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Trait Affective, Behavioral, and Cognitive Factors of Anxiety and Depressive Symptoms in Children and Adolescents: A Hierarchical Model

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A Dissertation

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The Department of Psychology

by
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For my family and friends; you have been instrumental in making my life and academic career what it is. I would be remiss if I did not acknowledge the several organizations that have directly and indirectly supported this process and its final product, chiefly Community Coffee Houses of Baton Rouge, Louisiana, Cup’s Coffees of Jackson, Mississippi, and Molten Java of Bethel, Connecticut. Your establishments have made outstanding coffees over the years.

Finally, I dedicate this work to the Vancouver Canucks for providing much needed entertainment during the writing of this document and for teaching me to persist against the odds.
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LIST OF ABBREVIATIONS

BIS – Behavioral Inhibition System/Behavioral Inhibition Scale
BAS – Behavioral Approach System/Behavioral Approach Scale
CASI – Children’s Anxiety Sensitivity Scale
CDI 2-S – Children’s Depression Inventory-Short Version
GAD – Generalized Anxiety Disorder
IHM – Integrative Hierarchical Model of Anxiety and Depression
IUS-C – Intolerance of Uncertainty Scale for Children
MDD – Major Depressive Disorder
OCD – Obsessive Compulsive Disorder
OCI-CV – Obsessive Compulsive Inventory-Child Version
PANAS-C – Positive and Negative Affectivity Scale for Children
PSWQ-C – Penn State Worry Questionnaire for Children
TCQ-A – Adolescent Thought Control Questionnaire
ABSTRACT

Researchers have proposed a vulnerabilities model that attempts to explain the similarities between the internalizing disorders via a hierarchical pathway of trait affectivity and cognitive factors among adults. The current study aimed to replicate and extend this model to symptoms of four internalizing disorders among youth: obsessions and compulsions, generalized anxiety/worry, social anxiety, and depression. Regression-based path analyses utilized data from a community sample of 105 youth aged 12-17 (67.6% female, 80% white, non-Hispanic). Results largely replicated prior models in the adult literature and overall supported a hierarchical paradigm. Trait negative affect and avoidant behavior predicted mid-tier cognitive vulnerabilities (anxiety sensitivity, intolerance of uncertainty, and thought suppression). Results were more mixed when predicting specific sets of internalizing symptoms with intolerance of uncertainty having the greatest impact on obsessive-compulsive and generalized anxiety symptoms. Specific pathways are discussed in terms of prior research and theory. The hypothesized model was compared to a fully identified version. While statistically significant, the difference in variance explained was not practically meaningful, indicating parsimony and theory driven relationships. Limitations and future directions based on this preliminary work are discussed.
1. INTRODUCTION

Internalizing disorders are among the most common psychological diagnoses in children and adolescents (Costello et al., 1996; Kessler & Walters, 1998; Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Merikangas et al., 2010). The internalizing disorders are those in which the primary cause of impairment is emotional in nature (i.e., anxiety or depression) as opposed to behavioral (i.e., hyperactivity, oppositional behavior, delinquency, etc.). According to the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5; American Psychiatric Association, 2013), the anxiety disorders include separation anxiety disorder, selective mutism, specific phobia, social anxiety disorder (formerly social phobia), panic disorder, agoraphobia, generalized anxiety disorder (GAD), substance/medication-induced anxiety disorder, and other- or un-specified anxiety disorders. In the 5th edition of the DSM, obsessive-compulsive disorder (OCD) was separated from the anxiety disorders and categorized within obsessive-compulsive spectrum disorders, though OCD is conceptualized as an anxiety disorder in the majority of the literature to date, based on the previous DSM edition. Internalizing mood disorders are major depressive disorder (MDD), persistent depressive disorder (formerly dysthymic disorder), the bipolar disorders, cyclothymic disorder, and other- or un-specified mood disorders. Typically, only MDD and persistent depressive disorder are the subject of internalizing research, due to conceptual overlap with externalizing features (i.e., mania) in the bipolar and cyclothymic disorders.

Cognitive-behavioral theories generally conceptualize the internalizing disorders as consisting of three symptom domains: physiology, behavior, and cognition. Lang (1979) described fear as consisting of familiar patterns of information processing including physiological arousal (i.e., activation of the autonomic nervous system), cognitive processing
(i.e., formation and activation of unrealistic beliefs and/or expectations), and behavioral prototypes (i.e., familiar patterns of behavioral responses to feared stimuli). Likewise, depressive disorders have been conceptualized as consisting of the same three general domains, with a primary emphasis on cognitive (e.g., distorted beliefs/schemas) and behavioral processes (e.g., withdrawal, inhibition; Beck, 1976).

Epidemiological and clinical research has often noted high rates of comorbidity among anxiety and depression in children and adolescents (Brady & Kendall, 1992; Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Strauss, Last, Hersen, & Kazden, 1988; Woolston et al., 1989). Similarly, clinical and psychometric investigations have found moderate-to-strong correlations between anxiety and depressive symptomatology measures, often leading to poor differentiation between the two constructs (for a review, see Clark & Watson, 1991). It has been suggested that such high rates of comorbidity and correlations among internalizing syndromes suggests underlying etiological, process, and/or symptom commonalities (Barlow, 2002; Chorpita & Barlow, 1998; Clark, 2005; Clark & Watson, 1991; Mennin, Heimberg, Fresco, & Ritter, 2008; Mineka, Watson, & Clark, 1998).

Models of adult psychopathology help to explain symptom overlap and high rates of comorbidity among the internalizing disorders. However, such models for children and adolescents have not been widely studied. Therefore, this study proposes to examine a hierarchical model of common factors of symptoms of four internalizing disorders: OCD, GAD, social anxiety disorder, and major depressive- and/or persistent depressive disorder. The proposed model is based upon the adult literature and includes aspects of several factors that contribute to adult psychopathology: trait affectivity, behavioral temperament, and cognitive traits. The proceeding literature review introduces the various theoretical and empirical bases for
such a model. The review begins by highlighting three theories that explain the often-noted similarities and comorbidities between the internalizing disorders: the Tripartite Model, the Integrative Hierarchical Model, and the Triple Vulnerabilities Model. Next, a behavioral trait (i.e., behavioral inhibition) and three specific cognitive traits, thought to be vulnerabilities for internalizing disorders, are presented (i.e., anxiety sensitivity, intolerance of uncertainty, and thought suppression). Following this, regression and structural models of adult internalizing symptomatology are presented. These structural models synthesize elements of the general and cognitive vulnerability theories and present an empirical heuristic for examining hierarchical trait and cognitive factors within the internalizing disorders. Finally, a rationale for the current study and a hypothesized path model is proposed that replicates and extends the adult literature to youth.

1.1 Tripartite Model of Internalizing Symptomatology

After noting symptom similarities and high rates of comorbidity between anxiety and depression, Clark and Watson (1991) outlined a hierarchical model to explain these relationships. Their three-factor model resembles a triangle with a general vulnerability factor on the top, leading to a second level consisting of two differentiating factors specific to either anxiety or depression. At the top of the model, an emotional factor (i.e., general distress) is common to both anxiety and depression. According to the model, the general distress factor is operationalized as a trait propensity to experience negative affective states (i.e., guilt, anger, sadness, fear, etc.). Following the general predictor, anxiety and depression are differentiated by the presence (or absence) of two other factors: physiological hyperarousal and propensity for positive affect (e.g., joy, pride, enthusiasm, alertness, etc.). An important distinction between the
Tripartite Model and other models of developmental psychopathology is that Clark and Watson did not specifically conceptualize negative affectivity, physiological hyperarousal, and positive affectivity as developmental psychopathology vulnerabilities, rather they viewed them as common and differentiating facets of internalizing emotional experiences. In contrast, others have discussed these factors as vulnerabilities in other models (i.e., predisposing traits, which can exist in the absence of an internalizing disorder).

1.1.1 Negative Affectivity

Negative affectivity is generally conceptualized as a trait, rather than state phenomenon (Clark, Watson, & Mineka, 1994); that is negative affectivity is the propensity to experience pervasive negative emotional states. Studies have demonstrated negative affectivity remains relatively stable over time (Brown, 2007; Lambert, McCreary, Joiner, Schmidt, & Ialongo, 2004; Lonigan, Phillips, & Hooe, 2003). Likewise, negative affectivity is considered analogous with Eysenck’s (1967) neuroticism personality dimension (Clark et al., 1994). Over the years since the Tripartite Model was proposed, several studies that jointly factor analyzed measures of anxiety and depression in children and adults have supported the existence of a higher-order factor (Joiner Catanzaro, & Laurent, 1996; Lambert et al., 2004; Prenoveau et al., 2010; Spence, 1997; Zinbarg & Barlow, 1996).

Predictive relationships have been routinely noted between negative affectivity/neuroticism and specific internalizing disorders, though results vary by methodology. Negative affectivity/neuroticism contributed significantly to social phobia symptomatology in adults in several studies (Brown, Chorpita, & Barlow, 1998; Kotov, Watson, Robles, & Schmidt, 2007; Norton & Mehta, 2007; Trull & Sher, 1994); however, a study among children (Chorpita,
Plummer, & Moffitt, 2000) did not find such a relationship. Negative affectivity has also been shown to contribute to OCD symptoms in adults (Brown et al., 1998; Kotov et al., 2007; Norton & Mehta, 2007; Sexton, Norton, Walker, & Norton, 2003) and children (Chorpita et al., 2000). However, a path model failed to replicate these relationships to OCD in a clinical sample of adults (Norton, Sexton, Walker, & Norton, 2005).

Researchers have also found that negative affectivity predicts GAD and depression symptoms; among adults, moderate-to-strong predictions of GAD symptoms have been noted (Brown et al., 1998; Kotov et al., 2007; Norton & Mehta, 2007; Norton et al., 2005; Sexton et al., 2003). However, among children, only a weak relationship has been found with worry, a common analog for GAD symptoms (Chorpita et al., 2000). Links between negative affectivity/neuroticism and depression have also been noted among adults, though the strength of the relationship varies (Brown et al., 1998; Norton & Mehta, 2007; Norton et al., 2005; Trull & Sher, 1994; Watson, Clark, & Carey, 1988). Depressive symptoms in children and adolescents likewise have variable relationships with negative affectivity; some studies have shown moderate-to-strong predictions of depressive symptoms (Anthony, Lonigan, Hooe, & Phillips, 2002; Jacques & Mash, 2004; Lonigan et al., 2003), while another did not find a relationship (Chorpita et al., 2000). Collectively, these studies suggest negative affectivity plays a role in social phobia, GAD, OCD, and depression symptoms in adults, but that these relationships are possibly less stable or less well researched in children and adolescents.

1.1.2 Positive Affectivity

One of the defining features of MDD and persistent depressive disorder is a significant loss of pleasure in daily activities or interests (American Psychiatric Association, 2013). It is not
surprising then that low positive affectivity was proposed as a specific component of depression according to the Tripartite Model (Clark & Watson, 1991). Positive affectivity is also seen as analogous to Eysenck’s (1967) extroversion personality dimension (Clark et al., 1994). Lack of positive affectivity is often conceptualized as anhedonia; therefore, the absence of positive affectivity is theorized to differentiate depression from anxiety. Consistent with the Tripartite Model, positive affectivity/extroversion has routinely been shown to negatively predict depressive symptoms among adults (Brown et al., 1998; Norton et al., 2005; Trull & Sher, 1994; van der Heiden et al., 2010; Watson et al., 1988). Similar relationships have been noted with children’s depression symptoms (Anthony et al., 2002; Chorpita et al., 2000; Hughes & Kendall, 2009; Jacques & Mash, 2004; Lonigan et al., 2003).

It has also been suggested that social phobia is characterized by low positive affectivity/extroversion due to associations with introverted and/or inhibited temperament (Clark et al., 1994); regression and structural equation models have supported this hypothesis in adults (Brown et al., 1998; Norton & Mehta, 2007; Trull & Sher, 1994; Watson et al., 1988; Watson, Gamez, & Simms, 2005). Similarly, researchers have found positive affectivity predicts social anxiety in children and adolescents (Chorpita et al., 2000; Hughes & Kendall, 2009). Therefore, studies of positive affectivity suggest associations with both depressive and social phobia symptoms in adults and children.

1.1.3 Physiological Hyperarousal

Physiological hyperarousal was originally modeled as a second-tier in the Tripartite Model; however, some studies have cast doubt on this configuration. Among a clinical sample of children and adolescents, Joiner et al. (1996) found a nonhierarchical, three-factor model had
the best fit. Similarly, another single-level model in children and adolescents had good structural fit with three-factors that approximated the components of the Tripartite Model (Chorpita, Albano, & Barlow, 1998). Evidence has also cast doubt on the proposal that physiological hyperarousal applies broadly to all anxiety domains. Among adults and children, studies have noted an adequate fit when hyperarousal is indicated only by specific anxiety symptoms, primarily the fear-based disorders (e.g., phobias, separation anxiety, panic, etc.; Brown et al., 1998; Chorpita et al., 1998; Chorpita et al., 2000; Joiner et al., 1996).

### 1.2 Integrative Hierarchical Model of Anxiety and Depression

The Integrative Hierarchical Model of Anxiety and Depression (IHM; Mineka et al., 1998) was born out of the Tripartite Model and the previously noted lack of support for the uniform relation of physiological hyperarousal to all anxiety domains. Within the IHM, the hierarchical nature of the Tripartite Model was maintained, however, the content of each level was revised. As its name suggests, the IHM integrated the general distress factor of Tripartite Model with the hypothesis that the various internalizing domains are closely related but ultimately distinct (see Brown et al., 1998; Zinbarg & Barlow, 1996). Overall, the IHM does not propose specific lower level factors, rather it suggests a basic structure where a multitude of symptoms or emotions are co-related among the internalizing disorders. Like the Tripartite Model, the IHM was not specifically proposed as a model of developmental psychopathology; rather, it described the structural similarities and differences between internalizing symptoms themselves.

The IHM is structured in at least two levels of common factors (though more are possible in the model). The first level is a general factor common to all internalizing disorders, and
largely remains operationalized by negative affectivity. While, Mineka and colleagues argued the general factor relates to all internalizing disorders, they suggested that it might do so with varying degrees of influence. The second level of the model consists of more specific factors that are unique to certain disorders, thus helping to differentiate them. While these differentiating traits were originally discussed as secondary factors, the model did not specify outright the number and structure of these traits (i.e., several, more narrowly defined lower-levels are possible). The unique combination of general and specific factors is then theorized to differentiate between the internalizing disorders. In their proposal, Mineka and colleagues (1998) integrated theoretical and more basic literature by proposing examples of potential secondary factors such as Beck’s Content-Specificity Hypothesis (i.e., anxiety is associated with future-based worries, whereas depression is associated with ruminative cognitions of the past; 1976) and information processing such as memory and attentional biases. However, these factors are not considered inherent to the model, rather they represent examples of factors that fit the notion of discriminating secondary traits.

An example of a model using the IHM structure is a study by Prenoveau and colleagues (2010). The authors performed a series of confirmatory factor analyses (CFA) using items from several measures of anxiety and depression in late adolescents. The best-fitting model was a three level structure, in which five specific anxiety symptom domains were indicated. Additionally, two mid-level latent factors – “anxious-misery” and “fears” – were supported; anxious-misery was indicated by items relating to positive affectivity (negative loading), depression, and social anxiety. The factor, termed “fears,” was indicated by items relating to specific phobias, social anxiety, and panic symptoms. Lastly, a higher-order factor, termed “general distress” was indicated by all items. This factor analytic approach described a model in
which a general factor is common to all internalizing psychopathology and two mid-level factors relate largely to underlying emotional similarities between the items (e.g., anxious-misery indicated depressive and introverted/low positive affect symptoms). Overall, the IHM was an improved upon the Tripartite Model in its ability to explain correlations and comorbidities of internalizing psychopathology by allowing for several convergent and divergent factors. While the IHM provides a general heuristic with which to conceptualize the internalizing disorders, the model does not describe etiological similarities per se.

1.3 Triple Vulnerabilities Model of Anxiety

One of the best-known comprehensive models of developmental psychopathology for the internalizing disorders is the Triple Vulnerabilities Model, which proposes a diathesis of biological, psychological, and environmental factors. Evidence from family and twin studies points to a heritable component of anxiety and depression, suggesting a common biological etiology (Kendler, Neale, Kessler, Heath, & Eaves, 1992a; 1992b; Last, Hersen, Kazdin, Francis, & Grubb, 1987; Murray & Sines, 1996; Turner, Beidel, & Costello, 1987). Barlow (2002) and Chorpita and Barlow (1998) integrated biological evidence with Clark and Watson’s conception of trait affectivity when they proposed the first vulnerability: a genetic or biological risk factor, which can include personality (i.e., neuroticism), propensity for negative affectivity, or behavioral inhibition/propensity for autonomic arousal. The second vulnerability is a trait psychological/cognitive factor, which interacts with biological traits. This second vulnerability is similar to Bandura’s (1988) self-efficacy theory and includes a propensity to believe in the uncontrollability or unpredictability of experiences, as well as an internal attributional style (i.e., personal responsibility for control is attributed to the self rather than externally). It has been
suggested that early environmental experience and biological vulnerabilities contribute to the development of a schema consisting of beliefs in decreased personal control and increased personal responsibility (Barlow, 2002; Chorpita & Barlow, 1998; Chorpita, Brown, & Barlow, 1998). The combination of the biological and psychological/cognitive vulnerabilities is thought to result in a general proclivity for anxiety that is narrowed in focus toward specific domains (i.e., the categorical anxiety disorders) via interaction with negative environmental experiences. Environmental experiences operate through social learning and conditioning such as behavioral modeling and negative verbal information transmission. The domains of social learning then determine the focus of the anxiety; for example, aversive social interactions may result in social phobia in persons with the biological and psychological predispositions.

The primary difference between the IHM and the Triple Vulnerabilities Model is the focus on common etiology rather than overlapping symptomatology. Where the IHM suggests a higher-order affective symptom experience, the Triple Vulnerabilities Model suggests an underlying biological etiology common to anxiety and depression. Negative affectivity/neuroticism is theorized as one such biological factor due to having relationships with the internalizing disorders, as well as being heritable traits (Baker, Cesa, Gatz, & Mellins, 1992; Jang, McCrae, Angleitner, Riemman, & Livesley, 1998; Lake, Eaves, Maes, Heath, & Martin, 2000; Neiss et al., 2005). Barlow (2002) also proposed that the behaviorally inhibited temperament represents another biological vulnerability in the Triple Vulnerabilities Model due to its grounding in neurobehavioral theory.
1.3.1 Behavioral Inhibition and Approach Systems

Gray (1970; 1982) proposed a neurobehavioral heuristic of personality, in which activation of differing limbic pathways of motivation is thought to produce trait behavioral responding. The first pathway is the behavioral inhibition system (BIS), which is activated by aversive stimuli (i.e., punishment or uncertainty/novelty) and serves to redirect attentional resources toward the stimulus by inhibiting ongoing behaviors and increasing arousal (e.g., freezing, autonomic activation). In contrast, the behavioral approach system (BAS) is primarily thought to respond to pleasurable or rewarding outcomes by increasing behavioral responses (e.g., approach, impulsivity, avoidance). According to the theory, the BIS and BAS function relatively independently of one another, a notion that has been empirically supported by weak or no correlations between the constructs (Campbell-Sills, Liverant, & Brown, 2004; Coplan, Wilson, Frohlick, & Zelenski, 2006; Johnson, Turner, Iwata, 2003; Jorm et al., 1999; Vervoort et al., 2010). In Gray’s view, the BIS and BAS underlie the various categories of personality or temperament styles and relate to anxiety and impulsivity.

Hirshfeld et al. (1992) demonstrated that children with stable inhibited behavior were more likely to have an anxiety disorder four-to-five years later compared to children whose inhibited behavior was not a stable trait. Similar relationships to anxiety have been replicated in other family and prospective studies (Biederman et al., 1993; Biederman et al., 2001; Rosenbaum et al., 1991; Rosenbaum et al., 1988). Behavioral inhibition has shown particularly consistent relationships with social anxiety; over the course of a four-year follow-up, inhibited children demonstrated increased social avoidance (i.e., solitary play) during laboratory peer-play observation (Asendorpf, 1991). Longitudinal modeling of parent-rated behavioral inhibition and
anxiety symptomatology in children further revealed that social anxiety symptomatology was significantly predicted by behavioral inhibition (Muris, van Brakel, Arntz, & Schouten, 2011).

Behavioral inhibition has also been linked with GAD and OCD. Among adults, self-reported neuroticism and propensity for BIS activation predicted GAD symptoms over a 2-year period, and this prediction was stronger than for social anxiety (Brown, 2007). Likewise, adults with clinical or subclinical OCD symptoms self-reported greater sensitivity to punishment compared to controls (Fullana et al., 2004). Sensitivity to punishment is synonymous with Gray’s (1982) conception of the BIS. Similarly, two studies have noted adolescents’ socially-oriented behavioral inhibition was moderately-to-strongly correlated with worry and GAD symptoms, but was inconsistently related to OCD symptoms (i.e., unrelated in one study and weakly related in another; Muris, Merckelbach, Schmidt, Gadet, & Bogie, 2001; Muris, Merckelbach, Wessel, & van de Ven, 1999). Children’s self-rated general propensity for BIS activation has also been correlated with GAD symptomatology ratings (Vervoort et al., 2010).

Limited evidence also suggests a relationship between behavioral inhibition and depressive symptomatology, though the relationship is not as robust as with anxiety. A 26-year longitudinal study demonstrated behaviorally inhibited children were at an increased risk for developing depression later in adolescence (Jaffee et al., 2002). Similarly, adolescents’ socially-oriented behavioral inhibition and general propensity for BIS activation demonstrated moderate-to-strong correlations with self-reported depressive symptoms (Coplan et al., 2006; Muris, Meesters, de Kanter, & Timmerman, 2005; Muris, Merckelbach et al., 2001; Muris et al., 1999; Vervoort et al., 2010). Linkages between BIS and depressive symptoms have been noted among adults (Brown, 2007; Campbell-Sills et al., 2004; Johnson et al., 2003; Jorm et al., 1999; Kasch, Rottenberg, Arnow, Gotlib, 2002).
The combination of high behavioral inhibition and low behavioral approach is a proposed vulnerability for depressive disorders, similar to the tripartite model’s combination of low positive affectivity and high negative affectivity (Depue & Iacono, 1989; Kasch et al., 2002). Lower self-reported BAS activation in adults predicted depression symptoms after 8 months even when controlling for initial depression severity (Kasch et al., 2002). Additionally, negative correlations between self-reported propensity for BAS activation and measures of depressive symptomatology have been noted in children (Coplan et al., 2006) and adults (Campbell-Sills et al., 2004; Kasch et al., 2002). However, other studies have found no correlations between BAS propensity and depressive symptoms (Johnson et al., 2003; Jorm et al., 1999; Muris et al., 2005; Vervoort et al., 2010). Overall, consistent evidence has been found to suggest that BIS activation is a trait vulnerability for internalizing disorders. Propensity for low BAS activation has theoretical support as a vulnerability for depression, though current evidence suggests this relationship may be weak and/or less consistent than propensity for BIS activation.

1.4 Cognitive Vulnerabilities

In addition to a biological predisposition, the Triple Vulnerabilities Model proposed a psychological vulnerability for anxiety conceptualized as a broad schema consisting of a lack of perceived control and low predictability of experiences. However, others have expanded this vulnerability to include specific cognitive factors that serve to enhance, specify, and maintain emotional reactions. A cognitive model of fear and phobia by Armfield (2006) resembled the Triple Vulnerabilities Model, though it expanded the general psychological factor to include cognitive traits such as propensity for disgust and perceived level of threat. Though the model
was developed specifically to explain the development of phobias, specific cognitive processes may also present vulnerabilities to other internalizing domains.

Riskind and Alloy (2006) outlined four characteristics used to determine a general cognitive vulnerability: 1) temporal precedence (i.e., presence of the trait before the disorder onset), 2) temporal stability (i.e., a trait versus state characteristic), 3) demonstration of construct validity to the disorder in question, and 4) if theorized to be specific to a disorder, demonstrated discriminant validity to other disorders. Three cognitive traits that meet these characteristics are 1) anxiety sensitivity, 2) intolerance of uncertainty, and 3) thought suppression. Each is discussed below and their relations to internalizing symptom domains are highlighted.

1.4.1 Anxiety Sensitivity

Anxiety sensitivity is defined as the tendency to be concerned with or fearful of physiological arousal due to possible physical and/or psychosocial consequences of the arousal (e.g., rapid heart rate could signal a heart attack, or sweating could be embarrassing; Reiss, 1991). Anxiety sensitivity is therefore, a propensity to misinterpret physiological arousal as threatening. Several researchers have proposed anxiety sensitivity as a cognitive vulnerability for internalizing disorders (Li & Zinbarg, 2007; Olatunji & Wolitzky-Taylor, 2009; Reiss, 1991; Taylor, Koch, & McNally, 1992). Supporting this notion, anxiety sensitivity has been shown to be partly heritable (Stein, Jang, & Livesley, 1999), has been predicted by negative affectivity (Norton et al., 2003; Norton & Mehta, 2007) and is moderately-to-strongly correlated with negative affectivity (Bernstein, Zvolensky, Vujanovic, & Moos, 2009; Joiner et al., 2002), neuroticism (Cox, Borger, Taylor, Fuentes, & Ross, 1999), and propensity for BIS activation (Li & Zinbarg, 2007). Therefore, anxiety sensitivity may be derived from general vulnerabilities.
As its name implies, anxiety sensitivity has shown moderate-to-strong correlations with children’s and adults’ self-reported non-specific anxiety symptoms (Joiner et al., 2002; Taylor, Koch, Woody, & McLean, 1996; Weems, Hammond-Laurence, Silverman, & Ginsburg, 1998) as well as specific anxiety symptoms. Studies have most often examined anxiety sensitivity’s relation and contribution to panic disorder, due to conceptual similarities with physiological phenomenology of panic attacks. Prospective studies have demonstrated anxiety sensitivity is a predictor of the development of panic symptomatology in adults and children (Hayward, Killen, Kraemer & Taylor, 2000; Li & Zinbarg, 2007; Schmidt, Lerew, & Jackson, 1997; Weems, Hayward, Killen, & Taylor, 2002). Anxiety sensitivity has also been investigated within social anxiety; socially phobic adults (without comorbid panic or post-traumatic stress disorders) have demonstrated higher anxiety sensitivity than non-anxious controls (Taylor et al., 1996). Likewise, a meta-analysis found that social phobia is associated with increased anxiety sensitivity compared to non-clinical controls and is consistent with levels in other internalizing domains, with the exception of panic and post-traumatic stress disorders (Olatunji & Wolitzky-Taylor, 2009). Specific domains of anxiety sensitivity have also contributed significant variance to social fears, (Belcher & Peters, 2009).

Relationships between anxiety sensitivity and OCD have been mixed. In one study using a clinical sample of adults, anxiety sensitivity explained a small amount of variance in OCD symptoms, after controlling for depression and other cognitive factors (Calamari, Rector, Woodard, Cohen, & Chik, 2008). Likewise, among a non-clinical undergraduate sample, OCD symptoms were predicted by anxiety sensitivity (Sexton et al., 2003). In contrast, two path models (Norton & Mehta, 2007; Norton et al., 2005) found no significant relationship between anxiety sensitivity and OCD symptoms. Still, elevated anxiety sensitivity has been noted among
adult OCD patients, compared with non-anxious controls (Taylor et al., 1992). A strength of this comparison is that comorbid panic and/or post-traumatic stress diagnoses were excluded from the other groups, lessening potential influences from comorbidity. Overall, meta-analytic findings suggested that anxiety sensitivity within OCD is greater than non-clinical controls and is comparable to most other internalizing disorders excluding panic disorder (Olatunji & Wolitzky-Taylor, 2009).

The majority of research examining anxiety sensitivity in those with GAD has found weak or no relationships between the constructs; three regression analyses, one with a sample of clinical adults, another with a community sample of adults, and the third with a community sample of children, found no predictive relationships to worry symptoms (Chorpita & Daleiden, 2000; Norton et al., 2005; Sexton et al., 2003). Likewise, only a moderate correlation between GAD symptoms and anxiety sensitivity has been noted in children (Weems, Hammond-Laurence, Silverman, & Ferguson, 1997). However, a meta-analysis of adult studies indicated anxiety sensitivity in GAD is greater compared to non-clinical controls but is generally equivalent to other, non-panic internalizing domains (Olatunji & Wolitzky-Taylor, 2009).

A limited number of studies have noted relationships between depressive symptomatology and anxiety sensitivity; predictive models in a clinical sample of adults noted no such relationship (Norton et al., 2005; Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995). Adolescents’ and children’s ratings of anxiety sensitivity, however, have demonstrated relationships with depression symptoms (Lewis et al., 2010; Weems et al., 1997). However, when anxiety symptoms were controlled for, these relationships were weak or non-significant (Joiner et al., 2002; Weems et al., 1997). Therefore, anxiety sensitivity does not appear to be a robust predictor of depression or GAD, but is relevant to social anxiety and OCD.
1.4.2 Intolerance Of Uncertainty

Like anxiety sensitivity, intolerance of uncertainty has been proposed as a general cognitive feature of several internalizing disorders (Boelen, Reijntjes, 2009; Carleton, Mulvogue et al., 2012; Gentes & Rusio, 2011; Koerner & Dugas, 2008; Obsessive Compulsive Cognitions Working Group, 1997). Intolerance of uncertainty is the tendency to perceive, interpret, and respond cognitively, emotionally, and behaviorally to uncertain or ambiguous situations as if they are negative or threatening (Dugas, Schwartz, & Francis, 2004). The construct is conceptually similar to the psychological vulnerability of the Triple Vulnerabilities Model (i.e., low perceived control and a general belief in the unpredictable). Intolerance of uncertainty is thought to be a stable, future-oriented cognitive trait, rather than being present-oriented and state dependent (Carleton, Mulvogue et al., 2012). The latent structure of intolerance of uncertainty has been shown to be dimensional in nature rather than categorical, supporting the notion of a continuous vulnerability trait (Carleton, Weeks et al., 2012). Measures of intolerance of uncertainty have demonstrated good reliability correlations over periods of up to 5 weeks, suggesting relative temporal stability of the trait (Dugas, Freeston, & Ladouceur, 1997; Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994). Further, among adults and adolescents, intolerance of uncertainty has been predicted by negative affectivity/neuroticism, making it a likely secondary vulnerability for internalizing psychopathology (Boelen, Vrinssen, & van Tulder, 2010; Norton & Mehta, 2007; Sexton et al., 2003; van der Heiden et al., 2010).

The largest body of research on intolerance of uncertainty has been applied to GAD; the construct is considered a key feature of the disorder (Koerner & Dugas, 2006). Among youth, a 5-year prospective follow-up found adolescents’ intolerance of uncertainty was predictive of
worry longitudinally (Dugas, Laugesen, & Bukowski; 2012). Similarly, experimental manipulation of situational uncertainty (Ladouceur, Gosselin, & Dugas, 2000) and increased intolerance of uncertainty self-talk (Rosen and Knäuper, 2009) resulted in greater self-reported worry, suggesting a causal relationship. Likewise, several non-causal studies among adults and children have demonstrated intolerance of uncertainty predicted GAD symptoms, even when controlling for trait negative affect/neuroticism in some studies (Boelen et al., 2010; Dugas et al., 2007; Freeston et al., 1994; Holaway, Heimberg, & Coles, 2006; Kertz & Woodruff-Borden, 2013; Norton & Mehta, 2007; Norton et al., 2005; Sexton et al., 2003; van der Heiden et al., 2010). Lastly, those with either primary or secondary GAD endorsed the greatest levels of intolerance of uncertainty compared with other anxiety disorders and non-clinical controls (Dugas, Marchand, & Ladouceur, 2005; Ladouceur et al., 1999). Overall, evidence suggests a moderate-to-strong relationship between intolerance of uncertainty and GAD in both adults and children.

Depression and GAD share considerable conceptual and statistical overlap (e.g., comorbidity, genetic risk, physiological symptoms, cognitive interference; see Mennin et al., 2008). Not surprisingly then, intolerance of uncertainty has been shown to have significant relationships with depression, though findings are mixed. Among adults, predictive relationships with depression have been found in some studies (Dugas et al., 1997; Dugas et al., 2004; McEvoy & Mahoney, 2011; Norton et al., 2005); however, two other studies, one with community adolescents and the other among undergraduates, have not supported such a relationship (Boelen et al., 2010; Norton & Mehta, 2007). A meta-analysis of correlational studies found a moderate overall relationship between intolerance of uncertainty and depression
that was comparable to those found in OCD and lower than that noted in GAD (Gentes & Ruscio, 2011).

Intolerance of uncertainty was identified as one of the major cognitive features of OCD (OCCWG, 1997). Obsessive-compulsive symptoms have significantly contributed to intolerance of uncertainty scores (Boelen & Reijntjes, 2009). Likewise, intolerance of uncertainty has predicted OCD symptoms among clinical and non-clinical adults (Fergus & Wu, 2010; Lind & Boschen, 2009; McEvoy & Mahoney, 2011; Norton & Mehta, 2007), even when controlling for worry, depression, and anxiety sensitivity (Steketee, Frost, & Cohen, 1998). Interestingly, analyses examining OCD symptom subtypes found checking and repeating compulsions predicted intolerance of uncertainty, where other OCD symptoms were not related (Tolin, Abramowitz, Brigidi, & Foa, 2003). A meta-analysis of correlational studies indicated intolerance of uncertainty is moderately-to-strongly related to OCD symptoms; however, this relationship was weaker compared to that found with GAD (Gentes & Ruscio, 2011). Therefore, intolerance of uncertainty appears to significantly contribute to OCD but may have a more specific relationship with symptom subtypes than the general relationship in GAD. To date, no information regarding intolerance of uncertainty in childhood OCD symptoms was found in the literature.

A recent development has been the examination of intolerance of uncertainty in social anxiety. While not specifically implicated in theories of social phobia, diagnostic conceptualizations of the disorder include the experience of fear of embarrassment or negative judgments from others (APA, 2000). Therefore, intolerance of uncertainty may play a role in social anxiety due to the unpredictable or ambiguous nature of social interactions. Studies have noted a predictive relationship between intolerance of uncertainty and social anxiety among
clinically referred (McEvoy & Mahoney, 2011) and non-clinical undergraduate samples (Boelen & Reijntjes, 2009; Carleton, Collimore, & Asmundson, 2010; Norton & Mehta, 2007; Whiting, Jenkins, May, Rudy, Davis, & Reuther, 2013). A similar relationship was found in adolescents as well (Boelen et al., 2010). Strengthening these findings is that these relationships were found when controlling for comorbid and/or correlated traits such as GAD and OCD symptoms, fear of negative evaluation, and trait affect. Interestingly, change in intolerance of uncertainty, resulting from a cognitive-behavioral group therapy with a focus on intolerance of uncertainty, predicted treatment response for social phobia, suggesting a causal relationship (Mahoney & McEvoy, 2012). Overall, the literature supports the role of intolerance of uncertainty as a cognitive trait associated to varying degrees with several internalizing domains including GAD, depression, OCD, and social phobia.

1.4.3 Thought Suppression

Thought suppression, or purposefully attempting to avoid and control unwanted thoughts, may be another cognitive vulnerability for internalizing problems. Deleterious effects of thought suppression and its associations to psychopathology were first hypothesized by Wegner, Schneider, Carter and White (1987). In their influential study, adult participants were told to either think of a white bear or avoid thinking of a white bear (suppression), followed by a period of engaging in the opposite thought condition. Participants who engaged in thought suppression demonstrated greater frequency of intrusive thoughts after ending suppression compared with those who did not engage in thought suppression. The authors termed this paradox, the Rebound Effect and hypothesized similar processes may occur within internalizing psychopathology, such that engagement in thought suppression leads to increased expression of unwanted thoughts.
Though results and methodologies vary, numerous experimental studies have supported the Rebound Effect in a variety of adult samples (Clark, Ball, & Pape, 1991; Clark, Winton, & Thynn, 1993; Janeck & Calamari, 1999; Lavy & van den Hout, 1990; Wegner & Gold, 1995).

Others have proposed a second effect, termed the Immediate Enhancement Effect, where the increase in target thought expression occurs during active suppression, due to increased vigilance for the thought (Wegner & Erber, 1992). Like the Rebound Effect, the Immediate Enhancement Effect has support in the literature but results vary by methodology (Harvey & Bryant, 1999; Janeck & Calamari, 1999; Lavy & van den Hout, 1990; Muris, de Jongh, Merkelbach, Postema, & Vet, 1998; Rassin, Merkelbach, & Muris, 1997; Salkovskis & Reynolds, 1994). A meta-analysis of experimental studies suggested limited support for the Immediate Enhancement Effect, overall, but small-to-moderate effect sizes for the Rebound Effect (Abramowitz, Tolin, Street, 2001). Regardless of the timing of the effect (i.e., during vs. following active suppression), thought suppression appears to increase the occurrence of unwanted thoughts.

The frequent experience of intrusive or negative thoughts is a diagnostic feature common to all internalizing disorders. Researchers have particularly noted similarities between intrusive or ruminative thought processes in OCD, GAD, and depression, suggesting common underlying cognitive processes for these disorders (Brown, Moras, Zinbarg & Barlow, 1993; Morillo, Belloch, & Gracia-Soriano, 2007; Tolin, Worhunsky, & Maltby, 2006; Turner, Beidel, & Stanley, 1992; Wells & Morrison, 1994). Similarly, an experimental study demonstrated thought suppression completely mediated the relationship between negative affectivity and intrusive thought experiences (Lynch, Schneider, Rosenthal, & Cheavens, 2007). Therefore, thought
suppression may be a secondary cognitive trait for disorders involving intrusive thought experiences as their core features.

Thought suppression has been conceptualized as being either adaptive or maladaptive depending upon the strategies used to control unwanted thoughts (Wells & Davies, 1994). Adaptive or neutral strategies for controlling unwanted thoughts are believed to include distraction from the thought, discussion of the thought with another person, and reappraising the thought’s validity, implications, or likelihood. Maladaptive strategies include worrying – either as a distraction from the target thought or as a means of problem solving – and self-punishment (i.e., performing an unpleasant action in response to the thought). As described below, maladaptive thought suppression has been associated with psychopathology, while adaptive strategies are either uncorrelated or negatively correlated with internalizing psychopathology.

Obsessive-compulsive disorder is conceptually defined partly by the experience of actively attempting to “ignore or suppress” (p. 462, APA, 2000) intrusive obsessions or compulsions. Likewise, cognitive models of OCD propose thought suppression is a principal feature of the disorder; Rachman (1998) outlined a cognitive model of obsessions in which distorted beliefs in the significance of intrusive thoughts leads to heightened aversiveness of the thought. Heightened aversiveness then prompts engagement in avoidance and/or neutralization actions, which includes thought suppression. Parkinson and Rachman (1981) noted that the Rebound Effect is thought to increase the frequency of intrusive thoughts but also may prevent habituation thereby maintaining the obsession. Salkovskis (1996) further suggested that overreliance upon thought suppression taxes cognitive resources, leading to poor inhibitory control of thoughts and compulsions, thus the rebound or immediate enhancement effects occur.
Tolin, Abramowitz, Przeworski, and Foa (2002) compared the frequency of thought intrusions between those with OCD, those with social anxiety, and non-anxious controls. The Immediate Enhancement Effect was evidenced among those with OCD but not among those with social anxiety or no anxiety. It is notable that the effect was only seen in those with OCD, suggesting thought suppression differentiates between OCD and social phobia. Studies have also found total engagement in thought suppression and engagement the maladaptive suppression strategies (i.e., worry and punishment) are related to OCD symptoms among clinical and non-clinical adults (Abramowitz, Whiteside, Kalsy, & Tolin, 2003; Amir, Cashman, & Foa, 1997; Tolin, Worhunsky, Brady, & Maltby, 2007; Wegner & Zanakos, 1994; Wells & Davies, 1994). Relatively no information on thought suppression in youth is currently known; until recently, a measure of thought suppression strategies had not been developed for youth. An initial investigation of such a measure among community adolescents supported associations between total thought suppression engagement, use of maladaptive thought suppression strategies, and OCD symptomatology (Whiting, May, Rudy, & Davis, 2014).

Thought suppression is also conceptually related to GAD; the principal feature of GAD is the repeated experience of intrusive thoughts in the form of uncontrollable worries (APA, 2000). As with OCD, several cognitive models of GAD have included thought suppression processes, often referred to as cognitive avoidance. Notably, Borkovec’s Avoidance Model of Worry and GAD theorized that worry functions to prevent exposure to feared stimuli, such as negatively valenced mental imagery (1994; Borkovec, Alcaine, & Behar, 2004). According to the model, worry is a less aversive and less activating linguistic representation of the more distressing visual cognition. Engaging in worry may then reduce arousal and distress. This type of avoidance prevents habituation and serves to maintain the anxiety. Other models of GAD have also
included cognitive avoidance and/or maladaptive emotional coping strategies similar to some thought suppression strategies (Dugas et al., 1997; Ellis & Hudson, 2010; Mennin et al., 2005; Wells, 1995).

Experimentally, Becker, Rink, Roth, and Margraf (1998) found significantly more intrusive, personally relevant worries following suppression of negative thoughts among adults with GAD compared to adults with social phobia and non-anxious controls. Interestingly, emotionally neutral thoughts did not produce group differences, suggesting only suppression of aversive thoughts is associated with GAD. Likewise, frequency of thought suppression and endorsement of maladaptive suppression strategies are related to worry symptoms in clinical and non-clinical adults (Coles & Heimberg, 2005; Wells & Carter, 2009; Wells & Davies, 1994). Among adolescents, total and maladaptive thought suppression, and avoidance of thought triggers each explained unique variance in worry (Gosselin et al., 2007). However, another study using a variant measure of thought suppression did not find a relationship between adolescents’ worry and broad thought suppression when other cognitive variables were included in the model (Laugesen, Dugas, & Bukowski, 2003). Therefore, the evidence suggests cognitive avoidance is a factor related to GAD, though the relationship among adolescents is not well explored.

Cognitive models of depression place great emphasis on intrusive rumination about negative past experiences and subsequent cognitive interpretations of these ruminations, such as attributions regarding self-concept and unrealistic expectations (i.e., catastrophizing, selective abstraction, misattribution of personal responsibility, etc.; Beck, 1976; Beck, Rush, Shaw, & Emery, 1979). Poor thought control via ineffective thought suppression processes are hypothesized to contribute to the maintenance of rumination and depression (Wenzlaff, Wegner, & Roper, 1988). An experimental study found depressed college students experienced the
Rebound Effect for negatively valenced thoughts, while no effect was seen among non-depressed participants (Wenzloff et al., 1988). The authors also noted adaptive thought suppression (i.e., thinking of something positive instead) reduced intrusions compared with negative distraction used more often by the depressed group. In two longitudinal studies, non-depressed adults, who endorsed frequently engaging in thought suppression, reported increased rumination and depression symptoms 7-10 weeks later; however, this effect only occurred when there was an interaction with stress (Beevers & Meyer, 2004; Wenzlaff & Luxton, 2003). Non-experimental research has found a relatively consistent relationship between depression symptoms and thought suppression frequency (Reynolds & Wells, 1999; Watkins & Moulds, 2009; Wegner & Zanakos, 1994; Wenzlaff & Rude, 2002). Likewise, comparisons between symptom measures show similar patterns of association between thought suppression and depression, GAD, OCD, and post-traumatic stress symptoms (McKay & Greisberg, 2002; Reynolds & Wells, 1999; Wells & Carter, 2009). Overall, the literature suggests thought suppression is a cognitive feature of depression, OCD, and GAD and may serve to increase intrusive thought experiences in these populations.

Interestingly, evidence suggests thought suppression is not associated with social phobia. Currently, the applicability of thought suppression to child and adolescent internalizing disorders is largely unknown due to lack of empirical study in this area. Likewise, thought suppression has not been included in comprehensive models of trait and cognitive vulnerabilities, even among adults (described below).
1.5 Synthesis of Hierarchical Models and Cognitive Vulnerabilities

The Tripartite, IHM, and Triple Vulnerabilities Models largely propose analogous heuristics for the structure of underlying common and differentiating factors of internalizing disorders. Overall, these models describe at least two levels of traits, the first of which are stable, general indicators, such as proclivity for positive and negative affect and temperament/personality traits (e.g., neuroticism, extroversion, inhibition.). The second tier consists of narrower traits, which are considered to be stable and broadly applicable to several, but not all, internalizing disorders. Examples of these mid-level factors vary by model; however, the most common factors examined in the literature are two cognitive processes discussed above: anxiety sensitivity and intolerance of uncertainty.

Sexton and colleagues (2003) incorporated elements of the previously described theoretical models and empirical studies of cognitive traits into a hierarchical regression-based path model. Unless performed prospectively, path analysis cannot directly infer causal or developmental relationships. Instead, their model is best conceptualized as examining the convergent and discriminating relationships of hypothesized vulnerability factors at a single moment in time. The hierarchical path model consisted of three levels (see page 1 of Addendum); in accordance with broad theoretical models, the first level included trait neuroticism, which was hypothesized to explain significant variance in higher-order cognitive factors as well as symptomatology directly (i.e., partial, but not full mediation).

The second level of the model consisted of two trait cognitive factors, thought of as vulnerabilities for internalizing disorders: anxiety sensitivity and intolerance of uncertainty. The inclusion of these variables as second-level predictor represents a synthesis of the theoretical and empirical literature. First, the IHM’s notion of converging but also discriminating common
factors was represented. The Triple Vulnerabilities Model’s proposed psychological vulnerability (i.e., increased beliefs in uncertainty) also relate directly to intolerance of uncertainty. Likewise, the vast literature demonstrating common relationships of cognitive traits in the internalizing disorders was replicated and summarized within a single model. The predicted relationships of anxiety sensitivity were based upon theory and empirical evidence; Sexton and colleagues predicted anxiety sensitivity and intolerance of uncertainty would differentially contribute to four anxiety domains: panic symptoms, health anxiety/hypochondriacal fears, OCD symptoms, and GAD/worry symptoms. Specific hypothesized paths included a link between anxiety sensitivity and panic symptoms as well as health anxiety. Intolerance of uncertainty was expected to contribute to GAD symptoms only.

Results of the path model among a sample of non-clinical undergraduates supported the hypotheses, with the exception that neuroticism was not a significant predictor of health anxiety. Further examination of a full predictive model (i.e., all possible paths are drawn) revealed a significant relationship between anxiety sensitivity and OCD symptoms that fully mediated the relationship with neuroticism. Intolerance of uncertainty remained only related to pathological worry (i.e., GAD symptoms).

Norton and colleagues (2005) replicated and expanded the Sexton et al. (2003) model, by including positive affectivity and depressive symptoms, as well as conducting the study within a clinical sample of adults with internalizing disorders. The expanded model incorporated the full distinction of the Tripartite Model’s negative and positive affectivity in addition to the elements of the Sexton et al. (2003) model (see page 2 of addendum). Accordingly, the new model hypothesized positive affectivity would contribute to depression symptoms in addition to negative affectivity. Based on some empirical evidence, positive affectivity was also expected to
contribute to GAD/worry symptomatology. A hypothesized path was also expected between intolerance of uncertainty and depressive symptoms. To replicate the previous model, negative affectivity was expected to relate to the same internalizing domains as found by Sexton et al., (2003).

Using the same regression-based methodology, the expanded model noted some important differences compared with the original. First, negative affectivity did not significantly predict anxiety sensitivity nor did it predict intolerance of uncertainty. Low positive affectivity predicted depression and GAD symptoms as expected. Likewise, anxiety sensitivity and intolerance of uncertainty maintained their predictive relationships with panic and health anxiety but not with OCD. Unlike the previous model, negative affectivity in the clinical sample contributed to health anxiety. Overall, the structural relationships in the Sexton et al. (2003) non-clinical sample were upheld in the Norton et al. (2005) clinical sample, suggesting differential contribution of intolerance of uncertainty and anxiety sensitivity to several internalizing domains.

There were three primary limitations to the replication and original models, however; first, social anxiety was not included as an outcome. Second, the regression-based methodologies used in both studies precluded modeling correlational relationships between the higher order predictor variables and limited the ability to examine overall goodness of fit of the models. Lastly, only single indicator variables were use in the regression models; using several instruments to indicate latent variables within a structural equation model, better ensures construct validity. Therefore, Norton and Mehta (2007) conducted a replication and extension of the Norton et al. (2005) model using structural equation modeling of latent variables (see page 3 of addendum). As with the model from the clinical sample, negative and positive affectivity,
anxiety sensitivity, and intolerance of uncertainty were differential indicators of internalizing psychopathology. However, the model included social anxiety symptoms in place of health anxiety/hypochondriacal fears. Hypothesized paths from the Norton and colleagues (2005) model were modeled, along with an additional path between intolerance of uncertainty and OCD symptoms, based on previous research. Paths were expected between negative affectivity and all internalizing symptom categories. Likewise positive affectivity was expected to contribute to depressive symptoms and social anxiety. Anxiety sensitivity was expected to contribute to panic, social anxiety, and OCD symptoms. Lastly, intolerance of uncertainty was modeled to contribute to all measured symptomatology.

Overall, the structural model produced a good fit for the observed data; however, some paths were not supported despite this good fit. Anxiety sensitivity did not contribute significantly to social anxiety as expected, nor did it contribute to OCD symptoms as previously found. Likewise, positive affectivity did not significantly contribute to GAD/worry or social anxiety symptoms.

The relationships across this series of three models are largely consistent for some variables and less so for others; negative affectivity/neuroticism consistently predicted GAD and depressive symptoms and positive affectivity was a consistent predictor of depressive symptoms. Intolerance of uncertainty was a consistent predictor of GAD and depression symptoms as well. Other paths varied across the models. Among non-clinical participants in the Sexton et al. (2003) and Norton and Mehta (2007) studies, negative affectivity predicted anxiety sensitivity and intolerance of uncertainty; however, this was not replicated in the clinical sample (Norton et al., 2005). Negative affectivity did not relate to OCD symptoms in two of the three models, suggesting a weak contribution to OCD overall. Intolerance of uncertainty predicted OCD
symptoms in the structural equation model by Norton and Mehta (2007) but not in the other undergraduate sample by Sexton et al. (2003), suggesting an unstable or weak relationship. Anxiety sensitivity predicted OCD symptoms in the first model, but was not replicated in the subsequent models. Lastly, as social anxiety was only modeled once, the stability of the relationships modeled is unknown.

These regression and structural models are important examples of the IHM heuristic as well as the growing consensus that anxiety sensitivity and intolerance of uncertainty have broad relations to internalizing disorders. However, two clear limitations of these models exist. First, these models have only been conducted within adult samples, limiting their generalizability to children and adolescents. Therefore, it is currently unknown if these structural relationships in adult psychopathology are adequate representations of childhood psychopathology. Second, other potentially important contributions to internalizing psychopathology have yet to be modeled in this way, namely BIS and BAS, as implicated in the Triple Vulnerabilities Model, and the cognitive variable, thought suppression.

1.6 Rationale

The Tripartite, Triple Vulnerability, and IHM are commonly cited theories for describing the similarities between internalizing disorders. Research has demonstrated support for trait affective and personality vulnerabilities among adults and children. However, models of behavioral inhibition/activation and secondary cognitive factors have been fewer, have focused primarily on anxiety sensitivity and intolerance of uncertainty, and have almost exclusively been conducted with adult samples. Relatively little empirical attention has been given to cognitive processes in child and adolescent internalizing psychopathology despite being conceptualized as
one of the major components of such disorders (see Davis, May, & Whiting, 2011). A greater understanding of the contribution and differential relationships of cognitive factors to child and adolescent psychopathology may lead to better informed interventions. For example, cognitive-behavioral treatments for GAD (Dugas & Ladouceur, 2000) and social anxiety (Mahoney & McEvory, 2012) in adults included specific elements for reducing intolerance of uncertainty and have been found to be effective treatments. Therefore, the current study proposes to expand the literature by replicating and extending the Norton and Mehta (2007) hierarchical model of general affective and trait cognitive predictors of internalizing symptomatology among a sample of adolescents. The aims of the proposed study are twofold: 1) test a direct replication of the adult model within a sample of youth, and 2) extend the replication model by including trait behavioral responding (i.e., BIS/BAS) as a higher-order predictor and explore thought suppression’s influence as an additional cognitive factor of internalizing symptoms.

1.7. Hypotheses

Figure 1 illustrates the proposed hierarchical model for the current study. In the model, outcome variables are intended to approximate the various primary symptoms of their respective disorders. In accordance with the various models and statistical findings described above, trait affect and behavioral predispositions are included as the first level of the proposed model, followed by cognitive factors. The hypothesized model was based upon the relationships modeled by Norton and Mehta (2007) due to finding an overall good structural fit, despite some inconsistencies with previous models.

Hypothesis 1: Negative affectivity and BIS propensity are hypothesized to have significant paths with all internalizing symptom domains.
Figure 1. Proposed Hypothesized Model. Notes: Solid lines denote positive coefficients; dashed lines denote negative coefficients.
Hypothesis 2: Negative affectivity and BIS propensity are hypothesized to predict anxiety sensitivity, intolerance of uncertainty, and frequency of thought suppression.

Hypothesis 3: Low positive affectivity and low BAS propensity are expected to contribute to social anxiety and depressive symptoms.

Hypothesis 4: Anxiety sensitivity is expected to contribute to social anxiety and OCD symptoms.

Hypothesis 5: Intolerance of uncertainty is hypothesized to relate to all internalizing symptom domains.

Hypothesis 6: Total frequency of thought suppression is expected to demonstrate pathways to OCD, GAD, and depression symptoms.

Hypothesis 7: The hypothesized model is expected to explain equivalent variance to that of a fully identified model (see Figure 2), indicating the hypothesized model is parsimonious while explaining the most amount of variance.

Hypothesis 8: A direct replication of the Norton and Mehta (2007) model (see Figure 3) is expected to demonstrate the same pattern of associations between trait affect, intolerance of uncertainty, anxiety sensitivity, and internalizing psychopathology, excluding panic disorder.
Figure 2. Proposed Fully Identified Model. Notes: thick, grey paths are not hypothesized but included in the fully identified model.
Figure 3. Proposed Replication Model. Notes: adapted from Norton and colleagues (2007). Solid lines denote positive coefficients; dashed lines denote negative coefficient.
2. METHODS

2.1 Sample Size Recommendations and A Priori Power Analysis

Several recommendations exist for the minimum sample size required for path analysis using structural equation modeling; a commonly used metric is a ratio of the number of subjects to free parameters. On the upper end, Kline (2011) and Tanaka (1987) have suggested an ideal ratio of 20:1, though this is often impractically high. An alternative for exploratory or preliminary investigations - such as the intent of this study - is to utilize regression-based path modeling (see section 3. Analytic Plan), which allows for smaller sample sizes. An a priori power analysis was conducted using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007). With 6 predictors (most complex planned regression equation) and a conservative small effect size of $f^2=0.25$ (Cohen, 1988), the a priori analysis revealed $n=90$ would achieve adequate power, set at minimum of .95. Using the observed effect sizes displayed in tables displayed in Chapter 4, the smallest observed power across all regression analyses was .97.

2.2 Sample and Recruitment

The only formal inclusion criterion was chronological age between 12 and 17 years inclusively. Adolescents were excluded from participation if they self-reported inadequate ability to speak or read English. No potential participant met this exclusion criterion. Participants were 105 community adolescents, 103 of which were recruited in Louisiana private schools and two from youth sporting events held in Connecticut (mean age = 15.61; SD = 1.63; range = 12-17). Girls comprised 67.6% of the sample. Participants predominantly self-identified as White/Caucasian (83.8%; $n=88$). The remainder identified as Black/African American (11.4%; $n=12$) or Asian (1.9%; $n=2$). Three participants did not report racial identity.
Independent of race, 3.8% (n=3) of participants self-identified as Hispanic. Diagnostically, 15.2% (n=15) of the sample self-reported a history of one or more psychological diagnoses as follows: attention-deficit/hyperactivity disorder (n=6), an anxiety disorder (n=8), depression (n=6), and Aspergers Syndrome (n=2). Independent of self-reported diagnoses, 19 participants reported a history of receiving psychological therapy or counseling.

2.3 Procedure

The Louisiana State University Institutional Review Board approved this study and its consenting/assenting process. Parents provided written informed consent for their child to participate and adolescents provided written informed assent. Individuals with questions could contact the investigators by email, which some parents did to clarify age requirements prior to providing consent. All students at the 6th grade level of participating schools were distributed consent forms, though some may not have turned 12 years of age yet. Those who contacted the investigators for age clarification were told those under 12 years old could not participate, but were allowed to enter the gift-card drawing to be equitable (though none did so). None contacted investigators regarding adverse events or concerns about participation. Participants with parental consent then provided written assent and completed anonymous questionnaire packets individually or in small groups.

A total of 12 schools in Louisiana were approached for recruitment and two agreed. An additional 48 schools were approached in Connecticut, and five in Mississippi, though all either declined or did not respond to several attempts by the investigator to make contact. One youth sports facility in Connecticut was also approached and permitted recruitment during public events. Packets were distributed in one semi-public middle and high school (i.e., state funded
but requiring tuition and application for acceptance), one private secular school for girls (grades 6-12), and one dual-enrollment high school psychology course at a public university in Louisiana. Participation was also offered to spectators during two youth sporting events in Connecticut. Packets were completed either at home, at the sporting events, or during class periods in schools. Questionnaires took approximately 30-45 minutes to complete. Measure packets were created in three different counterbalanced versions to minimize presentation order effects. As an incentive, participants were offered the opportunity to enter a drawing to win one of ten, $10 gift cards to an online music store. A random drawing, utilizing a random number generator, was held at the completion of data collection and the ten selected participants were each mailed a gift card.

2.4 Measures

Demographics questionnaire. A demographics questionnaire, created for this study, included questions regarding age, gender, grade level, and racial/ethnic self-identification. Additional questions regarding current and previous psychological diagnoses and psychological treatment were also included.

Positive and Negative Affectivity Scale for Children (PANAS-C). The PANAS-C (Laurent et al., 1999) is a 27-item self-report measure assessing children and adolescents’ propensity for experiencing differing emotional states. The measure asks respondents to rate the degree to which they generally experience positive and negative emotions on a 5-point Likert-type scale ranging from 1 (not at all or very slightly) to 5 (extremely). In addition to affective experience, the measure also contains items assessing physiological hyperarousal. The PANAS-C was developed as a downward extension of the Expanded Version of the Positive and Negative
Affectivity Scale for adults (Watson & Clark, 1994). Items from the original measure were tested among fourth and eighth grade children for comprehension; any items frequently indicated as unfamiliar or not understood were eliminated. The remaining items were examined in an exploratory factor analysis, which supported a two-factor structure measuring positive and negative affectivity. Good (> .80) or excellent (> .90) internal consistency of the positive and negative affectivity factors has consistently been found among samples of children ranging in age from 7 – 18 years (Chorpita & Daleiden, 2002; Hughes & Kendall, 2009; Joiner & Lonigan, 2000; Laurent et al., 1999). Additionally, good convergent validity for both factors has been noted (i.e., positive affectivity negatively correlates with depression, while negative affectivity positively correlates with anxiety and depression; Hughes & Kendall, 2009; Joiner & Lonigan, 2000; Laurent et al., 1999). Comparisons with the Affect and Arousal Scales (Chorpita & Daleiden, 2002), an alternative measure of children’s positive and negative affect, also indicated moderate-to-strong correlations between the conceptually similar subscales, suggesting both convergent and construct validity (Chorpita & Daleiden, 2002). In the current sample, the Positive Affectivity scale had an internal consistency of .88, while the Negative Affectivity scale was .89.

*Behavioral Inhibition and Behavioral Activation Scales for Children (BIS/BAS-C).* The BIS/BAS-C (Muris et al., 2005) was developed as a downward extension of the adult BIS/BAS (Carver & White, 1994). The BIS/BAS-C was designed to measure the sensitivity or propensity of BIS and BAS activation; to do this, the measure assesses affective reactions to reward and punishment, as well as self-reported behavioral responses to such stimuli. The 20-item BIS/BAS-C is rated on a 4-point Likert-type scale between 1 (not true at all of me) and 4 (very true of me), with higher scores indicating greater propensity for activation. A factor analysis
determined that the BIS/BAS-C has a two-factor structure, with BIS and BAS loading as separate factors (Bjørnebekk, 2009; Muris et al., 2005). Adequate (> .70) to good (> .80) internal consistency of the BIS/BAS-C factors has been found among samples of children and adolescents ranging in age from 8-18 years (Bjørnebekk, 2009; Muris et al., 2005; Vervoort et al., 2010). In the current sample, the BIS scale had an internal consistency of .77, while the BAS scale’s internal consistency was .84.

*Children’s Anxiety Sensitivity Index (CASI).* The CASI (Silverman, Fleisig, Rabian, & Peterson, 1991) is a downward extension of the adult Anxiety Sensitivity Index (Reiss, Peterson, Gursky, & McNally, 1986). The original CASI contained 18-items, which demonstrated a hierarchical factor structure with a single factor indicating general anxiety sensitivity and three sub-factors indicating fear of physiological arousal, fear of publically observable anxiety reactions, and fear of cognitive dyscontrol (Muris, Schmidt, Merckelbach, & Schouten, 2001; Silverman et al., 1999; Walsh, Stewart, McLaughlin, & Comeau, 2004; Wright et al., 2010). Internal consistency of the CASI has consistently been found to be in the good (> .80) range for the total score among children ranging in age from 6-17 years (Muris, Schmidt et al., 2001; Silverman, Ginsburg, & Goedhart, 1991; Silverman et al., 1999; Silverman, Goedhart, Barrett, & Turner, 2003; Walsh et al., 2004; Weems, Costa, Watts, Taylor, & Cannon, 2007; Weems et al., 1998). Likewise, convergence with measures of childhood anxiety and behavioral interoceptive tasks has been noted (Chorpita & Daleiden, 2000; Rabian, Embry, & MacIntyre, 1999; Silverman et al., 1991; Weems et al., 2007; Weems et al., 1998). While a revised version (CASI-R; Muris, 2002) has been developed and has promising psychometric properties, the CASI-R has not been widely tested or used in the literature to date. Therefore the original CASI was used in the current study. Internal consistency of the CASI in the current sample was .87.
**Intolerance of Uncertainty Scale for Children (IUSC).** The IUSC (Comer et al., 2009) is a 27-item self-report measure for children and adolescents. Items on the IUSC were adapted from the *Intolerance of Uncertainty Scale* for adults (Freeston et al., 1994); modifications were made to reduce the metacognitive and linguistic complexity of the measure to be appropriate for children (see Comer et al., 2009 for details). Items are rated on a 5-point Likert-type scale ranging from 1 (not at all) to 6 (very much), with higher scores indicating greater intolerance of uncertainty. The IUSC has not undergone factor analysis but was designed to yield a single score. The IUSC is a recently developed instrument and therefore, has limited psychometric testing. However, an initial examination of the measure indicated excellent (> .90) internal consistency for the total measure among clinical and non-clinical children and adolescents aged 7-17 years (Comer et al., 2009). Likewise, moderate-to-strong convergence was found with measures of anxiety, worry, and self-reported reassurance seeking behavior (Comer et al., 2009). Additionally, receiver operator curve analyses indicated that the IUSC had acceptable detection of children with anxiety and discrimination from community controls (Comer et al., 2009). The IUSC evidenced an internal consistency in the current sample of .93.

**Adolescent Thought Control Questionnaire (TCQ-A).** The TCQ-A (Whiting, May et al., 2014) is a 15-item measure of adolescents’ self-reported engagement in thought suppression. The TCQ-A is an adaptation of the Thought Control Questionnaire for adults (Wells & Davies, 1994). The TCQ and TCQ-A were designed to measure frequency of engaging in several thought suppression strategies. The measure has been examined among adolescents aged 12-18 years. Adolescents rate the frequency at which they use each thought suppression strategy on a 4-point Likert-type scale between 0 (never) and 3 (always). Exploratory and confirmatory factor analyses revealed a 5-factor structure for the TCQ-A, each measuring a unique thought
suppression strategy: distraction (e.g., “I think about something good or happy instead”), social (e.g., “I ask a friend if they have similar thoughts”), reappraisal (e.g., “I tell myself, ‘it’s okay, it’s just a thought.’”), worry (e.g., “I think about a smaller problem I’m having instead.”), and punishment (e.g., “I get mad or frustrated at myself for having the thought”; Whiting, May et al., 2014). Additionally, a total score is derived from summing all 15 items, which indicates overall frequency engaging in any thought suppression strategy; only the total score is proposed to be use in the current study. Initial psychometric analyses of the TCQ-A revealed adequate (.75) internal consistency for the total scale, moderate-to-strong test-retest correlations, and expected convergence and divergence with measures of anxiety and externalizing symptoms respectively (Whiting, May et al., 2014). However, the TCQ-A’s properties have yet to be replicated in other samples. The total score of the TCQ-A demonstrated questionable internal consistency of .64 (Kline, 1999). However, instruments that measure behaviors with low base-rates or those that measure a range of distinct behaviors like the TCQ-A may still be informative, even with questionable internal consistency, as Cronbach alpha is often low in such cases due to the uniqueness of individual items (Cortina, 1993; Kline, 1999; Streiner, 2003).

Social Anxiety Scale for Adolescents (SAS-A). The SAS-A (La Greca & Lopez, 1998) is a self-report measure of social anxiety for adolescents. The SAS-A is a modified version of the Social Anxiety Scale for Children-Revised (SASC-R; La Greca & Stone, 1993). Modifications for the adolescent version were limited to alternate wording of references to childlike behaviors/terminology (i.e., “playing” or “kids”) to terminology relevant to adolescents (i.e., “do things” or “others”; see La Greca & Lopez, 1998). Otherwise, the response options and number of items remained consistent with the SASC-R. The 18-item SAS-A is scored on a 5-point Likert-type scale from 1 (not at all) to 5 (all the time), with higher scores indicating greater social
anxiety. Four filler items are also included but do not contribute to the scoring (e.g., “I like to read”). A three-factor structure was found for the SAS-A: 1) fear of negative evaluation, 2) distress to new situations, and 3) generalized social avoidance (Inderbitzen-Nolan & Walters, 2000; La Greca & Lopez, 1998; Myers, Stein, & Aarons, 2002). Additionally, a total score can be obtained by summing all items. The SAS-A has been found to have adequate (> .70) to excellent (> .90) internal reliability of the subscales among adolescents in grades 6-12 (Inderbitzen-Nolan & Walters, 2000; La Greca & Lopez, 1998; Myers et al., 2002). Likewise, several studies have found convergence of the SAS-A and SASC-R with measures of anxiety, depression, peer acceptance, perceived social competence, friendships, self-esteem, and/or negative affectivity (Ginsburg, La Greca, & Silverman, 1998; Inderbitzen-Nolan & Walters, 2000; La Greca & Lopez, 1998; La Greca & Stone, 1993; Myers et al., 2002). In the current sample, the SAS-A demonstrated internal consistency of .93.

**Obsessive Compulsive Inventory-Child Version (OCI-CV).** The OCI-CV (Foa et al., 2010) is a 21-item self-report measure of obsessions and compulsions among children and adolescents. The OCI-CV is a downward extension of the Obsessive Compulsive Inventory for adults (Foa, Kozak, Salkovskis, Coles, & Amir, 1998). Items are rated on a 3-point Likert-type scale from 0 (never) to 3 (always), with higher scores indicating greater frequency of obsessions and compulsions. The measure yields a total score (sum of all items) as well as six subscale scores indicating OCD symptom subtypes: 1) washing, 2) hoarding, 3) doubting/checking, 4) ordering, 5) obsessing, and 6) neutralizing. Good (> .80) internal consistency of the total scale and each subscale has been noted in samples of children and adolescents ranging in age from 7 to 18 years (Foa et al., 2010; Whiting, May et al., 2014). The OCI-CV has demonstrated strong test-retest reliability correlations and has shown good convergence with parent, child, and
clinician administered measures of OCD and other anxiety constructs (Foa et al., 2010; Whiting, May et al., 2014). In the current sample, the OCI-CV total score demonstrated an internal consistency of .89.

**Penn State Worry Questionnaire for Children (PSWQ-C).** The 14-item PSWQ-C (Chorpita, Tracey, Brown, Collica, & Barlow, 1997) is a self-report questionnaire of general worries for children and adolescents, based upon the Penn State Worry Questionnaire for adults (Meyer, Miller, Metzger, & Borkovec, 1990). Items are rated on a 4-point Likert-type scale between 0 (not at all true) and 3 (always true), with higher scores indicating greater worry. Some items are reverse scored. The PSWQ-C has demonstrated a unifactoral structure (Chorpita et al., 1997; Muris, Meesters, & Gobel, 2001). Previous studies with children and adolescents between 6 and 18 years have found acceptable (>0.75) to excellent (≥0.90) internal consistency (Chorpita et al., 1997; Laugesen et al., 2003; Muris, Meesters et al., 2001; Muris, Meesters, Merckelbach, & Hülsenbeck, 2000). The PSWQ-C has excellent test-retest reliability and has demonstrated adequate convergent and divergent validity (Chorpita et al., 1997; Muris, Meesters et al., 2001). The PSWQ-C demonstrated internal consistency of .95 in the current sample.

**Children’s Depression Inventory 2: Self-Report-Short Form (CDI 2-S).** The CDI 2-S (Kovacs, 2010) is a brief version of the norm-referenced *Children’s Depression Inventory 2* (CDI 2; Kovacs, 2010), which was revised from the original CDI (Kovacs, 1992). The revised version includes updated normative scoring for children and adolescents between 7 and 17 years and also contains new and revised items that capture core features of depression in youth. The CDI 2-S contains 12-items rated on a 3-point scale; items are structured as three statements, which vary in either intensity or frequency of a symptom (e.g., “I am sad once in a while [0]”, “I am sad many times [1]”, and “I am sad all the time [2]”). Children and adolescents are asked to
read the statements for each item and choose the one that best fits their mood experience over the previous two weeks. The CDI 2 and CDI 2-S have demonstrated good (> .80) internal consistency for the total scores (Kovaks, 2010). Likewise, the CDI 2 has adequate convergent and discriminant validity (Kovaks, 2010). The CDI 2-S has also shown strong convergence with the total score of the long form (Kovaks, 2010). Internal consistency of the CDI 2-S in the current sample was .74.
3. ANALYTIC PLAN

3.1 Missing Values Procedure

Missing values were replaced by the within-subject means of completed items on the same subscale or from the total instrument for measures without subscales. Missing values were only replaced if fewer than 10% of items from the instrument were missing, though this method is effective with up to 20% missingness (Downey & King, 1998). Participants missing more than 10% of items on any one instrument were excluded from analyses that involved the incomplete measure(s). One or more missing data points from eleven participants were replaced by the above procedure. Nine participants had >10% missing on an individual subscale or measure, and those participants were excluded from analyses that utilized those missing variables.

3.2 Preliminary Analyses

Sample demographic influences were examined using multivariate analysis of variance. The examined sample characteristics were gender and race. Females reported higher means on the BIS subscale ($\eta^2 = .12$), PANAS-C-NA ($\eta^2 = .09$), CASI ($\eta^2 = .01$), PSWQ-C ($\eta^2 = .13$), and SAS-A ($\eta^2 = .01$; all $p$’s < .05). In terms of race, those who self-reported as Asian endorsed higher scores on the OCI-CV ($p < .05$; $\eta^2 = .07$) as compared to those who identified as White/Caucasian or Black/African American. However, it should be noted that the sample size for those identifying as Asian was prohibitively small to draw conclusions ($n=2$). No other significant racial discrepancies were found. Given the small effect sizes of these demographic differences and inadequate power if the sample were to be split, all subsequent analyses are
performed using the total sample, with potential gender differences noted as a limitation of this investigation.

Tests for outliers, linearity, and normality of the data were performed to ensure assumptions were met. Participant z-scores were examined for each measure, with a cut-off of +/- 3 used to define a substantial outlier. One participant was identified as an outlier on the IUSC and PANAS-C-NA and was therefore removed from analyses. A separate participant had a z-score of 3.11 on the CDI 2-S but was not an outlier on other measures; this participant was not removed, as the z-score was close in range to other participants who were just under the threshold. Mahalanobis Distances were used to test for multivariate outliers; no additional outliers were identified using this method. Therefore, the total sample used for analyses was $n=104$. Normality was partly defined as absolute skewness <2 and absolute kurtosis <7 (West, Finch, & Curran, 1998), which no instrument violated. However, using the Shapiro-Wilk test, several scores were identified as significantly non-normal. Therefore, all subsequent analyses were conducted using bootstrapping with 1000 samples, which is an adequate minimum number of samples for generating accurate confidence intervals (Efron & Tibshirani, 1993). Bootstrapping is a resampling procedure that guards against violations of normality.

### 3.3 Correlational Considerations for Model Revisions

All variables of interest were examined for correlations to demonstrate initial evidence supporting or disconfirming the hypothesized paths in the model. These results were considered for alterations of the model in consort with the literature review above. Correlational results supported the majority of hypothesized pathways, with the exception of paths between BAS and
social anxiety (SAS-S) and depression symptoms (CDI 2-S), and thought suppression (TCQ-A) and depression symptoms (see Table 1); therefore, those paths were removed.

Some additional correlations were also observed, such as positive relationships between BAS and thought suppression (TCQ-A), anxiety sensitivity (CASI) and worry (PSWQ-C), and thought suppression (TCQ-A) and social anxiety symptoms (SAS-A). As noted previously, a review of the literature does not offer strong support to add these pathways to the hypothesized model, despite the correlations found herein. Therefore no additional pathways were included.

3.4 Path Analysis Procedure

The final path model, the fully identified model, and the direct replication model were examined with regression-based modeling (Kline, 1998). Each endogenous variable (i.e., those that are indicated by another variable) was set as a dependent variable in separate regression equations. Standardized regression coefficients (i.e., beta weights) are equivalent to standardized path coefficients in this procedure.

As demonstrated in the preliminary correlational analyses, all symptom measures in the model hold some degree of interrelation. To improve independent estimation, symptom variables not of interest to a pathway are entered in the first block, followed by a second block containing the hypothesized predictor variables. For example, in the regression model predicting generalized anxiety symptoms (PSWQ-C), the SAS-A, OCI-CV, and CDI 2-S are entered first in block 1 to control for shared symptom variance. Then, the hypothesized constructs are entered into block 2 (PANAS-C-NA, BIS, IUSC, and TCQ-A).
Table 1. Correlations, Means, and Standard Deviations

<table>
<thead>
<tr>
<th></th>
<th>M (SD)</th>
<th>BIS</th>
<th>BAS</th>
<th>CASI</th>
<th>CDI 2-S</th>
<th>IUSC</th>
<th>OCI-CV</th>
<th>PANAS-C-NA</th>
<th>PANAS-C-PA</th>
<th>PSWQ-C</th>
<th>SAS-A</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIS</td>
<td>9.25 (3.25)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BAS</td>
<td>24.55 (6.99)</td>
<td>.57**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CASI</td>
<td>30.30 (6.63)</td>
<td>.36**</td>
<td>.133</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CDI 2-S</td>
<td>55.18 (9.64)</td>
<td>.35**</td>
<td>-.16</td>
<td>.36**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IUSC</td>
<td>57.09 (16.89)</td>
<td>.56**</td>
<td>.01</td>
<td>.58**</td>
<td>.44**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>OCI-CV</td>
<td>12.47 (7.22)</td>
<td>.45**</td>
<td>-.00</td>
<td>.52**</td>
<td>.41**</td>
<td>.64**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PANAS-C-NA</td>
<td>29.31 (9.42)</td>
<td>.42**</td>
<td>.14</td>
<td>.54**</td>
<td>.58**</td>
<td>.53**</td>
<td>.55**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PANAS-C-PA</td>
<td>38.30 (8.88)</td>
<td>-.18</td>
<td>.34*</td>
<td>-.21*</td>
<td>-.45**</td>
<td>-.26*</td>
<td>-.25*</td>
<td>-.31**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSWQ-C</td>
<td>17.11 (9.76)</td>
<td>.65**</td>
<td>.06</td>
<td>.56**</td>
<td>.45**</td>
<td>.67**</td>
<td>.59**</td>
<td>.67**</td>
<td>-.23*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAS-A Total</td>
<td>46.48 (14.67)</td>
<td>.67**</td>
<td>-.04</td>
<td>.58**</td>
<td>.47**</td>
<td>.56**</td>
<td>.46**</td>
<td>.54**</td>
<td>-.28*</td>
<td>.54**</td>
<td></td>
</tr>
<tr>
<td>TCQ-A Total</td>
<td>15.64 (4.69)</td>
<td>.35**</td>
<td>.29*</td>
<td>.36**</td>
<td>.06</td>
<td>.36**</td>
<td>.31**</td>
<td>.37**</td>
<td>.13</td>
<td>.34**</td>
<td>.36**</td>
</tr>
</tbody>
</table>

Notes: *p < .05; **p < .01; BIS = Behavioral Inhibition Scale; BAS = Behavioral Activation Scale; CASI = Children’s Anxiety Sensitivity Index; CDI 2-S = Children’s Depression Inventory-2 Short Form; IUSC = Intolerance of Uncertainty Scale for Children; PANAS-C = Positive and Negative Affectivity Scales; OCI-CV = Obsessive Compulsive Inventory-Child Version; PSWQ-C = Penn State Worry Questionnaire for Children; SAS-A = Social Anxiety Scale-Adolescent Version; TCQ-A = Adolescent Thought Control Questionnaire.
3.5 Model Comparison Procedures

To test hypothesis #7, a fully identified model is presented for comparison. A fully identified model is one in which paths are drawn from every predictor variable to every measure of symptomatology, regardless of theory. This contrast procedure is examines the parsimony of the hypothesized model and also further explores the non-hypothesized significant correlations noted in section 3.3.

The hypothesized and fully identified models are contrasted by an examination of the variance accounted for by each. This is accomplished by comparing the generalized squared multiple correlation of each model ($R^2_M$; see Kline, 1998). The $R^2_M$ for each is calculated by the following formula, where $q$ is the number of regression equations in the model:

$$R^2_M = 1 - (1 - R^2_1)(1 - R^2_2)...(1 - R^2_q)$$

Once the $R^2_M$ is calculated for each model, the following formula contrasts the explanatory power of each, where $Q$ represents the difference in variance explained, ranging from zero to one, with larger values indicating better fit of the hypothesized model:

$$Q = \frac{1 - R^2_{full}}{1 - R^2_{hypothesized}}$$

Next, a significance test is performed by converting $Q$ to a $\chi^2$ statistic with the degrees of freedom being the number of paths omitted from the hypothesized model as compared to the full model. The following formula is used to achieve this conversion, where $N$ is the sample size:

$$\chi^2_Q = -(N - df) \log Q$$

Lastly, to address hypothesis #8, the direct replication model is calculated using the regression-based modeling procedures discussed in section 3.4. To form the direct replication model, paths originally drawn in by Norton and Mehta (2007) are replicated and additional variables, such as BIS/BAS and thought suppression are removed.
4. RESULTS

4.1 Hypothesized Model Results

As expected, temperament traits (negative affectivity and behavioral inhibition) significantly and positively contributed to anxiety sensitivity (39% variance explained), intolerance of uncertainty (39% variance explained), and thought suppression (17% variance explained). Table 2 presents regressions on cognitive vulnerabilities and Figure 4 displays the revised hypothesized path model with standardized path coefficients (i.e., beta weights).

Results were less consistent with hypotheses when examining regressions on symptom level variables (see Table 3). Social anxiety symptoms were only predicted by behavioral inhibition (56% variance). Likewise, only intolerance of uncertainty contributed significantly to OCD symptoms (49% variance). Negative affectivity, behavioral inhibition, and intolerance of uncertainty were significantly related to worry (GAD symptoms) with 65% explained variance. Finally, depression symptoms were significantly explained (45%) by negative affectivity and lack of positive affectivity only.

Table 2. Regressions Predicting Cognitive Vulnerabilities of Hypothesized Path Model

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Predictors</th>
<th>Regression Coefficients</th>
<th></th>
<th></th>
<th>R²</th>
<th>f²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Unstandardized (std. err.)</td>
<td>Standardized (beta)</td>
<td>95% Confidence Interval</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. CASI</td>
<td>PANAS-C-NA</td>
<td>.23** (.08)</td>
<td>.32</td>
<td>.08 – .38</td>
<td>.39</td>
<td>.65</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>.83** (.20)</td>
<td>.40</td>
<td>.47 – 1.26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. IUSC</td>
<td>PANAS-C-NA</td>
<td>.52** (.18)</td>
<td>.29</td>
<td>.16 – .88</td>
<td>.39</td>
<td>.59</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>2.21** (.48)</td>
<td>.42</td>
<td>1.28 – 3.20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. TCQ-A</td>
<td>PANAS-C-NA</td>
<td>.12* (.06)</td>
<td>.25</td>
<td>-.00 – .23</td>
<td>.17</td>
<td>.20</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>.33* (.14)</td>
<td>.23</td>
<td>.04 – .59</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes: * p < .05; ** p < .01, f² = Cohen’s f² effect size; all values obtained using 1000 bootstrapped samples
Figure 4. Hypothesized Model. Notes: * p< .05, ** p<.01. Solid lines denote positive coefficients; dashed lines denote negative coefficients.
Table 3. Regressions Predicting Internalizing Domains of Hypothesized Path Model

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Predictors</th>
<th>Unstandardized Coefficients (std. err.)</th>
<th>Standardized Coefficients</th>
<th>95% Confidence Interval</th>
<th>$R^2$</th>
<th>$f^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. SAS-A</td>
<td>PANAS-C-NA</td>
<td>.14 (.18)</td>
<td>.09</td>
<td>- .21 – .48</td>
<td>.56</td>
<td>1.27</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>1.89** (.43)</td>
<td>.42</td>
<td>.99 – 1.74</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>- .09 (.13)</td>
<td>- .06</td>
<td>- .35 – .17</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CASI</td>
<td>.42 (.23)</td>
<td>.19</td>
<td>- .01 – .90</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.12 (.09)</td>
<td>.14</td>
<td>- .05 – .32</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. OCI-CV</td>
<td>PANAS-C-NA</td>
<td>.11 (.11)</td>
<td>.14</td>
<td>- .12 – .32</td>
<td>.49</td>
<td>.96</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>-.10 (.24)</td>
<td>-.04</td>
<td>- .57 – .36</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CASI</td>
<td>.13 (.13)</td>
<td>.13</td>
<td>- .13 – .36</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.15** (.05)</td>
<td>.36</td>
<td>.06 – .24</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCQ-A</td>
<td>.07 (.13)</td>
<td>.05</td>
<td>- .19 – .30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. PSWQ-C</td>
<td>PANAS-C-NA</td>
<td>.34** (.10)</td>
<td>.33</td>
<td>.14 – .52</td>
<td>.65</td>
<td>1.86</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>.94** (.30)</td>
<td>.31</td>
<td>.34 – 1.44</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.16** (.06)</td>
<td>.28</td>
<td>.05 – .28</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCQ-A</td>
<td>-.02 (.15)</td>
<td>-.01</td>
<td>- .35 – .23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. CDI 2-S</td>
<td>PANAS-C-NA</td>
<td>.38* (.16)</td>
<td>.34</td>
<td>.07 – .71</td>
<td>.45</td>
<td>.82</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>-.19 (.38)</td>
<td>-.06</td>
<td>-.95 – .55</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>-.29* (.12)</td>
<td>-.27</td>
<td>-.51 – .05</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.02 (.07)</td>
<td>.03</td>
<td>- .11 – .16</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes: * $p < .05$; ** $p < .01$, $f^2$ = Cohen’s $f^2$ effect size; presented results are from block 2, after controlling for shared symptom variance in the first block; all values were obtained using 1000 bootstrapped samples.

4.2 Fully Identified Model Results

To demonstrate parsimony of the hypothesized model, a fully identified model was evaluated in which all possible pathways were included in analyses (see Figure 5). Fully identified pathways indicating cognitive vulnerabilities are displayed in Table 4 below. Overall, results of the fully identified model were highly similar to the hypothesized model; negative affectivity and behavioral inhibition contributed 40% of the variance in anxiety sensitivity and intolerance of uncertainty scores separately. While not hypothesized initially, positive
Figure 5. Fully Identified Model. Notes: grey paths are those omitted from the hypothesized model. Path coefficients omitted for ease of reading; see tables 4 and 5 for standardized path coefficients.
affectivity added additional variance to thought suppression scores, along with negative
affectivity and behavioral inhibition (24% variance explained).

No additional pathways indicating symptom types were significant beyond those found in
the hypothesized model above. As before, only behavioral inhibition contributed positively and
significantly to social anxiety scores (57% variance explained). Only intolerance of uncertainty
explained significant variance in OCD symptoms (49%). Negative affectivity, behavioral
inhibition, and intolerance of uncertainty remained the only significant contributors to variance
in worry scores (65%). Finally, when fully identified, only negative affectivity explained
variance in depression scores (47%); lack of positive affectivity did not remain significant. Fully
identified pathways indicating symptom domains are displayed below in Table 5.

Table 4. Regressions Predicting Cognitive Vulnerabilities of Fully Identified Path Model

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Predictors</th>
<th>Regression Coefficients</th>
<th>95% Confidence Interval</th>
<th>R²</th>
<th>f²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Unstandardized (std. err.)</td>
<td>Standardized (beta)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. CASI</td>
<td>PANAS-C-NA</td>
<td>.22** (.07)</td>
<td>.31</td>
<td>.08 – .36</td>
<td>.40</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>.83** (.20)</td>
<td>.40</td>
<td>.44 – 1.22</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>-.04 (.07)</td>
<td>-.06</td>
<td>-.19 – .09</td>
<td></td>
</tr>
<tr>
<td>2. IUSC</td>
<td>PANAS-C-NA</td>
<td>.47** (.18)</td>
<td>.26</td>
<td>.09 – .80</td>
<td>.40</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>2.18** (.50)</td>
<td>.42</td>
<td>1.26 – 3.15</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>-.22 (.16)</td>
<td>-.12</td>
<td>-.52 – .07</td>
<td></td>
</tr>
<tr>
<td>3. TCQ-A</td>
<td>PANAS-C-NA</td>
<td>.15** (.06)</td>
<td>.31</td>
<td>.04 – .26</td>
<td>.24</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>.35* (.13)</td>
<td>.25</td>
<td>.06 – .57</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>.14** (.05)</td>
<td>.27</td>
<td>.05 – .24</td>
<td></td>
</tr>
</tbody>
</table>

Notes: * p < .05; ** p < .01, f² = Cohen’s effect size; all values obtained using 1000 bootstrapped samples.
Table 5. Regressions Predicting Internalizing Domains of Fully Identified Path Model

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Predictors</th>
<th>Unstandardized Coefficients (std. err.)</th>
<th>Standardized Coefficients</th>
<th>95% Confidence Interval</th>
<th>(R^2)</th>
<th>(f^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. SAS-A</td>
<td>PANAS-C-NA</td>
<td>.08 (.18)</td>
<td>.05</td>
<td>-.25 – .42</td>
<td>.57</td>
<td>1.33</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>1.82** (.48)</td>
<td>.40</td>
<td>.85 – 2.77</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>-.14 (.14)</td>
<td>-.09</td>
<td>-.43 – .12</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CASI</td>
<td>.39 (.22)</td>
<td>.18</td>
<td>-.03 – .84</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.10 (.10)</td>
<td>.12</td>
<td>-.09 – .30</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCQ-A</td>
<td>.40 (.25)</td>
<td>.12</td>
<td>-.14 – .84</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. OCI-CV</td>
<td>PANAS-C-NA</td>
<td>.10 (.11)</td>
<td>.14</td>
<td>-.08 – .28</td>
<td>.49</td>
<td>.96</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>-.09 (.26)</td>
<td>-.04</td>
<td>-.61 – .43</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>-.02 (.07)</td>
<td>-.04</td>
<td>-.17 – .11</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CASI</td>
<td>.13 (.13)</td>
<td>.12</td>
<td>-.10 – .36</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.15** (.05)</td>
<td>.35</td>
<td>.05 – .24</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCQ-A</td>
<td>.09 (.13)</td>
<td>.06</td>
<td>-.20 – .37</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. PSWQ-C</td>
<td>PANAS-C-NA</td>
<td>.33** (.10)</td>
<td>.33</td>
<td>.14 – .52</td>
<td>.65</td>
<td>1.86</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>.92** (.30)</td>
<td>.30</td>
<td>.26 – 1.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>.02 (.08)</td>
<td>.02</td>
<td>-.14 – .17</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CASI</td>
<td>.06 (.16)</td>
<td>.04</td>
<td>-.22 – .41</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.16** (.06)</td>
<td>.27</td>
<td>.03 – .28</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCQ-A</td>
<td>-.04 (.15)</td>
<td>-.02</td>
<td>-.35 – .23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. CDI 2-S</td>
<td>PANAS-C-NA</td>
<td>.42* (.15)</td>
<td>.42</td>
<td>.11 – .72</td>
<td>.47</td>
<td>.89</td>
</tr>
<tr>
<td></td>
<td>BIS</td>
<td>-.14 (.40)</td>
<td>-.05</td>
<td>-.97 – .63</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>-.23 (.12)</td>
<td>-.22</td>
<td>-.45 – .01</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CASI</td>
<td>.04 (.16)</td>
<td>-.03</td>
<td>-.35 – .26</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.03 (.07)</td>
<td>.06</td>
<td>-.10 – .18</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCQ-A</td>
<td>-.35 (.18)</td>
<td>-.17</td>
<td>-.75 – .02</td>
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</tr>
</tbody>
</table>

Notes: * \(p < .05\); ** \(p < .01\), \(f^2\) = Cohen’s \(f^2\) effect size; presented results are from block 2, after controlling for shared symptom variance in the first block; all values were obtained using 1000 bootstrapped samples.

4.3 Comparison of Variance Explained

The total variance explained by the hypothesized model (\(R^2_{hyp} = .99\)) was compared to that explained by the fully identified model (\(R^2_{full} = .99\)) via chi-square analysis as described in section 3.5 above. The chi-square was significant, \(\chi^2 (9) = 17.21, p < .05\), indicating the fully
identified model explained greater variance overall. The observed difference in variance explained was 0.22%.

### 4.4 Replication Model Results

To replicate prior work with adult samples, a third model was evaluated that most closely resembles the work of Norton and Mehta (2007). Regression values are presented in Table 6 and standardized path coefficients for this replication model are presented in Figure 6. Negative

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Predictors</th>
<th>Regression Coefficients</th>
<th>95% Confidence Interval</th>
<th>R²</th>
<th>f²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Unstandardized</td>
<td>Standardized (beta)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. CASI</td>
<td>PANAS-C-NA</td>
<td>.37** (.06)</td>
<td>.52</td>
<td>.24 – .50</td>
<td>.27</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. IUSC</td>
<td>PANAS-C-NA</td>
<td>.83** (.17)</td>
<td>.46</td>
<td>.50 – 1.15</td>
<td>.27</td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>-.25 (.18)</td>
<td>-.13</td>
<td>-.59 – .10</td>
<td>.48</td>
</tr>
<tr>
<td>3. SAS-A</td>
<td>PANAS-C-NA</td>
<td>.19 (.19)</td>
<td>.12</td>
<td>-.19 – .58</td>
<td>.48</td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>-.07 (.14)</td>
<td>-.05</td>
<td>-.35 – .20</td>
<td>.48</td>
</tr>
<tr>
<td></td>
<td>CASI</td>
<td>.64* (.25)</td>
<td>.29</td>
<td>.14 – 1.10</td>
<td>.48</td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.17 (.11)</td>
<td>.20</td>
<td>-.05 – .39</td>
<td>.48</td>
</tr>
<tr>
<td>4. OCI-CV</td>
<td>PANAS-C-NA</td>
<td>.12 (.11)</td>
<td>.15</td>
<td>-.10 – .32</td>
<td>.49</td>
</tr>
<tr>
<td></td>
<td>CASI</td>
<td>.13 (.12)</td>
<td>.12</td>
<td>-.11 – .36</td>
<td>.49</td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.15* (.05)</td>
<td>.36</td>
<td>.06 – .25</td>
<td>.49</td>
</tr>
<tr>
<td>5. PSWQ-C</td>
<td>PANAS-C-NA</td>
<td>.39** (.08)</td>
<td>.38</td>
<td>.24 – .56</td>
<td>.60</td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>.04 (.07)</td>
<td>.03</td>
<td>-.10 – .17</td>
<td>.60</td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.20** (.06)</td>
<td>.34</td>
<td>.08 – .32</td>
<td>.60</td>
</tr>
<tr>
<td>6. CDI 2-S</td>
<td>PANAS-C-NA</td>
<td>.38** (.15)</td>
<td>.37</td>
<td>.09 – .68</td>
<td>.45</td>
</tr>
<tr>
<td></td>
<td>PANAS-C-PA</td>
<td>-.29** (.11)</td>
<td>-.27</td>
<td>-.51 – -.07</td>
<td>.45</td>
</tr>
<tr>
<td></td>
<td>IUSC</td>
<td>.01 (.07)</td>
<td>.02</td>
<td>-.12 – .15</td>
<td>.45</td>
</tr>
</tbody>
</table>

Notes: * p < .05; ** p < .01, f² = Cohen’s f² effect size; all values obtained using 1000 bootstrapped samples; regressions 3-6 results are from block 2, after controlling for shared symptom variance in the first block.
Figure 6. Replication Model. Notes: Adapted from Norton and colleagues (2007).* p<.05, ** p<.01. Solid lines denote positive coefficients; dashed lines denote negative coefficients.
affectivity significantly contributed to cognitive vulnerabilities of anxiety sensitivity (27% variance explained) and intolerance of uncertainty (27% variance explained). Lack of positive affectivity did not significantly contribute to intolerance of uncertainty.

Symptom domain pathways were also only partially supported. Variance in social anxiety symptoms was significantly explained by anxiety sensitivity only (48%). Intolerance of uncertainty was the only significant predictor of OCD symptomatology (49% variance explained). Negative affectivity and intolerance of uncertainty predicted worry, though lack of positive affectivity was not significant (60% variance explained). Finally, variance in depression symptoms as significantly explained by negative affectivity and lack of positive affectivity (45%); intolerance of uncertainty did not significantly contribute to depression ratings.
5. DISCUSSION

Developmental psychopathology theory and prior modeling research with adult samples presents a tiered vulnerability heuristic for the development of internalizing symptoms, with trait affective and/or biological vulnerabilities at the top, leading to more specific cognitive vulnerabilities that differentially contribute to symptomatology. In the current study, such a heuristic was applied and tested in an adolescent sample, which has historically been an understudied developmental period, particularly in relation to cognitive aspects of psychopathology. Three models were tested, a hypothesized path model, a fully identified model of comparison, and a model that more directly replicates prior research with adults.

5.1 Negative Affectivity and BIS Hypothesized Pathways

Negative affectivity and BIS have a long history of research suggesting they are temperament or trait biological vulnerabilities for internalizing problems. Current findings partially support this view. The hypothesized path model supports that trait negative affect and behavioral inhibition may be higher-order vulnerabilities for the development of all three cognitive variables: anxiety sensitivity, intolerance of uncertainty, and thought suppression. Interestingly, BIS was more strongly related to the cognitive vulnerabilities than trait negative affect, though both explained significant variance. This is an important - albeit theoretically consistent - finding that has previously been absent in large-scale vulnerability path models. Cognitive-Behavioral Theory suggests behavioral avoidance contributes to increased anxious or depressive cognitions due to limited opportunities for habituation learning to occur (Foa & Kozak, 1986). Results are consistent with this view in that both trait avoidance behavior and propensity for negative affect contributed to trait maladaptive cognitions.
While results clearly demonstrate the contribution of biological vulnerabilities to cognitive ones, results were more mixed for the direct contribution to symptomatology. By definition, social anxiety, obsessions and compulsions, worries, and dysthymic mood involve the persistent or problematic experience of negative affect. Therefore, both trait affect and cognitive variables were expected to contribute directly to internalizing symptoms. This hypothesis was supported for symptoms of GAD and depression, but was not supported for social anxiety or OCD symptoms. However, again supporting the importance of trait behavioral avoidance/inhibition, BIS was the only significant contributor to social anxiety symptoms. The lack of direct higher-order biological vulnerabilities contributing to OCD symptoms suggests differentiation in the experience of those symptoms as compared to the others studied in this model. Potentially, cognitive vulnerabilities are a greater component of OCD in youth as compared to affect or inhibition, as evidenced by the sole significant contribution of intolerance of uncertainty. The doubting/checking subtype of OCD shows stronger relations with intolerance of uncertainty in adults compared to the other subtypes (Tolin et al., 2003). Within this sample, post-hoc examination of the individual subscales on the OCI-CV indicated the doubting/checking subtype was more prevalent than other subtypes (doubting/checking mean = 3.31; obsessions mean = 2.79; hoarding mean = 1.66; washing mean = 1.30; ordering mean = 2.85; neutralizing mean = 0.69). Therefore, it could be that intolerance of uncertainty is a stronger predictor of total OCD symptoms due to predominance of doubting/checking symptoms in this sample. It could also be that instead of merely inhibiting behavior, those with OCD symptoms engage in active, compulsive behaviors to manage obsessions.

A final alternative may be that OCD symptoms in this non-clinical sample, did not meet an adequate threshold for the other expected relationships to emerge. However, this does not
seem likely given that age of onset for OCD is bimodal with an increase in late childhood (around age 11; e.g., “early onset”) and again in early adulthood (around age 23; e.g., “late onset”; Delorme et al., 2005), meaning typical ages for onset are within or near the range of the current sample. Further, OCD symptoms have a dimensional quality, as opposed to taxonic (Haslam, Williams, Kyrios, & McKay, 2005; Olatunji, Williams, Haslam, Abramowitz, & Tolin, 2008), which would suggest adequacy to capture relationships, despite a non-clinical sample. Indeed, models among adult community and clinical samples had similar null or weak relationships with trait affectivity, though trait behavior was not assessed in those studies (Norton et al., 2005; Norton & Mehta, 2007).

Interestingly, negative affectivity contributed as expected to depression symptoms, but BIS did not. This is contrary to common behavioral experiences of depression such as withdrawal from social and leisure activities. It is possible that depression in adolescents may manifest as having a greater emotional component than avoidant behavior. However, the success of behavioral activation therapy for depression in adults does not lend support for this conclusion (for review, see Cuijpers, van Straten, & Warmerdam, 2007). Though, it remains possible behavioral activation for adolescents does not function similarly, as large-scale randomized clinical trials of pure behavioral activation (i.e., in the absence of other cognitive treatment elements) are absent in the literature. Instead, a structural equation modeling study found that the best fitting pathway between BIS and depression symptoms in youth is one in which BIS is a vulnerability for anxiety first, which then leads to depression symptoms (Muris, Mercklebach, Schmidt, Gadet, & Bogie, 2001).
5.2 Positive Affectivity and BAS Hypothesized Pathways

Initial correlational findings did not support the hypothesized contribution of behavioral activation to social anxiety and depression symptoms. Those unsupported hypotheses were therefore removed. Findings from the extant literature have been quite variable regarding the relationship between low behavioral activation and depression or social anxiety and the current findings suggest other factors, chiefly trait affectivity, are more important to these internalizing domains in youth.

Positive affectivity largely performed as expected, however. The combination of elevated negative affectivity and reduced positive affectivity significantly contributed to depression symptoms, as expected according to the Tripartite Model (Clark & Watson, 1991). Thus, along with negative affectivity, the lack of positive affectivity appears to uniquely differentiate depression from other internalizing syndromes.

While there is some evidence in the literature that low positive affectivity is also associated with social anxiety, such a relationship did not emerge in the current study. This null relationship is similar to findings by Treadwell and Kendall (1996) in which frequency of negative, but not positive, self-statements predicted children’s anxiety symptoms and also mediated cognitive-behavioral treatment gains. A possible explanation is that one’s level of positive affectivity is not a vulnerability for social anxiety in young people, but is a result of avoidance behaviors. Kashdan and Stager (2006) partially explain this hypothesis in an experience-sampling (e.g., ecological momentary sampling) study. Socially anxious individuals reported greater positive affect on days when their social anxiety was lowest, and the opposite trend for high-anxiety days. Likewise, the authors found low endorsement of positive daily events on high-anxiety days, suggesting that anxiety may lead to behavioral avoidance, thus
limiting opportunities for positive affect or experiences. In adolescents, the ability to avoid potentially positive daily experiences are reduced due to compulsory education. While still anxious, adolescents may have greater opportunities for everyday pleasurable events (e.g., classes, reading, physical education/activity, etc.), which could have influenced the null pathway in the current study.

5.3 Anxiety Sensitivity Hypothesized Pathways

Pathways between anxiety sensitivity and OCD and social anxiety symptoms were not supported by the current data. These null findings could be due to the multidimensionality of the anxiety sensitivity construct. The factor structure of the Anxiety Sensitivity Index (Reiss et al., 1986) and its various revisions for adult respondents have most often demonstrated a hierarchical factors structure with a single broad construct followed by three or four subfactors relating to physical, cognitive, and social consequences of anxiety sensations (Deacon, Abramowitz, Woods, & Tolin, 2003; Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004; Zinbarg, Barlow, & Brown, 1997). The total score does not differentiate the various anxiety constructs as well as the individual sub-factors (Deacon & Abramowitz, 2006). Specifically, the anxiety sensitivity social concerns dimension is strongly associated, unsurprisingly, with social phobia (Deacon & Abramowitz, 2001; McWilliams, Stewart, & MacPherson, 2000; Zinbarg et al., 1997) as well as socially observable OCD compulsions (e.g., checking, ordering/arranging); Raines, Oglesby, Capron, & Schmidt, 2014). Those with OCD demonstrate greater physical anxiety sensitivity concerns compared to those with other anxiety disorders, excepting panic disorder (Deacon & Abramowitz, 2001). Anxiety sensitivity cognitive concerns (i.e., concern that anxiety sensations indicate loss of mental faculty) is also associated with aggressive, religious, or sexual obsessions
and some neutralizing compulsions (Raines et al., 2014). The cognitive concerns dimension is also commonly associated with chronic worry specifically (Rector, Szacun-Shimizu, & Leybman, 2007).

The CASI (Silverman et al., 1991) has inconsistently demonstrated a similar structure to its adult counterpart in which there is a higher-order single factor (i.e., total score) and either three or four specific subfactors similar in nature to physiological, cognitive, and social concerns (Muris et al., 2001). Therefore, the null findings of the current investigation’s hypothesized model may be due to lack of specificity both in the anxiety sensitivity total score as well as specific domains of obsessions and compulsions. That is, a total score may obscure multidimensional relationships that have emerged in the adult literature. However, the exact facture structure of the CASI (i.e., three versus four subfactors) remains in question and has not been consistently replicated, thus limiting the ability to address this question in the current study.

5.4 Intolerance of Uncertainty Hypothesized Pathways

Two of the four hypothesized relationships between intolerance of uncertainty and internalizing psychopathology were supported. Consistent with cognitive-theory and published adult and youth studies, intolerance of uncertainty was associated with OCD and GAD symptomatology. This lends support to cognitive conceptualizations that suggest cognitive rigidity is a significant and overarching component of OCD and GAD symptom development and maintenance (Carleton, 2012; Dugas, Buher, & Ladouceur, 2004). Likewise, these pathways support the applicability of these cognitive frameworks to youth. Therefore, intolerance of uncertainty may warrant greater attention in therapeutic interventions for adolescents, as has been applied in treatment studies for adults. Dugas and Ladouceur (2000) developed a
cognitive-behavioral treatment for adults with GAD that specifically targets intolerance of uncertainty. Immediate and long-term outcomes (12 months) included significant reductions of intolerance of uncertainty itself and sustained remission of GAD. Interventions that target these relevant higher-order cognitive vulnerabilities have the potential to improve treatment response for youth due to equivalent pathways in adult and youth hierarchical path models such as this.

However, two additional hypothesized intolerance of uncertainty pathways – those leading to social anxiety and depressive symptoms – were not significant. Prior models, which served as the basis for the present investigation, found similar inconsistent relationships; intolerance of uncertainty was associated with depression in a clinical adult sample (Nortal et al., 2005) but that relationship was weaker among a non-clinical adult sample (Norton et al., 2007). Therefore, replication with clinically depressed youth is recommended before conclusions are drawn regarding the influence of intolerance of uncertainty to depressive disorders in adolescents.

Surprisingly, intolerance of uncertainty did not significantly contribute to adolescents’ reports of social anxiety. While a newer empirical line of research in comparison to studies of GAD and OCD, the relationship between intolerance of uncertainty and social anxiety in adults and emerging adults (undergraduates) appears rather robust (Boelen & Reijntjes, 2009; Carleton et al., 2010; Whiting et al., 2013). However, like anxiety sensitivity, there is evidence to suggest that the Inhibitory Anxiety subfactor of intolerance of uncertainty (i.e., uncertainty prevents taking action) largely drives this association with social anxiety (Carleton et al., 2010; Whiting et al., 2013). These subfactor differences have only been published among studies using measures of intolerance of uncertainty for adults. The IUSC (Comer et al., 2009), while adapted for youth,
has not undergone factor analysis and thus a breakdown of subfactor distinctions is premature for the current study but is a needed area of research.

5.5 Thought Suppression Hypothesized Pathways

The pathway between thought suppression and depression symptoms was removed for lack of correlational evidence in the current sample. This pathway was hypothesized based on extant research among adult samples; however, this relationship has been noted inconsistently in prior studies and is typically not of a strong magnitude (Muris, Merckelbach, & de Jong, 1993; Roemer & Borkovec, 1994). Therefore, it is a somewhat unsurprising null finding, though replication with clinically diagnosed youth is needed to make further determinations, due to an extremely limited body of research on this area in young people. Similarly, there is evidence to suggest moderating effects of stress on the relationship between depression and though suppression. Adults who engage in greater thought suppression and who have high levels of stress, demonstrate greater depression symptoms than do those with similarly high thought suppression engagement but low life stress (Beevers & Meyer, 2004). This pattern is consistent with Barlow’s Triple Vulnerabilities Model (2002). It could be that stress levels influenced the relationships tested here, though general stress was not measures to test this hypothesis.

Another possible explanation for null findings is the influence of measurement in the current procedures. Thought suppression, as measured by the TCQ-A (Whiting et al., 2014) and the analogous measure for adults, assesses several methods with which individuals attempt to control or suppress unwanted thoughts. Some methods are conceptualized as adaptive due to negative relationships with psychopathology (i.e., reappraisal and social), and others are either largely neutral (i.e., distraction), or maladaptive (i.e., punishment and worry; Wells & Carter,
2009; Wells & Davies, 1994; Whiting et al., 2014). It is possible that the use of the total score to capture overall thought suppression engagement obscured distinct positive and negative relationships, leading to nonsignificant pathways. The internal consistency of some individual subscales of the TCQ-A was prohibitively low for analyses beyond use of merely the total score, however. Further measure refinement and additional study in clinical samples of youth with internalizing disorders is needed before such analyses should be undertaken.

5.6 Discussion of Comparison to Fully Identified Model

A comparison of the amount of variance explained by the hypothesized model versus the fully identified model was not expected to be significant, indicating parsimony and supporting theory-based relationships in the hypothesized model. While tests resulted in a statistical difference in variance explained, the observed difference was considered not practically significant. This indicates the presumed null paths in the hypothesized model do not meaningfully contribute to the cognitive vulnerabilities and internalizing symptom domains studied here.

A notable exception was the pathway between positive affectivity and thought suppression, indicating that positive affectivity contributed to total thought suppression engagement. This positive relationship runs against expectations. For example, adults who use thought suppression for emotion regulation have increased negative affect and reduced positive affect, while those who engage in reappraisal of emotions show the opposite pattern (Gross & John, 2003). This pattern partly forms the basis for cognitive therapy interventions such as Acceptance and Commitment Theory. The reappraisal component, though, may explain the unexpected result seen herein. Due to limited study overall, and lower internal reliability of the
individual subscales of the TCQ-A in the current sample, the total score was used to represent overall frequency of thought suppression. However, it is likely that a specific relationship between positive affectivity and reappraisal or social thought control drove the unusual positive relationship noted in the fully identified model.

5.7 Replication Model Discussion

The hypothesized model was an extension of research conducted by Sexton et al. (2003) and Norton and colleagues (2005; 2007) in adult samples. As such, the hypothesized model included additional vulnerability constructs (e.g., BIS, thought suppression) believed to also be related to internalizing disorders. However, to more directly replicate prior research, a third model was examined that included only those constructs from published models (see Figure 6).

A minor difference between the hypothesized model, which included a behavioral vulnerability, and the replication model, which excluded that variable, was that anxiety sensitivity significantly contributed to social anxiety symptoms in the replication model. When trait propensity for behavioral avoidance is accounted for in the hypothesized model, this relationship disappears, however. This suggests a direct relationship between behavioral avoidance and sensitivity to physiological arousal. While modeled here as beginning with trait avoidant behavior, the cross-sectional nature of this design cannot exclude the possibility that the inverse relationship is true or that the relationship is bidirectional.

Overall however, the replication model performed similarly to the structural model presented by Norton et al. (2007) in adults. Direct comparison of pathway significance is difficult due to differing statistical methodologies (i.e., regression-based versus structural equation modeling). Broadly though, the cognitive vulnerabilities of anxiety sensitivity and
intolerance of uncertainty were predicted by trait negative affectivity in both models, supporting the hierarchical conceptualization of developmental vulnerabilities. The individual pathways leading to OCD, GAD, and depression symptoms varied slightly between studies but overall support hypotheses and vulnerabilities theory. While the sample size herein precludes use of structural statistical modeling, the similarities between this and the adult model support such application in future research.

5.8 Limitations and Future Directions

A strength of the current investigation is the conservative control employed for potential measurement overlap. However, this level of control may have eliminated relevant overlapping aspects of internalizing symptomatology from study. This work, with its tight control, provides groundwork for future investigations, which might utilize clinical populations to reduce the need for such rigid controls.

While attempts were made to sample from a broad community base, the current findings remain limited by sampling methodology. First, participants were from the community and do not necessarily represent those with clinical diagnoses of an internalizing disorder. Similarly, though distributed to males and females equally, is disproportionate number of females returned responses. Female adolescents are known to show higher rates of internalizing psychopathology broadly compared to males (Lewinsohn et al., 1998), and thus the higher proportion of females somewhat limits generalizability to male youth. Further, the sample size, while largely adequate for these preliminary model statistics, is prohibitively small to perform separate analyses by gender to address the gender imbalance limitation.
Due to the exploratory nature of this study (i.e., application to a new population with limited study), regression-based path modeling statistics were used. That is, regression based analyses allow for smaller sample size and thus, fewer required resources to conduct this study. However, this limits evaluation of overall model fit and direct comparison to the Norton et al. (2007) structural model. However, now that preliminary study has been conducted, alterations to the model and measurement procedures can be employed in future research (e.g., latent variable constructs using multiple measures for each variable). Likewise, this study serves as reasonable support for utilizing increased resources for study in clinical samples and overall larger samples to better speak to goodness of model fit.

While theory suggests a developmental pathway starting with trait affect and behavior, leading to cognitive vulnerabilities and psychopathology, this cross-sectional design cannot evaluate temporal development of psychopathology (i.e., causation). At this juncture, a prospective research design is premature. However, the adequate replication of the adult path model, indicates that adult models of affective, behavioral, and cognitive components of internalizing disorders likely adequately represent the experience of internalizing psychopathology in adolescents. This study therefore presents a foundation on which to refine the model and employ prospective methodology in the future.
6. REFERENCES


APPENDIX – INSTITUTIONAL REVIEW BOARD DOCUMENTS

ACTION ON PROTOCOL APPROVAL REQUEST

TO: Thompson Davis
Psychology

FROM: Robert C. Mathews
Chair, Institutional Review Board

DATE: February 14, 2013

RE: IRB# 3341

TITLE: Trait Affective, Behavioral, and Cognitive Factors of Anxiety and Depressive Symptoms in Children and Adolescents: A Hierarchical Model


Review type: Full ___ Expedited _X_ Review date: 2/15/2013

Risk Factor: Minimal ___ X ___ Uncertain _____ Greater Than Minimal _____

Approved ___ X ___ Disapproved ______

Approval Date: 2/15/2013 Approval Expiration Date: 2/14/2014

Re-review frequency: (annual unless otherwise stated)

Number of subjects approved: 400

Protocol Matches Scope of Work in Grant proposal: (if applicable) ______

By: Robert C. Mathews, Chairman

PRINCIPAL INVESTIGATOR: PLEASE READ THE FOLLOWING – Continuing approval is CONDITIONAL on:

1. Adherence to the approved protocol, familiarity with, and adherence to the ethical standards of the Belmont Report, and LSU’s Assurance of Compliance with DHHS regulations for the protection of human subjects.
2. Prior approval of a change in protocol, including revision of the consent documents or an increase in the number of subjects over that approved.
3. Obtaining renewed approval (or submittal of a termination report), prior to the approval expiration date, upon request by the IRB office (irrespective of when the project actually begins); notification of project termination.
4. Retention of documentation of informed consent and study records for at least 3 years after the study ends.
5. Continuing attention to the physical and psychological well-being and informed consent of the individual participants, including notification of new information that might affect consent.
6. A prompt report to the IRB of any adverse event affecting a participant potentially arising from the study.
8. SPECIAL NOTE: All investigators and support staff have access to copies of the Belmont Report, LSU’s Assurance with DHHS (45 CFR 46) and FDA regulations governing use of human subjects, and other relevant documents in print in this office or on our World Wide Web site at http://www.lsu.edu/rb

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Application for Approval of Projects Which Use Human Subjects

This application is used for projects/studies that cannot be reviewed through the exemption process.

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A Complete Application Includes All of the Following:
(A) Two copies of this completed form and two copies of part B thru F.
(B) A brief project description (adequate to evaluate risks to subjects and to explain your responses to Parts 1&2)
(C) Copies of all instruments to be used.
   "If this proposal is part of a grant proposal, include a copy of the proposal and all recruitment material.
(D) The consent form that you will use in the study (see part 3 for more information.)
(E) Certificate of Completion of Human Subjects Protection Training for all personnel involved in the project, including students who are involved with testing or handling data, unless already on file with the IRB. Training link: (http://phrp.nhrtraining.com/users/login.php)
(F) IRB Security of Data Agreement: (http://research.lsu.edu/files/item26774.pdf)

1) Principal Investigator* Thompson E. Davis III
   "Must be an LSU Faculty Member
   Rank: PhD

   Dept: Psychology Ph: 225-578-1500 E-mail: sed@lsu.edu

2) Co-Investigator(s) please include department, rank, phone and e-mail for each.
   Reanna (Sara) E. Whitting, MA
   203-516-1550 sara.whitting@gmail.com

3) Project Title:
   Trait Affective, Behavioral, and Cognitive Factors of Anxiety and Depressive Symptoms in Children and Adolescents: A Hierarchical Model

4) Proposal Start Date: January 15, 2013
5) Proposed Duration Months: 12

6) Number of Subjects Requested: 400
7) LSU Proposal #: 334

8) Funding Sought From: N/A

ASSURANCE OF PRINCIPAL INVESTIGATOR named above
I accept personal responsibility for the conduct of this study (including ensuring compliance of co-investigators/co-workers) in accordance with the documents submitted herewith and the following guidelines for human subject protection: The Belmont Report, LSU’s Assurance (FWA00003892) with OHRP and 45 CFR 46 (http://www1.od.nih.gov/orhrp.html). I also understand that copies of all consent forms must be maintained at LSU for three years after the completion of the project. If I leave LSU before that time, the consent forms should be preserved in the Departmental Office.

Signature of PI Date

ASSURANCE OF STUDENT/PROJECT COORDINATOR named above. If multiple Co-Investigators, please create a “signature page” for all Co-Investigators to sign. Attach the “signature page” to the application.

I agree to adhere to the terms of this document and am familiar with the documents referenced above.

Signature of Co-PI (s) Date
PARENTAL CONSENT FORM

Project Title: Trait Affective, Behavioral, and Cognitive Factors of Anxiety and Depressive Symptoms in Children and Adolescents: A Hierarchical Model

Performance Site:
Physical Address: Psychological Services Center, LSU, 31 Johnston Hall, Baton Rouge, LA 70803.
Mailing Address: Psychological Services Center, 230 Audubon Hall, Baton Rouge, LA 70803

Investigator: The following investigators are available for questions Monday-Friday, 10:00 a.m.-4:00 p.m.
Thompson Davis III, Ph.D.
Psychology Department, LSU
(225) 578-1494

Reanna (Sara) E. Whiting, M.A.
Psychology Department, LSU
(225) 578-1494

Purpose of the Study: The purpose of this research project is to see how anxiety and depression symptoms are related to specific traits. The traits being examined are adolescents' emotional reactions, behavioral reactions, and thinking styles.

Inclusion Criteria: Adolescents 12-17 years of age whose parents have given consent to participate in the study.

Exclusion Criteria: Adolescents who do not meet the age requirements, are unable to provide assent or whose parents have not consented for participation; non-English speakers; adolescents who do not understand the questionnaires or who have a medical or developmental condition that would prevent their ability to complete the study will be excluded from participating.

Maximum Number of Subjects: The maximum number of participants will be 400.

Study Procedures/Description of the Study: Adolescent participants will be asked to complete questionnaires for the investigators during one 30-minute session at school or in local clinics.

Benefits: Adolescent participants will be entered into a random drawing to win one of ten, $10 gift cards to the Apple iTunes Store online. The drawing will be conducted at the end of data collection and winners will be notified and sent a gift card by postal mail.

Risks/Discomforts: No other risk or discomfort is anticipated other than those associated with completing questionnaires.

Right to Refuse: Participation is voluntary and an adolescent will become part of the study only if both child and parent agree to the child's participation. At any time, either the child or parent may withdraw from the study without penalty or loss of any benefit to which they might otherwise be entitled at that point.

Privacy: Records with identifying information will be kept in a locked facility. Electronic data will be entered without identifying information. Summary results of the study may be published, but no names or identifying information will be included for publication. Participant identity will remain confidential unless disclosure is required by law (e.g., suspected or reported ongoing child abuse or neglect). I understand

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that the investigators are required by law to report any reasonable suspicions of abuse or neglect.

Withdrawal: Participants may withdraw from the study at any time. Parents wishing to withdraw should contact the principal investigator or co-investigators in writing as soon as this decision has been made.

Removal: Participants may be removed from the study without consent if they are believed to be a danger to themselves or others and/or if the investigators believe removal and assessment elsewhere would be in the best clinical interest of the participants.

Unforeseeable Risks: There may be unforeseeable risks to participants of this study as a result of participating, however, steps are taken to minimize any potential foreseeable risks and discomfort.

Study-related illness or injury: In case of medical emergency and in case further psychological attention is needed, we have listed resources below:

**Medical Services**
911 (for emergencies)

**Mental Health Services**
911 (for emergencies)
Psychological Services Center, LSU, (225) 578-1494

Signatures:

The study has been discussed with me and all my questions have been answered. I may direct additional questions regarding study specifics to the investigators. If I have questions about subjects' rights or other concerns, I can contact Robert C. Mathews, Chairman, LSU Institutional Review Board, (225) 578-8992. I agree to participate in the study described above and acknowledge the researchers' obligation to provide me with a copy of this consent form if signed by me.

Parent/guardian Signature _______________________________ Date ____________

*Reader of the consent form, please sign the statement below if the consent form was read to the parent because he/she is unable to read:*

The parent/guardian has indicated to me that he/she is unable to read. I certify that I have read this consent form to the parent/guardian and explained that by completing the signature line above, he/she has agreed to participate and has given permission for the child to participate in the study.

Signature of Reader _______________________________ Date ____________

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Adolescent Assent Form

I, ________________________, agree to be in this study that looks at how my emotions, behaviors, and thoughts may be related to different problems like anxiety, worry, or sadness. I will be asked to answer questions about emotions that I tend to have, ways I tend to behave, and ways I tend to think. I will also be asked about whether I experience problems like anxiety, worry, or sadness. I agree to do my best to answer these questions honestly. I can decide to stop being in the study at any time without getting in trouble.

Adolescent Signature ______________ Date __________ Age __________

Witness Signature* __________________ Date __________

(*Witness must be present for the assent process, not just the signature by the minor.)
Project Report and Continuation Application
(Complete and return to IRB, 130 David Boyd Hall. Direct questions to IRB Chairman Robert Mathews 578-8692.)

IRB#: 3341 Your Current Approval Expires On: 2/14/2014
Review type: Expedited Risk Factor: Minimal Date Sent: 12/2/2013
Pt: Thompson Davis Dept: Psychology Phone: 8-1500
Student/Co-Investigator: Regina Whiting
Project Title: Trait Affective, Behavioral, and Cognitive Factors of Anxiety and Depressive Symptoms in Children and Adolescents: A Hierarchical Model
Number of Subjects Authorized: 400

Please read the entire application. Missing information will delay approval!

I. PROJECT FUNDED BY: LSU proposal #:

II. PROJECT STATUS: Check the appropriate blank(s); and complete the following:

1. Active, subject enrollment continuing; # subjects enrolled: 50
2. Active, subject enrollment complete; # subjects enrolled:
3. Active, subject enrollment complete; work with subjects continues.
4. Active, work with subjects complete; data analysis in progress.
5. Project start postponed
6. Project complete; end date __/__/___
7. Project cancelled: no human subjects used.

III. PROTOCOL: (Check one). Protocol continues as previously approved
Changes are requested
* List (on separate sheet) any changes to approved protocol.

IV. UNEXPECTED PROBLEMS: (did anything occur that increased risks to participants):

State number of events since study inception: ___ since last report ___
If such events occurred, describe them and how they affect risks in your study, in an attached report.
Have there been any previously unreported events? Y/N ___
(If YES, attach report describing event and any corrective action).

V. CONSENT FORM AND RISK/BENEFIT RATIO:
Does new knowledge or adverse events change the risk/benefit ratio? Y/N ___
Is a corresponding change in the consent form needed? Y/N ___

VI. ATTACH A BRIEF, FACTUAL SUMMARY of project progress/results to show continued participation of subjects is justified; or to provide a final report on project findings.

VII. ATTACH CURRENT CONSENT FORM (only if subject enrollment is continuing), and check the appropriate blank:

1. Form is unchanged since last approved
2. Approval of revision requested here with: (identify changes)

Signature of Principal Investigator: Date: 12/18/13

IRB Action: Continuation approved; Approval Expires: 12/19/14
Disapproved
File closed

Signed Date: 12/20/13

Form date: April 16, 2008
Adolescent Assent Form

I, __________________________, agree to be in this study that looks at how my emotions, behaviors, and thoughts may be related to different problems like anxiety, worry, or sadness. I will be asked to answer questions about emotions that I tend to have, ways I tend to behave, and ways I tend to think. I will also be asked about whether I experience problems like anxiety, worry, or sadness. I agree to do my best to answer these questions honestly. I can decide to stop being in the study at any time without getting in trouble.

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Witness Signature* __________________________ Date ___________

(*Witness must be present for the assent process, not just the signature by the minor.)

STUDY APPROVED BY:
Dr. Robert C. Mathews, Chairman
Institutional Review Board
Louisiana State University
130 David Boyd Hall
225-578-8692 / www.lsu.edu/irb
Approval Expires: 12/19/2014
PARENTAL CONSENT FORM

Project Title: Trait Affective, Behavioral, and Cognitive Factors of Anxiety and Depressive Symptoms in Children and Adolescents: A Hierarchical Model

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  - 911 (for emergencies)

- **Mental Health Services**
  - 911 (for emergencies)
  - Psychological Services Center, LSU, (225) 578-1494

**Signatures:**

The study has been discussed with me and all my questions have been answered. I may direct additional questions regarding study specifics to the investigators. If I have questions about subjects' rights or other concerns, I can contact Robert C. Mathews, Chairman, LSU Institutional Review Board, (225)578-8692. I agree to participate in the study described above and acknowledge the researchers’ obligation to provide me with a copy of this consent form if signed by me.

Parent/guardian Signature ____________________________ Date ________________

*Reader of the consent form, please sign the statement below if the consent form was read to the parent because he/she is unable to read:*

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Signature of Reader ____________________________ Date ________________

**STUDY APPROVED BY:**
Dr. Robert C. Mathews, Chairman
Institutional Review Board
Louisiana State University
130 David Boyd Hall
225-578-8692 / www.lsu.edu/jrb
VITA

Reanna (Sara) E. Whiting is a native of Bethel, Connecticut. She graduated *cum laude* with her Bachelor’s of Arts in psychology from Smith College in 2006. Following work as a research assistant for federally and industry sponsored clinical trials at Massachusetts General Hospital and Hartford Hospital/Institute of Living, Sara completed a Master’s of Arts in clinical psychology at Louisiana State University in 2012 under the mentorship of Thompson E. Davis III. She completed her predoctoral clinical internship at the University of Mississippi Medical Center/G.V. (Sonny) Montgomery VA Medical Center Consortium where she specialized in pediatric neuropsychology. She is now a Postdoctoral Fellow in pediatric neuropsychology at Baylor University College of Medicine/Texas Children’s Hospital.