problems may be mediated by interpretive and judgment biases. Theoretically, such a developmental progression is possible: a biologically-based anxious predisposition may lead to experiencing events with fear and avoidance (Kagan, 2008); in turn, these negatively-laden experiences may be more easily recalled from memory during subsequent individual-environment transactions, thereby resulting in erroneous interpretations and judgments of those situations (Weems & Watts, 2005). This process may ultimately culminate in the emergence of significant anxiety problems. A model that tests such a

path would be a welcome addition to the study of etiological factors implicated in the development anxiety.

With this in mind, this introduction addresses: (1) the relevance of anxiety problems in the U.S., (2) the leading cognitive information processing model of anxiety in clinical psychology, (3) interpretive biases that may be associated with anxiety, (4) judgment biases that may be associated with anxiety; (5) anxiety sensitivity and behavioral inhibition and their relation to anxiety; and (6) the three conceptual models proposed: a cumulative risk model, a multiple risk-factor model, and a mediational model that implicate interpretive and judgment biases, anxiety sensitivity, and behavioral inhibition to explain their effects on anxiety symptoms.

1.2 Overview and Significance

Anxiety disorders are the most common form of psychopathology in the U.S. It is estimated that, in any given year, an estimated 40 million adults age 18 or older (i.e., 18.1% of the population) meet criteria for an anxiety disorder (Kessler, Chiu, Demler, & Walters, 2005). Furthermore, in 1990, a third (i.e., $42.3 billion) of the country's mental health budget was spent treating individuals with anxiety disorders (Greenberg et al., 1999). The landscape looks quite similar for children, with anxiety disorders being among the most common mental disorders in this population (Weiss & Last, 2001). Depending on the assessment strategy, age of the sample, and type of disorder included, childhood anxiety disorder prevalence rates in the community vacillate between 2.4% (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003) and 17% (Kashani & Orvaschel, 1988).

Anxiety and phobic disorders significantly impact children's school functioning (Beidel, Turner, & Morris, 1999), peer interactions, and mastery of developmental tasks (e.g., sleeping

alone) (e.g., Kearney, 2005; Silverman & Kurtines, 1996), and research evidence suggests that childhood anxiety disorders may follow a chronic course (e.g., Last, Perrin, Hersen, & Kazdin, 1996; Otto, Pollack, Rosenbaum, Sachs, & Asher, 1994). In adults, anxiety is also often the cause of significant functional impairment in work, social, and leisure domains (Wittchen, Stein, & Kessler, 1999). The benefits of increasing our understanding of anxiety and its disorders thus seem apparent, particularly from public health, prevention, and economic standpoints.

1.3 Anxiety and Distorted Thinking

Efforts to increase our understanding of anxiety have been fueled by the widespread finding in practice and research that those who suffer from anxiety evince distorted thinking (Beck, 1976; Ingram & Kendall, 1987).[2] From a theoretical standpoint, Beck's (1976) cognitive theory of emotional disorders has been seminal to the understanding of the cognitive component of anxiety. Specifically, Beck (Beck et al., 1985) noted that individuals with pathological anxiety make systematic errors in interpretation of their experience (i.e., *cognitive distortions*), which often go unchallenged and lead to considerable psychological distress (Beck et al.). Thus, the manner in which information is cognitively processed was posited as crucial in the etiology, maintenance, and treatment of anxiety disorders (Beck & Clark, 1997; Eysenck, 1992; Wells & Matthews, 1994).

Research in this area has outlined 4 types of cognitive biases/distortions in children (Vasey & MacLeod, 2001; Weems & Watts, 2005) and adults (Craske & Pontillo, 2001; McNally, 1996) with anxiety, all of which evolve around a theme of personal threat: memory biases (e.g., tendency to recall threatening or negative information), attention biases (selective attention to threatening stimuli in the context of other, nonthreatening stimuli), judgment biases

(e.g., tendency to perceive deficits in self-coping ability), and interpretive biases (i.e., interpreting neutral or ambiguous events as threatening).

Although the role of memory and selective attention in anxiety has been extensively studied (e.g., Gotlib & MacLeod, 1997), comparatively less research has been devoted to the study of judgment and interpretive biases (Vasey & Dadds, 2001; Weems & Watts, 2005). In addition, little is known about the relation between judgment and interpretive biases and biologically-based risk factors implicated in anxiety and their cumulative impact on anxiety symptoms. Specifically, more knowledge is needed regarding (1) the potentially cumulative risk posed by anxiety sensitivity and behavioral inhibition in conjunction with interpretive and judgment biases to the prediction of anxiety problems; this can clarify whether the number of risks, and not their content, is what is relevant; (2) the way in which these risks incrementally predict anxiety problems, which can potentially identify specific risk factors critical to the expression of anxiety symptoms and clarify the relative contribution of specific risk factors in the context of others; and (3) interpretive and judgment biases as mediators of the relation between anxiety sensitivity and behavioral inhibition, and anxiety symptoms, which can potentially identify candidates for prevention before biases develop. The present investigation seeks to shed light on these issues, an important endeavor considering the causal role of anxiety sensitivity (Schmidt et al., 1997), behavioral inhibition (Biederman et al., 2001), and cognitive biases in pathological anxiety (Mathews & MacLeod, 2002; Yiend & Mathews, 2002).

1.4. A Cognitive Information Processing Model of Anxiety

As mentioned above, Beck and colleagues (Beck et al., 1985; Beck & Clark, 1997) articulated a three-stage, schema-driven information processing model of anxiety that "considers the erroneous or biased interpretation of stimuli as dangerous or threatening to an individual's

physical or psychological well-being, a core feature of anxiety disorders" (Beck & Clark, 1997, p. 50-51). In addition to biased interpretation of stimuli as threatening, the model argues that anxiety-prone individuals also underestimate their coping resources to deal with potential threats and ignore safety signals in the environment (Lazarus, 1966). Thus, an anxiety-prone individual may perceive threat in a relatively innocuous situation *and* also underestimate his/her capacity to cope effectively with its demands. In order to understand the nature of these distortions and when they emerge, it is necessary to briefly discuss each of the model's stages, with particular attention to Stage II—when biases are presumably activated in the information processing chain.

1.4.1. Stage I: Initial registration. The first step in the processing of threat material involves the activation of the *orienting mode* (Beck, 1996), the structure of the information processing system that governs the very fast and automatic recognition of the stimulus. It is hypothesized that processing at this stage is automatic (e.g., involuntary, rapid, requiring minimal energy) and stimulus-driven. The orienting mode serves as a rapid screening device (analogous to a metal detector; e.g., "threat" or "no threat"), assigning priority to the stimulus and orienting attentional resources to it. Its main goal is to guarantee the survival of the individual. Beck (Beck & Clark, 1997) as well as others (Mathews & MacLeod, 1994; McNally, 1995) further argued that processing at this stage is likely undifferentiated; the goal is to simply assign valence (neutral, positive, negative) and personal relevance to the stimulus. Empirical evidence supports this contention (Mogg, Bradley, Williams, & Mathews, 1993), suggesting that hypersensitivity to negative stimuli in general may characterize the anxiety response at this initial step of information processing.

1.4.2. Stage II: Immediate preparation. After the orienting mode has identified a stimulus as negative, the organism begins to prepare to respond to it (Beck, 1996; Beck et al.,

1985). This involves the activation of a *primal mode*, the part in the information processing system that contains interconnected schemas of cognitive, affective, behavioral, and physiological patterns of response (or sets) passed down through evolution to immediately cope (e.g., guarantee survival, security, procreation, and so on) with the stimulus (Beck & Clark, 1997). There are thus a variety of primal modes (e.g., depressive mode, erotic mode) that are activated according to the stimulus presented (e.g., threatening stimulus activates the "danger or threat mode"). In anxiety, the threat mode is activated to help guarantee survival and involves the selection of the appropriate schema to deal with the threat. The schema, in turn, will govern the appropriate responses: narrowing of cognition on the threat (i.e., "tunnel vision"), feelings of fear, avoidant/escaping behavior, and physiological hyperarousal.

The activation of primal modes involves both automatic and controlled processes (Beck & Clark, 1997); their activation is rapid, involuntary, and inflexible. At the same time, *primary threat analysis* also begins during this stage—a controlled, semantic-linguistic characterization of the threat. In other words, the individual begins to assign meaning to the threat. Two products result from this primary threat appraisal and activation of the primal mode: (1) a narrowing of cognitive processing, which leads to biases and distortions (a focus of this investigation), and (2) negative automatic thoughts. It is at this stage that the anxiety-prone individual often focuses excessively on potentially threatening aspects of a situation while ignoring other positive aspects or personal assets, entertains the worst possible outcome, attributes to him/herself excessive responsibility for a possible negative outcome, and attributes a single negative event as representative of all similar future events. In addition, automatic thoughts and images involving themes of danger or threat occur, which the individual is aware of but cannot control easily.

As mentioned above, Stage II in Beck's model (Beck et al., 1985) provides the conceptual framework for the occurrence of judgment and interpretive biases associated with anxiety symptoms. Noticeably, the literature lacks a coherent model that articulates the interrelation between these biases and biologically-based anxiety precursors in the development of anxiety symptoms (e.g., Kagan et al., 1988; Silverman & Weems, 1999). Investigating their relations may suggest where and how to intervene in a chain that may culminate in problematic anxiety, given the temporal precedence of biologically-based anxiety precursors in relation to the emergence of cognitive biases. The goal of the present investigation is to shed light on these issues.

1.4.3. Stage III: Secondary elaboration. In this final stage of the information processing model of anxiety, controlled, elaborated processing of the threat takes place. Other schemas involving personal concerns are activated, "a contextualized processing involving self-in-relation-to-the-world" (Beck & Clark, 1997, p. 53). During this *secondary appraisal process*, the individual carefully examines his/her current coping resources and the likelihood of dealing effectively with the threat (Lazarus, 1966). This controlled consideration of personal resources and attributes of the threat, Beck argued, involves the activation of the *metacognitive mode*. The activation of this mode can lead to an escalation (or decrease) of anxiety symptoms if personal resources and attributes of the threat are unrealistically (or realistically) appraised. Important aspects of Stage III elaborative process are worry (Borkovec, Robinson, Pruzinsky, & DePree, 1983), and the search for safety signals in the environment.

As mentioned above, Beck's model does not clearly articulate the interrelation between interpretive and judgment biases, and their association with anxiety. In addition, the model does not mention the potential relation between cognitive biases and biologically-based anxiety

precursors; yet, a number of constructs (e.g., anxiety sensitivity, behavioral inhibition) have

gained great attention in the field of anxiety. To the extent that anxiety sensitivity and behavioral

inhibition, as well as cognitive biases incrementally increase risk for anxiety, intervention

targeting these early developmental precursors may be necessary to prevent the emergence of

interpretive and judgment biases.

Finally, Beck's model does not consider cognitive biases as a potential mediator of the

relation between biologically-based anxiety precursors and anxiety symptoms; as mentioned

above, however, individuals born with a predisposition to anxious apprehension (e.g., behavioral

inhibition, anxiety sensitivity) may learn to interpret and judge the world and themselves in

distorted ways, which can lead to developing anxiety problems. Testing such a mediational

model is a necessary step that can inform efforts aimed at preventing interpretive and judgment

biases from firmly rooting in cognition. The present study tests a model that includes these

previously missing pieces. It is now prudent to specifically review the evidence on interpretive

and judgment biases/distortions.

1.5. Interpretive Biases

Ambiguity is often the status quo in life. An explosive noise may signal fireworks or a

discharging weapon. The steps of someone walking in the hallway may be those of a friend, or

those of an approaching burglar. People who consistently interpret ambiguous situations or

stimuli as threatening are said to be at risk for higher anxiety (Beck et al., 1985; Mathews &

MacLeod, 2002; McNally, 1996).

In an early study (Butler & Mathews, 1983) investigating interpretive biases in anxiety,

12 generalized anxiety disorder (GAD) patients, 12 major depressive disorder (MDD) patients,

and 12 controls were presented with 10 ambiguous scenarios (e.g., you wake up with a startle in

the middle of night, thinking you heard a noise, but all is quiet") and asked to respond to an open-ended question (e.g., "what do you think woke you up?"). Patients and controls were then instructed to arrange three hypothetical explanations to each scenario in the order in which they would be most likely to come to mind in a similar situation. Only one of the three explanations provided for each scenario was purposely threatening (e.g., "it could be a burglar"). Findings showed that, relative to controls, anxious patients were more likely to interpret ambiguous scenarios as threatening. This study, however, did not examine biased interpretations in the context of other risk factors for anxiety (i.e., behavioral inhibition, anxiety sensitivity), which would have provided a more detailed analysis on the relative contribution of interpretive biases in relation to other developmental precursors of anxiety. In addition to a small sample, Butler and Mathews's (1983) focus on clinical groups also limits conclusions about interpretive biases' role in anxiety among individuals with nonclinical anxiety.

Using a sample of agoraphobic patients with panic attacks, McNally and Foa (1987) extended Butler and Mathews's (1983) findings by adding ambiguous situations involving *internal* stimuli (e.g., "you feel discomfort in your chest area…why?") to Butler and Mathews's questionnaire. The authors hypothesized that threatening interpretations in agoraphobic patients should show specificity to internal rather than external sensations, in light of the high anxiety sensitivity associated with panic attacks (Reiss & McNally, 1985). Contrary to hypotheses, the authors found that agoraphobic patients were more likely to interpret *both* external and internal stimuli as threatening relative to controls—an important finding suggesting that interpretation biases among high-anxiety individuals may be more pervasive than once thought. Again, although important, this study restricted its analyses to a single risk factor for anxiety, leaving

unanswered the question of the relevance of interpretive biases in the context of other risk

factors. In addition, generalizability of findings to nonclinical populations is also a concern.

Interpretive biases have also been examined across anxiety disorder groups for purposes

of comparability and to answer questions raised about the specificity of these biases (see e.g.,

Amir, Foa, & Coles, 1998, 2000). In a sample of 12 panic disorder (PD) patients, 12 social

phobics (SP), and 12 controls (Harvey, Richards, Dziadosz, & Swindell, 1993), threatening

interpretations to ambiguous stimuli, either internal or external, were more characteristic of both

disordered groups relative to controls. Findings were consistent with the assertion that anxiety

disorders, in general, are characterized by a predisposition to interpret ambiguous stimuli as

threatening (Williams, Watts, MacLeod, & Mathews, 1988). In addition, while both PD and SP

patients were more likely to choose threatening explanations to external stimuli, PD patients

were more likely than SP patients to choose threatening explanations for interoceptive stimuli.

Although anxiety sensitivity was indeed assessed in this study and both anxiety disordered

groups scored significantly higher than controls on this construct, a cumulative risk index for all

risk factors was not considered nor a test of incremental prediction—important extensions over

unitary risk-factor models that are in dire need. As with previous studies, this study's focus on a

small clinical sample also limits the findings' generalizability to nonclinical groups. Examining

these relations in a nonclinical group is important if we are to understand how these processes

operate under "normal" circumstances. At the same time, inasmuch as findings with a

nonclinical sample reflect relations found with clinical populations, identifying those at risk for

future, more serious pathology may be facilitated.

Other methodologies have also been used to study interpretive biases (see, e.g.,

Blanchette & Richards, 2003; Calvo, Eysenck, & Castillo, 1997; Calvo, Eysenck, & Estevez,

1994; Richards & French, 1992). Stoler and McNally (1991) asked agoraphobics, recovered agoraphobics, and controls to complete sentence stems with the first thought that came to mind. Sentence stems were either ambiguous or unambiguous. For example, in the stem "Knowing that entering the store would produce a sure fit, I…" the word fit could be interpreted as referring to clothing or to an anxiety attack (McNally, 1996). Consistent with studies reviewed earlier, results showed that agoraphobics were more likely to interpret stems as threatening relative to controls.

In an effort to circumvent a possible response bias effect in previous studies, Mathews, Richards, and Eysenck (1989) instructed GAD patients, recovered GAD patients, and controls to listen to ambiguous homophones (e.g., *dye* or *die*) and to write down the word they heard. Consistent with their hypothesis, anxious individuals wrote down threatening spellings more frequently than controls. GAD patients showed the highest number of threatening spellings, followed by recovered GADs and lastly healthy controls. Similar results were also found using samples of low and high trait anxiety individuals (Eysenck, MacLeod, & Mathews, 1987; Mogg, Bradley, Miller, Potts, Glenwright, & Kentish, 1994; Richards & Millwood, 1989).

Eysenck and colleagues (Eysenck, Mogg, May, Richards, & Mathews, 1991) also asked participants (3 groups: GADs, recovered GADs, and controls; $n = 16$ in each group) to listen carefully to a series of ambiguous sentences (e.g., "At the refugee camp, the week would soon be finished") presented quickly. Participants were then shown a number of sentences on a computer screen that were threatening (e.g., "at the camp, the sick would soon be dead") or nonthreatening (e.g., "at the camp, the weekend had nearly arrived") disambiguations of the sentences they heard earlier and asked to decide whether the visually displayed sentence had the same meaning as the one heard earlier. Anxious patients were more likely than controls to endorse the threatening rather than nonthreatening interpretation sentences, and this bias was for stimuli of

varying nature (e.g., social, physical). Similar results were also found using low and high trait individuals (MacLeod & Cohen, 1993).

In contrast to the focus on clinical groups evident across studies reviewed thus far, some investigators have indeed looked into specific interpretive biases among nonclinical groups; their results have been encouraging (for a review, see Weems & Watts, 2005). This work has focused primarily on samples of children and adolescents using the Children's Negative Cognitive Error Questionnaire (CNCEQ; Leitenberg, Leonard, & Carroll-Wilson, 1986). The CNCEQ measures 4 types of interpretive biases hypothesized by Beck (Beck et al., 1985) to be related to anxiety: catastrophizing, overgeneralizing, personalizing, and selective abstraction. Leitenberg et al. (1986) used the CNCEQ in a sample of children with high ($n = 95$; 67 girls, 28 boys) and low ($n = 106$; 40 girls, 66 boys) evaluation anxiety. They found that children with high evaluation anxiety displayed significantly higher cognitive errors on all four subscales relative to children with low evaluation anxiety. This early study provided support for the study of these 4 specific interpretive biases and their relation to anxious outcomes.

Building on this earlier work (Leitenberg et al., 1986), differential relations between each of the interpretive biases, as assessed by the CNCEQ, and anxiety and depression were explored (Epkins, 1996). A socially anxious group ($n = 14$), a dysphoric group ($n = 13$), a mixed socially anxious-dysphoric group ($n = 14$), and a control group ($n = 14$) were compared on all subscales of the CNCEQ. Epkins (1996) hypothesized that selective abstraction would be more strongly associated with depression than anxiety because of depression's characteristic focus on the negative aspects of situations. Conversely, personalization was hypothesized to be more strongly related to anxiety because of anxiety's focus on personal danger/ threat. The mixed group scored highest on all subscales, and both the socially anxious and dysphoric groups scored higher than

controls (Epkins, 1996). Personalizing *and* overgeneralizing were significantly elevated in the social anxiety group relative to the dysphoric group. Although selective abstraction was higher in the dysphoric group relative to the socially anxious group, the difference was not significant. This study suggested a significant link between different interpretive biases (Beck et al., 1985) and anxiety, with some specific relations highlighted.

Others have looked at the relations between these four interpretive biases and broader internalizing and externalizing symptoms, respectively (Leung & Poon, 2001; Leung & Wong, 1998), and found similar results. In a sample of 405 high school students (189 boys, 216 girls; mean age = 15 years) in Hong Kong, all four interpretive biases assessed by the CNCEQ were significantly related to internalizing and externalizing problems, although the relation to internalizing problems was stronger. In addition, results showed that personalizing, catastrophizing, and selective abstraction were significantly related to internalizing problems at a quadratic, U-shaped, rate. The relation between interpretive biases and externalizing problems was linear. Because of the high correlation between internalizing and externalizing problems in the study ($r = .58$), the same regression analyses were conducted controlling for each other. Internalizing symptoms significantly predicted all four interpretive biases after controlling for externalizing symptoms, although the opposite was not true. Results of this study suggested that the relation between internalizing problems and interpretive biases may be curvilinear, and that the relation between externalizing problems and interpretive biases may be explained by the comorbidity between internalizing and externalizing problems.

In a sample of 581 high school students (ages 12- 18; mean age = 14.7; 54.1% female) the same investigative group (Leung & Poon, 2001) examined similar interpretive biases using a modified version of the CNCEQ, the Children's Cognitive Distortions Questionnaire (CCDQ;

see Leung & Poon, 2001), that assesses personalizing, catastrophizing, and external attributions. Each of the CCDQ's subscales contains items related to three kinds of problems: anxiety, depression, and aggression. Significant specificity between the content of interpretive biases (about anxiety, depression, and aggression, respectively) and participants' levels of anxiety, depression, and aggression, respectively, was found. Thus, results of this study provided some evidence about the unique relation between specific interpretive biases and emotional problems, but also suggested that the content, and not necessarily the type, of bias is what may be specific to each emotional problem.

Weems and colleagues (Weems, Berman, Silverman, & Saavedra, 2001) found that all interpretive biases (except selective abstraction) were significantly related to measures of trait and manifest anxiety even after controlling for depression scores (rs ranged from .39 to .43) in a clinical sample of children and adolescents with anxiety disorders ($N = 251$; age range = 6 to 16 years). Age was a significant moderator, with weaker (but significant) correlations between anxiety and interpretive biases found in children ages 6-to-11 relative to adolescents ages 12-17 (gender was not a significant moderator). Weems et al. (2001) further examined the strength of the relation between biases and anxiety and depression, respectively. Somewhat consistent with previous results (Epkins, 1996), selective abstraction was the strongest predictor of depression while catastrophizing and overgeneralizing were the strongest predictors of anxiety.

Recently, Weems and colleagues (Weems et al., 2007) examined links between interpretive biases and anxiety in a community sample of ethnically diverse children and adolescents ($N = 145$; age range = 6-17 years; 55% female). A number of findings from this study are worth highlighting: (1) CNCEQ's four interpretive biases (i.e., catastrophizing, overgeneralizing, personalization, and selective abstraction) were significantly related to anxiety

and depression; (2) anxiety sensitivity, as measured by the Childhood Anxiety Sensitivity Index (CASI; Silverman et al., 1991), was significantly related to the four interpretive biases; (3) controlling for age and gender, the CASI, CNCEQ's interpretive biases, and anxiety control beliefs (Anxiety Control Questionnaire for Children [ACQ-C]; Weems et al., 2003) significantly predicted anxiety symptoms, together accounting for 32% of the variance; (4) each construct uniquely (i.e., after controlling for the remaining cognitive indices) predicted anxiety symptoms, with CASI scores accounting for the largest percentage of the variance (12%); (5) similar results were found with depression as the dependent variable, with selective abstraction accounting for the largest percentage of the variance (8%); (6) although the relation between selective abstraction and depression was specific (i.e., remained significant after controlling for anxiety), the relation between catastrophizing, overgeneralizing, and personalization was nonspecific to both anxiety and depression. Contrary to an early study (Weems et al., 2001), age did not moderate these relations, nor did gender or ethnicity.

Taken together, these studies suggest that, relative to controls, clinically anxious individuals tend to resolve ambiguity by favoring threatening interpretations over nonthreatening or neutral interpretations. At the same time, interpretive biases are also associated with anxiety in studies with nonclinical groups (e.g., Leitenberg et al., 1986; Leung & Poon, 2001; Leung & Wong, 1998; Weems et al., 2007). The overwhelming focus on clinical populations (e.g., Mathews et al., 1989), however, has a number of limitations. First, generalizability of findings to nonclinical populations with less severe levels of anxiety is problematic, given our interest in identifying the role such factors in the early development of anxiety. Second, studies on clinical samples may suffer from response bias. That is, patients may provide responses that they believe are consistent with their diagnosis. These limitations coupled with a lack of a developmental

framework in much of this research emphasize the need for studies with nonclinical populations (Cicchetti, 2006). In addition, the current literature is characterized by a lack of consideration of the relation of interpretive biases to anxiety problems *in the context* of other anxiety risk factors, despite overwhelming evidence that both cumulative (e.g., Rutter, 1979; Sameroff et al., 1993) and multiple risk-factor (e.g., Deater-Deckard et al., 1998) investigations may have better explanatory value and allow for examination of the incremental contribution of specific risk factors. Finally, little is known about the role of interpretive and judgment biases in mediating the relation between anxiety-related temperamental variables and anxiety symptoms—an important first step in outlining a potential developmental progression from biologically-based anxiety precursors to cognitive biases to anxiety symptoms.

1.6. Judgment Biases

Before reviewing the literature on judgment biases, it is important to first note that there is no general agreement as to how judgment and interpretive biases differ conceptually. Indeed, one can easily see how catastrophizing (e.g., "I have failed this test, I know I will end up as a bum in the streets") can also be construed as an error in judgment. Despite the lack of agreement, authors (Weems & Watts, 2005) have suggested that greater conceptual clarity can be achieved by considering where the object of the bias lies.

If the bias is centered on a specific context or event and involves negative interpretations of a neutral, ambiguous or potentially threatening stimulus, it is said to be an interpretive bias. On the other hand, if the focus of the bias is on the person making the judgment and on a perceived personal deficit or lack of ability (e.g., in terms of skills, coping ability, and the like), it is said to be a judgment bias (Weems & Watts, 2005; Weems et al., 2007). The present investigation utilizes this distinction for the sake of parsimony with the existing literature and to

allow for future comparison with published studies. However, it is recognized that the debate between what constitutes a judgment versus an interpretive biases has not subsided, and that utilizing the aforementioned conceptual distinction by no means implies that the issue has been definitively resolved (see, e.g., Weems et al.).

One construct in the area of anxiety involving judgments of one's ability is *perceived control*. Perceptions of control or lack thereof, are central to some etiological formulations on the nature of anxiety (Barlow, 2002; Chorpita & Barlow, 1998). These theoretical models suggest that perceptions of little control over internal emotions/sensations as well as external threats (fear-eliciting situations, events, places)—coupled with a heightened physiological reactivity— predispose individuals to experience pathological anxiety (Barlow, 2002; Rapee, Craske, Brown, & Barlow, 1996). Specifically, Barlow suggested that emotional reactions may lead some to pathological anxiety because they view these sensations as being out of control. In addition, empirical evidence suggests that repeated exposure to uncontrollable negative events may lead to pathological anxiety (Barlow, 2002), providing further support for the role of control in anxiety.

Notably, control has also been conceptualized more broadly, with a focus on perceived control in relation to all aspects of life (Rotter, 1966). These early conceptualizations argued that people differ in the extent to which they believe reinforcement is the result of their own actions (i.e., internal locus of control) or random external forces (i.e., external locus of control). The studies stemming from this model suggested that high perceptions of external control may cause someone to feel anxious because of the belief that there is no control over reinforcement (Watson, 1967).

Thus, early models on locus of control (Rotter, 1966, 1975) focused on perceived control over *reinforcement*, whereas Barlow's (2002) model focuses on perceived control over *internal*

sensations and external threats. While somewhat different in focus, both constructs (i.e., locus of

control and perceived control) have found that judgments about control are central to the

experience of anxiety. Nevertheless, several authors (Rapee et al., 1996) have questioned the

usefulness of measuring perceived control in relation to all areas of life. These concerns were

supported, in part, by results showing the multidimensional nature of Rotter's (1966) construct

(Berrenberg, 1987**;** Harper, Oei, Mendalgio, & Evans, 1990) and because people's perceptions of

control vary according to the area of life being assessed. Thus, with respect to anxiety, it has

been suggested that measuring perceived control of anxiety-specific phenomena may be more

useful (Barlow, 2002).

One of the first studies to examine the relation between control and anxiety was that of

Sanderson, Rapee, and Barlow (1989). The authors examined the effects of experimentally

granting an illusion of control to patients (*N* = 20) with PD with agoraphobia who were to

experience laboratory-induced panic attacks. All patients were asked to breathe 5.5% carbon

dioxide-enriched air for 20-minutes and fill out a panic attacks questionnaire afterwards. Only

half of the patients, however, were made to believe they had some control over the carbon

dioxide inhalation (by using a bogus handle). In support of the illusion-of-control effects, 20% of

patients in the illusion-of-control group reported panic attacks versus 80% in the no-illusion

group. The sense of control affected both cognitive and somatic symptoms of panic (see also

Chorpita & Barlow, 1998**;** McNally, 1990). Rapee et al. (1996) also found that greater perceived

control over anxiety-specific phenomena, as measured by the Anxiety Control Questionnaire

(ACQ; Rapee et al.) was significantly negatively related with self-reported anxiety, depression,

and stress symptoms (as measured by the Depression, Anxiety, and Stress Scales [DASS];

Lovibond & Lovibond, 1994) in a nonclinical subsample of 71 undergraduates that were part of a

larger study. Consistent with Rapee et al.'s findings, one study compared children and adolescents with anxiety disorders ($N = 86$; ages 9 to 17) to controls ($N = 31$) and found that the former group reported significantly lower perceptions of control over anxiety-related events relative to the latter group (Weems et al., 2003).

Consistent with studies showing a relation between external locus of control and anxiety in children (Ollendick, 1979; Rawson, 1992), investigators (Ginsburg, Lambert, & Drake, 2004) have found that both higher [general] external locus of control and lower perceived control over anxiety-specific phenomena were significantly, concurrently related to higher panic symptoms in a community sample of African-American adolescents ($N = 109$; mean age = 15.75 years). However, only perceived control over anxiety-specific situations predicted panic symptoms 6 months later. Ginsburg et al.'s (2004) study suggests that both types of judgment biases are important to the experience of anxiety symptoms, with perceived control over anxiety-specific symptoms prospectively contributing to the development of anxiety symptoms. A study reviewed earlier (Weems et al., 2007) also showed that controlling for age and gender, anxiety control beliefs (ACQ-C; Weems et al., 2003), an index of judgment biases, significantly predicted anxiety symptoms.

Taken together, results from these studies suggest that both an external locus of control *and* a low perception of control over anxiety-related events are important judgment biases associated with anxiety symptoms, with the latter being particularly salient to the phenomenology of clinical anxiety among diagnosed groups. Also evident is that, with few exceptions (e.g., Ginsburg et al., 2004), the literature has utilized clinical samples; thus, little is known about the nature of these relations among nonclinical populations. As mentioned above, it is important to examine whether these relations exist among individuals who have not crossed

into the diagnostic group for at least two reasons. First, diagnostic groups represent a particularly skewed section of the population, thereby limiting the extent to which findings with clinical groups extend to the broader population. Second, to the extent that judgment biases are present and associated with anxiety symptoms in community samples undergoing normative albeit potentially stressful life transitions (e.g., college), results may suggest judgment biases as a potential target for intervention to help cope (i.e., reduce anxiety) during such transitions.

In addition, greater understanding of the role of judgment biases in the context of other risk factors for anxiety (i.e., behavioral inhibition, anxiety sensitivity, and interpretive biases) may help understand their contribution as part of a constellation of risk factors cumulatively or incrementally predicting anxiety symptoms. The ways in which various risks aggregate may have implications for the timing and focus of preventive interventions. Investigating the role of judgment biases as a mediator of the relation between anxiety-related temperamental variables and anxiety symptoms may also inform these efforts.

1.7. Anxiety Sensitivity, Behavioral Inhibition, and Anxiety

As stated earlier, a central question of this investigation is to determine whether anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases cumulatively or incrementally increase risk for anxiety problems; a second goal is to test whether interpretive and judgment biases mediate the relation of anxiety sensitivity and behavioral inhibition to anxiety problems. It is thus pertinent to review each of these constructs, before summarizing and outlining this study's specific hypotheses.

1.7.1. Anxiety sensitivity. Anxiety sensitivity, or the fear of anxiety-related sensations arising from a belief that these sensations have negative social, physical, and psychological consequences, is a risk factor for the development of anxiety disorders (Reiss & McNally, 1985).

An individual with high anxiety sensitivity may interpret a racing heart as an impending heart attack, or stomach discomfort as a sign of a fatal health condition. Anxiety sensitivity has been hypothesized as a risk factor for the development of anxiety disorders, especially panic disorder, by increasing preexisting levels of anxiety. The ensuing increase in anxiety and its symptoms is further interpreted by an individual with high anxiety sensitivity as a sign of danger, which, in turn, generates more anxiety. This positive feedback mechanism results in an escalating response that may culminate in a full-blown panic attack (Reiss, 1991).

Early research (e.g., Brown & Cash, 1990; Schmidt, Lerew, & Jackson, 1999) focused on anxiety sensitivity as a vulnerability factor for panic attacks, in part, because catastrophic misinterpretations of bodily sensations were theoretically posited by Clark (1986) as necessary for the occurrence of a panic attack. Nevertheless, correlational (see Cox, Borger, & Enns, 1999; Rabian, Peterson, Richters, & Jensen, 1993) and longitudinal (Maller & Reiss, 1992) research with both adults and adolescents showed that anxiety sensitivity was also elevated in individuals with non-panic anxiety disorders. For example, Taylor, Koch, and McNally (1992) found that Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986) scores were elevated in all anxiety disordered groups—except simple phobia—in comparison to controls. Thus, despite having been initially conceptualized as a risk factor solely for panic, researchers now agree that the construct is relevant across the anxiety disorder spectrum.

There is a vast literature supporting the relation between anxiety sensitivity and anxiety, both as a category and as a dimension (e.g., Maller & Reiss, 1992; Taylor et al., 1992). In a sample of 313 primarily white patients who presented for anxiety treatment at a university hospital, elevated anxiety sensitivity levels were found among adult patients with *DSM-III-R* (American Psychiatric Association, 1987) anxiety diagnoses relative to healthy controls (Taylor

et al.). Findings remained significant even when controlling for trait anxiety, supporting anxiety sensitivity's unique role in anxiety disorders above and beyond that of trait anxiety. Similarly, a 3-year longitudinal study of 47 college students (Maller & Reiss, 1992) showed that ASI scores obtained at initial assessment predicted occurrence of anxiety disorders and frequency and intensity of panic attacks in the third year. In a nonclinical sample of 1401 cadets undergoing a 5-week military training (Schmidt et al., 1997), ASI scores at the beginning of training predicted development of spontaneous panic attacks and anxiety symptoms at the end of the training. Among nonclinical undergraduate students with no history of panic attacks (Rapee & Medoro, 1994), ASI scores accounted for a significant amount of variance in anxious responding to a voluntary hyperventilation task above and beyond that accounted for by trait anxiety scores. More recently, an 11-year follow-up study by Plehn and Peterson (2002) found that ASI scores predicted panic symptoms as well as self-reported panic attacks (see also Telch, Silverman, & Schmidt, 1996).

The relation between anxiety sensitivity and anxiety has also been found in studies with children. In an outpatient clinical sample of 40 youths (ages 8-17; 24 females), half diagnosed with PD with and without agoraphobia ($n = 18$ and $n = 2$, respectively) and half diagnosed with an anxiety disorder (other than PD), the PD group had significantly higher CASI scores than the anxiety disorder group (Kearney, Albano, Eisen, Allan, & Barlow, 1997). Similarly, a study of adolescent girls ($N = 1,013$; age range = 11.6 to 16.2) classified into three groups—a PD, a panic attacks, and a panic-free group—found that adolescent girls with PD scored significantly higher on the ASI than the other two groups (Hayward et al., 1997). Although anxiety sensitivity scores tended to be particularly high in youths with PD, findings also showed that they were elevated among the other anxiety disordered groups.

A significant correlation ($r = .42$) between the Panic Attack Questionnaire (*PAQ*; Norton, Dorward, & Cox, 1986) and the CASI was also found in a nonclinical sample of adolescents (age range = 14 to 18 years; Lau, Calamari, & Waraczynski, 1996). More recently, CASI scores predicted scores on the PAQ while controlling for the Revised Children's Manifest Anxiety Scale (*RCMAS*; Reynolds & Richmond, 1985) physiological subscale in sample of 114 children and adolescents (age range = 11-18 years; Calamari et al., 2001). In testing incremental validity of the CASI in non-clinical ($n = 72$; mean age = 13.3 years) and clinical ($n = 33$; mean age = 10.6 years) samples of children and adolescents, one study (Silverman et al., 1991) found that the CASI explained an additional 48% and 35% of the variance (for the non-clinical and clinical samples, respectively) on the Fear Survey Schedule for Children- Revised (FSSC-R; Ollendick, 1983) after controlling for the State-Trait Anxiety Inventory for Children (STAIC; Spielberger, 1973) (see also, Chorpita, Albano, & Barlow, 1996; Weems, Hammond-Laurence, Silverman, & Ginsburg, 1998). In a 4-year prospective study of 2,365 nonclinical adolescents (mean age = 15.4 years; $SD = 0.9$ years), anxiety sensitivity, as measured by the ASI, predicted the onset of 4-symptom panic attacks during a 4-year period after controlling for past or concurrent major depression (Hayward, Killen, Kraemer, & Taylor, 2000; see also Weems et al., 2002). Also, CASI scores accounted for a significant, albeit small (4%), proportion of variance in post-task state anxiety after controlling for pre-task state anxiety and fear in a behavioral challenge study of 56 nonclinical children (ages 8-to-11; 34 females; Rabian, Embry, & MacIntyre, 1999).

Taken together, these investigations suggest that, first, there is a significant correlation between anxiety sensitivity and measures of anxiety in nonclinical and clinical samples; second, anxiety sensitivity explains variance above and beyond other measures of anxiety (e.g., trait anxiety) and has a distinctive, reliable, and valid role in anxiety; and third, anxiety sensitivity is

causally implicated in the development of anxiety as indicated by results from longitudinal (Hayward et al., 2000; Schmidt et al., 1997; Weems et al., 2002) as well as challenge studies (e.g., Rabian et al., 1999).

Although anxiety sensitivity is defined as a risk factor for anxiety with a strong biological origin (Reiss & McNally, 1985; Stein et al., 1999; Taylor et al., 2008), how it leads to pathological anxiety is not entirely understood. Whereas it is entirely possible that anxiety sensitivity itself may lead to pathological anxiety in some cases, it is likely that anxiety sensitivity covaries with other anxiety precursors, such as behavioral inhibition, and affects other cognitive processes emerging later in development, namely interpretive and judgment biases. Hence, important questions to answer are whether (a) anxiety sensitivity may contribute to anxiety symptoms cumulatively in conjunction with other risks; (b) anxiety sensitivity may offer incremental prediction to anxiety symptoms in the context of other risks, and (c) whether the relation of anxiety sensitivity to anxiety symptoms is mediated by interpretive and judgment biases. To the extent that individuals biologically predisposed to view anxiety as a feared and highly uncomfortable experience (i.e., high anxiety sensitivity), life experiences that cause anxiety may be viewed negatively and progressively lead to the emergence of interpretive and judgment biases; interpretive and judgment biases, in turn, may lead to significant anxiety symptoms (Beck et al., 1985). The present investigation attempts to shed light on these questions.

1.7.2. Behavioral Inhibition. Behavioral inhibition has been defined as a biologically-based temperamental trait characterized by a general tendency to react cautiously and with restraint to novel stimuli or situations (e.g., persons, objects, or places; Kagan, Reznick, & Snidman, 1987). In contrast, behaviorally uninhibited children are said to have "an affectively

spontaneous approach" to the unfamiliar (Kagan, 2008; p. 163). Kagan (2008) suggests that

although it has not yet been proven, behavioral inhibition, as well as all temperamental traits, is

heritable. Children who are born with high behavioral inhibition share some of the same

behavioral, affective, and physiological characteristics seen in individuals with anxiety disorders.

For example, behaviorally inhibited children show greater avoidance of novel situations, fear and

clinginess to parental figures and higher levels of physiological arousal (Kagan, 2008; Kagan et

al., 1988), some or all of which are seen in children who suffer from anxiety (Weems &

Silverman, 2008).

　　In concordance with this proposition, empirical evidence has shown that children

identified as behaviorally inhibited were significantly more likely to receive an anxiety disorder

diagnosis 3 years later relative to control children (i.e., without behavioral inhibition)

(Biederman et al., 1993). Additionally, relative to children with no behavioral inhibition,

behaviorally inhibited children are more likely to come from a family in which one or both

parents have or had an anxiety disorder (Rosenbaum, Biederman, Hirshfeld, & Bolduc, 1991).

Twin studies have shown high heritability for behavioral inhibition (DiLalla, Kagan, & Reznick,

1994) and its behavioral, affective, and physiological signs have been found to be moderately

stable across childhood (Kagan et al., 1988) and related to later anxious symptoms (Kagan &

Snidman, 2004). The associations between behavioral inhibition and anxiety disorder symptoms

have been replicated extensively by other researchers (see, e.g., Muris, 2006; Muris, Meesters, &

Spinder, 2003). At the same time, only a small proportion of behaviorally inhibited children go

on to develop a full blown anxiety disorder. For example, Biederman et al. (2001) compared 64

children with behavioral inhibition to 152 children without (age range for both groups = 5-6

years) and found that only 17% of behaviorally inhibited children met criteria for social anxiety

disorder, relative to 5% of children without behavioral inhibition (note that the difference between groups was, however, significant). These results suggest that although behavioral inhibition is an important risk factor for the development of anxiety disorders, it does not explain most of the variance in anxiety disorder rates. Thus, the first model posited by the present investigation that incorporates the effects of behavioral inhibition as part of a cumulative risk model may clarify whether its contribution, in the context of other risk factors, is generic. The second model, on the other hand, may be better able to account for when and how behavioral inhibition incrementally increases risk for anxiety. Finally, the third model may clarify whether an indirect path between behavioral inhibition and anxiety symptoms, that is, through interpretive and judgment biases, exists.

Behavioral inhibition has significantly contributed to our understanding of anxiety disorders in children and adults (Kagan, 2008; Kagan et al., 1987). Indeed, several studies (e.g., Muris, Merckelbach, Schmidt, Gadet, & Bogie, 2001; Muris et al., 2003) have found that behavioral inhibition may lead to anxiety and depression symptoms in adolescence. At the same time, there is a dearth of knowledge with respect to how this construct relates to other risk factors for anxiety, such as anxiety sensitivity and interpretive and judgment biases. A model that considers the joint effects of these risk factors may be better able to explain their effects on anxiety symptoms (Rutter, 1979); in addition, as a construct characterized by a general tendency to react cautiously and with restraint to novel stimuli or situations, it is possible that behavioral inhibition leads to biased interpretations and judgments of potentially threatening stimuli—once the appropriate cognitive structures are in place to make those attributions—which can lead to anxiety problems. In other words, young children who are high on behavioral inhibition may be more likely to, later in development, engage in erroneous interpretation of stimuli and make

faulty judgments about their ability to deal with those stimuli, which may lead to problematic anxiety symptoms. The present investigation thus posits an indirect link between behavioral inhibition and anxiety symptoms through interpretive and judgment biases.

1.8. The Present Study

Anxiety sensitivity, behavioral inhibition, and cognitive biases each alone place men and women at significant risk for anxiety problems; their accumulation most likely increases this risk. However, whether the increase is cumulative, or whether each risk incrementally increases risk for anxiety as part of a multivariate process, awaits empirical testing. The present study is also concerned with the purported pathway from anxiety sensitivity and behavioral inhibition to anxiety symptoms. Theorizing and the available literature propose interpretive and judgment biases as potential mediators of the link between anxiety sensitivity and behavioral inhibition, and anxiety symptoms. However, no study to date has tested this proposition. Thus, an investigation is warranted to examine (1) the cumulative impact of anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases on anxiety symptoms, (2) the incremental value added by each of these risks to the prediction of anxiety symptoms and (3) the extent to which interpretive and judgment biases account for the link between anxiety sensitivity and behavioral inhibition, and anxiety symptoms.

A preliminary goal of the present investigation was to corroborate prior findings linking anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases with anxiety symptoms. It was hypothesized that anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases would be associated with increased anxiety symptoms.

The first major goal of the present investigation was to examine the cumulative risk posed by anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases to

anxiety symptoms. In line with cumulative risk models (Rutter, 1979; Sameroff et al., 1993) it was hypothesized that the accumulation of these risk factors would significantly predict anxiety symptoms. The second major goal was to examine the degree to which the risks considered increment each other in the prediction of anxiety symptoms. Based on available research (Biederman et al., 1993; Weems et al., 2007), it was hypothesized that each risk factor would offer a unique contribution to the prediction of anxiety symptoms, thereby lending support to an incremental, multiple risk-factor model where content of risk is vital to the development of anxiety. Thus, it was also hypothesized that the amount of variance in anxiety symptoms accounted for by the multiple risk-factor model would be greater than that of the cumulative risk model.

The third major goal of the present investigation was to examine via structural equation modeling (SEM) direct and indirect pathways linking anxiety sensitivity and behavioral inhibition with anxiety symptoms. It was hypothesized that anxiety sensitivity, behavioral inhibition, and judgment and interpretive biases would directly predict anxiety symptoms. In addition, it was hypothesized that judgment and interpretive biases would be directly predicted by anxiety sensitivity and behavioral inhibition. Thus, as shown in Figure 1, it was expected that interpretive and judgment biases would mediate the pathways linking anxiety sensitivity and behavioral inhibition with anxiety symptoms.

Finally, as an exploratory aim and with the goal of serving to guide for future work, a cluster analysis was conducted to determine whether homogenous groups of participants with specific profiles of risk factors were found within the larger sample. The aim of this person-oriented analysis (Bergman & Magnusson, 1997) is to identify homogenous clusters in which members are similar to each other while distinct from individuals in other clusters (Aldenderfer

& Blashfield, 1984; Clogg, 1995; DiStefano & Kamphaus, 2006). Because these analyses were strictly exploratory, no firm hypotheses were made.

2. Method

2.1 Participants

The initial sample consisted of 892 undergraduate students from introductory psychology classes at a large state university located in the northeastern United States. Twenty-two students completed demographic questions but did not complete any of the remaining questionnaires; thus, they were excluded from the sample. Prior to analyses, all variables of interest were examined for missing values, accuracy, and normality. Missing values of individual items were replaced through linear-trend-at-point substitution, as recommended when missing values occur in fewer than 5% of cases (Tabachnick & Fidell, 2007). After totaling scores, one case had a missing value on the BSI-Depression subscale, and 3 cases had a missing value on the ACQ-Total, ACQ-External, and ACQ-Internal subscales. Because missing values on total scores were so few (i.e., $n = 4$ cases), they were also replaced with the linear-trend-at-point (Tabachnick & Fidell, 2007). All but two measures were well within acceptable levels of skewness and kurtosis (all < │1│). The BMWS and the BSI-mixed anxiety composite were transformed with a square root transformation to improve skewness. Transformations significantly improved normality. Eight cases with ages > 24 were found to be univariate outliers (z scores > 3.29, $p < .001$ two-tailed; Tabachnick & Fidell, 2007); no other univariate or multivariate outliers were found. All eight outliers were excluded. Although age was obviously positively skewed due to the nature of the sample (first year college students), it was not transformed to avoid problems with interpretability of solutions that included age as a variable (Tabachnick & Fidell, 2007).

The final sample consisted of 862 undergraduate students with a mean age of 18.75 years (*SD* = 1.04; age range = 18 – 24). Seventy percent were female (70.5%, *n* = 608; mean age = 18.62 years, *SD* = 0.92; age range = 18 – 23) and 29.0% male (*n* = 250; mean age = 19.07, *SD* = 1.25; age range = 18 -24). Four participants (0.5%) did not report their sex. Eighty-two percent (82.8%) of the sample identified as White American, 7.0% as Asian American, 4.6% as African American, 4.2% as Hispanic/Latino(a), 0.8% as Pacific Islander, and 0.2% as American Indian. Three participants (0.3%) did not report their ethnicity. In light of the very low *n* of some ethnic groups, ethnicity was collapsed into 2 groups (i.e., White American [*n* = 714; 82.8%] and Minorities [*n* = 145; 16.8%]). The overwhelming majority (98.8%) of participants were single, never married. Twenty-nine percent of the sample worked part-time, and only 1.7% worked full time. The remaining participants (68.7%) were full-time students only. For detailed demographic information of the sample, see Table 1.

2.2 Measures

2.2.1 Risk factors.

2.2.1.1 Interpretive biases.

2.2.1.1.1. Positive and Negative Cognitive Error Questionnaire (PNCEQ; Henriques & Leitenberg, 2002). Based on the Negative Cognitive Error Questionnaire (NCEQ; Lefebvre, 1981), the PNCEQ is a 32-item self-report questionnaire that assesses the degree to which adults interpret events in an overly negative and overly positive manner. Each item consists of a vignette describing an event, followed by a thought about that event. Half of the items describe academic situations and half describe social situations. Participants are asked to rate, on a 5-point Likert scale ranging from 1 ("Not at all what I would think") to 5 ("Almost exactly what I would think"), the extent to which the thought presented is similar to a thought they might have if they

were in that situation. The PNCEQ has two subscales, a negative cognitive error subscale

(NCES) and a positive cognitive error subscale (PCES), each consisting of 16 items. A sample

item for the NCES includes "Some new friends asked you to join them for an evening on the

town. You agree to go. The next day you heard that some people did not have that good of time.

You think: 'They probably didn't enjoy themselves because I wasn't good company.'" A sample

item for the PCES includes "A paper you turned in for a history course was selected by the

instructor as an example of how a good paper should be written. You are about to do a

presentation in a business class. You think: 'I'll probably get an excellent grade for this too.'"

Total NCES and PCES scores, respectively, are computed by summing responses to items in

each subscale. McKenna (1987) found Cronbach's alphas of .86 and .83 for the negative

cognitive error and positive cognitive error subscales, respectively, and 11-week test-retest

reliability of .72 (see Henriques & Leitenberg, 2002). The NCES total score is further comprised

of 4 subscales measuring 4 types of interpretive biases: catastrophizing ($\alpha = .72$), personalizing

($\alpha = .62$), overgeneralizing ($\alpha = .75$), and selective abstraction ($\alpha = .62$). In this study, the NCES

total score ($\alpha = .89$) was used for analyses involving hypothesis 1. For the SEM analyses,

however, the NCES subscales were used as indicators of the interpretive biases latent construct.

2.2.1.2 Judgment Biases

2.2.1.2.1 Anxiety Control Questionnaire (ACQ; Rapee et al., 1996). The ACQ is a 30-

item self-report questionnaire that assesses the degree of perceived control over external events

(ACQ-External subscale, 16 items, $\alpha = .82$; e.g., "There is little I can do to change frightening

events") and control over internal emotional reactions (ACQ-Internal subscale, 14 items, $\alpha = .87$;

e.g., "I can usually relax when I want") in adults. Participants rate on a 6-point Likert scale (0 =

strongly disagree; 5 = strongly agree) the degree to which they agree with each statement

presented, and total scores are computed by adding all items (after reverse scoring appropriate items). In order to facilitate interpretation of findings and ensure that all estimates would have a positive sign, in this study, higher scores on the ACQ indicate *lower* perceived control. Rapee et al. reported Cronbach's alphas in a clinical sample of .87, .83, and .80 for the total scale, external events, and internal reactions subscales, respectively. Similar internal consistency estimates were obtained with a nonclinical undergraduate sample (Rapee et al.; Zebb & Moore, 1999). One-week and one-month test-retest reliability estimates were .88 and .82, respectively (Rapee et al.). The ACQ has also shown good discriminant and convergent validity as well as sensitivity to change after treatment (Rapee et al.). The ACQ Total Score ($\alpha = .91$) was used to test hypothesis 1 and 2. For SEM analyses, the two subscales served as indicators for the judgment biases latent construct.

2.2.1.3 Anxiety Sensitivity

2.2.1.3. 1 Anxiety Sensitivity Index-3 (ASI-3; Taylor et al., 2007). The ASI-3 is an 18-item self-report questionnaire that assesses fear of anxiety related sensations in adults. Based on the well-established ASI (Reiss et al., 1986) and ASI-R (Taylor & Cox, 1998), the ASI-3 asks participants to respond on a 5-point Likert scale ranging from 0 ("very little") to 4 ("very much") the extent to which they agree with statements assessing their fears about normal anxiety reactions, such as inability to concentrate, blushing, sweating, and increased heart rate. Scores range from 0 to 72 and are calculated by adding all items. A multi-sample ($N = 2,361$) factor analysis by Taylor et al. suggested a Total Score and three subscales for the ASI-3, namely: (1) physical concerns (6 items, $\alpha = .89$; e.g., "When my stomach is upset, I worry that I might be seriously ill"), (2) cognitive concerns (6 items, $\alpha = .88$; "When my thoughts seem to speed up, I worry that I might be going crazy"), and (3) social concerns (6 items, $\alpha = .80$; e.g., "I worry that

other people may notice my anxiety"). Taylor et al. reported good internal consistency for the ASI-3 subscales across six samples from different countries (alphas' range = .73-.91). Evidence for convergent validity with, and improved performance over, previous anxiety sensitivity measures was also found (Taylor et al.). Although test-retest reliability is not currently available, it is likely acceptable based on the measure's strong grounding on the ASI (Reiss et al.) and ASI-R (see Deacon, Abramowitz, Woods, & Tolin, 2003; Taylor & Cox, 1998). The ASI-3 Total Score ($\alpha = .91$) was used in this study to test hypothesis 1 and 2. For the SEM analyses, however, the three subscales served as indicators of the anxiety sensitivity latent construct.

2.2.1.4 Behavioral Inhibition

2.2.1.4.1 Adult Measure of Behavioral Inhibition (AMBI; Gladstone & Parker, 2005). The AMBI is a 16-item self-report measure of behavioral reticence, wariness, and fearfulness in response to unfamiliar stimuli (i.e., behavioral inhibition to the unfamiliar; Garcia-Coll, Kagan, & Reznick, 1984). Respondents are asked to indicate on a Likert scale ranging from 0 ("no/hardly ever") to 2 ("yes/most of the time") the degree to which they experience a variety of signs of behavioral inhibition. Total scores range from 0 to 48 and are computed by summing responses to each item (items negative for behavioral inhibition are reverse scored prior to totaling scores). The AMBI ($\alpha = .79$) yields four subscales: (1) non-approach (3 items, $\alpha = .73$; e.g., "do you tend to introduce yourself to new people?"), (2) fearful inhibition (7 items, $\alpha = .75$; e.g., "do you tend to observe strangers from a distance first, before being able to mix in?"), (3) risk avoidance (3 items, $\alpha = .21$; e.g., "If physically able, would you enjoy adventure holidays with some element of risk?"), and (4) low sociability (3 items, $\alpha = .58$; e.g., "Do you prefer your own company to the company of others?"). Gladstone and Parker (2005) reported a Cronbach's alpha of .87 for the AMBI total score; subscale alphas ranged from .52 (risk avoidance) to .86

(fearful inhibition). The AMBI showed adequate construct and discriminant validity (Gladstone

& Parker, 2005), as well as a 20-week test-retest reliability coefficient of .69. The AMBI Total

Score was used in this study to test hypothesis 1 and 2. For the SEM analyses, however, the four

subscales served as indicators of the behavioral inhibition latent construct.

2.2.2 Anxiety outcomes. Four measures were used to assess anxiety-related symptoms:

the Brief Symptom Inventory (BSI; Derogatis & Spencer, 1982), the State-Trait Anxiety

Inventory-Trait Version (STAI-T; Spielberger, Gorsuch, & Lushene, 1970), the Brief Measure of

Worry Severity (BMWS; Gladstone, Parker, Mitchell, Malhi, Wilhelm, & Austin, 2005) and the

Negative Affectivity scale of Positive and Negative Affectivity Scale (PANAS-NA; Watson,

Clark, & Tellegen, 1988). The BSI was used as a measure of *Diagnostic and Statistical Manual*

*of Mental Disorder*s (4th edition, text revision [DSM-IV-TR]; American Psychological

Association, 2000) anxiety symptoms, while the BMWS, the PANAS-NA, and STAI-T were

used as broader measures of chronic, cross-situational anxiety and anxiety-related distress.

2.2.2.1 Clinical anxiety.

2.2.2.1.1. Brief Symptom Inventory (BSI; Derogatis & Spencer, 1982). The BSI, a shorter

version of the Symptom Checklist-90-R (SCL-90-R; Derogatis, 1983), is a 53-item self-report

measure designed to assess presence of psychological symptoms in clinical and community

samples. Respondents are asked to rate on a 5-point Likert scale (0 = "not at all;" 4 =

"extremely") the extent to which a particular symptom has caused distress "during the past 7

days including today." The BSI yields scores for 9 symptom dimensions, namely: somatization,

obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety,

paranoid ideation, and psychoticism. For the present study, the anxiety (6 items, α = .84; e.g.,

"nervousness or shakiness inside," "suddenly scared for no reason"), somatization (7 items, α =

.84; e.g., "pains in heart or chest," "hot or cold spells"), obsessive-compulsive (6 items, α = .84; e.g., "having to check and double-check what you do," "feeling blocked in getting things done"), and phobic anxiety (5 items, α = .76; e.g., "feeling afraid in open spaces or on the streets," having to avoid certain things, places, or activities because they frighten you") symptom dimensions were used to assess a broad array of anxiety symptoms. A "mixed anxiety" composite score was computed by averaging scores across these 4 dimensions (α = .94). Derogatis (1993) reported very good internal consistency estimates (α ranging from .77 [phobic anxiety] to .83[obsessive-compulsive]) as well as good two-week test-retest reliability (ranging from .68 [somatization] to .91 [phobic anxiety]) for the BSI subscales used in this study. Adequate convergent validity with related MMPI scales was also reported (Derogatis, 1993).

2.2.2.2 Trait anxiety.

2.2.2.2.1. State-Trait Anxiety Inventory-Trait Version (STAI-T; Spielberger et al., 1970). The STAI-T is a 20-item self-report measure designed to assess chronic, cross-situational anxiety. Respondents are asked to rate on a 4-point Likert scale—ranging from 1 ("almost never") to 4 ("almost always")—how frequently they experience a variety of anxiety symptoms. Sample items include "I worry too much over something that really doesn't matter" and "I feel nervous and restless." Total scores range from 20 to 80 and are computed by adding all items. Several studies report that the STAI-T has good internal consistency (Cronbach's alpha > .87; Barnes, Harp, & Jung, 2002; Hishinuma et al., 2000; α = .92 in this study) and good test-retest reliability across a 3-month period (r = .73 and .77 for males and females, respectively; Spielberger et al.). In addition, The STAI-T has evidenced good convergent validity with other measures of trait anxiety (Spielberger, 1983) as well as good construct validity (Metzger, 1976).

2.2.2.3. Negative affectivity.

2.2.2.3.1 Positive and Negative Affectivity Scale (PANAS; Watson et al., 1988). The

PANAS is a widely used 20-item self-report measure of positive and negative affect.

Respondents are asked to rate on a Likert scale ranging from 1 ("very slightly or not at all") to 5

("very much") the extent to which they have experienced a particular emotion within a specific

time period. Several time frames have been used with the PANAS; in this study, "during the past

few weeks" was the time frame used. The PANAS yields a positive affectivity scale (PANAS-

PA, 10 items, α = .86; e.g., "determined," "interested"), measuring pleasurable engagement with

the environment, and a negative affectivity scale (PANAS-NA, 10 items, α = .87; e.g.,

"distressed," "irritable"), measuring overall subjective distress and negative engagement. High

scores on each scale indicate high PA and high NA, respectively. Total scores for each scale

range from 10 to 50 and are obtained by summing responses to items on each scale. Watson et al.

reported 8-week test-retest reliability estimates of .68 and .71 for the PA and NA scales,

respectively. Crawford and Henry (2004) reported adequate construct validity and internal

consistencies of .89 and .85 for the PA and NA scales, respectively, in a large ($N = 1{,}003$)

nonclinical sample. The PANAS-NA scale was used in this study.

2.2.2.4. Worry.

2.2.2.4.1. Brief Measure of Worry Severity (BMWS; Gladstone et al., 2005). The BMWS

is an 8-item self-report measure of worry severity and dysfunction in which respondents are

asked to indicate on Likert scale ranging from 0 ("not true at all") to 3 ("definitely true") the

extent to which each item is accurate in describing their usual experience of worrying. Sample

items include "when I think I should be finished worrying about something, I find myself

worrying about the same thing, over and over" and "when I worry, it interferes with my ability to

make decisions or solve problems." Total scores range from 0 – 24 and are computed by summing responses to each item (α = .89). Gladstone et al. reported an internal consistency estimate of .92 for the BMWS, and evidence of good construct and clinical discriminant validity.

2.2.3 Cumulative risk computation.

In order to test the cumulative risk hypothesis, each of the fours risk variables was converted into a dichotomous coding (0 = absence of risk; 1 = presence of risk), and an overall cumulative risk variable was computed by summing the total number of risk factors for each participant. The resulting cumulative risk variable thus ranged from 0 (i.e., no risk) to 4 (i.e., risk criteria met across the 4 risk variables). Because all 4 variables were continuous, the risk criterion was empirical such that participants scoring one standard deviation above the mean of the sample distribution for a specific risk variable were placed in the risk category (Corapci, 2008).

2.3. Procedure

Participants were recruited through the subject pool of the department of psychology and received course credit for their participation. All questionnaires were completed online. The link containing all questionnaires was listed in the department of psychology subject pool online system. Before completing the battery of questionnaires, interested participants were provided with an informed consent form and informed that they could refrain from participation or leave questions unanswered at any point during the study without adverse consequences.

3. Results

3.1. Preliminary Analyses

Descriptive statistics were computed for each measure at the time of assessment (see Table 2). We used univariate analysis of variance (ANOVA) to test gender and ethnic group differences, respectively, in these scores.

3.1.1. Description of behavioral inhibition. Behavioral inhibition (AMBI-Total) scores were significantly higher for males ($M = 14.74$, $SD = 4.96$) than females ($M = 13.54$, $SD = 5.21$), $F(1, 851) = 5.71$, $p < .05$. No significant ethnic differences was found for behavioral inhibition ($p = .34$). The interaction between gender and ethnic group was not significant for ($p > .90$).

3.1.2. Description of anxiety sensitivity. No significant gender differences were found in anxiety sensitivity (ASI-3-Total) scores ($p = .17$). However, significant ethnic differences were found in anxiety sensitivity scores, $F(1, 851) = 4.70$, $p < .05$; minorities ($M = 17.49$, $SD = 13.39$) scored significantly higher than whites ($M = 15.29$, $SD = 11.32$). The interaction between gender and ethnic group was not significant ($p > .50$).

3.1.3. Description of judgment biases. ACQ Total scores were significantly higher for females ($M = 55.33$, $SD = 19.56$) than males ($M = 50.49$, $SD = 18.73$), $F(1, 851) = 4.64$, $p < .05$. Note that, in this study, higher scores on the ACQ represent judgments of less control. Similarly, females scored significantly higher on the ACQ-Internal reactions subscale, $F(1, 851) = 4.51$, $p < .05$ (Females $M = 26.85$, $SD = 11.13$; Males $M = 24.24$, $SD = 10.52$). No gender differences were found for the ACQ-External Events subscale. Thus, females judged themselves to have less total control, as well as less control over internal reactions, than males. Significant ethnic differences were found for the ACQ-External subscale, $F(1, 851) = 8.93$, $p < .01$; minorities reported significantly less control over external events ($M = 29.98$, $SD = 10.36$) than White

Americans ($M = 27.37$, $SD = 10.06$). No significant ethnic differences were found for the ACQ-Total ($p = .06$) or ACQ-Internal events ($p = .60$). The interactions between gender and ethnic group were not significant (all $ps > .50$).

3.1.4. Description of interpretive biases. No significant gender or ethnic differences were found for the NCEQ total score (all $ps > .30$). However, significant differences were found in two of the four NCEQ subscales: Personalizing and Selective Abstraction. Males ($M = 8.60$, $SD = 2.84$; $F [1, 851] = 7.10$, $p < .01$) and minorities ($M = 8.68$, $SD = 2.82$; $F [1, 851] = 6.63$, $p < .01$) reported a significantly higher tendency to personalize than females ($M = 7.96$, $SD = 2.71$) and White Americans ($M = 8.05$, $SD = 2.74$), respectively. Minorities ($M = 7.53$, $SD = 2.74$) also reported a significantly higher tendency to selectively abstract negative information than White Americans ($M = 7.08$, $SD = 2.54$), $F (1, 851) = 5.20$, $p < .05$. The interactions between gender and ethnic group were not significant (all $ps > .20$).

3.1.5. Description of anxiety problems. Significant gender differences were found in 2 of the 4 anxiety measures; females scored significantly higher than males on the PANAS-NA (Males $M = 18.79$, $SD = 6.17$; Females $M = 20.61$, $SD = 6.94$; $F [1, 854] = 5.45$, $p < .05$), and BMWS (Males $M = 2.72$, $SD = 3.35$; Females $M = 3.72$, $SD = 4.17$; $F [1, 850] = 6.20$, $p < .01$). No significant gender differences were found for the STAI-T ($p = .26$) or BSI-mixed anxiety composite ($p = .13$). No significant ethnic differences were found for any of the anxiety measures (all $ps > .24$). The interactions between gender and ethnic group were not significant for any of the anxiety measures (all $ps > .42$).

3.2. Individual Risk Variables and Anxiety Problems

A preliminary aim of this investigation was to test whether the anxiety sensitivity, behavioral inhibition, and the cognitive risk variables (interpretive and judgment biases) were

associated with anxiety problems. Bivariate Pearson product-moment correlations were computed between the risk variables and between the risk variables and anxiety problems (see Table 3).

The correlation between anxiety sensitivity and behavioral inhibition was significant and in the expected direction (r [862] = .36, $p < .001$), as was the correlation between interpretive and judgment biases (r [862] = .54, $p < .001$). The intercorrelations between the four types of interpretive biases (catastrophizing, personalizing, overgeneralizing, and selective abstraction) were also significant (rs [862] from .56 to .71, $p < .001$). The two types of judgment biases (ACQ-External and ACQ-Internal) were also significantly intercorrelated, r (862) = .68, $p < .001$. Finally, anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases were all significantly intercorrelated in the expected direction (rs [862] from .36 to .56, $p < .001$).

Thus, results indicated that (a) high behavioral inhibition was associated with high anxiety sensitivity; (b) an increased tendency to engage in interpretive biases was associated with an increased tendency to engage in judgment biases; and (c) elevations in anxiety sensitivity and behavioral inhibition were associated with elevations in interpretive and judgment biases.

Next, the risk variables were correlated with anxiety problems. All risk variables were significantly correlated with anxiety measures. As shown in Table 3, higher behavioral inhibition and higher anxiety sensitivity were significantly correlated with higher anxiety symptoms as measured by the BSI-mixed anxiety composite, BMWS, PANAS-NA, and STAI-T (rs [862] from .31 to .63, $p < .001$). Similarly, interpretive biases were significantly correlated with anxiety symptoms (rs [862] from .48 to .59, $p < .001$). Finally, judgment biases as measured by the ACQ-Total were also significantly correlated with anxiety symptoms (rs [862] from 56 to

.69, $p < .001$). Taken together then, higher anxiety symptoms were associated with an inhibited temperament, high anxiety sensitivity, and an increased tendency to engage in interpretive and judgment biases (see Table 3).

3.3. Multiple Risk Factors and Anxiety Problems

As stated above, anxiety research consistently suggests that anxiety sensitivity and a behaviorally inhibited temperament may lead to anxiety problems; similarly, research suggests that certain cognitive biases may place individuals at risk for anxiety. However, little is known about how these constructs covary to predict anxiety. In order to shed light on this issue, risk variables were entered into a regression equation to test the incremental value added by each of these risks to the prediction of anxiety symptoms. In these regression models, the significant contribution of each risk variable (e.g., anxiety sensitivity) after controlling for the other (e.g., behavioral inhibition) reflects the unique contribution of that variable to the prediction of anxiety problems. Entering risk variables as continuous variables also allows examination of the role of variability, or content, of each measure to the prediction of anxiety.

Two hierarchical regression models were used to specifically test this question; in the first model, the statistical prediction of each risk variable was estimated in the following hierarchical regression equation: ethnicity and gender, risk factor of interest, interactions.[3] This model estimated the amount of variance accounted for by each factor without attention to the correlation with the other risk factors. In the second model, the *unique* contribution of each risk variable and interactions with gender and ethnicity was estimated in the following hierarchical regression equation: ethnicity and gender, remaining risk factors, the risk factor of interest, interactions. This model estimated the unique contribution of each risk factor after taking into

account the covariation with the other risks. Separate models were tested for the BSI-mixed anxiety composite, BMWS, PANAS-NA, and STAI-T.

As shown in Table 4, the 4 risk variables predicted 65% of the variance in PANAS-NA scores, 56% of the variance in STAI-T scores, 52% of the variance in BSI-mixed anxiety composite scores, and 40% of the variance in BMWS scores. Anxiety sensitivity predicted 24 – 41% of the variance in anxiety symptoms (across BSI-composite, BMWS, PANAS-NA, and STAI-T; "initial" regression model), and 1 – 10% of this variance was unique to anxiety sensitivity after all other risk variables were controlled for ("unique" regression model; see Table 4). Behavioral inhibition predicted 12-21% of the variance in anxiety symptoms ("initial" regression model), and 1 – 2% of this variance was unique to behavioral inhibition after all other risk variables were controlled for ("unique" regression model). However, behavioral inhibition did not offer *unique* prediction to worry and negative affectivity scores once the other variables were controlled for.

Interpretive biases predicted 24 – 35% of the variance in anxiety symptoms ("initial" regression model), and 1 – 3% of this variance was unique to interpretive biases after all other risk variables were controlled for ("unique" regression model). Finally, judgment biases predicted 30 – 47% of the variance in anxiety symptoms ("initial" regression model), and 5 – 12% of this variance was unique to judgment biases after all other risk variables were controlled for ("unique" regression model) (all ps < .001).

3.4. Cumulative Risk and Anxiety Problems

As a first step, the prevalence of each dichotomous risk variable was computed (see Method for a detailed description of the dichotomization process) for the entire sample. Table 5 shows the percentage of participants meeting the risk criterion for each of the risk variables. The

total number of risk factors was also computed. On average, participants had 0.62 risk factors

(range = 0 – 4; SD = 0.99). Sixty-four percent (63.6%) of the sample did not meet risk criteria;

twenty percent of the sample had 1 risk factor, 14.5% had 2 – 3 risk factors, and 2% had 4 risk

factors. Regarding individual risk factors, 16.8% met risk criteria for anxiety sensitivity, 12.6%

for behavioral inhibition, 15.5% for judgment biases, and 17.1% for interpretive biases. No

significant gender differences were found in number of risks (Males $M = 0.63$, $SD = 1.00$;

Females $M = 0.62$, $SD = 0.98$). No ethnic differences were found either (Minorities $M = 0.66$, SD

$= 1.06$; Whites $M = 0.61$, $SD = 0.97$) (see Table 4).

Anxiety sensitivity risk was significantly correlated with behavioral inhibition risk, r

$(862) = .16$, $p < .001$, judgment biases risk, $r (862) = .39$, $p < .001$, interpretive biases risk, r

$(862) = .37$, $p < .001$ and total risk, $r (862) = .72$, $p < .001$. Thus, meeting the risk criteria for one

of the dichotomous-risk variables was associated with increased risk on the other risk variables.

Each of the dichotomous-risk variables, as well as the total cumulative-risk variables, were

significantly correlated with all anxiety outcomes, with rs (862) ranging from .23 to .57, $p <$

$.001$. Thus, more risk factors were associated with higher anxiety symptoms.

In order to test the statistical prediction of anxiety problems from each dichotomous-risk

variable, two regression models were used (see Table 6). In the first model, the statistical

prediction of each dichotomous-risk variable and interactions with gender and ethnicity was

estimated in the following hierarchical regression equation: ethnicity and gender, dichotomous-

risk variable of interest, interactions. This model estimated the amount of variance accounted for

by each dichotomous-risk variable without attention to the covariation with the other

dichotomous-risk variables. In the second model, the *unique* contribution of each dichotomous-

risk variable and interactions with gender and ethnicity was estimated in the following

hierarchical regression equation: ethnicity and gender, the remaining dichotomous-risk variables, the dichotomous-risk variable of interest, interactions. This model estimated the *unique* contribution of each dichotomous-risk variable after taking into account the correlation with the other dichotomous-risk variables. Separate models were tested the BSI-mixed anxiety composite, BMWS, PANAS-NA, and STAI-T.

As shown in Table 6, the dichotomous-risk variables predicted 34% of the variance in BSI-mixed anxiety composite scores, 33% of the variance in STAI-T scores, 27% of the variance in PANAS-NA scores, and 24% of the variance in BMWS scores (ps < .001). Anxiety sensitivity predicted 11 – 22% of the variance in anxiety symptoms (across BSI-mixed anxiety composite, BMWS, PANAS-NA, and STAI-T; "initial" regression model), and 2 – 6% of this variance was unique to anxiety sensitivity after all other dichotomous risk variables were controlled for ("unique" regression model). Behavioral inhibition predicted 6 – 10% of the variance in anxiety symptoms ("initial" regression model), and 1 – 8% of this variance was unique to behavioral inhibition after all other dichotomous risk variables were controlled for ("unique" regression model). Interpretive biases predicted 13 – 20% of the variance in anxiety symptoms ("initial" regression model), and 2 – 4% of this variance was unique to interpretive biases after all other dichotomous risk variables were controlled for ("unique" regression model). Finally, judgment biases predicted 14 – 21% of the variance in anxiety symptoms ("initial" regression model), and 4 – 6% of this variance was unique to judgment biases after all other dichotomous risk variables were controlled for ("unique" regression model) (all ps < .001).

The predictive ability of the total number of risk factors was also estimated. The total cumulative-risk variable predicted 33% of the variance in STAI-T scores, 33% of the variance in

BSI-mixed anxiety composite scores, 26% of the variance in PANAS-NA scores, and 24% of the variance in BMWS scores (ps < .001).

Finally, we wanted to examine whether the variance predicted by each continuous risk variable was due to the total number of risk factors present. Thus, the predictive ability of each continuous risk variable, after controlling for the total number of risk factors, was estimated. To the extent that a continuous risk variable predicts anxiety problems above and beyond the total number of risk factors, it would serve as evidence that the content of risk, rather than the sheer number of risk factors, is crucial to anxiety outcomes. A hierarchical regression equation model with the following terms was used: ethnicity and gender, total cumulative risk, continuous risk variable of interest. Four separate models were tested, one for each continuous risk variable (anxiety sensitivity, behavioral inhibition, judgment biases, and interpretive biases). Anxiety sensitivity as a continuous risk variable *uniquely* predicted 3 – 12% of the variance in anxiety outcomes (across BSI-mixed anxiety composite, BMWS, PANAS-NA, and STAI-T) after controlling for the total number of risk factors. Similarly, behavioral inhibition, judgment biases, and interpretive biases as continuous variables uniquely predicted 1 – 3%, 9 – 18%, and 3 – 7% of the variance, respectively, in anxiety outcomes (across all anxiety outcomes) after controlling for the total number of risk factors (ps < .001).

Thus, anxiety symptoms were not solely explained by total cumulative risk; each continuous risk variable significantly predicted anxiety (across BSI-mixed anxiety composite, BMWS, PANAS-NA, and STAI-T) even after the total number of risk factors was statistically controlled, thereby lending support to the hypothesis that the content of risk is a crucial to the prediction of anxiety outcomes.

3.5. Structural Equation Modeling of Direct and Indirect Pathways Linking Anxiety Sensitivity, Behavioral Inhibition, and Interpretive and Judgment Biases, to Anxiety Symptoms

The third major goal of the present investigation was to examine via structural equation modeling (SEM) direct and indirect pathways linking anxiety sensitivity, behavioral inhibition, and cognitive risk (interpretive and judgment biases) with anxiety symptoms. In Figure 1, ovals represent latent variables, and rectangles represent measured variables. Absence of a line connecting variables implies no hypothesized direct effect. Lines with an arrow at both ends indicate a covariance between variables with no hypothesized direction of effect. In general, we tested a model positing that the effects of behavioral inhibition and anxiety sensitivity risk on anxiety outcomes are mediated by cognitive risk. Specifically, we hypothesized that behavioral inhibition and anxiety sensitivity risk influenced anxiety outcomes both directly and indirectly. The indirect effect operated through interpretive and judgment biases, whereby increased behavioral inhibition and anxiety sensitivity risk were associated with increased interpretive and judgment biases. The model also hypothesized that interpretive and judgment biases influenced anxiety outcomes directly. The hypothesized model was tested through structural equation modeling (SEM) performed with LISREL 8.80 software (Jöreskog & Sörbom, 2007).

Table 3 shows the intercorrelations among all variables used in the SEM analyses. Indicators of each construct were significantly correlated ($p < .001$). SEM analyses were conducted in 3 steps. In the first step, the full mediational model was tested, which included all direct and indirect pathways linking the 5 constructs of interest. In the second step, we estimated the total unmediated effect of anxiety sensitivity and behavioral inhibition on anxiety. By constraining to zero the indirect paths linking each mediator—interpretive biases and judgment

biases—to anxiety, the total effects of anxiety sensitivity and behavioral inhibition on anxiety were forced to operate through the direct path. Chi-square difference tests were then conducted between the full mediational model and the total effects model to assess the benefit of adding the indirect paths. A significant increase in model chi-square and substantial increase in the direct effects of anxiety sensitivity and behavioral inhibition on anxiety would provide evidence of indirect pathways from these constructs to anxiety as hypothesized in the full mediational model (P. Molenaar, personal communication, March 3, 2010; R. Nix, personal communication, February 23, 2010).

In the third step, the full mediational was tested simultaneously for males and females to evaluate whether the hypothesized links in the model were moderated by sex. After estimating paths for the two groups simultaneously, the model was then fitted with the constraint that the estimates for the two groups were identical. If sex moderated the relations hypothesized in the full mediational model, the chi-square value for the model with the equality constraint would be significantly larger than the chi-square of the previous model without constraints.

Figure 2 shows the latent path coefficients for the full mediational model. Factor loadings for the full mediational model are shown in the Appendix. Higher anxiety sensitivity and higher behavioral inhibition both predicted higher interpretive biases, higher judgment biases, and higher anxiety symptoms. Also, higher interpretive and judgment biases predicted higher anxiety symptoms. The full mediational model indicated excellent fit (P. Molenaar, personal communication, March 3, 2010), χ^2 (109) = 710.26, $p < .01$ (Standardized RMR = .049, CFI = .97, NNFI = .96).

The effects of anxiety sensitivity and behavioral inhibition on anxiety symptoms were partially mediated by the intervening effects of interpretive and judgment biases. When the

mediating paths from these variables to anxiety symptoms were constrained to zero in Step 2, the model chi-square increased significantly, $\Delta\chi^2(4) = 512.94$, $p < .01$. The path coefficient linking anxiety sensitivity and anxiety symptoms was .25 (t [109] = 5.88, $p < .01$) in the full mediational model including the indirect effects. This value increased to .31 (t [113] = 6.61, $p < .01$) when all indirect effects were constrained to zero. Similarly, the path coefficient linking behavioral inhibition and anxiety symptoms was .10 (t [109] = 3.11, $p < .01$) in the full mediational model, but this value increased to .13 (t [113] = 3.22, $p < .01$) when indirect effects were constrained to zero (see Figure 3). Taken together, the findings from Step 2 suggest that not allowing interpretive and judgment biases to mediate the link between anxiety sensitivity and behavioral inhibition, on the one hand, and anxiety symptoms, on the other hand, resulted in a significantly worse model. Indeed, together interpretive and judgment biases accounted for 19% (i.e., 1 - $\left| .25/.31 \right|$ = 0.19) of the total effect of anxiety sensitivity on anxiety symptoms, and 23% (i.e., 1 - $\left| .10/.13 \right|$ = 0.23) of the total effect of behavioral inhibition on anxiety symptoms—results in support of partial mediation (P. Molenaar, personal communication, March 3, 2010).

In the third and final step, the full mediational model was tested for males and females simultaneously in an SEM multi-group format, allowing the *pattern* of free and fixed estimates to be the same across groups. Then, the model was fitted with the constraint that the pattern be the same *and* that free estimates be equal across both groups (i.e., invariance). Results showed that the relations between anxiety sensitivity, behavioral inhibition, interpretive biases, judgment biases, and anxiety problems were not moderated by sex. There was not a statistically significant difference between models with and without equality constraints, $\Delta\chi^2 (44) = 67.76$, $p = ns$. Thus, we found no support for sex moderation of the relations between latent constructs.

3.6. Patterns of Risk and Relations to Anxiety Outcomes

With the goal of serving to guide future work, we wanted to explore whether homogenous groups of participants with specific profiles of risk factors were found within the larger sample, and to examine the extent to which these groups were similar or differed in anxiety outcomes. Thus, we conducted a cluster analyses on participants who met risk criteria on at least one of the anxiety outcomes (i.e., above the sample mean for total number of risk factors; see, e.g., Deater-Deckard et al., 1998, Sameroff et al., 1993). Following recommendations by DiStefano and Kamphaus (2006) and Davis, DiStefano, and Schutz (2008) we conducted a cluster analysis that combined Ward's (1963) hierarchical clustering method and K-means algorithms, in order to overcome the limitations inherent in using one of these methods alone (DiStefano & Kamphaus, 2006). We repeated the same procedure on random 50% and 60% subsamples from the total sample to determine the stability of the cluster solution. To examine the external validity of the cluster solution found, clusters were compared across all anxiety outcomes using ANOVA. When an omnibus ANOVA showed significant differences between clusters, Tukey's post-hoc tests were used to identify between-cluster differences and similarities.

Hierarchical clustering (Ward method) suggested that there might be 3 or 4 clusters in the data. Based on these preliminary results, subsequent k-means clustering was conducted in order to test the stability of three- and four-cluster solutions. Because interpretation of clusters based on measures with different scales is difficult, all measures were standardized (z-scores) to facilitate understanding of the magnitude of results.

Based on the four risk variables used for these analyses (anxiety sensitivity, behavioral inhibition, interpretive biases, and cognitive biases), results from our cluster analysis yielded

four clusters with samples sizes large enough for statistical analyses (see Table 7). The cluster

solution was reliably reproduced on random 50% and 60% subsamples from the total sample.

The full sample and each of the random subsamples were compared in terms of the magnitude

(z-score) of each risk factor within clusters. No significant differences were found. The pattern of

risk for each cluster was also plotted for both random subsamples. Visual inspection of these

plots confirmed a similar pattern to that of the full sample (see Figure 4). Thus, the solution was

considered stable.

3.6.1. Cluster 1. Individuals in this group ($n = 73$) were primarily characterized by high

levels of behavioral inhibition and average levels of anxiety sensitivity, interpretive biases, and

judgment biases relative to other groups (see Figure 4). Thus, the primary characteristic of

Cluster 1 might be interpreted as higher-than-average fear of novel stimuli and a tendency to

respond with restraint when confronted with these events.

3.6.2. Cluster 2. This group ($n = 105$) was comprised of individuals high on anxiety

sensitivity, with moderate levels of interpretive and judgment biases, and lower ratings of

behavioral inhibition. Thus, these individuals were not particularly apprehensive or withdrawn in

novel situations, but did have strong beliefs regarding the negative consequences of anxiety-

related sensations. Not surprisingly, their reports of the degree to which they feel a lack control

of internal sensations and external events were elevated. In sum, this group had intense levels of

anxiety sensitivity and moderate cognitive biases.

3.6.3. Cluster 3. Individuals in this group ($n = 63$) scored high across the four risk factors

assessed. Indeed, this group had the highest scores on three of the four risk variables (i.e.,

anxiety sensitivity, interpretive biases, and judgment biases; see Table 7). Thus, compared to

individuals in other clusters, those in cluster 3 interpreted events in a significantly biased manner

(e.g., catastrophize events, personalize negative outcomes), were afraid of anxiety-related sensations, perceived low control, and reacted with apprehension to novel stimuli.

3.6.4. Cluster 4. Those in cluster 4 ($n = 73$) reported moderate levels of interpretive and judgment biases and comparatively low levels of behavioral inhibition and anxiety sensitivity. Indeed, this group had the lowest scores for anxiety sensitivity. Thus, this group was primarily characterized by purely cognitive biases that increase risk for anxiety.

Cluster membership was unrelated to gender, χ^2 (N = 3) = 6.02, $p = .11$, and ethnicity χ^2 (N = 3) = 2.67, $p = .45$. Finally, we tested whether cluster membership was related to differences in anxiety outcomes. Group differences would suggest that particular patterns of risk factors may be differentially related to anxiety outcomes, while group similarities would suggest that different constellations of risk factors are related to similar anxiety outcomes—a principle known as equifinality (Achenbach, 1982). As shown in Table 7, significant group differences emerged for the four anxiety outcomes. Not surprisingly, individuals in group 3 reported higher BSI-mixed anxiety, worry, negative affectivity, and trait anxiety symptoms than individuals in the remaining three groups.

Individuals in group 2 also reported higher BSI-mixed anxiety symptoms than those in groups 1 and 4, and higher negative affectivity and worry symptoms than those in group 1. However, groups 1 and 4 did not differ in their levels of worry, BSI-mixed anxiety, trait anxiety, or negative affectivity. The lack of significant differences in each of the four anxiety outcomes, despite significant differences in each of the four risks for these two groups provides evidence of equifinality. It is thus possible that the risk posed by significantly higher levels of behavioral inhibition in group 1 is somehow offset by the significantly higher cognitive risks present in group 4 (see Figure 4), thereby leading both groups to comparable levels of anxiety problems.

4. Discussion

Anxiety problems in late adolescence and early adulthood are predicted by a number of risk factors, including biologically-based anxiety precursors, such as anxiety sensitivity (Schmidt et al., 1997; Weems et al., 2002, 2007) and behavioral inhibition (Muris et al., 2003), and cognitive risk factors, such as interpretive and judgment biases (Leung & Poon, 2001; Rapee et al., 1996; Weems et al., 2007). Less is known, however, about how these risk factors covary to predict anxiety problems and the nature of the direct and indirect pathways linking these constructs. The present investigation attempted to shed light on these issues in a large sample of emerging adults.

4.1. Individual Risk Variables and Anxiety Problems

We first sought to corroborate prior findings linking anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases with anxiety outcomes. We hypothesized that higher scores across these four constructs would be associated with increased anxiety problems. Consistent with our first hypothesis, results showed that all 4 risk factors were interrelated, and that higher anxiety problems were associated with higher anxiety sensitivity, a behaviorally inhibited temperament, and a higher tendency to engage in interpretive and judgment biases. These results are also consistent with the literature linking each of these constructs with anxiety and other forms of internalizing problems (Gladstone et al., 2005; Rabian et al., 1999; Weems et al., 2007). A significant contribution of this study is the examination of these four risks factors in tandem and the measurement of anxiety outcomes through multiple indicators—including *DSM-IV-TR* (APA, 2000) constructs, worry, and broader trait anxiety and negative affectivity. In addition, the nature of the risks assessed varied conceptually, from biologically-based precursors hypothesized to emerge early in development (Kagan, 2008; Reiss & McNally, 1985) to

cognitive risk factors predominantly thought of as emerging once higher-order cognitive capacities are in place. Thus, methodologically, this study provided a stronger test of these relations in comparison to studies assessing risk in one domain. Conceptually, this investigation extends available research by examining risk with a developmental lens. Indeed, the cross-sectional nature of this study notwithstanding, results showed that these domains were interrelated, providing preliminary evidence for the developmentally informed model that was subsequently tested.

4.2. Multiple Risk Factors and Anxiety Problems

We also attempted to clarify how these risks operate in combination with each other, and examined the incremental and cumulative risk posed by anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases to anxiety outcomes. This was done by testing two competing models; a multiple-risk model that accounted for the dimensional severity and content of risk, and a cumulative-risk model—wherein each of the risk variables was dichotomized and the number of risks totalized.

Overall, statistical prediction from the multiple-risk model was very good. The four risk variables (including gender and ethnicity) accounted for about 53% of the variance in anxiety problems (range = 40 – 65% across anxiety outcomes). Averaging across anxiety outcomes, behavioral inhibition and anxiety sensitivity accounted for about 15% and 29% of the variance in anxiety problems, respectively. The average prediction of anxiety symptoms from interpretive and judgment biases was higher; about 28% and 36% of the variance in anxiety problems was predicted by interpretive and judgment biases, respectively. These findings are important in a number of ways. First, these results were found in a nonclinical population with normally distributed scores across most measures. We would expect that statistical prediction would be

even stronger in clinical populations—although replication of our findings in such samples is needed. Second, our findings indicate that anxiety sensitivity, and interpretive and judgment biases, relative to behavioral inhibition, are stronger predictors of anxiety outcomes, suggesting that these constructs may be particularly good targets for intervention. Specifically, findings suggest that normalizing the experience of anxiety symptoms, reducing the tendency to interpret events negatively—such as the tendency to catastrophize, overgeneralize, personalize and selectively abstract negative information—and augmenting regulatory capacities (with particular attention to augmenting control), are promising points of intervention to reduce anxiety.

In addition, our results suggest that these risk factors increment each other to predict anxiety outcomes. We found that each of the four risk factors *uniquely* contributed to the prediction of anxiety outcomes in the context of other risks. Judgment biases uniquely accounted for up to 12% of the variance after all other risk factors were statistically controlled for, with anxiety sensitivity (up to 10%), interpretive biases (up to 3%), and behavioral inhibition (up to 2%) also offering unique predictions. These results strongly suggest that the development of anxiety problems is not only determined by biologically-based anxiety precursors which theory suggests develop early in life—such as behavioral inhibition and anxiety sensitivity; cognitive risk factors, such as inaccurate judgments of one's ability to control internal and external events, as well as biased interpretations of events, also play a vital role in anxiety problems. Thus, conceptual models aimed at furthering our understanding of the development of anxiety problems should, at a minimum, taking into account these four risks as important developmental processes.

Indeed, in the field of clinical psychology, research on the development of anxiety has overwhelmingly focused on the study of risk factors in isolation (Schmidt et al., 1997; Weems &

Silverman, 2008) and considerably less attention has been paid to covariation among risk processes (Weems & Stickle, 2005). Several developmental psychopathologists in the field of anxiety (Vasey & Dadds, 2001) have, particularly over the past decade, argued for the need to expand current conceptualizations of anxiety from unitary models of risk to conceptual models that take into account the multifactorial nature of specific pathological outcomes. In this vein, our conceptual framework attempted to address this issue by testing a multi-factorial model to the prediction of anxiety symptoms. The value of such a model was supported by our results. The multiple risk-factor model findings showed that each of the risk factors considered offered unique contributions to the prediction of anxiety symptoms.

From a developmental psychopathology perspective (Achenbach, 1982), these findings support the conceptualization of anxiety as a multivariate process wherein different risks combine to incrementally predict anxiety outcomes, rather than a model in which risks are considered empirically redundant. From a clinical perspective, results also argue in favor of systematic assessment of these risk domains for a more comprehensive case conceptualization that can shed light on the appropriateness of different intervention avenues. With the current focus being on moving from efficacy and effectiveness research to developing evidence-based explanations of treatment (i.e., mediators of treatment) and personalizing delivery (e.g., moderators of treatment) of mental health services (La Greca, Silverman, & Lochman, 2009), findings from this study contribute to efforts to specifically understand the simultaneous role of specific risks in the development of anxiety. This, in turn, can directly aid those who are attempting to deliver more individualized, evidence-based treatments. In addition, our findings highlight the need to continue to move beyond conceptualizations of anxiety problems and favorable treatment outcomes as either "presence" or "absence" of diagnostic labels and to

instead think in terms of presence or absence of contributing factors to the development of anxiety.

4.3. Cumulative Risk and Anxiety Problems

The cumulative-risk model also offered significant contribution to the prediction of anxiety outcomes, although estimates were substantially lower than those of the multiple-risk model. Together with gender and ethnicity, the dichotomous-risk variables predicted an average of 28.5% of the variance in anxiety outcomes (24 – 34% across anxiety measures), with each offering a unique contribution to the prediction of anxiety problems (up to 8%). The total number of risk factors also significantly predicted anxiety problems; indeed. 24 – 33% of the variance in anxiety problems was predicted by the total number of risk factors, regardless of which ones they were. Importantly, each individual risk factor, when entered as a continuous predictor, significantly predicted anxiety problems above and beyond the total number of risk factors, with judgment biases uniquely predicting up to18% of the variance, and anxiety sensitivity (up to12%), interpretive biases (up to 7%) and behavioral inhibition (up to 3%) also contributing significantly.

These findings provide strong support for the superiority of a multiple-risk factor model (on average accounting for 53% of the variance in internalizing symptoms) relative to a cumulative risk model (on average predicting 28.5% of the variance in internalizing symptoms). While several authors have argued for the latter as a more parsimonious model (Rutter, 1979; Sameroff et al., 1993), it is clear that a significant amount of statistical prediction of individual differences in internalizing symptoms is lost if the content and dimensional severity of risk factors are disregarded. Results from this study clearly suggest that the complexity inherent in models that consider content, process, and severity significantly benefits our understanding of

the development of anxiety problems and outweighs the potential benefits of a more parsimonious, cumulative –risk model. This is not to say that a purely cumulative model does not have value or explanatory power. Indeed, the total number of risk factors predicted over a quarter of the variance in anxiety problems, regardless of which factors they were. Thus, there is support for the cumulative stress hypothesis—more risks, in general, place individuals at risk for anxiety problems. However, prediction is significantly dampened by this model and, more importantly, little knowledge is gained with respect to which mechanisms may be responsible for elevated anxiety in some groups. The latter clearly would limit efforts to improve the specificity of available interventions. A similar conclusion has also been reached in the area of externalizing problems, where the content and process of risk was more informative for the development of externalizing problems (Deater-Deckard et al., 1998) than a cumulative model.

It is also worth noting that the unique contribution of judgment biases, in this study indexed by control beliefs about internal sensations and external events, was substantial regardless of model. Therefore, these results strongly support the role of control beliefs in the development of anxiety. This argument is not new—others have argued for control beliefs as a key factor in the development of anxiety (Barlow, 2002; Chorpita & Barlow, 1998; Rapee et al., 1996). However, that control beliefs played such a key role *in the context of* other important anxiety-related risk factors warrants particular attention. From an intervention standpoint, these findings suggest that encouraging greater control over internal sensations and external events may be particularly worthwhile in efforts to prevent anxiety problems—efforts that can begin in childhood. Indeed, several lines of research aimed at improving socioemotional competence in children have found success in reducing anxiety problems. While the focus of these interventions has not been solely and specifically on increasing control over anxiety, these interventions do

focus on teaching competence building strategies that do generalize to the domain of anxiety and its management.

Teaching optimistic thinking skills in schools, for example, was associated with subsequent reductions in depressive attributions and the use of worry as a coping mechanism (Cunningham, Brandon, & Frydenberg, 2002). Consistent with other investigations (see Greenberg & Kusché, 2006, for a review), the preschool Promoting Alternative Thinking Strategies (PATHS) curriculum (Kusché & Greenberg, 1994), aimed at reducing or preventing behavioral and emotional problems in children, specifically found reductions in anxiety and social withdrawal in low-income children post-intervention (Domitrovich, Cortes, & Greenberg, 2007). Notably, one of the lessons in PATHS specifically includes the teaching of self-control strategies. Although the self-control strategies taught are not specific to anxiety, findings suggest that the self-control skills learned generalize to outcomes such as anxiety (Domitrovich et al., 2007; see Greenberg & Kusché, 2006). Helping clients, both children and adults, expand their repertoire of regulatory strategies is likely to give them greater confidence in their ability to specifically control internal reactions and responses to external events that seem out of their control—an aspect closely linked to anxiety problems (Chorpita & Barlow, 1998).

At the same time, it is important to note that the prominent role of judgment biases, in particular anxiety control beliefs, in our study may be partly explained by the content of the measure we used, the ACQ (Rapee et al., 1996). The ACQ contains several items that include the term 'anxiety,' potentially priming participants to respond in the affirmative in the context of other questions also assessing anxiety-related events. This, coupled with shared method variance, may have resulted in stronger relations between this measure and the other measures used. Future studies should consider gathering of data from multiple informants and through multiple

methods to address these limitations, as well as utilizing measures of judgment biases that more neutrally assess the construct without explicit reference to anxiety. However, as mentioned above, theorists (Rapee et al.) have argued for the utilization of the measure we used if the goal is to specifically identify those at risk for anxiety.

4.4. Structural Equation Modeling of Direct and Indirect Pathways Linking Anxiety Sensitivity, Behavioral Inhibition, and Interpretive and Judgment Biases, to Anxiety Symptoms

This investigation also examined via structural equation modeling (SEM) direct and indirect pathways linking anxiety sensitivity, behavioral inhibition, and cognitive risk, with anxiety outcomes. Consistent with our hypothesis, the full mediational model positing interpretive and judgment biases as partial mediators between anxiety sensitivity and behavioral inhibition, and anxiety symptoms provided excellent fit to our data. Significant direct and indirect relations were found between behavioral inhibition, anxiety sensitivity, and anxiety problems. As hypothesized, men and women with high anxiety sensitivity and high behavioral inhibition tended to report higher anxiety problems in part because they engaged in higher interpretive and judgment biases. In turn, their increased biases in interpretation and judgment were directly associated with higher anxiety outcomes. Results from the SEM revealed that interpretive and judgment biases together accounted for 19% and 23% of the total effect of anxiety sensitivity and behavioral inhibition, respectively, on anxiety outcomes. These findings suggest that individual differences in anxiety sensitivity and behavioral inhibition associated with anxiety problems are, in part, due to differences in how individuals interpret the world around them and judge their ability to control internal sensations and external events.

This has important implications, in light of the different developmental time points during which these constructs theoretically emerge. Anxiety sensitivity (Reiss & McNally, 1985) is conceptualized as a risk factor for the development of anxiety with strong biological origins. Indeed, genetic studies suggest that 55% of the variance in anxiety sensitivity is heritable (Stein et al., 1999). Similarly, behavioral inhibition and the physiological reactivity associated with this behavioral pattern of response are thought to be highly heritable (Kagan, 2008). Thus, it is reasonable to predict that these risks have temporal precedence over interpretive and judgment biases—arguably reliant on higher-order cognitive capacities that are not in place until later in development. Interventions can therefore be designed to alter biologically-based precursors like behavioral inhibition and anxiety sensitivity before cognitive biases develop and lead to an increase of risk for more serious pathology. For example, behaviorally inhibited children can be identified early for interventions designed to make the child more comfortable in novel situations. Components of available evidence-based treatments for social anxiety (Beidel, Turner, & Morris, 2000) may be specifically used to decrease behavioral inhibition in young children. At the same time, the temporal relations between behavioral inhibition and anxiety sensitivity are not fully known. Longitudinal investigations are needed to clarify how these constructs covary over time, which can provide additional details as to when and how to intervene.

At the same time, intervention at the level of cognition may be more prudent if interpretive and judgment biases are already present, and because traits such as behavioral inhibition have been found to be rather stable (Kagan, 2008). Cognitive constructs may be relatively more malleable; indeed, some evidence is beginning to accumulate suggesting that cognitive behavioral therapy effects for anxiety may be mediated by changes in cognition

(Treadwell & Kendall, 1996; Kendall & Treadwell, 2007), specifically by increasing the ratio of

positive versus negative self-statements anxious children make. However, positive behavioral

change from CBT is not always associated with cognitive change (Durlak, Fuhrman, &

Lampman, 1991). With the exception of these very few studies, there is a scarcity of research on

whether treatment actually modifies cognitive structures. Similarly, how changes in interpretive

and judgment biases may relate to changes in behavioral inhibition and anxiety sensitivity over

time remains unknown. Research in this area can outline which specific constructs should be

targeted in treatment as potential mechanisms for change. Longitudinal data can significantly

inform these efforts and address the limitation of directionality of effects inherent in this cross-

sectional study. Similarly, in light of the ethnic composition of our sample testing of these

hypotheses in a more ethnically diverse sample is a necessary step; however, based on the lack of

significant interactions including ethnicity we would not expect drastic ethnic differences.

    While our findings suggested that both interpretive and judgment biases partially

mediated the relations between developmental precursors of anxiety and anxiety outcomes, the

mediating effect of judgment biases was clearly stronger than that of interpretive biases (see

Figure 3). There is significant empirical evidence to support these results. Judgment biases were

conceptualized in this study as the degree to which participants judge themselves to be in control

of internal sensations as well as external events. As reviewed above, a substantial body of

literature (Barlow, 2002) has specifically documented the relevance of control processes to

internalizing outcomes. This research has established that judgments of increased control of

internal and external events serve as protective factors that decrease the likelihood of

experiencing internalizing difficulties. Our findings are consistent with this literature. At the

same time, our findings expand the available literature by shedding light into how developmental

precursors of internalizing outcomes, that is, anxiety sensitivity and behavioral inhibition, may covary with judgment biases to lead to anxiety problems. Specifically, our findings support a developmentally informed model wherein behavioral inhibition to unfamiliar stimuli, as well as a biologically-based fear of anxiety sensations, may lead individuals to avoid situations that may trigger discomfort. As a result of this avoidance, individuals may not acquire the necessary strategies to successfully cope with these challenges. Repeated transactions of this nature and the feelings associated may ultimately result in the development of judgment biases; that is, the individual comes to believe internal sensations and external events are out of personal control, which, in turn, may lead to anxiety problems (Chorpita & Barlow, 1998).

In parallel to this process, our findings suggest that these same transactions may also result in the development of interpretive biases. Behavioral inhibition in response to novel stimuli, coupled with a disproportionate fear of the anxiety-related sensations, leads to avoidance (Kagan, 2008; Weems & Silverman, 2008). Although adaptive in the short term because of its anxiety –reducing effect, avoidance prevents individuals from facing the fear stimulus and realizing that the catastrophic outcomes imagined most likely do not occur (Borkovec, 2002). In other words, their threat interpretations go unchallenged, unmet by potentially disconfirming evidence that the perceived threat is not real. This schema of biased interpretations is thus strengthened and generalized to multiple situations; in time, the result is problematic anxiety symptoms (Beck et al., 1985). There is also empirical evidence to support the path from behavioral inhibition and anxiety sensitivity to interpretive biases. Several studies have shown that behavioral inhibition prospectively predicts the development of anxiety disorders, particularly social anxiety disorder—a disorder wherein biased interpretations of social situations (e.g., threatening, catastrophic) are hypothesized to be causal (Beidel & Turner, 2007). Similarly,

anxiety sensitivity has been established as precursor of several anxiety disorders, including panic disorder—a disorder in which negative interpretations of interoceptive cues are hypothesized to be causal (Clark, 1986). Thus, the hypothesized mediational model fits within the available research on the development of anxiety; furthermore, it extends current research by considering interpretive *and* judgment biases as a part of a more comprehensive model that explains the effects of biologically-based developmental precursors of anxiety on a wide range of internalizing outcomes. Notably, parental modeling of cognitive biases (e.g., "I don't think you should play with those children…who knows if they are good kids?"), and not solely avoidance and its consequences on skill acquisition, may lead to development of both judgment and interpretive biases. Indeed, research suggests that parents can influence children's interpretations of events (Barrett, Rapee, Dadds, & Ryan, 1996; Dadds & Barrett, 1996). Our model would be strengthened by incorporation and measurement of parental modeling of negative interpretations of events and parents' own perceptions of control as contributors to participants' own levels of interpretive and judgment biases. The same is true for anxiety sensitivity and behavioral inhibition. Importantly, our model operated in the same way for males and females, suggesting similar mechanisms across gender.

4.5. Patterns of Risk and Relations to Anxiety Outcomes

Finally, we wanted to explore whether homogenous groups of participants with specific profiles of risk factors were found within the larger sample. Cluster analyses yielded four clusters reliably reproduced on random 50% and 60% subsamples from the total sample. We found evidence for both multifinality and equifinality. Evidence for diverse outcomes (i.e., multifinality) was supported by significant differences in anxiety outcomes across some clusters. In general, higher risk was associated with higher anxiety problems—a finding consistent with

our earlier results of significant correlations between the total number of risks and anxiety outcomes. However, specific constellations of risk were also linked with specific outcomes. For example, membership in group 2, characterized primarily by intense levels of anxiety sensitivity and moderate cognitive biases, was specifically related to high *DSM-IV* anxiety symptoms (BSI-mixed anxiety composite). This finding supports the strong links of anxiety sensitivity with anxiety disorders identified by many (Cox et al., 1999; Taylor et al., 1992).

Notably, clusters 1 and 4, despite being comprised of clearly different patterns of risk, had comparable levels of anxiety across the four anxiety outcomes (see Figure 4), suggesting that similar anxiety outcomes have diverse pathways (i.e., equifinality). Our findings suggest that problematic anxiety for some may be the result of intense of levels of behavioral inhibition only (cluster 1), whereas for others it may be the result of moderate-to-high levels of interpretive and judgment biases. These results suggest that there are different subgroups of individuals at risk for anxiety, and that different risk patterns may lead to similar anxiety outcomes. However, whether these different subgroups and risk constellations may predict different outcomes over time remains an open question. Unfortunately, the cross-sectional nature of this study did not allow for examination of this question. Longitudinal research can clarify the correlates and determinants of these potentially different pathways.

Taken together, results from cluster analyses suggest that individuals seeking services for anxiety, although they may look similar based on symptom presentation, may differ in important ways regarding the patterns of risk that led to their anxiety symptoms. This highlights the need for comprehensive clinical assessment that takes into account the complex risk histories that may place individuals along different developmental trajectories leading to anxiety (Weems & Stickle, 2005). While the core elements for the treatment of anxiety may remain the same

regardless of the risk constellation that brought individuals in for treatment, efforts to try to personalize treatment and identify mechanisms of change will certainly be informed by efforts to better understand the specific causes and risk covariations that lead to problematic anxiety. Our findings suggest that risk factors for anxiety may differ across individuals and subgroups in important ways, despite comparable between-group levels of anxiety. Because the same risk factors may aggregate differently among individuals and result in similar or different outcomes, identification and understanding of subgroups and/or subtypes of individuals at risk for anxiety seems a particularly promising endeavor as we continue in the quest to advance our understanding into the causes of anxiety.

5. Concluding remarks

The present study aimed to advance understanding of the cumulative, incremental, and mediated influences of anxiety sensitivity, behavioral inhibition, and interpretive and judgment biases on anxiety outcomes. The cumulative and multiple-risk factor models proposed significantly predicted anxiety outcomes, although the statistical prediction offered by the latter model was superior. This model provided statistical support for the value of risk content in the prediction of anxiety symptoms, as evinced by the significant contribution of each risk factor to anxiety outcomes after statistically controlling for the total number of risks. Using SEM, we found that interpretive and judgment biases partially mediated pathways linking anxiety sensitivity and behavioral inhibition with anxiety outcomes; our SEM analyses further revealed that judgment biases, and in particular control beliefs, have a prominent mediating role between these constructs. Our model represents a welcome addition to the study of risk factors for the development of anxiety, arguing in favor of a developmentally informed pathway wherein biologically-based risk factors theoretically associated with early stages of development lead to

cognitive biases which, in turn, lead to anxiety outcomes. Additionally, identification of specific subgroups of individuals with different constellations of risk and somewhat different levels of anxiety argue for the need to expand our efforts to understand different trajectories leading to the expression of anxiety—efforts that can be facilitated by replication of this model with longitudinal data. This can not only expand our understanding into the causes of anxiety but also clarify the timing at which constructs like those included in this study may be targeted to prevent problematic anxiety from developing.

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APPENDIX

Standardized Factor Loadings for the Full Mediational Model

Measure	Loading
Anxiety sensitivity	
Physical concerns	.72
Social concerns	.67
Cognitive concerns	.84
Behavioral inhibition	
Fearful inhibition	.87
Non-approach	.44
Low sociability	.44
Risk avoidance	.43
Interpretive biases	
Catastrophizing	.84
Personalizing	.76
Overgeneralizing	.83
Selective abstraction	.72
Judgment biases	
External control	.75
Internal control	.91
Anxiety	
Worry	.74
Negative affectivity	.81
Trait anxiety	.85
Mixed anxiety	.81

Table 1

Demographic characteristics of the sample ($N = 862$)

Variable	N	%
Age (Mean, SD)		
Males	19.07 (1.25)	
Females	18.62 (0.92)	
Gender		
Males	250	29.0
Females	608	70.5
Ethnicity		
White	714	82.8
Ethnic minority	145	16.8
Marital status		
Single, never married	852	98.8
Married	3	0.3
Living with partner	7	0.8
Education		
Some college	830	96.3
2-year college degree	8	0.9
4-year college degree	22	2.6
Employment		
Not working/unemployed	592	68.7
Part-time	252	29.2
Full-time	15	1.7
Education-father		
Less than High School	9	1.0
High School/GED	141	16.4
Some college	81	9.4
2-year college degree	62	7.2
4-year college degree	267	31.0
Some graduate courses	30	3.5
Master degree	170	19.7
Doctoral/Professional degree	97	11.3
Education-mother		
Less than High School	9	1.0
High School/GED	159	18.4
Some college	82	9.5
2-year college degree	108	12.5
4-year college degree	300	34.8
Some graduate courses	34	3.9
Master degree	141	16.4
Doctoral/Professional degree	24	2.8

Table 2

Risk variables, means and standard deviations for the total sample ($N = 862$) as well as by sex and ethnic group

Variable	Total		Sex				Ethnicity			
			Males		Females		White-American		Minorities	
	M	SD	M	SD	M	SD	M	SD	M	SD
Variable and subscales					Anxiety sensitivity					
ASI-Total	15.69	11.71	16.47	11.35	15.32	11.81	15.29	11.32	17.49	13.39
Cognitive concerns	2.98	4.05	3.22	4.07	2.85	4.01	2.77	3.83	3.92	4.91
Physical concerns	4.12	4.71	4.03	4.62	4.15	4.74	3.99	4.62	4.69	5.09
Social concerns	8.58	5.19	9.20	4.89	8.32	5.29	8.52	5.13	8.85	5.49
					Behavioral inhibition					
AMBI-Total	13.88	5.16	14.74	4.96	13.54	5.21	13.81	5.22	14.26	4.83
Non-approach	2.86	1.62	3.19	1.55	2.74	1.63	2.85	1.63	2.94	1.58
Fearful inhibition	5.95	3.01	6.38	2.98	5.78	3.01	5.97	3.01	5.92	3.03
Risk avoidance	2.87	1.11	2.87	1.10	2.87	1.11	2.87	1.12	2.90	1.08
Low sociability	2.14	1.36	2.26	1.36	2.09	1.35	2.08	1.34	2.42	1.42
					Interpretive biases					
NCES-Total	32.36	9.99	32.75	10.13	32.21	9.96	32.23	9.85	33.03	10.75
Catastrophizing	7.55	3.10	7.55	3.11	7.55	3.11	7.53	3.08	7.61	3.25
Overgeneralizing	9.51	3.35	9.22	3.13	9.63	3.44	9.57	3.30	9.21	3.59
Personalizing	8.15	2.76	8.60	2.84	7.96	2.71	8.05	2.74	8.68	2.82
Selective Abstraction	7.15	2.58	7.37	2.66	7.07	2.54	7.08	2.54	7.53	2.74
					Judgment biases					
ACQ-Total	53.97	19.42	50.49	18.73	55.33	19.56	53.37	19.29	56.37	19.74
ACQ-Internal	26.12	11.00	24.24	10.52	26.85	11.13	26.00	10.92	26.39	11.27
ACQ-External	27.85	10.16	26.26	9.82	28.47	10.25	27.37	10.06	29.98	10.36
					Anxiety Outcomes					
BMWS	3.42	3.96	2.72	3.35	3.72	4.17	3.37	3.94	3.57	4.00
BSI-mixed	0.60	0.56	0.51	0.49	0.63	0.58	0.59	0.55	0.62	0.61
PANAS-NA	20.09	6.77	18.79	6.17	20.61	6.94	20.08	6.63	19.84	7.25
STAI-T	39.38	9.52	38.81	8.79	39.61	9.82	39.17	9.38	40.19	10.16

Note. AMBI-Total = Adult Measure of Behavioral Inhibition Total Score; ASI-3-Total = Anxiety Sensitivity Index-3 Total Score;

ACQ-Total = Anxiety Control Questionnaire Total Score; NCES = Negative Cognitive Error Scale of the Positive and Negative

Cognitive Error Questionnaire; BMWS = Brief Measure of Worry Severity (square root transformed); BSI-mixed = Brief Symptom

Inventory Mixed Anxiety Composite (square root transformed); PANAS-NA = Positive and Negative Affectivity Scale, Negative

Affectivity Subscale; STAI-T = State-Trait Anxiety Inventory-Trait Scale.

Table 3

Intercorrelations among all variables and those included in SEM analyses

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Anxiety sensitivity																					
1. ASI-3-Total	----																				
2. Cognitive concerns	.84	----																			
3. Physical concerns	.86	.67	----																		
4. Social concerns	.82	.50	.50	----																	
Behavioral inhibition																					
5. AMBI-Total	.36	.27	.25	.37	----																
6. Non-approach	.07	.02	.01	.13	.65	----															
7. Fearful inhibition	.43	.32	.29	.46	.87	.39	----														
8. Risk avoidance	.13	.07	.10	.14	.60	.28	.36	----													
9. Low sociability	.22	.22	.20	.15	.60	.20	.35	.33	----												
Interpretive biases																					
10. NCES	.56	.52	.43	.47	.41	.19	.44	.16	.23	----											
11. Castrophizing	.50	.45	.35	.44	.39	.18	.43	.13	.21	.87	----										
12. Personalizing	.50	.44	.38	.44	.33	.16	.37	.11	.18	.83	.60	----									
13. Overgeneralizing	.46	.39	.35	.41	.35	.18	.38	.17	.15	.88	.71	.65	----								
14. Selective Abstract.	.46	.49	.37	.30	.31	.13	.30	.11	.24	.80	.62	.58	.56	----							
Judgment biases																					
15. ACQ-Total	.52	.53	.43	.36	.39	.19	.42	.18	.21	.54	.49	.42	.47	.45	----						
16. ACQ-External	.46	.49	.39	.30	.38	.21	.40	.16	.21	.50	.44	.41	.43	.41	.91	----					
17. ACQ-Internal	.49	.47	.39	.37	.34	.13	.38	.16	.18	.50	.46	.36	.44	.41	.92	.68	----				
Anxiety outcomes																					
18. BSI-mixed	.63	.58	.51	.52	.38	.08	.45	.16	.24	.50	.46	.38	.48	.38	.60	.50	.59	----			
19. BMWS	.49	.43	.35	.43	.31	.09	.35	.16	.15	.51	.45	.40	.48	.37	.56	.41	.60	.59	----		
20. PANAS-NA	.49	.47	.37	.39	.32	.09	.36	.13	.21	.48	.43	.37	.46	.36	.59	.47	.60	.71	.61	----	
21. STAI-T	.52	.52	.36	.43	.44	.21	.47	.18	.25	.59	.58	.43	.52	.45	.69	.56	.70	.65	.63	.69	----

Note. All correlations with magnitudes > .08 were significant at $p < .01$. Correlations with magnitudes of .05 - .08 were significant at p

< .05. All other correlations ($n = 2$) were nonsignificant. AMBI-Total = Adult Measure of Behavioral Inhibition Total Score; ASI-3-

Total = Anxiety Sensitivity Index-3 Total Score; ACQ-Total = Anxiety Control Questionnaire Total Score; NCES = Negative

Cognitive Error Scale of the Positive and Negative Cognitive Error Questionnaire; BMWS = Brief Measure of Worry Severity (square root transformed); BSI-mixed = Brief Symptom Inventory Mixed Anxiety Composite (square root transformed); PANAS-NA = Positive and Negative Affectivity Scale, Negative Affectivity Subscale; STAI-T = State-Trait Anxiety Inventory-Trait Scale.

Table 4

Demographic and continuous risk variables predicting anxiety outcomes: Initial and unique regression models

Variable	BSI-mixed Total $R^2 = .52$[a]				BMWS Total $R^2 = .40$[a]				PANAS-NA Total $R^2 = .65$[a]				STAI-T Total $R^2 = .56$[a]			
	β		ΔR^2		β		ΔR^2		β		ΔR^2		β		ΔR^2	
	I	*U*	*I*	*U*	*I*	*U*	*I*	*U*	*I*	*U*	*I*	*U*	*I*	*U*	*I*	*U*
Demographics	----	----	.01[a]	----	----	----	.01[c]	----	----	----	.01[b]	----	----	----	.00	----
Gender	.10[a]	----	----	----	.10[b]	----	----	----	.12[a]	----	----	----	.04	----	----	----
Ethnicity	.00	----	----	----	-.02	----	----	----	.01	----	----	----	-.04	----	----	----
Risk variables																
ASI-3-Total	.64[a]	.41[a]	.41[a]	.10[a]	.49[a]	.20[a]	.24[a]	.02[a]	.50[a]	.19[a]	.24[a]	.02[a]	.52[a]	.10[a]	.27[a]	.01[a]
AMBI-Total	.40[a]	.10[a]	.16[a]	.01[a]	.32[a]	.03	.10[a]	.00	.35[a]	.07[c]	.12[a]	.00	.46[a]	.14[a]	.21[a]	.02[a]
ACQ-Total	.59[a]	.30[a]	.35[a]	.05[a]	.55[a]	.32[a]	.30[a]	.06[a]	.58[a]	.36[a]	.33[a]	.08[a]	.69[a]	.45[a]	.47[a]	.12[a]
NCES	.51[a]	.08[c]	.26[a]	.00	.51[a]	.21[a]	.26[a]	.03[a]	.49[a]	.16[a]	.24[a]	.01[a]	.59[a]	.23[a]	.35[a]	.03[a]

Note. I = initial regression model. U = unique regression model. AMBI-Total = Adult Measure of Behavioral Inhibition Total Score; ASI-3-Total = Anxiety Sensitivity Index-3 Total Score; ACQ-Total = Anxiety Control Questionnaire Total Score; NCES = Negative Cognitive Error Scale of the Positive and Negative Cognitive Error Questionnaire. BMWS = Brief Measure of Worry Severity (square root transformed); BSI-mixed = Brief Symptom Inventory Mixed Anxiety Composite (square root transformed); PANAS-NA = Positive and Negative Affectivity Scale, Negative Affectivity Subscale; STAI-T = State-Trait Anxiety Inventory-Trait Scale.

[a] $p < .001.$ [b] $p < .01.$ [c] $p < .05.$ [d] $p < .10$

Table 5

Percentage of cases meeting the dichotomous risk criterion for the total sample as well as by sex

and ethnic group

Variable	Total		Sex				Ethnicity			
			Males		Females		White-American		Minorities	
	N	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*
1. AMBI-Total	109	12.6	39	15.6	70	11.5	95	13.3	14	9.7
2. ASI-3-Total	145	16.8	45	18.0	99	16.3	114	16.0	30	20.7
3. ACQ-Total	134	15.5	27	10.8	106	17.4	108	15.1	23	15.9
4. NCES	147	17.1	47	18.8	100	16.4	118	16.5	29	20.0

Note. AMBI-Total = Adult Measure of Behavioral Inhibition Total Score; ASI-3-Total = Anxiety

Sensitivity Index-3 Total Score; ACQ-Total = Anxiety Control Questionnaire Total Score;

NCES = Negative Cognitive Error Scale of the Positive and Negative Cognitive Error

Questionnaire.

Table 6

Demographic and dichotomous risk variables predicting anxiety outcomes: Initial and unique regression models

Variable	BSI-mixed Total R^2 = .34[a]				BMWS Total R^2 = .24[a]				PANAS-NA Total R^2 = .27[a]				STAI-T Total R^2 = .33[a]			
	β		ΔR^2		β		ΔR^2		β		ΔR^2		β		ΔR^2	
	I	*U*	*I*	*U*	*I*	*U*	*I*	*U*	*I*	*U*	*I*	*U*	*I*	*U*	*I*	*U*
Demographics	----	----	.01[a]	----	----	----	.01[c]	----	----	----	.01[b]	----	----	----	.00	----
Gender	.10[a]	----	----	----	.10[b]	----	----	----	.12[a]	----	----	----	.04	----	----	----
Ethnicity	.00	----	----	----	-.02	----	----	----	.01	----	----	----	-.04	----	----	----
Dichotomous Risk																
ASI-3-Total	.45[a]	.27[a]	.22[a]	.06[a]	.34[a]	.15[a]	.11[a]	.02[a]	.35[a]	.15[a]	.12[a]	.02[a]	.38[a]	.16[a]	.14[a]	.02[a]
AMBI-Total	.28[a]	.14[a]	.09[a]	.02[a]	.24[a]	.11[a]	.06[a]	.01[a]	.25[a]	.11[a]	.06[a]	.01[a]	.31[a]	.30[a]	.10[a]	.08[a]
ACQ-Total	.45[a]	.27[a]	.21[a]	.06[a]	.38[a]	.22[a]	.14[a]	.04[a]	.42[a]	.28[a]	.17[a]	.06[a]	.46[a]	.28[a]	.21[a]	.06[a]
NCES	.36[a]	.14[a]	.14[a]	.02[a]	.36[a]	.21[a]	.13[a]	.03[a]	.22[a]	.18[a]	.20[a]	.03[a]	.41[a]	.22[a]	.17[a]	.04[a]

Note. I = initial regression model. U = unique regression model. AMBI-Total = Adult Measure of Behavioral Inhibition Total Score; ASI-3-Total = Anxiety Sensitivity Index-3 Total Score; ACQ-Total = Anxiety Control Questionnaire Total Score; NCES = Negative Cognitive Error Scale of the Positive and Negative Cognitive Error Questionnaire. BMWS = Brief Measure of Worry Severity (square root transformed); BSI-mixed = Brief Symptom Inventory Mixed Anxiety Composite (square root transformed); PANAS-NA = Positive and Negative Affectivity Scale, Negative Affectivity Subscale; STAI-T = State-Trait Anxiety Inventory-Trait Scale.

[a] $p < .001$. [b] $p < .01$. [c] $p < .05$. [d] $p < .10$

Table 7

Mean scores for risk factors and anxiety outcomes for the four-cluster solution, and additional characteristics of each group ($N = 314$)

	Cluster 1 ($n = 73$)		Cluster 2 ($n = 105$)		Cluster 3 ($n = 63$)		Cluster 4 ($n = 73$)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Risk factors								
AMBI-Total	1.66[a]	0.54	-0.06[c]	0.71	1.32[b]	0.82	0.00[c]	0.72
ASI-3-Total	0.00[c]	0.67	1.51[b]	0.53	1.78[a]	0.88	-0.31[d]	0.55
ACQ-Total	0.06[c]	0.84	0.81[b]	0.76	1.31[a]	0.72	0.81[b]	0.74
NCES	0.22[c]	0.79	0.55[b]	0.80	1.90[a]	0.72	0.84[b]	1.01
Anxiety outcomes								
BMWS	1.68[c]	1.12	2.18[b]	1.09	2.65[a]	0.89	1.92[bc]	1.27
BSI-mixed	0.78[c]	0.27	0.96[b]	0.36	1.18[a]	0.32	0.73[c]	0.34
PANAS-NA	21.67[c]	6.11	24.56[b]	7.04	27.94[a]	5.86	22.54[bc]	7.41
STAI-T	42.78[b]	7.82	44.79[b]	8.53	52.52[a]	8.03	44.41[b]	8.21
Demographics								
Age	18.81	1.28	18.78	0.88	18.62	0.99	18.76	1.07
Males	35.6%		33.3%		28.6%		19.2%	
Females	64.4%		64.8%		71.4%		80.8%	
White American	87.7%		78.1%		81.0%		84.9%	
Minority	12.3%		20.0%		19.0%		13.7%	

Note. Risk factor variables are presented in standardized form for consistency with graph. AMBI-Total = Adult Measure of Behavioral Inhibition Total Score; ASI-3-Total = Anxiety Sensitivity Index-3 Total Score; ACQ-Total = Anxiety Control Questionnaire Total Score; NCES = Negative Cognitive Error Scale of the Positive and Negative Cognitive Error Questionnaire; BMWS = Brief Measure of Worry Severity (square root transformed); BSI-mixed = Brief Symptom Inventory Mixed Anxiety Composite (square root

transformed); PANAS-NA = Positive and Negative Affectivity Scale, Negative Affectivity Subscale; STAI-T = State-Trait Anxiety Inventory-Trait Scale.

Means not sharing superscripts are significantly different from each other.
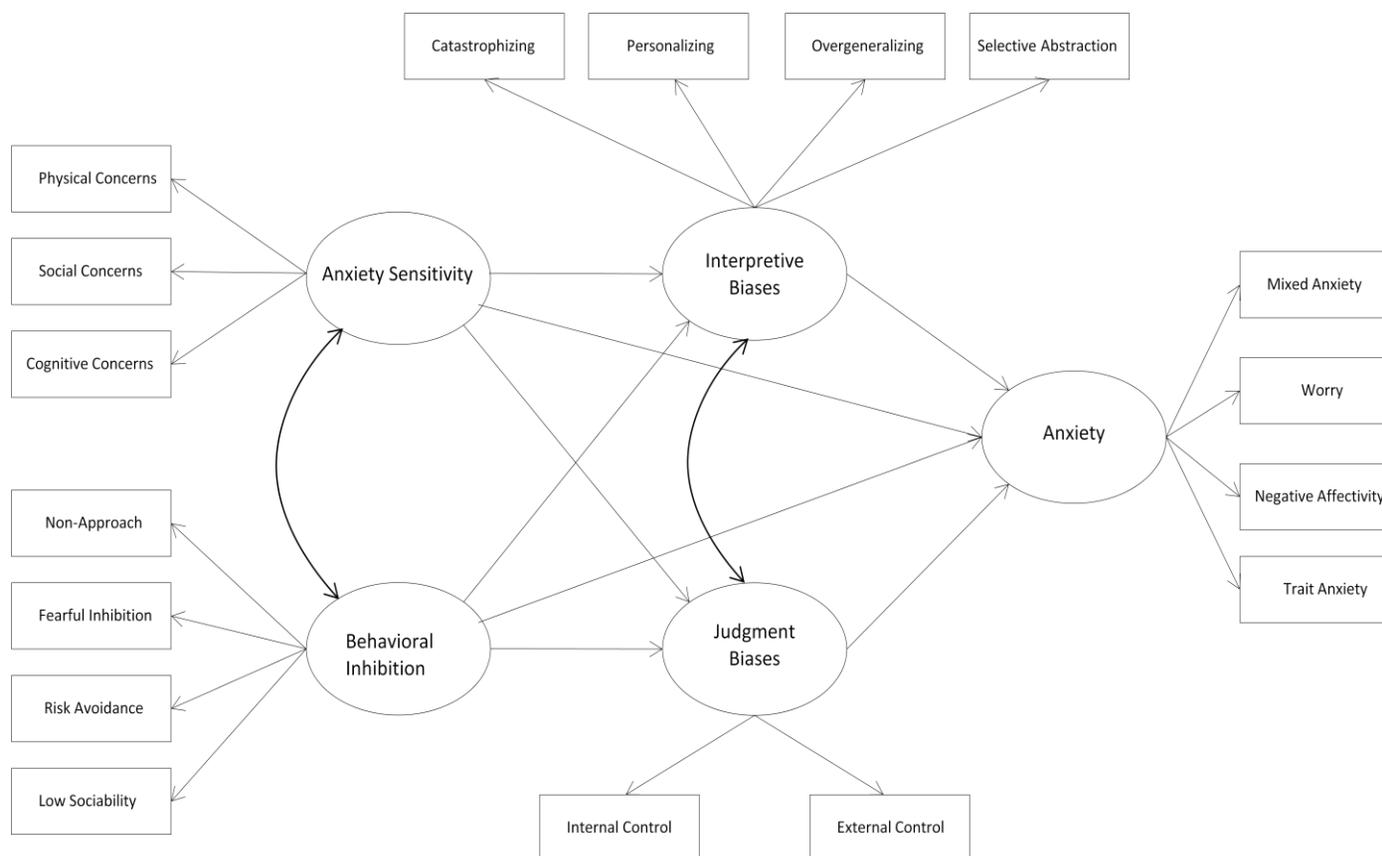
Figure 1. Hypothesized mediational model showing direct and indirect pathways from anxiety sensitivity and behavioral inhibition to anxiety symptoms. The model also shows interpretive and judgment biases as mediators of the links between anxiety sensitivity and behavioral inhibition, and anxiety.
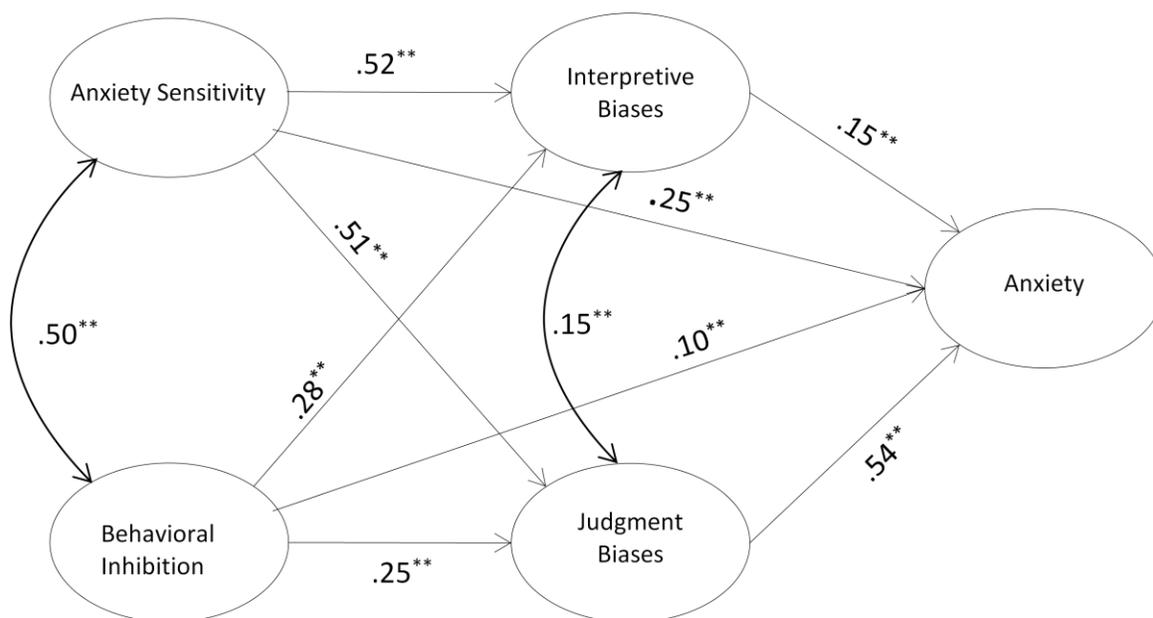
Figure 2. Full mediational model: Paths linking anxiety sensitivity and behavioral inhibition, with anxiety symptoms through interpretive and judgment biases. Latent path coefficients are presented in standardized units.
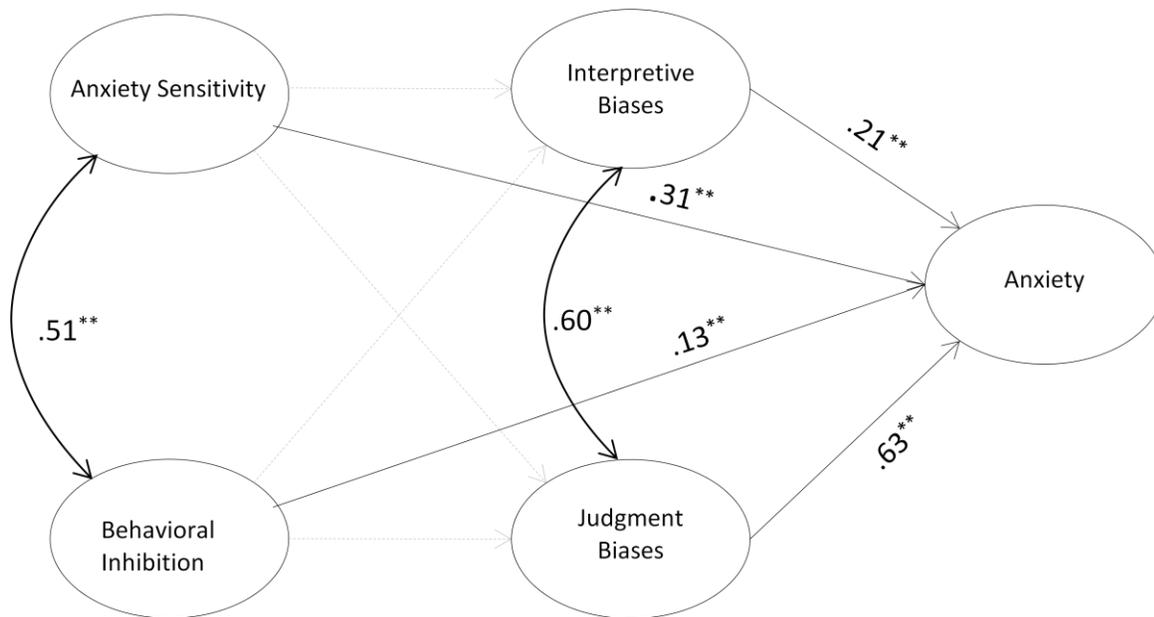
Figure 3. Step 2 of mediational analyses: Indirect paths linking both mediators to anxiety are constrained to zero and shown as dashed, gray arrows. Latent path coefficients are presented in standardized units.
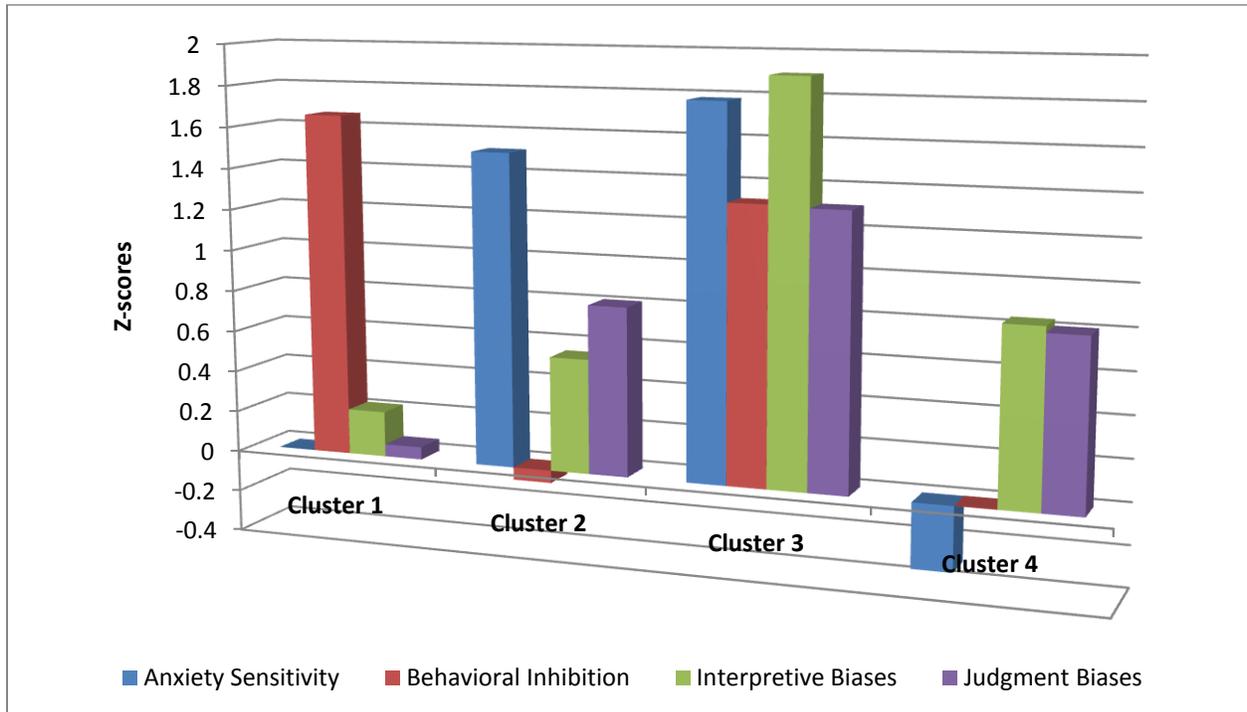
Figure 4. An examination of patterns of risk for the final, four-cluster solution. To assist with

comparison, scores on the four risk factors were converted to standardized scores (z-scores).

Higher scores on each of the risks mean increased risk.

Footnotes

[1] There is vast support for the conception of anxiety sensitivity and behavioral inhibition as biologically-based precursors of anxiety. Indeed, an increased sensitivity to physiological cues (i.e., anxiety sensitivity) has been described by many (e.g., Clark, Watson, & Mineka, 1994; Fowles, 1993) as a risk factor for the development of anxiety problems. Such proposition has been supported by empirical evidence (e.g., Maller & Reiss, 1992; Schmidt, Lerew, & Jackson, 1997). The evidence (Clark et al., 1994) further suggests that the construct may be part of a complex biological sensitivity (e.g., autonomic hyperarousal) that increases risk for anxiety. McNally (1990) and Reiss (1991) also posited anxiety sensitivity as a construct with biological origins. In a similar vein, a vast literature (see Degnan & Fox, 2007, for an excellent review) describes behavioral inhibition as a stable temperamental trait with strong biological origins (Kagan, 2008; Degnan & Fox, 2007) that predicts social reticence (Fox, Henderson, Marshall, Nichols, & Ghera, 2005), and the development of anxiety and its disorders (e.g., Biederman et al., 2001; Coplan, Wilson, Frohlick, & Zelenski, 2006; Gar, Hudson, & Rapee, 2005; Gladstone, Parker, Mitchell, Wilhelm, & Malhi, 2005; van Brakel, Muris, Bogels, & Thomassen, 2006). At the same time, the authors are keenly aware of other biologically-based indices, such as heightened physiological arousal and negative affectivity (Clark et al.), cardiac reactivity (Garcia Coll, Kagan, & Reznick, 1984) and electroencephalogram (EEG) asymmetry (Davidson & Fox, 1982; Calkins, Fox, & Marshall, 1996), implicated in anxiety.

[2] These models, however, recognize that what we term "anxiety" is the result of more than just cognition (Clark & Beck, 1988); it is instead the product of complex and interconnected cognitive (e.g., derealization), affective (e.g., feelings of apprehension), physiological (e.g., autonomic hyperarousal), and behavioral (e.g., escape) responses and systems. It is with the

cognitive component, however, that the present study is primarily concerned. That is, the cognitive distortions, biases, and fear beliefs that are integral to the anxiety response.

[3] Continuous variables that were part of an interaction term were centered around their mean to prevent multicollinearity problems (Cohen, Cohen, West, & Aiken, 2003). All interaction terms, however, were not significant for these as well as subsequent analyses (ps > .12). As a result, interaction terms are not presented in the Tables in order to simplify presentation of findings.

Vita

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