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Individual Differences in Anxiety Sensitivity: The Role of Emotion Regulation and Alexithymia

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The Role of Emotion Regulation and Alexithymia

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Abstract

The literature has shown anxiety sensitivity to be a significant risk factor in the development of pathological anxiety. Recent theoretical models have also emphasized the additional importance of emotion regulation in predicting the development of anxiety disorders. The present study examined the interactive influence of anxiety sensitivity and emotion regulatory strategies on anxiety symptoms in an ethnically diverse sample recruited in Singapore in order to determine the most appropriate anxiety prevention strategies to pursue. Results indicate that emotion regulation skills had a much greater effect on anxiety levels in this non-clinical sample than anxiety sensitivity and, second, that emotion regulation skills partially mediated the effect of anxiety sensitivity on anxiety such that emotion regulation accounted for 77% of the impact. Therefore, instruction in emotion regulation skills provides potentially a far more effective means of preventing anxiety than the targeting of anxiety sensitivity in the cultural groups studied. A second aim of the study was to determine if anxiety sensitivity varies across cultures, and if the difference is accounted for by an individual's awareness, understanding, and ability to communicate his or her feelings (alexithymia). Anxiety sensitivity and alexithymia did not, in fact, vary across the Chinese, Malay, and Indian students sampled. However, Singapore participants in general reported far higher anxiety sensitivity and trait anxiety scores than similar American and international samples. Further, Indian participants had significantly lower anxiety and emotion regulation difficulties, including alexithymia, than Chinese and Malay participants.

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Chapter 1: Review of Related Literature and Aims

Anxiety disorders are the most prevalent among all classes of psychopathology (Kessler, Chiu, Demler, & Walters, 2005), representing an annual cost of more than $42 billion to the American economy (Greenberg et al., 1999). Anxiety disorders are often significantly disabling in core aspects of life, tend to affect functioning, maintain a chronic fluctuating presence, and are associated with high rates of psychiatric hospitalizations, substance abuse, and physical health problems (Kessler et al., 2005; Sareen, Cox, Clara, & Asmundson, 2005). Given the high prevalence and scale of the negative impact of anxiety disorders, the public health and economic burden created by the need to deliver empirically supported treatments or to identify successful prevention efforts for all individuals suffering from anxiety psychopathology is vast.

In the wake of the larger national debate on health-care reform and the fact that significant progress has been made toward the establishment of empirically based treatments, the time is ripe for clinical psychology to move more serioU.S.ly toward the development of preventative treatments (Zvolensky, Schmidt, Bernstein, & Keough, 2006). The potential for effective prevention of anxiety-related psychopathology is further supported by the large body of research that demonstrates a significant overlap among disorders. This overlap is most evident from the high rates of current and lifetime comorbidity (e.g. Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Kessler et al., 1998). Brown et al. (2001) collected data from 1127 patients who were diagnosed with a principal anxiety or mood disorder and found that 55% had at least one additional anxiety or depressive disorder at the time of assessment. This rate increases to 76% when considering additional diagnoses occurring at any time during the patient's life. Given the diagnostic overlap, Zvolensky et al. (2006) argue that good preventative

programs can be developed by beginning with a sound understanding of risk factors that may be common across disorders.

To demonstrate the importance of understanding risk factors, Mineka and Zinbarg (2006) provide the illustration of two pairs of patients who have undergone very similar traumatic experiences but who experience vastly different outcomes:

> *Emily and Marian both had traumatic experiences with dogs. Emily was hiking with her own dog when another dog attacked her and bit her on the wrist. She was terrified. The wound became badly infected and very painful, requiring medical treatment. Marian was walking in the fields when she became terrified by three large, growling dogs that chased her to a fence. One began tearing at her pant legs, but their owner fortunately intervened before she was physically injured. Why did Marian but not Emily go on to develop dog phobia when only Emily was actually bitten by a dog?*
>
> *Ahmet and Hasan were both male Turkish citizens in their 30s who were arrested, imprisoned, and tortured. Ahmet was imprisoned for several years and experienced a great deal of torture; Hasan was imprisoned for several weeks and experienced far less torture (Bas¸og˘lu, 1997). Why did Hasan but not Ahmet develop posttraumatic stress disorder? What accounts for such individual differences?*

Although learning theory provides some understanding of how anxiety disorders develop, there is far less understanding of what causes the disorders to develop in some people and not in others with similar experiences. While an understanding of risk factors could play a great part in the effort to develop preventative programs, such risk-factor research is somewhat

underdeveloped. Efforts to understand risk factors have considered a number of variables, including neuroticism and a general elevation of negative affect (Clark & Watson, 1991).

One promising construct that has received more than its fair share of attention is anxiety sensitivity. A vast literature has developed around anxiety sensitivity in recent years, and evidence has accumulated implicating the anxiety sensitivity construct as a risk factor for the development of panic and other anxiety disorders. Although conceptualized to explain variances in who does and does not develop an anxiety disorder, anxiety sensitivity has been found not only to be specific to anxiety but also to be a predictive factor in other disorders including substance abuse, depression, and eating disorders (Olatunji & Wolitzky-Taylor, 2009). However, significantly less attention has been focused on examining its relations with and distinctiveness from other psychological constructs that focus on a sensitivity to or intolerance of aversive somatic and affective states. In other words, although anxiety sensitivity is an important predictor of the development of anxiety and other disorders, its role in this context can be better understood by carefully considering its complex interactions with other individual-difference characteristics (Zvolensky, Felder, Leen-Feldner, & McLeish, 2005).

One individual-difference characteristic that has gained widespread scholarly interest is emotion regulation. A number of *third wave* behavior therapies (e.g., Acceptance and Commitment Therapy and Dialectic Behavior Therapy) attempt to address difficulties in emotion regulation by educating clients about their emotions, teaching them emotional acceptance and impulse control, and promoting responses that are non-avoidant and in the service of one's values and goals (see Linehan, 1993; Hayes, 2004). Evidence is accumulating on the efficacy of interventions targeting emotion regulation on anxiety and other disorders (Powers, Zum, Vording & Emmelkamp, 2009).

While some attempts have been made to look at the relative contributions of anxiety sensitivity and emotion regulation to anxiety disorders (Tull, Stipelman, Salters-Pedneault, & Gratz, 2009; McDermott et al., 2009; Tull et al., 2007; Mennin, Heimberg, Turk, & Fresco, 2002), these efforts have been limited in number and have been lacking diversity in their sample groups. Many questions remain unanswered, particularly if emotion regulation difficulties compound the effects of anxiety sensitivity in predicting psychopathology, the contribution of emotion regulation to anxiety sensitivity itself, and the value of anxiety sensitivity over and above that of emotion regulation as a predictor of anxiety. Further, emotion regulation itself has been conceptualized as a confluence of factors, and it is unclear which particular factor makes the greatest contribution.

This study examined the relationship between anxiety, anxiety sensitivity, and emotion regulation to understand their relative roles as predictors of psychopathology symptoms among students of differing ethnicities. The study was conducted in a sample of Singaporean individuals of three major ethnicities (Chinese, Malay, and Indian) to extend current findings about the effect of anxiety sensitivity and emotion regulation on anxiety to populations outside of North America. Hierarchical linear regressions were conducted to determine the direction of influence of emotion regulation on anxiety sensitivity and anxiety. The study also aimed to examine the role of alexithymia (which has been conceptualized as a subfactor of emotional dysregulation) in creating differences in the anxiety sensitivity levels of individuals across cultures. Alexithymia has been demonstrated to be elevated in Chinese individuals relative to individuals of other cultures and has been found to be closely correlated with anxiety sensitivity. Therefore, the sample included Chinese individuals as well as individuals from two other cultures to allow for a comparison of mean anxiety sensitivity and alexithymia levels across cultures. It is hoped that

the results will inform efforts to turn an understanding of risk factors into valuable prevention protocols.

The following sections present the literature on the key constructs of this study. Various aspects of anxiety sensitivity are outlined first, including its history, its role in anxiety disorders, and the genetic, familial, and cultural influences that determine an individual's level of anxiety sensitivity. Next, emotion regulation and its key components as conceptualized by Gratz and Roemer (2004) are described, focusing especially on emotional acceptance, the ability to control impulses and act in a goal-directed manner, and the ability to identify and understand one's emotions (the conceptual opposite of alexithymia). Last, the sparse literature that assesses the impact of anxiety sensitivity and the subfactors of emotion regulation on anxiety disorders is examined before key research questions and the hypotheses of this study are presented.

Anxiety Sensitivity

Anxiety sensitivity is defined as the fear of anxiety-related sensations, arising from the belief that these are dangerous in and of themselves. Anxiety disorders are often defined by their eliciting stimuli (Taylor 1995). For example, agoraphobia is defined as the fear of open, public places, and social phobia is defined as the fear of social encounters. While these definitions tell us what elicits these fears, they say little about their causes. To shed light on this, various theoreticians over the last century have commented on the role of the fear of anxiety or the *fear of fear* in the etiology of anxiety disorders (Fenichel, 1945; Freud, 1895). Freud's personal experience with panic attacks may have led him to remark that, in the case of agoraphobia, the recollection of an anxiety attack often drives us to avoid situations from which we cannot escape (Freud, 1895). Fenichel (1945) proposed that many *anxiety hysterias* develop out of defensive processes in which the anxiety caused by unconscious conflict is focalized on anxiety sensations.

Rosenberg (1949) took the idea further and conceptualized the capacity to tolerate anxiety sensations as an individual difference variable, while Frankl (1959) argued that individuals who have been humiliated by their blushing or stuttering often end up worrying so much about their recurrence that the fear of such anxiety symptoms produces the very symptoms that they fear might occur.

Although much has been said of the role of the *fear of fear*, the clinical value of identifying and treating it has been most clearly demonstrated in recent years in the area of panic attacks and agoraphobia. When Reiss and McNally (1985) reviewed the historical literature on the fear of anxiety sensations and modern studies on panic and related disorders, they found striking similarities in the conceptualizations of anxiety sensitivity or a fear of fear across theoretical orientations. Drawing from psychodynamic, cognitive, and learning theories, they defined anxiety sensitivity as the fear of anxiety-related sensations, based on the belief that these have harmful physical, psychological, and/or social consequences. Since Reiss and McNally's paper, more than 600 articles have been published on the topic of anxiety sensitivity, relating it to the etiology and treatment of anxiety and other disorders including depression, pain, and substance abuse.

In these studies, anxiety sensitivity has been conceptualized as an individual difference variable that increases the intensity of emotional reactions (particularly anxiety). Persons with high levels of anxiety sensitivity are more likely to misinterpret unexplained physical symptoms of anxiety and to see them as dangerous. To illustrate, a person who is phobic about driving would experience anxiety when required to drive. If that person had elevated anxiety sensitivity, then he or she would also become anxious about being anxious. Thus, the fear of driving would be amplified. Although there is some controversy over the exact factor structure, it appears that

there are at least three basic dimensions of anxiety sensitivity: (a) fear of publicly observable anxiety reactions (e.g., fear of sweating from beliefs that this will lead to embarrassment), (b) fear of cognitive dyscontrol (e.g., fear of concentration difficulties arising from beliefs that such difficulties are the harbingers of insanity), and (c) fear of somatic sensations (e.g., fear of palpitations arising from beliefs that cardiac sensations lead to heart attacks; Taylor, 1995).

Although this definition is most often attributed to Reiss's expectancy theory (1991), Reiss himself acknowledges that the definition above and the growing literature surrounding anxiety sensitivity has its roots in a number of places, namely in early psychodynamic theory (most prominently with Frankl, 1959), in cognitive theory, and in learning theory. According to this theory, expectations about the danger of various stimuli are typically learned through conditioning or other learning processes, but the individual's underlying level of anxiety sensitivity is the factor that helps to explain differences in the development and maintenance of different levels of fears between people with similar experiences (Reiss, 1991; Reiss & McNally, 1985). Therefore, if someone is high in anxiety sensitivity, she is more likely to develop a fear or phobia in a situation that activates this fundamental fear. In this way, anxiety sensitivity has come to be thought of as a dispositional variable that functions as an *anxiety amplifier* (Reiss 1991).

While expectancy theory takes a trait-like perspective, this is not universally adopted in the literature. Behavioral or learning theories stress classical and operant conditioning for the development and maintenance of the construct respectively. Unlike expectancy theory (Reiss, 1991), which postulates that individuals differ in anxiety sensitivity prior to the experience of a panic attack, in their work on agoraphobia, Goldstein and Chambless (1978) made the assumption that the fear of fear results from having experienced a panic attack. After even one

attack, these bodily sensations may become conditioned to elicit the fear associated with the panic attack. For instance, if a person experiences shortness of breath during the attack, he may experience fear the next time he is short of breath, even if only from climbing a flight of stairs, because this sensation has become a conditioned stimulus for fear. Avoiding activities that produce the feared sensation is operantly conditioned in that it is reinforced by escape (negative reinforcement) as well as possibly by positive social reinforcement in the form of attention (Goldstein & Chambless, 1978). Once the fear of anxiety-related sensations has developed, the avoidant behavior prevents the person from coming into contact with the sensations related to anxiety (e.g., increased heart rate, shortness of breath), and the individual does not learn that anxiety sensations are not inherently dangerous.

While behavioral models focus on classical and operant conditioning, cognitive models of anxiety focus on the role of cognitive misinterpretations in the development of anxiety. In writing on expectancy theory, Reiss (1991) acknowledges that anxiety sensitivity is defined in terms of irrational beliefs, and so is similar to the cognitive model of panic attributable to Clark (1986) and can be traced to Beck's (1979) writings on depression. The cognitive model of anxiety sensitivity revolves around the misinterpretation of bodily sensations as signs of danger. A cycle of escalating anxiety begins when a person perceives a stimulus as a threat. The stimulus causes anxiety and the misinterpretation of bodily sensations as signs of danger, which in turn leads to more anxiety-relevant internal sensations. Eventually, the experience of a panic attack or other traumatic consequences reinforces the catastrophic misinterpretations, thus maintaining the person's fear of the anxiety-related bodily sensations.

The roles of nature and nurture. Given the differing perspectives on whether anxiety sensitivity is a trait or a form of learned behavior, a number of studies have been conducted to

determine the relative role of each. Chief amongst these studies are twin research studies, which indicate that anxiety sensitivity may be heritable, with a genetic contribution of about 45% (Jang et al., 1999; Stein, Jang & Livesley, 1999). Along with the twin studies, studies examining the link between anxiety sensitivity and psychopathology in parents and children provide another source of information for the familial aggregation of anxiety sensitivity. However, the results have been mixed and provide little indication of whether transmission is mainly genetic, as suggested by expectancy theory, or environmental (East, Berman, & Stoppelbein, 2007), as suggested by cognitive and behavioral theories. For example, Van Beek and Griez (2003) found that adult first-degree relatives of patients with panic disorder had higher anxiety sensitivity than normal controls. Similarly, children of parents with panic disorder experienced more fear of physical sensations than children of parents with no known psychopathology. Pollock et al. (2002) further found an association between parental substance abuse and adolescent anxiety sensitivity and determined that anxiety sensitivity was more related to anxiety in the children of parents with anxiety disorders. In contrast, Mannuzza, Klein, and Moulton (2002) found no anxiety sensitivity difference in children of patients with anxiety or mood disorders compared to children of healthy controls.

The mixed nature of the findings is further complicated by apparent gender differences in transmission. Tsao et al. (2005a) found that parental anxiety, especially social concerns, accounted for substantial incremental variance in daughters' anxiety sensitivity. In a subsequent study, Tsao, Lu, Kim, and Zeltzer (2006) found parent anxiety sensitivity to be related to the pain intensity experienced by children in a laboratory task via its contribution to anxiety sensitivity in girls, but not boys. In contrast, East et al. (2007) found that the relationship between parental and child anxiety sensitivity was limited to father and child. Looking to provide clarity on the issue

of gender and transmission, Taylor, Jang, Stewart, and Stein (2008) conducted possibly the largest, most comprehensive behavioral-genetic study of anxiety sensitivity Using 438 twin pairs and found that heritability in women significantly increased with anxiety sensitivity scores. They concluded that although severe forms of anxiety sensitivity are more strongly influenced by genetic factors than milder forms, genes tell only half the story. For men, transmission is influenced by environmental, but not genetic, factors.

Consistent with cognitive and behavioral theories, the parenting/family environment has similarly been implicated in other studies examining parental and offspring anxiety sensitivity. For instance, college students with elevated levels of anxiety sensitivity reported receiving more reinforcement for *sick-role* behaviors as children than those with low levels of anxiety sensitivity (Watt, Stewart, & Cox, 1998). In particular, they reported receiving more attention from their parents and were more likely to indicate that their parents expressed concern in response to somatic symptoms. While the familial and twin studies indicate that parents may transmit the specific genotype that predisposes their children toward higher biological arousal (DiLalla, Kagan & Reznick, 1994; Stein et al., 1999; Stein, Schork, & Gelernter 2001), the role of learning in the development of anxiety sensitivity cannot be ignored.

Relationship between anxiety sensitivity and psychopathology. Although conceptualized initially as a causal mechanism for panic, the anxiety sensitivity construct has now been implicated in a number of different pathologies, including anxiety disorders, depression, substance abuse, and chronic pain. Deacon and Abramowitz (2006a) showed in a cross-sectional study that across anxiety diagnoses, individuals with panic disorder had the highest level of anxiety sensitivity, although their scores were only slightly higher than individuals with social phobia and generalized anxiety disorder. In the next subsection, a

summary of the findings on the relationship between anxiety sensitivity and anxiety and other disorders is presented. Before doing so, it is worth noting that factor analytic research on the Anxiety Sensitivity Index and subsequent measures has demonstrated that anxiety sensitivity consists of a higher order factor (i.e., global anxiety sensitivity) and lower order dimensions (Zinbarg, Mohlman, & Hong, 1999) of fear of physical, cognitive, and social anxiety symptoms. Although global anxiety sensitivity is thought to serve as a shared vulnerability factor, the lower-order dimensions have been shown to have varying levels of correspondence to different disorders.

Anxiety sensitivity and panic disorder. Of all the anxiety disorders, prospective research has strongly supported the power of anxiety sensitivity to predict the development of panic disorder in both adults and children (Li & Zinbarg, 2007; Maller & Reiss, 1992; Schmidt, Lerew, & Jackson, 1997, 1999). Although the role of the overall (global) level of anxiety sensitivity is clear, there has been less consensus on a specific subfactor that is most predictive. In one study, Air Force recruits were monitored during a very stressful five-week basic training period (Schmidt, Lerew, & Jackson, 1997, 1999), and their levels of anxiety sensitivity were found to predict the development of spontaneous panic attacks, with participants' responses related to the mental concerns factor (fear of cognitive dyscontrol) of anxiety sensitivity being the best predictor. In another year-long study, Li and Zinbarg (2007) similarly found that the mental concerns factor, but not other subfactors, was a significant predictor of panic onset, contributing 16% of the total variance. These results run somewhat counter to earlier studies in which the elevation of the physical concern aspects of anxiety sensitivity in panic disorder was suggested to be key (Taylor, Koch, McNally & Crockett, 1991; Zinbarg, Barlow, & Brown, 1997; Zinbarg, Brown, Barlow, & Rapee, 2001). Overall, the results appear to underscore the importance of

anxiety sensitivity in panic attacks and show that there may be utility in employing a multidimensional approach that expressly recognizes the role of lower-order factors in panic vulnerability.

Anxiety sensitivity and PTSD. The link between anxiety sensitivity and PTSD has been demonstrated following exposure to natural disasters (Hagh-Shenas, Goodarzi, Dehbozorgi, & Farashbandi, 2005), work place injuries (Asmundson, Norton, Allerdings, Norton, & Larsen, 1998), motor vehicle accidents (Fedoroff, Taylor, Asmundson, & Koch, 2000), childbirth (Keogh, Ayers, & Francis, 2002), and interpersonal violence (Lang, Kennedy, & Stein, 2002). For example, Lang et al. (2002) found significantly higher total scores on the Anxiety Sensitivity Index among women who developed PTSD in response to intimate partner violence than both those experiencing such violence who did not develop PTSD and women with no trauma history. Overall, although the anxiety sensitivity levels of people who develop PTSD tend to be high, they are somewhat lower than in those with panic disorder, but far higher than in normal controls (Taylor, Koch, & McNally, 1992). Anxiety sensitivity levels are correlated also with the severity of PTSD symptoms (Fedoroff, Taylor, Asmundson, & Koch, 2000). There is evidence to suggest that anxiety sensitivity, particularly the physical concerns facet of the construct, is more elevated among women who develop PTSD than among men (Peterson & Plehn, 1999; Stewart, Karp, Pihl, & Peterson 1997) and that treatment-resistant individuals are characterized by high levels of anxiety sensitivity (Bryant & Panasetis, 2001). In an investigation of survivors of motor vehicle accidents, Fedoroff, Taylor, Asmundson, and Koch (2000) found that not only did persons higher in anxiety sensitivity report more severe PTSD symptoms following the accident, but the reductions in PTSD were also associated with reductions in anxiety sensitivity following a 12-week cognitive-behavioral PTSD treatment program. In keeping with these findings, treatments

for PTSD that address the underlying anxiety sensitivity are being piloted, and this appears to be a promising strategy for dealing with treatment-resistant PTSD (Wald & Taylor, 2008).

Anxiety sensitivity and social phobia. Anxiety Sensitivity, specifically the social concern subfactor, has been implicated in social phobia (Norton, Cox, Hewitt, & McLeod, 1997; Vriends et al., 2007). Individuals with this disorder have been found to score significantly higher on this subfactor than patients with panic disorder without agoraphobia, obsessive-compulsive disorder, and specific phobia (Zinbarg, Barlow, & Brown, 1997). In addition, Vriends et al. (2007) showed that individuals with lower Anxiety Sensitivity Index scores showed greater recovery from social phobia over an 18-month period than those with higher levels. In explaining these findings, the authors proposed that anxiety sensitivity might promote a focus on internal symptoms that the socially anxious person may perceive as being obvious to others. However, Zinbarg et al. (1997) also found that individuals with social phobia did not score significantly higher on the social concerns subfactor than patients with panic disorder with agoraphobia and patients with generalized anxiety disorder. Whether this component of anxiety sensitivity is specifically related to social anxiety disorder requires further study.

Anxiety sensitivity and obsessive-compulsive disorder (OCD). Disgust sensitivity has been found to be a key predictor of contamination fears in individuals with OCD, but Cisler, Reardon, Williams, and Lohr (2007) have recently demonstrated that anxiety sensitivity (more specifically, the physical concerns factor) also predicts contamination fear independently of this construct. These and other similar results (Olatunji et al., 2005) suggest that individuals with high anxiety sensitivity who tend to fear aversive sensations may experience their disgust response as more aversive. The relationship between anxiety sensitivity and OCD has also been examined in relation to hoarding, and anxiety sensitivity was found to be a significant predictor

(Coles, Frost, Heimberg, & Steketee, 2003). Following learning theory, the authors speculated that individuals with OCD experience negative emotions at the thought of purging hoarded items, and that the fear of anxiety heightens this experience such that hoarding becomes more and more reinforcing. More data are needed before firmer conclusions can be reached.

Anxiety sensitivity and depression. Although anxiety sensitivity research was originally predicated on the theoretical relationship between anxiety sensitivity and anxiety disorders, an unanticipated connection has been found between anxiety sensitivity and depression. Anxiety sensitivity has been shown to be elevated in individuals with major depressive disorder (MDD), and depressed patients tend to score in the same range as individuals with other non-panic anxiety disorders (Cox, Enns, & Taylor, 2001; Taylor, Koch, Woody, & McLean, 1996). Unfortunately, the literature in this area is mixed and the link between anxiety sensitivity and depression has not always been consistently demonstrated. For example, Otto, Pollack, Fava, Uccello, and Rosenbaum (1995) found that while participant Anxiety Sensitivity Index scores were related to factors such as anxiety sensitivity and somatic symptoms, these scores were not related to depression severity. Similarly, although Muris, Schmidt, Merckelbach, and Schouten (2001) found a significant correlation between anxiety sensitivity and depression, this correlation was no longer statistically significant when trait anxiety was included in the analysis.

Another area of controversy relates to the specific subfactor of anxiety sensitivity that is most significantly related to depression. A number of studies have identified the fear of cognitive dyscontrol to be key (Cox, Enns, & Taylor, 2001; Taylor, Koch, Woody, & McLean, 1996), but again, this finding is far from unanimous, and others have found the physical concern factor to be more predictive (e.g., Grant, Beck, & Davila, 2007). Further, it has been suggested that the association between anxiety sensitivity and depression may be the result of covariation between

anxiety and depression (Schmidt, Lerew, & Jackson, 1997; 1999). However, there is also evidence that depressed individuals report high levels of anxiety sensitivity even when they do not have comorbid anxiety disorders (Cox, Enns, Freeman, & Walker, 2001). For example, Weems, Hammond-Laurence, Silverman, and Ginsburg (1998) found a significant correlation between anxiety sensitivity and childhood depression that remained significant when general anxiety was parceled out. Overall, the mixed nature of the findings creates concern. Published articles tend to be those that show positive results, and the fact that several non-significant findings have made it to print indicates that the relationship may be even less robust than it currently appears in the literature.

Anxiety sensitivity and substance abuse. Anxiety sensitivity has been identified in both cross-sectional and prospective studies as a risk factor for alcohol (Schmidt, Buckner, & Keough, 2007; Stewart, Peterson, & Pihl, 1995; Watt, Stewart, Birch, & Bernier, 2006) and other substance abuse problems including cigarette smoking (Stewart, Karp, Pihl, & Peterson, 1997) and heroin use (Lejuez, Paulson, Daughters, Bornovalova, & Zvolensky, 2006). For example, several studies have suggested that high anxiety sensitivity is associated with greater alcohol consumption (Stewart, Peterson, & Pihl, 1995; Watt, Stewart, Birch, & Bernnier, 2006) and greater frequency of excessive drinking (Stewart, Zvolensky, & Eifert, 2001). These findings have been explained using tension reduction theory (Conger, 1956), which states that individuals habitually engage in maladaptive behaviors for the negative reinforcement value they serve in dampening negative affect (DeHaas, Calamari, & Blair, 2002). In other words, substances are used by individuals high in anxiety sensitivity as a means of self-medication (Lejuez, Paulson, Daughters, Bornovalova, & Zvolensky, 2006; Zvolensky, Feldner, Leen-Feldner, & Yartz, 2005). Unfortunately, the use of such substances for emotion regulation is thought to have long-

term negative consequences in that they preclude the natural course of reduction in emotional intensity that accompanies exposure to the undesired sensations, and they increase the risk of developing substance abuse disorders (Campbell-Sills, Barlow, Brown, & Hoffmann, 2006a, 2006b; Feldner, Zvolensky, Stickle, Bonn-Miller, & Leen-Feldner, 2006).

While high anxiety sensitivity is associated with substance abuse, not all substance abusers have high anxiety sensitivity. Those who have high anxiety sensitivity tend to report more coping motivations for their substance abuse than low anxiety sensitivity substance abusers do. In particular, individuals with high levels of anxiety sensitivity are more likely to report that they drank alcohol in order to cope with negative affect, while those with low levels of anxiety sensitivity are more likely to endorse social reasons for drinking (Stewart & Zetlin, 1995). In addition, individuals high on anxiety sensitivity are likely to choose substances that dull their sensitivity rather than those that heighten them. Norton et al. (1997) showed that male participants who scored in the high range on the Anxiety Sensitivity Index preferred alcohol and other depressants that dampen or reduce their anxiety sensations, while participants in the low range preferred marijuana. In particular, there is evidence that individuals who regularly use heroin exhibit higher levels of anxiety sensitivity than all other substance abusers (Lejuez et al. 2006). These findings were replicated by Stewart et al. (1997), who argued that people high in anxiety sensitivity avoid marijuana because of some of the physical and psychological side effects of the drug, including cognitive dyscontrol and physiological arousal. The coping motive for substance abuse in high anxiety sensitivity individuals is significant because it highlights the importance of addressing the underlying anxiety sensitivity in order to treat the substance abuse. Studies are being piloted that examine the potential of anxiety sensitivity-targeted treatments in addressing substance abuse.

Differences in anxiety sensitivity across cultures. The cross-cultural literature on anxiety sensitivity has looked primarily at differences in levels of anxiety sensitivity and in the factor structure of anxiety sensitivity in different cultures and found significant variations in both. In particular, cross-cultural studies of anxiety sensitivity have demonstrated generally elevated levels. For example, non-clinical Native American college students reported significantly higher overall Anxiety Sensitivity Index scores than their same-age counterparts from the majority (Caucasian) culture (Zvolensky et al., 2003) as did urban African-American elementary school children (Carter, Marin, & Murrel, 1999) and members of a Russian community (Kotov et al., 2005). Similarly, Taylor et al. (2007) found in their assessment of the validity and reliability of the Anxiety Sensitivity Index, 3rd Edition (ASI-3), that non-clinical participants from France (M = 16.4), Mexico (M = 15.2), and Spain (M = 14.2) displayed moderately higher levels of anxiety sensitivity than participants from the U.S. and Canada (M = 12.8). Only participants from the Netherlands (M = 10.7) displayed lower ASI-3 levels than the U.S. and Canadian samples.

When we turn to clinical samples, it appears also that Anxiety Sensitivity Index scores are more elevated in non Anglo-American cultures. Hinton, Pich, Safren, Pollack, and McNally (2005) found that the Anxiety Sensitivity Index scores of a group of Cambodian refugees who suffered panic disorder were elevated, with a mean of 42.4, compared to the average score of approximately 36.6 amongst North American panic patients (Taylor et al., 1992). In contrast, Sandin, Chorot, and McNally (1996) found anxiety sensitivity scores to be lower (M = 32.4) in Spanish panic patients than in North American samples.

Alexithymia has been posited to be a possible reason for the greater levels of anxiety sensitivity, and a number of studies have demonstrated moderate correlations between the two

constructs (e.g., Devine, Stewart, &Watt, 1999). The construct of alexithymia is conceptually similar to the constructs of lack of awareness and clarity/understanding of emotional responses that is a key component of Gratz and Roemer's (2004) popularly used conception of emotion dysregulation. Not only are emotional identification and understanding key aspects of the ability to effectively regulate emotion, prior research has also found that alexithymia and anxiety sensitivity are significantly correlated (Celikel & Saatcioglu, 2007; Cox et al., 1994; Cox et al., 1995; Zeitlin, McNally & Cassiday, 1993). While individuals high in anxiety sensitivity tend to misinterpret anxiety sensations and view them as dangerous, alexithymic subjects are thought to misinterpret emotional arousal in general, and therefore alexithymia may play a contributory role to the development of anxiety sensitivity.

The following section outlines the construct of emotion regulation, the association between emotion regulation (including alexithymia) and anxiety, and the relationship between emotion regulation and anxiety sensitivity.

Emotion Regulation

As outlined above, a vast literature has developed around anxiety sensitivity in recent years, and evidence has accumulated implicating the construct as a risk factor for the development of panic and a number of anxiety and related disorders. This impact is seen both in studies conducted in North America and those that extend their assessment to international locales. However, although strongly predictive of anxiety, anxiety sensitivity does not completely explain the variability in anxiety symptoms (Schmidt et al., 1997). The scientific activity on anxiety sensitivity has principally been focused on *main effect* types of questions (Kashdan, Zvolensky, & McLeish, 2007); how anxiety sensitivity may interplay with other processes relevant to anxiety psychopathology is less well documented. Numerous scholars have

suggested that the role of anxiety sensitivity may be more complex than suggested purely by linear main effect models (Hayes & Feldman, 2004; Zvolensky, Feldner, et al., 2005). It has been posited that a better understanding of what causes the development of anxiety disorders may require a more careful consideration of the complex interactions of anxiety sensitivity with other individual-difference characteristics (Zvolensky, Felder, Leen-Feldner, & McLeish, 2005). Thus, it may be worth attending to other psychological factors that might also be promisingly predictive of anxiety disorders and that may also have a role in the development of anxiety sensitivity.

To this end, one promising area of work is the growing literature on emotion regulation (Barlow, Allen, & Choate, 2004; Kashdan et al. 2006; Linehan, 1993; Orsillo & Roemer, 2005). Emotion regulation refers to the process by which individuals identify, evaluate, and use strategies to control or influence the occurrence, experience, intensity, and expressions of emotions (Richards & Gross, 2000). In another definition, emotion regulation refers to behaviors, skills, and strategies, whether conscious or unconscious, automatic or effortful, that serve to modulate, inhibit, and enhance emotional experiences and expressions (Calkins, 2004)

Barlow (1988) was among the first to conceive of pathological anxiety as essentially representing a problem with the regulation of emotion, particularly the regulation of fear. Writers in this area suggest that anxious individuals may have difficulty identifying and understanding their emotions and may be overly concerned about the experience and expression of their feelings such that they maladaptively attempt to regulate (e.g., ignore, or suppress) them. However, such attempts may lead to a significant increase in the emotion that is the subject of regulation and result in a vicious cycle (Barlow et al., 2004; Gross & Levenson, 1997). The empirical data support this theoretical conception and indicate that suppression and inhibition

may play a significant role in many forms of psychopathology (e.g., Gross & Muñoz, 1995; Rottenberg & Gross, 2003). For example, emotion dysregulation and disruption has been associated with depression (e.g., Rude & McCarthy, 2003), panic disorder (Baker et al., 2004), posttraumatic stress disorder (Cloitre, Scarvalone, & Difede, 1997), and, perhaps most notably, borderline personality disorder (Linehan, 1993).

Among the studies in this area, Turk et al. (2005) compared the presence of emotion dysregulation in social phobia and in generalized anxiety disorder. Utilizing clinical analogue groups and a cross-sectional, retrospective, self-report design, the authors found evidence for the presence of deficits in emotion regulation across these conditions when compared to non-anxious controls. Other studies have demonstrated that emotion regulation difficulties may also partially explain the high rates of PTSD among those seeking treatment for substance use disorders. Indeed, emotion regulation has been found to mediate the relationship between PTSD symptom severity and negative drug use (Staiger et al., 2009). The emotion regulation functions of these substances are critical in understanding why many people with anxiety psychopathology use substances and subsequently develop anxiety problems. Looking specifically at the construct of avoidance, Holahan et al. (2007) found in a longitudinal study spanning 10 years that avoidance-based coping mechanisms predicted increases in depressive symptoms.

In a recent meta-analysis, Aldao, Nolen-Hoeksema, and Schweizer (2009) examined the relationship between six emotion-regulation strategies (acceptance, avoidance, problem solving, reappraisal, rumination, and suppression) and symptoms of four psychopathologies (anxiety, depression, eating, and substance-related disorders). They combined 241 effect sizes from 114 studies and found, overall, a medium to large effect size for avoidance, problem solving, and suppression, and small to medium for reappraisal and acceptance.

Although there has been a dramatic increase in the number of articles available through PsycINFO mentioning emotion regulation and its relation to anxiety and other forms of psychopathology, there remains a great deal of confusion about what emotion regulation is and is not. One challenge within the emotion regulation literature is that this concept may encompass a broad range of response topographies (Cisler, Olatunji, Feldner, & Forsyth, 2010), and it remains difficult to present a clear synopsis of which disorders are most related to emotion regulation difficulties and which emotion regulation strategies are most closely linked to specific forms of psychopathology.

Acknowledging this problem, Gratz and Roemer (2004) extensively reviewed the emotion regulation literature and identified several distinct but related domains involved in adaptive emotion regulation. Using a factor analytic strategy to differentiate distinct subfactors, they developed a scale entitled the Difficulties in Emotion Regulation Scale that operationalizes and measures emotion dysregulation as a higher-order construct involving the multiple, internally consistent lower-order dimensions. The domains they identified were (a) awareness and understanding of emotions, (b) acceptance of emotions, (c) ability to control impulsive behaviors and engage in goal-directed behaviors when experiencing negative emotions, and (d) access to and flexible use of situationally appropriate emotion regulation strategies. According to the authors, difficulties may appear in one or all of the above domains.

The role of emotion regulation as conceptualized by Gratz and Roemer (2004) in anxiety has been demonstrated in several studies, including one by Salters-Pedneault, Roemer, Tull, Rucker, and Mennin (2006), which assessed emotion regulation deficits and generalized anxiety disorder-related outcomes in an analogue sample. Consistent with the authors' hypotheses, general emotion dysregulation was associated with reports of chronic worry and with analogue

generalized anxiety disorder status. Also, specific regulation deficits, including emotional clarity, acceptance of emotions, ability to engage in goal directed behaviors when distressed, impulse control, and access to effective regulation strategies, were associated with worry and analogue generalized anxiety disorder above and beyond variance contributed by negative affectivity. The authors concluded that emotion regulation explains incremental variance in generalized anxiety disorder symptomatology over and above intensity of worry (Tull et al., 2009).

While studies adopting the Difficulties in Emotional Regulation Scale are slowly accumulating, the specific subfactors of this scale have had a longer history in the emotion regulation literature, and some studies have focused specifically on them. In the following sections, the key subfactors of emotion regulation are reviewed, including the theoretical and empirical literature that demonstrates the link to anxiety pathology, namely emotional acceptance, the ability to control impulses and behave in a goal directed manner, and the ability to identify and understand emotions (alexithymia).

Emotional acceptance. The general discussion on the role of emotion regulation has centered largely on the factor of emotional acceptance or its polar opposite, emotional avoidance or suppression. Emotional avoidance is a process involving excessive negative evaluations of unwanted private thoughts, feelings, and sensations, an unwillingness to experience these private events, and deliberate efforts to control or escape from them (Hayes, 1994; Hayes, Strosahl, & Wilson, 1999), even when the attempt to do so causes psychological harm (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). The problem of emotional avoidance forms a key target of Acceptance and Commitment Therapy (ACT).

According to Relational Frame Theory, which ACT is derived from (Hayes, Barnes-Holmes, & Roche, 2001), human beings, unlike animals, do not always have the option of

avoiding pain simply by avoiding a feared physical stimulus. Hayes (2004) gives the example that painful memories of a recently dead spouse might be cued by pictures, depressed mood, a comment in a conversation, a beautiful sunset, or myriad other cues. Unable to control pain by simply avoiding one or two specific situations or environments, humans begin to try to avoid the painful thoughts and feelings themselves. Unfortunately, many of these means (e.g., suppression) come to cue the avoided event because they strengthen the underlying associations ("don't think of John" will serve as a cue for the loss of John and the psychological experiences associated with it; Hayes, 2004).

Such an unwillingness to remain in contact with negatively evaluated private events and chronic attempts to alter the form of these events or contexts in which they arise is proposed to be a stronger contributor to psychopathology than the content (e.g., intensity, frequency, negative valence) of private psychological and emotional experiences (Hayes et al., 1999). Emotional avoidance is thus thought to play an important role in various psychological disorders, and empirical studies have shown it to be a factor in depression (DeGenova, Patton, Jurich, & MacDermid, 1994), substance abuse (Ireland, McMahon, Malow, & Kouzekanani, 1994), the sequelae of child sexual abuse (Leitenberg, Greenwald, & Cado, 1992), and many other areas. In particular, emotional avoidance has been implicated as a causal mechanism in anxiety disorders (Hayes et al. 1996; Orsillo & Roemer, 2005).

Several studies have shown the role of emotional acceptance/avoidance in the treatment of anxiety disorders, particularly that acceptance tends to have the unexpected effect of reducing anxiety levels in anxious individuals (see Powers, Vording, & Emmelkamp, 2009). For example, the effects of a computerized ACT-based acceptance teaching protocol on tolerance of the exposure to carbon dioxide (CO2)-enriched air (Feldner, Zvolensky, Eifert, & Spira, 2003) was

examined with 48 college students. One group was instructed to accept their feelings during the carbon dioxide inhalation, while the other was told to suppress them. Individuals in the suppression condition reported greater levels of anxiety. In a similar study, 60 highly anxious females were randomly assigned to a 10-minute acceptance condition, a control condition (controlling psychological experiences by abdominal breathing), or a no-instruction condition (Eifert & Heffner, 2003). Compared to the control and non-instruction participants, those in the acceptance condition were less avoidant behaviorally and reported less intense fear and cognitive symptoms. Participants in the acceptance group also reported greater willingness to return to the CO2-inhalation study than those in comparison groups. In a third such study (Levitt, Brown, Orsillo, & Barlow, 2004), sixty patients with panic disorder were randomly assigned one of three 10-minute audiotaped interventions: acceptance, suppression, and distraction. Once again, the acceptance group showed significantly greater levels of willingness to participate in the biological challenge and lower level of anxiety than those in comparison groups.

Interestingly, other studies have found that suppression or avoidance appears to bring about adverse consequences, including increases in physiological arousal, impaired memory for social information, and weaker social ties with interaction partners (Butler & Gross, 2004; Gross & Levenson, 1993, 1997; Richards & Gross, 2000). For example, Karekla, Forsyth, and Kelly (2004) made 54 healthy undergraduates, who were either high (n = 27) or low (n = 27) in experiential avoidance, undergo twelve 20-second long inhalations of 20% carbon dioxide-enriched air while their physiological reactions were being monitored. Physiological measures included skin conductance and heart rate. Participants were also asked to report subjective reactions such as subjective units of distress and the number and severity of panic symptoms they were experiencing. Not surprisingly, individuals high in experiential avoidance endorsed

more panic symptoms, more severe cognitive symptoms, and more fear, panic, and uncontrollability than their less avoidant counterparts did. However, the magnitude of autonomic response did not discriminate between groups, and the group that developed greater panic or cognitive symptoms did not do so specifically because they had greater autonomic responses. Similarly, the observed effects were not accounted for by differences in anxiety sensitivity. These results were replicated in a very similar study by Spira, Zvolensky, Eifert, and Feldner (2004). Overall, the literature shows that suppression or emotional avoidance is a psychological vulnerability for anxiety and that it tends to yield more of the very distress avoidant individuals wish to avoid (John & Gross, 2004).

Impulse control and the ability to engage in goal directed action. Related to the ability to accept and experience difficult emotions, no matter how intense, is the ability to look beyond immediate urges to reduce painful affect, control impulses (e.g., to escape or avoid a situation), and engage in goal-directed actions (see Linehan, 1993; Melnick & Hinshaw, 2000) despite these internal experiences. Individuals unwilling to experience difficult emotions are often unable to control impulses (often to flee the aversive thought, situation, or sensation) and act in the service of their goals because of preoccupation with managing or avoiding the painful emotions (Kashdan, Barrios, Forsyth & Sterger, 2005). As opposed to focusing on decreasing unwanted internal events, the individual is taught in ACT protocols to act despite the pain they fear will result in the service of valued goals and directions.

Although also widely adopted in third wave behavior therapy treatment packages that have shown promising results, the literature examining the role of impulse control and goal-directed behavior on anxiety disorders is sparse. Most studies do not dismantle the effects of these elements from other elements of ACT and Dialectical Behavior Therapy protocols, but

presumably, when acting in a goal-directed manner, the individual would be forced to confront much of the same stimuli that would have to be tolerated under traditional behavioral interventions (e.g., staying in a social situation to further one's goal of increasing friendships, despite the intense social anxiety one feels).

The current study provides specific information on the role of impulse control and acting in a goal-directed manner in anxiety, relative to anxiety sensitivity and other subfactors of emotion regulation.

Emotional awareness and clarity (alexithymia). While the role of emotional acceptance has been highlighted in a number of relatively recent treatment studies, the role of an awareness and understanding of emotions has a longer history in the clinical psychology literature. Gratz and Roemer's factors of a lack of emotional awareness and understanding are closely related to the construct of alexithymia, which can be traced to MacLean (1949). He observed that a large proportion of patients with psychosomatic complaints had problems with psychoanalysis because it appeared that their emotions do not generally reach the level of full conscious and verbal identification. Sifneos (1972) further described the psychological features of these patients, saying that the patients manifested either a total unawareness of feelings or an almost complete incapacity to put into words what they were experiencing. Sifneos (1972) coined the term alexithymia (a = lack, lexis = word, thymos = mood or emotion) for this complex set of features.

There is empirical evidence that alexithymia is associated not just with difficulties in discriminating among different emotional states (Bagby, Parker, Taylor, & Acklin, 1993), but also with a limited ability to think about and use emotions to cope with stressful situations. For example, alexithymic individuals sometimes display outbursts of rage or of sobbing but are

unable to elaborate further on what they are feeling (Nemiah, 1978). Unable to label their feelings and to use them as signals of inner conflict or of responses to external situations, alexithymic individuals tend to focus on the physical symptoms of emotional arousal and on other normal bodily sensations, which they often amplify and misinterpret as signs of physical disease (Barsky and Klerman, 1983). Without the feeling component, their emotions are predominantly physical responses and are so vague and undifferentiated that they only call attention to themselves rather than to what they signal (Krystal, 1979).

Alexithymia and psychopathology. Although less than 15% of the population is likely to be alexithymic, the tendency may deserve greater attention as it has been negatively correlated with positive affect (Prince & Berenbaum, 1993) and with life satisfaction (Schmitz, 2000; Valkamo et al., 2001) and positively associated with negative affect (DeGroot, Rondin, & Olmsted, 1995; Prince & Berenbaum, 1993). Alexithymic features have been associated with depressive disorders (Bankier, Aigner, & Bach, 2001; Honkalampi et al., 2000) and has been found to be predictive of depression at a 6-year follow-up assessment (Honkalampi et al., 2007). Alexithymia has also been found to be associated with cardiovascular mortality. In a 20-year study of more than 2000 Finnish men, it was found that risk of death from cardiovascular disease increased by 1.2% for each 1-point increase in Toronto Alexithymia Scale-26 (TAS-26) scores.

In substance abuse patients, alexithymia rates have been found to range from 39% to 54.5% (Taylor, Bagby, & Parker, 1997; Corcos & Speranza, 2003). Troisi et al. (1998) measured alexithymia in young cannabis-abusing/dependent subjects and found the diagnosis to be relevant to 30% of their sample of 88. This is about twice the rate of alexithymia reported recently by Säkkinen et al. (2007) who found a prevalence of 14.6% among boys and 17.3%

among girls in a sample of Finish non-clinical adolescents. These findings have led some to speculate that substances are used to compensate for deficits in emotional self-awareness.

Alexithymia has also been implicated in the anxiety disorders. Grabe, Spitzer, and Freyburger (2004) found that alexithymia dimension of difficulties in identifying feelings was significantly associated with all nine subscales of psychopathology assessed by the Symptom Checklist-90-Revised. In particular, the authors found a strong significant relationship between alexithymia and anxiety symptoms, a finding that replicated earlier observations by Haviland, Hendryx, Shaw, and Henry (1994). Zeitlin, McNally and Cassiday (1993) further investigated the alexithymia construct in 27 panic disorder patients and 31 individuals with obsessive-compulsive disorder and classified 67% of the panic patients as alexithymic. Further, panic patients scored significantly higher on the Toronto Alexithymia Scale than obsessional patients. A similar study by Parker, Taylor, and Bagby (1993) found that the prevalence of alexithymia in 30 panic disorder patients was 46.7%, as compared with only 12.5% in 32 simple phobia patients, based on scale cutoff scores. This study recommended that alexithymia should be part of the assessment of every panic patient. More longitudinal studies would be invaluable in establishing the role of alexithymia in the development of anxiety disorders. Nonetheless, these results indicate that like anxiety sensitivity, alexithymia too may be a risk factor for anxiety.

Family and cultural influences of alexithymia. Some evidence indicates that alexithymia is associated with social environments that do not foster the ability to identify and communicate emotions (Berenbaum & James, 1994; Fukunishi et al., 1997; Kench & Irwin, 2000; Lumley, Mader, Gramzow, & Papineau, 1996). Fukunishi et al. (1997) found that in a sample of Japanese college students, retrospective reports of perceived inadequate care from mothers (but not fathers) during childhood were associated with higher levels of alexithymia,

specifically with difficulty communicating emotions. More recently, retrospective reports of Australian college students found that only one aspect of the childhood family environment predicted alexithymia, namely family expressiveness (i.e., the degree to which family members are allowed and encouraged to express their opinions and feelings to each other; Kench & Irwin, 2000). This construct was negatively correlated with alexithymia. While family upbringing is thought to play an important role in the etiology of alexithymia, the role of the society at large in influencing individual family cultures cannot be ignored. The alexithymia research is growing, and an area of particular interest is the experience of non-native English speakers who appear to demonstrate generally elevated levels of alexithymia relative to Anglo-American samples (e.g., Dion, 1996; Lee, Berenbaum & Raghavan, 2002).

Numerous investigators (e.g., Ekman, 1971; Markus & Kitayama, 1994; Mesquita, 2001) have reported that culture plays a central role in shaping how emotions are experienced and expressed. For example, Eastern cultures typically have more rules than Western cultures restricting the open experience and expression of emotions (Argyle, Henderson, Bond, Iizuka, & Contarello, 1986). Parents and families tend to endorse socialization goals and child-rearing practices that are consistent within these cultural dimensions (e.g., Cole & Tamang, 1998). As a result, for example, Matsumoto and Kishimoto (1983) found that Japanese children were less likely to recognize facial expressions of anger than were American children. The authors proposed that Japanese children are socialized from an early age to avoid the expression of emotions like anger; therefore, Japanese parents may not have exposed their children to the situations surrounding anger as much as American parents.

Testing the notion of cultural differences in alexithymia, Lee, Berenbaum, and Raghavan (2002) looked at mean levels of alexithymia in three cultures, namely European American, Asian

American, and Malaysian college students (of Chinese, Malay, and Indian ethnicities). Asian groups both in the U.S. and Malaysian samples had higher alexithymia levels than the European American group. Similarly, Dion (1996) reported that among Canadian college student participants, native Chinese language speakers had higher mean levels of alexithymia than did native English and native European language speakers. It is possible that language influences alexithymia scores along with culture as the participants of European origin for whom English was not a first language scored higher on alexithymia than the American students, but lower than the students of Chinese origin. Both Dion (1996) and Lee, Berenbaum, and Raghavan (2002) found that somatization was more strongly associated with alexithymia in the Asian groups than in the European-American/Canadian groups.

Zhu et al. (2006) similarly examined differences in alexithymia across cultures after translating and back-translating the Toronto Alexithymia Scale-20 to create a Chinese version of the scale with good psychometric properties. Their findings corroborated earlier findings in that the mean alexithymia levels of students and patients sampled in China were much higher than students and patients in European-Canadian samples. Dion (1996) provided a sociocultural explanation for the differences in alexithymia levels across the cultures he studied, saying that the personality trait of alexithymia is fostered among ethnic Chinese because the Chinese culture strongly encourages the use of somatic idioms for understanding and describing one's emotional state. Because *psychological mindedness* relates inversely to alexithymia, these cultural pressures would lead ethnic Chinese individuals to score higher on an alexithymia scale.

Not only are emotional identification and understanding key aspects of the ability to effectively regulate emotion, studies looking at the relationship between alexithymia and anxiety sensitivity have found them also to be significantly correlated (Celikel & Saatcioglu, 2007; Cox

et al., 1994; Cox et al., 1995; Zeitlin, McNally, & Cassiday, 1993). While individuals high on anxiety sensitivity tend to misinterpret anxiety sensations and view them as dangerous, alexithymic subjects are thought to misinterpret emotional arousal in general, and therefore alexithymia may play a contributory role to the development of anxiety sensitivity.

Relative Roles of Emotion Regulation and Anxiety Sensitivity on Anxiety

As outlined above, there has been increasing scholarly interest in forwarding the scientific study of emotion dysregulation (Barlow, Allen, & Choate, 2004; Kashdan & Steger, 2006; Linehan, 1993; Orsillo & Roemer, 2005; Rottenberg & Gross, 2003). These perspectives tend to stress the tolerance of negative affect states (without the need to change or escape them). The question arises of whether the presence of high anxiety sensitivity (conceptualized as a trait) is as problematic if someone also has the ability to attend to, identify, and accept their emotional responses such that they can continue to act in a goal-directed manner (Brown, Lejuez, Kahler, Strong, & Zvolensky, 2005). Insofar as people high in anxiety sensitivity are able to identify emotions and emotionally accept aversive anxious states or thoughts, while inhibiting difficult impulses, they may be able to forestall escalation of problematic anxiety experiences.

Although the empirical literature on the specific link between anxiety sensitivity and emotion regulation is limited, some recent works provide preliminary indication that individual differences in how people regulate anxiety may affect the link between anxiety sensitivity and the development of anxiety disorders (Olatunji, Forsyth, & Feldner, 2007). The combined role of anxiety sensitivity and emotion regulation deficits has been demonstrated in panic disorder (Tull, Stipelman, Salters-Pedneault, & Gratz, 2009), generalized anxiety disorder (Mennin, Heimberg, Turk, & Fresco, 2002), PTSD (McDermott et al., 2009; Tull et al., 2007) and in the experience of

distressing but non-pathological levels of anxiety in non-clinical samples (Vukonavoic, Zvolensky, & Bernstein, 2007).

It has been shown, for example, that anxiety sensitivity interacts with emotional avoidance to delay recovery from an anxiety-relevant laboratory stressor (Feldner, Zvolensky, Stickle, Bonn-Miller, & Leen-Feldner, 2006). Kashdan, Zvolensky, and McLeish (2008) also found that among participants high in anxiety sensitivity, anxious arousal and worry were heightened in the presence of less acceptance of emotional distress. These findings suggest that among those with high anxiety sensitivity, emotion regulation difficulties exacerbate the impact of anxiety sensitivity on anxiety. Inflexible emotion regulatory strategies may thus positively moderate the relationship between anxiety sensitivity and anxiety and shift individuals from normative to clinical levels.

Taking a slightly different view, Gratz, Tull, and Gunderson (2007) found that although borderline personality disorder outpatients reported higher levels of anxiety sensitivity than non-personality-disordered outpatients (such that anxiety sensitivity reliably distinguished between these two groups), the relationship between anxiety sensitivity and borderline personality disorder was mediated by experiential avoidance.

Though such data are indicative of a significant role of emotion regulation together with anxiety sensitivity in anxiety and other pathologies (Kashdan, 2007; Mennin, 2005), empirical data that more comprehensively documents the role of each are not currently available. Most studies have looked at the value of emotional avoidance, but emotion regulation involves more than just emotional avoidance or acceptance. Further, while some attempts have been made to look at the relative roles of anxiety sensitivity and emotion regulation in psychopathology, these efforts have been limited to North-American samples, and the direction of the relationship is

unclear (Vujanovic, Zvolensky, & Bernstein, 2007). Many questions remain unanswered, particularly if emotion regulation difficulties compound the effects of anxiety sensitivity on psychopathology as has been shown in some studies, or if emotion regulation directly impacts anxiety sensitivity itself and is thus a possible cause of individual differences in anxiety sensitivity. The latter question has not been tested.

Similarly, the question arises of whether emotion regulation alone serves as a better predictor of anxiety disorders than anxiety sensitivity. Examining the issue of relative contribution of anxiety sensitivity versus emotion regulation strategies, McDermott, Tull, Gratz, Daughters, and Lejuez (2009) found that although the social concerns scale of Anxiety Sensitivity Index-3 contributed to the difference between PTSD and non-PTSD patients in a sample of crack-cocaine addicts, this contribution did not remain a reliable predictor of PTSD status once emotion regulation difficulties were included in the model. Thus, findings suggest that at least within this sample of inner-city crack/cocaine users, anxiety sensitivity may play a less fundamental role in probable PTSD than emotion regulation difficulties. No other studies have been identified that explore this relation in other populations. Further, emotion regulation itself has been conceptualized (Gratz & Roemer, 2004) as a confluence of factors, and few studies have examined the relative contribution of each.

Turning specifically to the emotion regulation subfactor of identifying and understanding emotion relevant stimuli, a strong positive and significant correlation has been found between anxiety sensitivity and alexithymia scores in both clinical and non-clinical samples (Cox et al., 1994; Cox et al. 1995; Zeitlin, McNally, & Cassiday, 1993). Looking at this correlation, Devine, Stewart, and Watt (1999) sought to examine if anxiety sensitivity and alexithymia were distinct constructs or if the correlations were the result of conceptual overlaps. Consistent with

predictions, an extreme group of high anxiety sensitivity participants (nonclinical university students) displayed greater total scores on the alexithymia measure than a group of low anxiety sensitivity participants. Moreover, after redundant items were removed from the data, high anxiety sensitivity students continued to display greater total scores on alexithymia than low anxiety sensitivity students. In the correlational analyses, the removal of redundant items did result in a significant reduction in the magnitude of the relation between anxiety sensitivity scores and alexithymia, but anxiety sensitivity was still highly correlated with alexithymia dimensions of difficulty identifying and describing emotions, even following the removal of redundant items. The findings thus provide support for a true relation between anxiety sensitivity and alexithymia, and challenge Cox and colleagues (1995), who argued that the relation between the two constructs is merely an artifact of conceptual and psychometric overlap between measures. Although not yet explicitly tested via a longitudinal study, the positive correlations between alexithymia and anxiety sensitivity scores observed in the study support the idea that elevated levels of alexithymia or a general inability to identify and understand emotions may be a predisposing factor for the development of high anxiety sensitivity. If so, treating underlying alexithymia may be a viable means of addressing anxiety sensitivity and symptoms of anxiety.

In particular, alexithymia has been posited to be a possible reason for the greater levels of anxiety sensitivity in some cultures, and a number of studies have demonstrated moderate correlations between the two constructs (e.g., Zahradnik et al., 2009). This study thus aims also to examine the role of the emotion regulation factor of alexithymia in creating differences in the anxiety sensitivity levels of individuals across cultures. Given the relationship between the alexithymia construct of difficulty identifying and describing emotions and anxiety sensitivity, and findings that both alexithymia and anxiety sensitivity scores vary across cultures, this study

examined whether differences in alexithymia account for a significant proportion of the variance in anxiety sensitivity across cultures. Additionally, this study examined the contribution of alexithymia to anxiety sensitivity and anxiety levels of individuals from three different cultures. It was hypothesized that cultures highest in alexithymia will also display the greatest anxiety sensitivity levels and vice versa. It is hoped that results will inform efforts to understand risk factors and influence valuable prevention protocols.

Research Questions and Hypotheses

In summary, empirical studies have shown that anxiety sensitivity significantly predicts anxiety and other psychopathology. However, a better understanding of the role of anxiety sensitivity in the development of anxiety disorders requires a more integrative understanding of the complex interactions between anxiety sensitivity and other individual difference variables. The overarching aim of the present investigation was to examine the relationships between anxiety sensitivity and anxiety, and the specific facets of emotion regulation in a sample outside that which has normally been studied. The key research questions of this study were (a) what is the role of emotion regulation and anxiety sensitivity in anxiety? and (b) what is the role of alexithymia in the relationship between anxiety sensitivity and anxiety across cultures?

In relation to the first research question, studies have indicated that the presence of emotion regulation difficulties and high anxiety sensitivity results in greater expression of anxiety. This study investigated whether emotion regulation moderates the relationship between high anxiety sensitivity and anxiety and which specific emotion regulation difficulty has the most impact.

The literature on the co-occurrence of high anxiety sensitivity and poor emotion regulation suggests that one may be influencing the other. Individuals high in anxiety sensitivity

find it difficult to regulate their emotions, and that emotion regulation may account for the variance in anxiety sensitivity. As outlined above, the relationship between anxiety sensitivity and borderline personality disorder is mediated by avoidance, and the social concerns subfactor of anxiety sensitivity is no longer significant in differentiating cocaine Users with and without PTSD when emotion regulation difficulties are accounted for. Therefore, it is possible that anxiety sensitivity has a weaker direct effect on anxiety once emotion regulation is accounted for, and the second hypothesis was that emotion regulation mediates the relationship between anxiety sensitivity and anxiety.

Finally, the current body of literature indicates that anxiety sensitivity varies across cultures but offers little indication of why this is so. Among the emotion regulation subfactors, two factors (lack of awareness of emotions and lack of emotional clarity) are very similar to alexithymia, which is known to vary across cultures. A secondary aim of this study, therefore, was to explore the role of alexithymia in anxiety sensitivity across cultures. Specifically, this study investigated whether the differences in anxiety sensitivity across cultures can be accounted for by differing levels of alexithymia and whether other emotion regulation subfactors play a role in explaining the differences.

Given the above research questions, the following hypotheses were tested:

1. All emotion regulation subfactors will positively moderate the relationship between anxiety sensitivity and anxiety, with emotional avoidance having the largest effect.

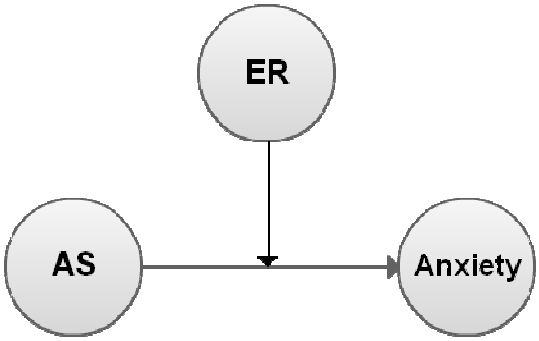

Figure 1. Moderation model

2. Emotion regulation will mediate the relationship between anxiety sensitivity and anxiety (once emotional regulation is accounted for, anxiety sensitivity will have no direct effect on anxiety).

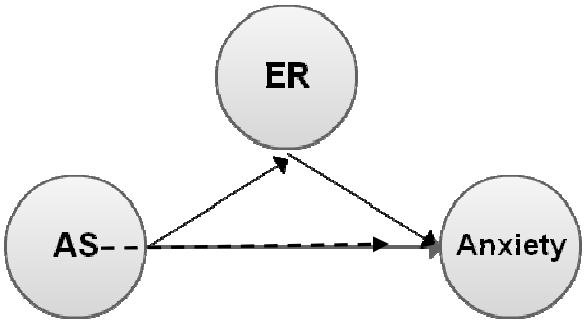

Figure 2. Mediation model

3. Anxiety sensitivity will differ across cultures, with the Chinese culture having the highest level of anxiety sensitivity, followed by the Malay culture and the Indian culture.
4. Alexithymia will partially mediate the relationship between culture and anxiety sensitivity (after controlling for alexithymia, culture will be a less significant predictor of anxiety sensitivity).

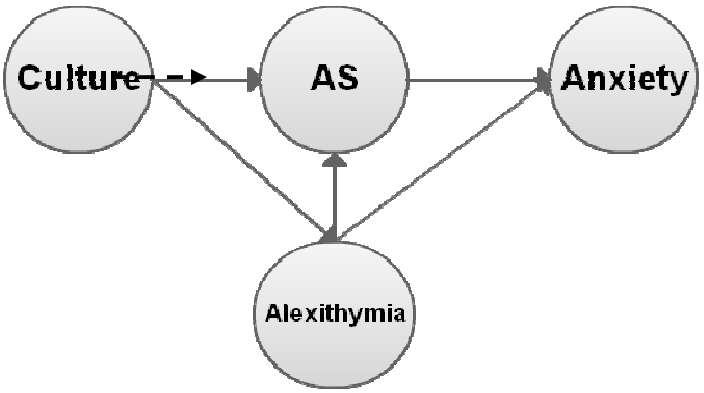

Figure 3. Path analysis of culture, anxiety sensitivity, alexithymia, and anxiety.

Chapter 2: Method

Participants

Singapore college students ($N = 312$) were recruited in person and asked to fill out their surveys either online or on paper. Singapore is a multi-ethnic society whose residents are from three main ethnicities: Chinese, Indian, and Malay. The medium of instruction in all institutions of learning in Singapore is English, and all students are required to pass an English level proficiency exam before admission. Thus, the study included a sample that is demographically similar to previous studies of anxiety sensitivity, emotion regulation, and alexithymia in non-clinical populations (Kashdan, Zvolensky & McLeisgh, 2007), with respect to gender, language preference, education, and income background, but differing mainly in culture and ethnicity.

The disadvantage of using college students is that the question arises first of whether they are a suitable representation of clinical populations and, second, whether they are suitable representatives of non-clinical samples in general because of cohort effects related to their unique life-experiences. Nonetheless, the use of the non-clinical sample of university students is not inappropriate, given that anxiety sensitivity by itself is not a clinical disorder and that a wide range of scores can be found within this type of population. For example, Coles, Frost, Heimberg, and Steketee (2003) Used non-clinical college students to study the impact of anxiety sensitivity on the hoarding behaviors associated with the development of obsessive-compulsive disorder and showed a strong association. Similarly Salters-Pedneaul et al. (2006) examined the relationship between emotion regulation difficulties and generalized anxiety disorder in a non-clinical sample of college students and found significant results.

Design

The study involved a cross-sectional survey of non-clinical, college student participants from a culture outside North America. Cross-sectional designs are efficient in that they allow economical and replicable measurement of multiple variables in a sample of enough size to provide sufficient power for rich data analysis. However, a primary limitation of cross-sectional designs is weak internal validity due to an inability to establish causal directions of resulting data and questionable ecological validity due to the use of self-report questionnaires (Bryman, 2001). Nonetheless, the use of a cross-cultural sample in this study is helpful in that it extends the findings of studies in the U.S. that have used largely student populations to a population very similar in terms of education and income but that differs by nationality and cultural background.

Power Analysis

A power analysis was conducted based on a medium effect size of .4 (Cohen, 1992), and 7 predictors: ethnicity, anxiety sensitivity, and the following emotion regulation and alexithymia variables (a) lack of awareness of emotional responses, (b) lack of clarity of emotional responses, (c) nonacceptance of emotional responses, (d) difficulties controlling impulses when experiencing negative emotions, and (e) difficulties engaging in goal-directed behaviors when experiencing negative emotions). Based on the power tables listed in Kraemer and Theimann (1988), a sample size of 104 was recommended. Taking into account the need for an adequate sample in each ethnic sub-group, a total of 312 participants were recruited.

Method of Recruitment

The survey was introduced during lectures, and students were informed that their participation was entirely voluntary but that it would help improve the identification of mental health issues and risk factors among Singaporeans. Participants were also informed that the study

was anonymous and that the researchers involved would not be able to trace responses to individual students. Although the survey was initially intended to be offered online to a list of willing participants, many students expressed a preference for a pen and paper version, and so this was made available during the next lecture period.

Procedures

The first page of the survey provided brief information on the objectives and time requirement of the study, along with the contact details of the researchers and the head of the Institutional Review Board who approved the dissemination of the survey. Informed consent was sought prior to the start of the online survey as well as on the first page of the pen and paper versions, and students had to either click on a button to confirm that they had read and understood the information provided or sign the informed consent before they completed the first measure. Fifty-two students completed the online version, while the rest completed pen and paper versions. The informed consent included explanations of the following topics: the purpose of the study, confidentiality, the voluntary nature of participation (participants could stop filling out the survey at any time), foreseeable risks (uncomfortable thoughts or emotions), benefits of participation, (no direct benefits, but of service to research and prevention of anxiety disorders in Singapore), and contact numbers for the experimenter and Institutional Review Board. Confidentiality was described by informing participants that their responses would not be connected to identifying information in any way. An email address was provided to the participants to which they could direct requests for results, but no participants requested this information. Identifying information was not collected for any other purpose.

Instruments

Demographic questions. Questions were included in the survey to elicit participant demographics, specifically ethnicity, age, gender, and socio-economic status as a means of assessing the generalizability of the data to the broader Singapore population and previous studies with American and international samples. In addition, the collection of demographic information allowed for potentially controlling confounds of the hypothesized relationships between emotion regulation, alexithymia, and anxiety sensitivity (see Results section for details).

Anxiety Sensitivity Index – 3 (ASI-3). The ASI-3 (Taylor et al., 2007) is the most recent attempt to create a multidimensional anxiety sensitivity measure with a more stable factor structure. Taylor et al (2007) selected items from the earlier Anxiety Sensitivity Index – Revised (ASI-R, Taylor & Cox, 1998a) with emphasis on content validity in relation to the three most commonly seen subscales (physical, cognitive, and social concerns) and eliminated 12 items because it was unclear where they mapped. Further three questions were eliminated because of similarity to other items. The resulting pool consisted of six items for each of three subscales. The ASI-3 is only two items longer than the Anxiety Sensitivity Index (ASI), making it less cumbersome than the Anxiety Sensitivity Profile (ASP; Taylor & Cox, 1998b), which has 60 questions, and ASI-R, which has 36 items. Although data on the use of the ASI-3 are limited to the original published study, Taylor et al. made extensive efforts to test the new instrument on a large multinational sample, and the results of this appear extremely positive.

Internal consistency was calculated according to subscale, with the reported alpha ranging from .76 (French sample) to .86 (Canadian sample) for the physical concerns subscale, .81 (The Netherlands) to .91 (Canadian sample) for cognitive concerns, and .73 (Mexican sample) to .86 (Canadian sample) for the social concerns subscale (Taylor et al, 2003). Internal consistency of

each factor was subsequently found by MacDermott et al. (2009) and Escocard et al. (2008) to be excellent, ranging from .86 for the social concerns subfactor to .93 for the physical concerns factor.

The factorial validity of the ASI-3 (three factors) was supported by a confirmatory factor analysis of six replication samples, including non-clinical samples from the U.S., Canada, France, Mexico, the Netherlands, and Spain ($N = 4494$). The instrument demonstrated a stable, three-factor structure across gender in seven different samples. Escocard et al. (2008) found the same factor structure in their study of 585 Brazilian patients with a primary anxiety disorder diagnosis. These are encouraging because assessments of the instrument in both clinical and non-clinical populations from a number of countries and languages appear to support the correspondence with its theoretical base.

To ensure that studies using the improved ASI-3 are comparable to the substantial body of studies using the original ASI, Taylor et al. (2007) assessed the criterion-related validity by inter-correlating participants' scores on the subscales of the ASI-3 with their scores on the original ASI subscales after correcting for less-than-perfect reliability. Each ASI-3 subscale measured the same content domain as the ASI counterpart as indicated by correlations that approached unity. Predictive validity was measured by examining scores on the ASI-3 subscales across different groups, specifically individuals with panic disorder, obsessive-compulsive disorder, social anxiety disorder, and generalized anxiety disorder. Results were as expected, with the physical and cognitive concerns subscale being most closely related to panic disorder, while the social concerns subscale was most closely associated with social anxiety disorder. In Escocard's study (2008), panic disorder patients produced significantly higher overall ASI-3 scores. The fact that the correlations were seen in an international sample indicates that the ASI-

3 has good cultural validity, and these results speak well of the general validity and factor stability of the ASI-3. It appears from more recently published works that that the ASI-3 is fast gaining acceptance.

Toronto Alexithymia Scale-20 (TAS-20; Bagby, Parker, & Taylor, 1994). The TAS-20 is currently the most frequently used method in the measurement of alexithymia. It measures the following three facets: (a) difficulty identifying feelings and distinguishing them from bodily sensations (ID; seven items, such as *I have feelings that I can't quite identify*); (b) difficulty communicating or describing emotions to others (COM; five items, such as *It is difficult for me to find the right words for my feelings*); and (c) an externally oriented style of thinking (EXT; eight items, such as *I prefer talking to people about their daily activities rather than their feelings*). Twenty items are answered using a 5-point scale to indicate the extent to which the respondent agrees with each statement, ranging from one (*strongly disagree*) to five (*strongly agree*), with total scores of 20 to 100. Items 4, 5, 10, 18, and 19 are negatively keyed. Connelly and Denny (2007) published TAS-20 cut-off scores for non-alexithymia (≤ 52), borderline (52–60), and alexithymia (> 60). The TAS-20 has been found to have modest internal consistency, test-retest reliability, and good convergent and discriminant validity (Bagby, Parker, & Taylor, 1994; Bagby, Taylor, & Parker, 1994; Parker, Bagby, Taylor, Endler, & Schmitz, 1993). In addition, the three-factor structure of alexithymia has been found to be replicable across different cultural groups, including samples in the United States (e.g., Bagby, Parker, & Taylor, 1994) and in Asia (for a review, see Taylor et al., 1997). Although the three factors correspond to the main features of alexithymia as defined by Nemiah and Sifneos (1970a) and Nemiah et al. (1976), it is the ID and COM factors that were the main concern of this study. The internal consistencies of the ID and COM facets were moderate to good for each cultural group in this study, with alphas

ranging from .75 to .84 across the three groups. In contrast, the alphas for the EXT facet were considerably lower, ranging from .52 to .61 across the three groups.

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). Gratz and Roemer's conception of emotion regulation and the DERS scale was chosen for the purposes of this study because it offers a more comprehensive account of emotion regulation than other conceptions that tend to stress one or two specific subfactors, and because the identified subfactors are the specific focus of new empirically supported treatments purported to address difficulties in emotion regulation, specifically, ACT and Dialectical Behavior Therapy.

The DERS (Gratz & Roemer, 2004) is a 36-item self-report questionnaire that assesses clinically relevant difficulties in emotion regulation. The DERS was chosen over other emotion regulation scales such as the Acceptance and Action Questionnaire (AAQ; Hayes et al., 2002) because of its better alpha levels, the more comprehensive integration of multiple facets of emotion regulation, and the good factor loading of these subscales to the overall construct. Six subscale scores can be computed from the 36 items, namely (a) acceptance - nonacceptance of emotions (6 items; e.g., *When I'm upset, I feel guilty for feeling that way*), (b) goals - difficulties engaging in goal-directed behavior when distressed (5 items; e.g., *When I'm upset, I have difficulty concentrating*), (c) impulse - impulse control difficulties (6 items; e.g., *When I'm upset, I become out of control*), (d) awareness - lack of emotional awareness (6 items; e.g., *I pay attention to how I feel* [reversed]), (e) strategies - limited access to emotion regulation strategies (8 items; *When I'm upset, it takes me a long time to feel better*), and (f) clarity - lack of emotional clarity (5 items; *I am confused about how I feel*). Items are scored on a 5-point scale ranging from one (*almost never*) to five (*almost always*). Subscale scores are obtained by summing the corresponding items, and higher scores indicate more difficulty regulating

emotions. The authors describe good psychometric properties for all subscales, such as adequate to good internal consistency (.80) and stability (.69) and significant correlations with other emotion regulation measures (Gratz & Roemer, 2004). Both the overall DERS score and subscale scores have been found to have high internal consistency within both clinical (Fox et al., 2007; Gratz et al., 2008) and nonclinical populations (Gratz & Roemer, 2004; Johnson et al., 2008). Support for the construct and predictive validity of DERS scores within both clinical and nonclinical populations have also been found (Fox et al., 2007; Gratz et al., 2006, 2009; Gratz, Bornovalova, Delany-Brumsey, Nick, & Lejuez, 2007; Gratz & Roemer, 2004, 2008;).

The State-Trait Anxiety Inventory (STAI). The STAI is a research instrument for the study of anxiety in adults. It is a self-report assessment device, which includes separate measures of state and trait anxiety. According to the author, state anxiety reflects a transitory emotional state or condition of the human organism that is characterized by subjective, consciously perceived feelings of tension and apprehension, and heightened autonomic nervous system activity (Speilberger, 1983). State anxiety may fluctuate over time and can vary in intensity. In contrast, trait anxiety denotes more stable differences between individuals' anxiety proneness and refers to a general tendency to respond with anxiety to perceived threats in the environment.

The STAI consists of two forms: the Trait (STAI-Y1) and State (STAI-Y2). It was chosen over other measures of anxiety disorders because it allows for a general measure of anxiety that is not specific to particular diagnoses. Such an approach is desirable given the non-clinical nature of the sample. Both the Trait and State forms consist of 20 sometimes-overlapping questions, with the key distinction being that the State form indicates that participants answer in relation to how they feel at *this moment,* while those in the Trait form are to be completed according to how they *generally* feel. Questions on the State form include *"I feel at ease; I feel*

upset; I am presently worrying over possible misfortunes," while those in the Trait form include "*I feel that difficulties are piling up so that I cannot overcome them; I worry too much over something that really doesn't matter; I lack self-confidence.*" On both scales, participants indicate their agreement with the statement on a four-point scale ranging from "*Almost Never"* to *"Almost Always*." Scores on the STAI have a direct interpretation: high scores on their respective scales mean more trait or state anxiety, and low scores mean less. The stability of the STAI scales was assessed on male and female samples of high school and college students for test-retest intervals ranging from one hour to 104 days (Speilberger, 1983). The magnitude of the reliability coefficients decreased as a function of interval length. For the Trait-anxiety scale, the coefficients ranged from .65 to .86, whereas the range for the State-anxiety scale was .16 to .62 (Speilberger, 1983). This low level of stability for the State-anxiety scale is expected since responses to the items on this scale are thought to reflect the influence of whatever transient situational factors exist at the time of testing. Correlations are presented in the manual between this scale and other measures of trait-anxiety: the Taylor Manifest Anxiety Scale (.80) and the Multiple Affect Adjective Check List (.52).

Cognitive-Somatic Anxiety Questionnaire (CSAQ). The Cognitive-Somatic Anxiety Questionnaire (CSAQ; Schwartz et al., 1978) is a 14-item self-report inventory divided into two 7-item scales (Cognitive and Somatic) that appear to reflect cognitive or somatic anxiety. DeGood and Tait (1987) found good internal consistency (.81) and reasonably low correlations between the CSAQ with State (.22 - .34) and Trait (.13 - .50) anxiety, respectively, indicating that the CSAQ taps on different components of the anxiety construct. There appears to be considerable overlap between the Cognitive and the Somatic scales (r =. 62), but for the purposes of the current analyses, only the somatic anxiety scale was of interest. Questions in the subscale

include *"I feel tense in my stomach,"* and *"I feel jittery in my body,"* and participants indicate agreement on a five-point scale ranging from *"Not at all*" to "*Very much so.*"

Statistical Analyses

The data were screened for missing values, errors, and significant outliers as well as analyzed to determine whether such data are random or whether significant patterns exist. Descriptive statistics (means and standard deviations) are presented to provide an overview of the sample's characteristics, covering key demographic variables including age, socio-economic status (SES), ethnicity, and mean scores of participants on each of the instruments and subscales by ethnicity. Initial analysis of the data included Pearson product-moment correlation coefficients between all variables. To determine the appropriateness of the regression analyses, assumptions of normality, linearity, collinearity, and homoscedasticity were assessed. The correlation matrix and the variance inflation factors were also examined to assess multicollinearity. A Durbin-Watson test was performed to ensure independent errors. Bivariate scatterplots and standardized residual plots were also evaluated to test the assumptions of linearity and homoscedasticity.

Hypothesis 1. To test Hypothesis 1 (All emotion regulation subfactors positively moderate the relationship between anxiety sensitivity and anxiety, with emotional avoidance having the largest effect), moderation analyses using a series of hierarchical multiple regressions were conducted in the procedure outlined by Baron and Kenny (1986): a) An interaction term was created for the predictor variable and anxiety; b) hierarchical regression analysis was performed with control variables and anxiety sensitivity in the first block, emotion regulation in the second, and the interaction term in the third; c) the results of the hierarchical regression were examined to determine whether the interaction term is significant after controlling for the

predictor and proposed moderator. A moderated relationship was determined to be indicated if the interaction between the predictor variable (anxiety sensitivity) and the proposed moderator (emotional regulation) is significantly related to the outcome variable (anxiety; Baron & Kenny, 1986). The same procedure was replicated with subfactors of emotion regulation to ascertain whether any of them significantly moderates the relationship between anxiety sensitivity and anxiety.

Hypothesis 2. Hypothesis 2 states that emotion regulation mediates the relationship between anxiety sensitivity and anxiety (i.e., once emotional regulation is accounted for, anxiety sensitivity has a weaker direct effect on anxiety; see Figure 2). This hypothesis was tested using a series of hierarchical multiple regressions predicting anxiety sensitivity. The following paths were tested: (a) anxiety sensitivity on anxiety, (b) anxiety sensitivity on emotion regulation, (c) emotion regulation on anxiety, (d) anxiety sensitivity and emotional regulation on anxiety. The tests for mediation were conducted in the method outlined by Baron and Kenny (1986) and Kenny et al. (1998). Additionally, the Sobel test (Sobel, 1982) was used to test the hypothesis of no difference between total effect and direct effect.

Hypothesis 3. Hypothesis 3 states that anxiety sensitivity differs across cultures, with the Chinese culture having the highest level of anxiety sensitivity, followed by the Malay culture and the Indian culture. An analysis of variance was conducted to test whether different cultures display significantly different levels of anxiety sensitivity.

Hypothesis 4. Hypothesis 4 states that alexithymia partially mediates the relationship between culture and anxiety sensitivity (after controlling for alexithymia, culture is a less significant predictor of anxiety sensitivity). Plans were to test this hypothesis using a path analysis.

Chapter 3: Results

Demographic Data

The sample comprised 312 tertiary students between the ages of 18 and 46. The mean age was 20, and the modal age was 19. Fifty-eight percent of the participants were female, and 56% reported that their household incomes were below $30,000 per year (the lowest category in the questionnaire); 18% reported that their household income was between $30,000 and $60,000; and 17% had household incomes higher than $60,000. Some participants (8%) declined to provide this information.

Reflecting Singapore's racial make-up, 83% of participants were of ethnic Chinese descent, 6% were of Indian descent, 9% were of Malay descent, and 2% placed themselves in the *Other*s category[1]. A majority, 61%, of the entire sample reported that they identified with their ethnic identity either *closely* or *very closely*, while a further 28% reported identifying *somewhat closely* with their ethnic group.

The primary language that participants used when speaking to their friends and family was assessed. Nearly half (40%) used English and a combination of other languages equally often in their interactions with friends and family; 35% used only English; and 25% indicated that they used a language/languages other than English as their primary language. Table 1 provides a summary of the demographic data.

Descriptive Statistics and Differences across Ethnic Groups

Table 2 summarizes the reliability (Cohen's alpha), mean, and standard deviation for each measure for the entire sample, as well as for each ethnic group. One-way ANOVAs were

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[1] Singapore's ethnic composition is Chinese form 75.2%, Malays form 13.6%, Indians form 8.8%, while Eurasians and other groups form 2.4% (www.singstat.gov.sg). The *Others* category refers to Singapore residents who do not consider themselves to be Malay, Chinese or Indian.

used to assess whether each ethnic group's mean score on the various measures differed significantly from the other groups.

Table 1

Demographic Data

Variable	n	Frequency (%)
Age		
18	76	24%
19-20	126	40%
21-22	57	18%
>23	53	18%
Gender		
Male	131	42%
Female	181	58%
Income		
<$30,000	175	55.6%
$30-60,000	56	17.8%
>$60,000	53	17.6%
Ethnicity		
Chinese	259	83%
Indian	18	6%
Malay	26	9%
Ethnic Identification		
Very Closely/Closely	191	61%
Somewhat Closely/A little Closely	111	35%
Not at all	10	3%
Singapore Identity		
Very Closely/Closely	187	59%
Somewhat Closely	88	28%
Not at all	37	12%
Primary Language		
English only	109	35%
English and ethnic languages	125	40%
Ethnic languages only	78	25%

Note. $N = 312$.

Table 2

Reliability, means, and standard deviations of measures, and evaluation of differences across ethnic groups.

		Full Group		Chinese (n=260)		Indian (n=18)		Malay (n=26)		Between Group ANOVAs		
Variable	Alpha	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	*df*	*F*	*p*
ASI-3 Total	.93	37.58	12.95	37.69	12.63	31.56	8.99	38.38	13.32	3	1.43	.23
- Physical	.86	11.83	5.58	11.91	5.63	9.33	3.46	12.42	5.41	3	1.48	.22
- Social	.78	11.19	4.85	14.94	4.70	13.39	3.85	14.08	4.86	3	.94	.42
- Cognitive	.88	14.82	4.75	11.13	4.69	9.00	2.93	12.00	4.76	3	.20	.20
DERS Total	.93	85.06	20.12	85.05	19.81	71.22	4.13	92.42	15.35	3	4.26	.01**
- Awareness	.58	16.59	3.58	16.66	3.44	14.22	3.17	17.81	3.84	3	3.78	.01**
- Clarity	.80	11.48	4.09	11.52	4.19	8.78	2.51	12.27	2.46	3	3.19	.02*
- Impulse	.82	13.62	5.10	13.70	5.16	11.33	4.04	14.15	4.26	3	1.46	.23
- Goals	.84	13.84	4.48	13.65	4.37	13.17	5.46	15.69	3.76	3	1.87	.13
- Strategies	.86	17.25	6.06	17.24	5.99	13.89	5.56	19.15	5.90	3	2.83	.04*
- Acceptance	.86	12.61	4.78	12.64	4.72	9.83	3.22	13.35	4.67	3	2.51	.06
TAS Total	.83	52.34	10.41	52.55	10.30	43.61	9.59	55.85	8.72	3	5.55	.00**
- COM	.82	13.45	3.63	13.48	3.51	10.61	3.91	14.65	3.31	3	4.94	.00**
- ID	.83	17.65	5.52	17.82	5.40	12.33	4.83	19.27	4.89	3	7.00	.00**
State Anxiety	.91	40.41	9.13	40.56	8.91	34.06	9.96	42.42	6.72	3	3.55	.01**
Trait Anxiety	.91	44.15	9.65	44.27	9.12	37.78	11.70	46.58	6.95	3	3.45	.02*
Somatic Anxiety	.87	14.93	5.34	14.99	5.23	13.06	5.01	14.77	5.22	3	.77	.51

Note: ASI-3= Anxiety Sensitivity Index -3, DERS = Difficulties in Emotion Regulation Scale, TAS = Toronto Alexithymia Scale.
* = $p < .05$ and ** = $p < .01$. Bonferroni-corrected post hoc test results are presented in the paragraphs below.

Significant differences across ethnic groups were found for certain measures. One-way ANOVAS indicated that the alexithymia total (TAS Total) and subscales of difficulty in identifying feelings (ID) and difficulty communicating or describing feelings (COM), the difficulty in emotion regulation scale (DERS Total), and the DERS subscales of lack of emotional awareness (Awareness), lack of emotional clarity (Clarity), and limited access to emotion regulation strategies (Strategies) all differed across ethnic groups (all $p < .05$). Trait and state anxiety also differed significantly across ethnic groups ($p < .05$). Contrary to Hypothesis 3, anxiety sensitivity (ASI-3 Total) and its cognitive, physical, and social concerns subscales (ASI Cog, ASI Phys, ASI Soc) did not differ across ethnic groups ($p > .05$). Similarly, somatic anxiety, a measure of more physical attributions of anxiety as measured by the subscale of the CSAQ, did not differ along ethnic lines. Results of post-hoc tests are outlined in the following section, and quoted *p*-values are SPSS Bonferroni-adjusted to meet significance at a $p = .003$ level (.05/15).

Malay participants had the highest levels of state and trait anxiety (state anxiety: $M = 42.42$ $SD = 6.72$; trait anxiety: $M = 46.58$ $SD = 6.95$), but posthoc analysis (Bonferroni adjusted) demonstrated that their mean scores were not significantly higher than their Chinese peers ($p > .05$). Both Chinese ($p < .05$) and Malay ($p = .01$) participants had significantly higher state anxiety than their Indian peers. Chinese ($p < .05$) and Malay ($p = .01$) participants also had significantly higher trait anxiety than their Indian peers. On both of these measures, Indian participants demonstrated the lowest scores (state anxiety: $M = 34.06$, $SD = 9.96$; trait anxiety: $M = 37.78$, $SD = 11.7$)

Further, Bonferroni corrected post hoc analysis demonstrated that Chinese (DERS total: $M = 85.05$, $SD = 19.81$, $p < .05$) and Malay participants (DERS total: $M = 92.42$, $SD = 15.35$, $p < .01$) had significantly greater difficulties in overall emotion regulation than the Indian participants, who had much lower DERS scores ($M = 71.22$, $SD = 4.13$). The DERS scores of the Chinese and Malay groups did not differ significantly from each other ($p = .40$). This same trend of Indian participants having significantly lower scores ($p<.05$) than Chinese and Malay participants was apparent in the DERS subscales of Awareness (Chinese: $M =16.66$, $SD = 3.44$; Malay: $M = 17.81$, $SD = 3.84$; Indian: $M = 14.22$, $SD = 3.17$) and Clarity (Chinese: $M = 11.52$, $SD = 4.19$; Malay: $M = 12.27$, $SD = 2.46$; Indian: $M = 8.78$, $SD = 2.51$). Malay participants had the highest scores on the subscale of difficulty accessing emotion regulation strategies ($M = 13.89$, $SD = 5.56$), but post hoc tests demonstrated that this difference was only significant when compared to the Indians who had a far lower level of difficulty on this construct ($p < .05$). Malays and Chinese did not differ significantly on this subscale ($p = .71$).

The post hoc analysis also indicated that Indian participants had significantly lower scores in alexithymia (TAS Total; $M = 43.61$, $SD = 9.59$, $p < .01$), difficulty in identifying feelings (ID; $M = 12.33$, $SD = 4.83$, $p < .01$) and difficulty describing feelings (COM; $M = 10.61$, $SD = 3.91$). Chinese and Malay participants did not differ from each other on these subscales ($p = .39$).

The same variables were then examined by comparing those most comfortable in English to those most comfortable in their ethnic language. Interestingly, there were no Indians or Malays who spoke only Malay or an Indian regional language; therefore, comparisons were solely between Chinese individuals those who spoke mainly Mandarin

and Chinese individuals who spoke mainly English. Trait anxiety differed significantly ($p < .05$) between English and Mandarin speakers. Those who spoke only Mandarin ($M = 46.61$, $SD = 8.98$) had significantly higher trait anxiety scores than those who spoke English ($M = 43.24$, $SD = 9.98$, $p < .05$). Similarly, ethnic Chinese who spoke only Mandarin had significantly ($p<.05$) higher COM ($M = 66.14$, $SD = 9.98$) than those who spoke English ($M = 62.69$, $SD = 12.38$). Finally, participants who spoke only Mandarin had significantly greater ($p<.05$) levels of somatic anxiety ($M = 16.11$, $SD = 5.09$) scores than their English-speaking ethnic Chinese counterparts ($M = 14.55$, $SD = 5.23$, $p < .05$).

Similar analyses were conducted across the variables of ethnic identification, identification with the Singapore culture, gender, and income, but no significant differences were found.

Correlational Analyses

The literature indicates that anxiety sensitivity and emotion regulation difficulty are associated with high levels of anxiety. Correlation analyses revealed that emotion regulation (DERS Total) was correlated moderately with both state and trait anxiety ($r = .67$, $r = .69$), and ASI Total was somewhat less associated with anxiety (state anxiety: $r = .41$, trait anxiety: $r = .45$). ASI Total and DERS Total ($r = .56$) were also moderately correlated to each other. DERS Total had a higher correlation with alexithymia ($r = .62$), than the anxiety sensitivity correlation with the same variable ($r = .52$).

Among the DERS subscales, the lack of emotional clarity (Clarity) was most highly associated with both state ($r = .53$) and trait ($r = .51$) anxiety. The DERS subscale of difficulty with impulse control (Impulse) was moderately associated with both state ($r = .53$) and trait anxiety ($r = .56$) and somatic anxiety (CSAQ, $r = .51$). The subscale of

lack of access to emotion regulation strategies (Strategies) was also significantly correlated with state, trait, and somatic anxiety (r = .62, .65 and .54 respectively). Finally, the cognitive symptoms subscale was the ASI subscale with the highest correlation with DERS Total (r = .57).

It was hypothesized that alexithymia would positively predict anxiety sensitivity and anxiety. The alexithymia scale (TAS Total) correlated moderately with ASI Total (r = .52). Alexithymia (TAS Total) was correlated slightly more positively with trait anxiety (r = .55) and state anxiety (r = .51) than anxiety sensitivity (ASI Total) was with the same measures (state anxiety: r = .41, trait anxiety: r = .45), although not as strongly as the correlations between these and the DERS (r = .67 and .69 respectively). Among the alexithymia subscales, the scale of difficulty in identifying feelings (ID) correlated moderately with state and trait anxiety (r = .51 and .55), as did the scale of difficulty describing or communicating feelings (COM, r = .51, .55)

As predicted in the literature, ASI Total (r = .46) and DERS Total (r = .57) were significantly correlated with somatic anxiety (as measured by the somatic subscale of the CSAQ), although moderately so. Being a measure of one form or expression of anxiety, the somatic anxiety was moderately correlated with anxiety in general (State Anxiety: r = .55, Trait Anxiety: r = .60).

Table 3

Zero-order relations between all variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1.ASI Total	1	.82**	.86**	.91**	.52**	.49**	.55**	.56**	.00	.40**	.45**	.39**	.52**	.49**	.41**	.45**	.46**
2.ASI Physical	-	1	.60**	.69**	.42**	.40**	.45**	.44**	.01	.31**	.37**	.27**	.41**	.38**	.33**	.37**	.37**
3.ASI Social	-	-	-	.67**	.43**	.41**	.45**	.45**	-.06	.33**	.33**	.36**	.42**	.42**	.32**	.37**	.37**
4. ASI Cognitive	-	-	-	1	.50**	.48**	.53**	.57**	.04	.40**	.47**	.38**	.53**	.47**	.41**	.45**	.49**
5. TAS Total	-	-	-	-	1	.99**	.90**	.62**	.29**	.53**	.45**	.38**	.49**	.41**	.51**	.55**	.44**
6. COM	-	-	-	-	-	1	.85**	.59**	.32**	.49**	.43**	.36	.37**	.37**	.50**	.54**	.42**
7. ID	-	-	-	-	-	-	1	.66**	.23**	.56**	.49**	.43	.43**	.48**	.51**	.55**	.45**
8. DERS Total	-	-	-	-	-	-	-	1	.24**	.66**	.77**	.77**	.90**	.75**	.67**	.69**	.57**
9. Awareness	-	-	-	-	-	-	-	-	1	.27**	.00	.01	.03	-.05	.23**	.18**	.07
10. Clarity	-	-	-	-	-	-	-	-	-	1	.45**	.45**	.50**	.43**	.53**	.51**	.44**
11. Impulse	-	-	-	-	-	-	-	-	-	-	1	.58**	.72**	.53**	.53**	.56**	.51**
12. Goals	-	-	-	-	-	-	-	-	-	-	-	1	.67**	.47**	.47**	.51**	.37**
13. Strategies	-	-	-	-	-	-	-	-	-	-	-	-	1	.64**	.62**	.65**	.54**
14. Acceptance	-	-	-	-	-	-	-	-	-	-	-	-	-	1	.40**	.45**	.43**
15. State Anxiety	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	.84**	.55**
16. Trait Anxiety	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	.60**
17. Somatic Anxiety	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1

Note. ** = $p < .01$; all p-values were two-tailed.

Research Question 1

The first research question was: what is the role of emotion regulation and anxiety sensitivity in anxiety disorders? Answering this question and the related hypotheses required the use of regression analysis. Therefore, before performing the regressions, the data were examined to assess whether they met the requirements for this kind of analysis. The results of this assessment are presented below. Further, in all assessments, trait anxiety was adopted as the primary measure of anxiety for three reasons: a) this is a more stable form of anxiety, b) state and trait anxiety are very highly correlated ($r = .84$, $p < .01$), and, c) analyses using state, trait, and somatic anxiety produced very similar results. For the sake of efficiency, anxiety in the following sections refers to trait anxiety, unless otherwise specified.

Hypothesis 1 was tested by performing a series of hierarchical multiple regressions to determine whether emotion regulation positively moderates the relationship between anxiety sensitivity and anxiety and whether emotional avoidance is the strongest moderator among the sub-scales of emotion regulation. Next, whether emotion regulation mediates the relationship between anxiety sensitivity and anxiety (Hypothesis 2) was tested by performing a series of multiple regressions and applying Baron and Kenny's (1986) mediation test.

Suitability of Regression Analyses. The data were screened for missing values, errors, and significant outliers. A total of five participants provided incomplete responses, with an additional two providing scores that were significant outliers. These two participants were removed from the final database for statistical analyses. Missing data were found to be random and were substituted with neutral scores for the particular item

(e.g., 3 on a Likert scale of 1-5). To determine the appropriateness of the regression analyses, assumptions of normality, linearity, multicollinearity, and homoscedasticity were assessed. Q-Q plots were examined, along with the skewness and kurtosis of the variables. The data did not appear to deviate substantially from a normal distribution. The correlation matrix and the variance inflation factors were examined, and no indications of multicollinearity were found (Tolerance > .02, Variance Inflation Factor < 5). The Durbin-Watson test was performed and indicated independent errors (d > 1.5). Bivariate scatterplots and standardized residual plots were also evaluated to test the assumptions of linearity and homoscedasticity, and these were supported. Thus, the assumptions of normality, linearity, collinearity, and homoscedasticity were met.

Multivariate analyses of the direct impact of emotion regulation, anxiety sensitivity, and alexithymia on anxiety. The role of anxiety sensitivity, emotional regulation, and alexithymia was first examined in predicting anxiety. ASI-3 Total was found to be a significant predictor of anxiety (Step 1), accounting for 22% of the variance in anxiety ($R^2 = 22$, $\beta = .44$, $F\ (2,312) = 39.97$, $p < .01$). Adding DERS Total into the model explained a further 24% of the variance ($R^2 = 46$, $\beta = .63$, $F\ (3,312) = 79.95$, $p < .01$).

However, when DERS Total was incorporated in the model (Step 2), ASI-3 Total was no longer a significant predictor of anxiety ($\beta = .10$, $p = .06$). This indicates that the DERS Total is a better predictor of anxiety than anxiety sensitivity. Although not directly related to the current hypothesis, alexithymia (TAS Total) was then included in the model (Step 3) to assess the relative role of this construct in relation to the other main constructs of the study. Although alexithymia explained only a further 2% of the variance, it was

highly significant (β = .20, $p < .01$), as was emotion regulation (β = .53, $p < .01$). Again, anxiety sensitivity was no longer a significant predictor.

Table 4

Regression Models for ASI Total and DERS Total on Trait Anxiety

Step	Predictor	R2	B	SE	Beta	*t*	*p*
1	(Constant)	.19	.00	.48	44.13	92.08	.00
	ASI Total		.33	.04	.44	8.64	.00
2	(Constant)	.46	16.34	1.76	.00	9.31	.00
	ASI Total		.07	.04	.10	1.95	.06
	DERS Total		.29	.02	.62	12.31	.00
3	(Constant)		12.15	2.07	.00	5.88	.00
	ASI Total	.48		.04	.05	.93	.35
	DERS Total		.25	.03	.53	9.48	.00
	TAS Total		.18	.05	.20	3.66	.00

To further examine the relationships between anxiety sensitivity and emotion regulation difficulties with anxiety symptoms, a series of regressions were computed to test the association between the ASI-3 subscales and trait anxiety while controlling for DERS subscales and, conversely, the association between the DERS subscales and anxiety while controlling for each ASI-3 subscale. Table 5 displays the results of these analyses.

In the first model, the ASI-3 subscales were entered in Step 1. The cognitive subscale of the ASI-3 was the only one that significantly predicted anxiety, indicating that the association between anxiety sensitivity and anxiety in our sample was largely dependent on this subscale. The DERS subscales were entered in Step 2. They explained significant additional variance in anxiety scores (change in $R^2 = .26$, F (10,312) = 27.65, $p = .01$).

Table 5

Hierarchical Regression Models for ASI-3 subscales and DERS subscales on Anxiety

Model 1	R^2	B	SE	β	*t*	*p*
Step 1	.22					
Constant		32.43	1.59	.00	20.38	.00
ASI Physical		.21	.12	.12	1.67	.10
ASI Social		.17	.14	.08	1.22	.22
ASI Cognitive		.60	.15	.30	3.91	.00
Step 2	.48					
Constant		16.12	2.46	.00	6.55	.00
ASI Physical		.13	.10	.07	1.25	.21
ASI Social		.08	.12	.04	.67	.50
ASI Cognitive		.06	.13	.03	.47	.64
Awareness		.35	.12	.13	3.02	.00
Clarity		.36	.12	.15	2.92	.00
Impulse		.23	.11	.12	2.03	.04
Goals		.16	.12	.08	1.32	.19
Strategies		.58	.11	.37	5.09	.00
Acceptance		-.05	.11	-.02	-.45	.65

Model 2	R^2	B	SE	β	*t*	*p*
Step 1	.48					
Constant		17.42	2.31	.00	7.56	.00
Awareness		.34	.12	.13	2.93	.00
Clarity		.40	.12	.17	3.27	.00
Impulse Control		.25	.11	.14	2.25	.03
Goals		.16	.12	.70	1.30	.20
Strategies		.63	.11	.40	5.58	.00
Acceptance		.00	.11	.00	.02	.99
Step 2	.49					
Constant		16.12	2.46	.00	6.55	.00
Awareness		.35	.12	.13	3.02	.00
Clarity		.36	.12	.15	2.92	.00
Impulse		.23	.11	.12	2.03	.04
Goals		.16	.12	.08	1.32	.19
Strategies		.58	.11	.37	5.09	.00
Acceptance		-.05	.11	-.02	-.45	.65
ASI Physical		.13	.10	.07	1.25	.21
ASI Social		.08	.12	.04	.67	.50
ASI Cognitive		.06	.03	.03	.47	.64

In the second model, the order of entry was reversed. The DERS subscales were entered in Step 1 and accounted for a total 48% of the variance in anxiety ($R^2 = .48$, $p < .01$). In particular, the subscales of strategies, awareness, clarity, and impulse control were significant predictors, with the strategies subscale having the highest impact ($\beta = .36$, $p < .01$). Introducing the ASI-3 subscales in Step 2 did not explain significant additional variance in anxiety scores (change in $R^2 = .01$, $p > .05$). Again, after controlling for DERS scores, the ASI-3 subscales were not significantly impacting anxiety (Model 1). However, the same four DERS subscales remained significant, even after controlling for the ASI-3 subscales (Model 2). Although the beta values differed slightly across the models, the significance levels of the individual predictors remained the same regardless of the order in which either set of subscales (ASI-3 or DERS) were entered. This indicates that the DERS subscales were a far more robust predictor of anxiety than ASI-3 subscales and that the DERS subscales of Strategies, Awareness, and Clarity have the best predictive value.

Moderation Analysis. The next multiple regression examined whether DERS moderates the impact of anxiety sensitivity on anxiety, with emotional avoidance having the largest effect (Hypothesis 1). Moderation analysis was carried out by examining the interaction terms after mean-centering the independent variables and taking into account the main effects (Baron and Kenny, 1986). The results indicated that the DERS Total was not a significant moderator of the relationship between anxiety sensitivity and trait anxiety (see Table 7).

Table 6

Regression predicting the impact of the interaction between mean centered ASI-3 Total and DERS Total (ASI-3 X DERS) on Trait Anxiety

Predictor	R^2	B	SE	β	*t*	*p*
(Constant)	.46	44.43	.45	.00	99.67	.00
ASI-3 Total		.08	.04	.11	2.17	.03
DERS Total		.30	.02	.63	12.40	.00
ASI X DERS		.00	.00	-.06	-1.40	.16

When the emotion regulation subscales were introduced as moderators (see Table 8), only Clarity had a significant effect (β = -.13, p = .01). Contrary to the hypotheses, greater emotional clarity (lower score on the Clarity subscale) interacts modestly with anxiety sensitivity to predict higher levels of anxiety.

Mediation Analysis. Having examined the direct effects and the possibility that emotional regulation moderates the relationship between anxiety sensitivity and anxiety, a series of regressions were conducted to explore the possibility that emotion regulation mediates the relationship between anxiety sensitivity and anxiety (Hypothesis 2). According to Baron and Kenny (1986), a variable may be considered a mediator to the extent to which it carries the influence of a given independent variable (IV) to a given dependent variable (DV). Mediation occurs when (1) the IV significantly affects the mediator, (2) the IV significantly affects the DV in the absence of the mediator, (3) the mediator has a significant unique effect on the DV, and (4) the effect of the IV on the DV shrinks upon the addition of the mediator to the model. MacKinnon, Warsi, and Dwyer (1995) introduced statistical methods to assess mediation, and their version of the "Sobel test" is utilized.

Table 7

Hierarchical regressions predicting the impact of the interaction between mean centered ASI Total and DERS Subscales on anxiety.

Predictor	R2	B	SE	β	*t*	*p*
ASI-3Total X Lack of Emotional Awareness						
Constant	.48	44.13	.47	.00	94.07	.00
ASI-3		.33	.04	.44	8.75	.00
Awareness		.51	.13	.19	3.85	.00
ASI-3 X Awareness		-.01	.01	-.05	-.97	.33
ASI-3 Total X Lack of Emotional Clarity						
Constant	.33	44.65	.48	.00	93.92	.00
ASI-3		.22	.04	.30	5.84	.00
Clarity		1.01	.13	.43	7.92	.00
ASI-3 X Clarity		-.02	.01	-.13	-2.56	.01
ASI-3 Total X Lack of Impulse Control						
Constant	.34	44.05	.47	.00	93.38	.00
ASI-3		.18	.04	.25	4.62	.00
Impulse		.80	.10	.43	8.25	.00
ASI-3 X Impulse		.00	.01	.02	.49	.63
ASI-3 Total X Goals						
Constant	.32	44.14	.47	.00	93.45	.00
ASI-3		.22	.04	.30	5.75	.00
Goals		.81	.11	.38	7.44	.00
ASI-3 X Goals		.00	.01	-.01	-.11	.91
ASI-3 Total X Lack of Emotional Regulation Strategies						
Constant	.42	44.46	.45	.00	97.75	.00
ASI-3		.12	.04	.17	3.26	.00
Strategies		.89	.08	.57	11.08	.00
ASI-3 X Strategies		-.01	.01	-.07	-1.61	.11
ASI-3 Total X Acceptance						
Constant	.25	44.41	.52	.00	85.83	.00
ASI-3		.24	.04	.32	5.65	.00
Strategies		.56	.11	.28	4.88	.00
ASI-3 X Strategies		-.01	.01	-.06	-1.20	.23

A model in which ASI-3 Total was the predictor, DERS Total the mediator, and anxiety the outcome was examined. In other words, to what extent an individual's

emotion regulation ability accounted for differences in the association between their anxiety sensitivity and their level of anxiety was tested. Consistent with the guidelines of Kenny et al. (2003), anxiety symptoms on ASI-3 (Path C in Table 9) were regressed first. The results indicated that anxiety symptoms were significantly predicted by ASI-3. Then regression of DERS Total difficulties on ASI-3 (Path A in Figure 4 and Table 9) was completed. The results indicated that ASI-3 difficulties significantly predicted emotion regulation (DERS Total) difficulties. The effect of DERS Total difficulties on anxiety (Path B in Figure 4 and Table 9) was significant, indicating that DERS Total difficulties significantly predicted anxiety symptoms.

Next, anxiety symptoms on DERS Total and ASI-3 difficulties were regressed simultaneously. The effect of ASI-3 on anxiety (Path C in Figure 4 and Table 9) was significant at the .05 level, indicating that ASI-3 affects anxiety symptoms above and beyond the effect accounted for by difficulties in emotion regulation and that the effect of anxiety sensitivity on anxiety is not fully mediated by emotion regulation. However, according to the conditions laid out by Baron and Kenny (1986), the results may indicate a partial mediation. A Sobel test was run to statistically determine whether the mediator variable significantly carries the influence of the IV to the DV. The Sobel Test statistic was found to be significant ($t = 8.48$, $p < .01$), indicating that emotion regulation does partially mediate the relationship between anxiety sensitivity and anxiety. It was further found that 78% of the total effect of anxiety sensitivity on anxiety is mediated by emotion regulation.

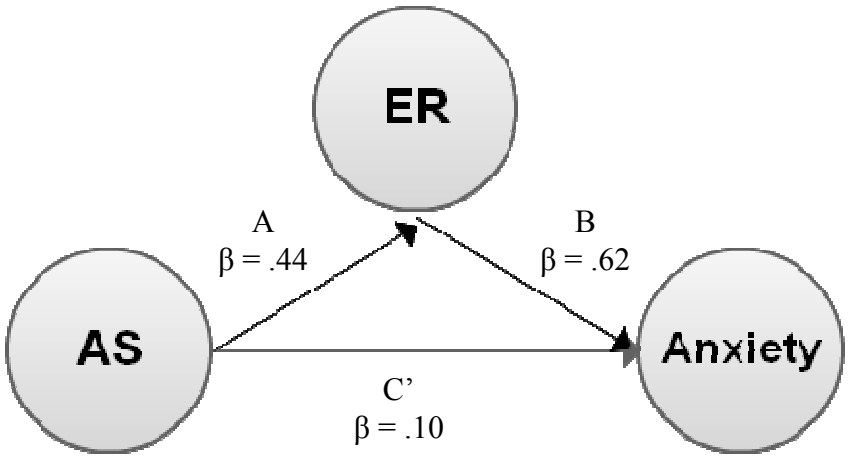

Figure 4. Mediating role of emotion regulation

Table 8

Summary of Multilevel Regression Analyses for the Mediational Model

Path	Predictor variable	Outcome variable	B	SE	β	*t*	*p*	*Sobel Test*
C	ASI-3	Trait Anxiety	.33	.04	.44	8.64	.00	$t = 8.48$ $p < .01$
A	ASI-3	DERS	.87	.07	.55	11.70	.00	Percentage of
B	DERS (after controlling for AS)	Trait Anxiety	.07	.02	.62	12.31	.00	total effect mediated:
C'	AS (after controlling for DERS)	Trait Anxiety	.06	.04	.10	1.95	.05	77.76

The procedure outlined above was repeated for each of the DERS subscales (Table 10). All DERS subscales, except for Awareness, partially mediated the effect of anxiety sensitivity on anxiety.

Table 9

Summary of Multilevel Regression Analyses for the Mediational Model with DERS Subscales as potential mediators

Step	Path	Predictor variable	Outcome variable	B	SE	β	*t*	*p*	*Sobel Test*
Awareness as a Mediator									
1	C	AS	Trait Anxiety	.33	.04	.44	8.64	.00	$t = .43$
2	A	AS	Awareness	.01	.02	.02	.43	.67	$p = .67$
3	B	Awareness (after controlling for AS)	Trait Anxiety	.51	.13	.19	3.85	.00	Percentage of total
	C’	AS (after controlling for Awareness)	Trait Anxiety	.32	.04	.44	8.74	.00	effect mediated: 1.02
Goals as a Mediator									
1	C	AS	Trait Anxiety			.44	8.64	.00	$t = 5.22$
2	A	AS	Goals	.14	.02	.38	7.32	.00	$p < .01$
3	B	Goals (after controlling for AS)	Trait Anxiety	.80	.11	.38	7.46	.00	Percentage of total
	C’	AS (after controlling for Goals)	Trait Anxiety	.22	.04	.30	5.79	.00	effect mediated: 33.11
Clarity as a Mediator									
1	C	AS	Trait Anxiety	.33	.04	.44	8.64	.00	Sobel = 5.23
2	A	AS	Clarity	.12	.02	.39	7.36	.00	$p < .01$
3	B	Clarity (after controlling for AS)	Trait Anxiety	.89	.12	.38	7.44	.00	Percentage of total
	C’	AS (after controlling for Clarity)	Trait Anxiety	.22	.04	.29	5.77	.00	effect mediated: 33.24
Impulse Control as Mediator									
1	C	AS	Trait Anxiety	.33	.04	.44	8.64	.00	Sobel = 5.93
2	A	AS	Impulse	.17	.02	.44	8.54	.00	$p < .01$
3	B	Impulse (after controlling for AS)	Trait Anxiety	.79	.10	.42	8.26	.00	Percentage of total
	C’	AS (after controlling for Impulse)	Trait Anxiety	.19	.04	.26	4.97	.00	effect mediated: 42.08

Table 9 continued:

Step	Path	Predictor variable	Outcome variable	B	SE	β	*t*	*p*	*Sobel Test*
Strategies as a Mediator									
1	C	AS	Trait Anxiety	.33	.04	.44	8.64	.00	Sobel = 7.60
2	A	AS	Strategies	.24	.02	.52	10.58	.00	$p < .01$
3	B	Strategies (after controlling for AS)	Trait Anxiety	.87	.08	.55	10.94	.00	Percentage of total
	C’	AS (after controlling for Strategies)	Trait Anxiety	.12	.04	.16	3.07	.00	effect mediated: 64.76
Acceptance as a Mediator									
1	C	AS	Trait Anxiety	.33	.04	.44	8.64	.00	Sobel = 4.27
2	A	AS	Acceptance	.18	.02	.48	9.68	.00	$p < .01$
3	B	Acceptance (after controlling for AS)	Trait Anxiety	.54	.11	.27	4.75	.00	Percentage of total
	C’	AS (after controlling for Acceptance)	Trait Anxiety	.23	.04	.31	5.54	.00	effect mediated: 29.27

All emotion regulation subscales except Awareness were found to be mediators. The DERS subscale of limited access to emotion regulation strategies was the strongest mediator, with 64% of the total effect mediated, followed by impulse control difficulties (42% of the total effect mediated).

Although not hypothesized, an alternative mediation analysis was run to reveal the underlying relationships in the data. This series of regressions were carried out to examine the possibility that anxiety sensitivity mediated the relationship between emotion regulation and anxiety (Table 11). The results indicate that the direct effect (path C) of DERS on anxiety is not greatly reduced after including anxiety sensitivity in the model (path C'), making it unlikely that any mediation is taking place.

Table 10

Summary of Multilevel Regression Analyses for the Mediational Model (Anxiety Sensitivity as a mediator of the relationship between Emotion Regulation and Anxiety).

Path	Predictor variable	Outcome variable	B	SE	β	*t*	*p*
C	DERS	Trait Anxiety	.32	.02	.67	16.01	.00
A	DERS	AS	.35	.03	.55	11.70	.00
B	AS (after controlling for DERS)	Trait Anxiety	.07	.04	.10	1.95	.05
C'	DERS (after controlling for ASI)	Trait Anxiety	.29	.02	.62	12.31	.00

Research Question 2

Hypothesis 3. The third hypothesis states that anxiety sensitivity differs across cultures, with the Chinese culture having the highest level of anxiety sensitivity, followed by the Malay culture and the Indian culture. Analyses of variance were conducted to test

whether different cultures displayed significantly different levels of anxiety sensitivity (Table 2), and no significant differences were found.

Hypothesis 4. Hypothesis 4 states that alexithymia partially mediates the relationship between culture and anxiety sensitivity. However, since anxiety sensitivity and culture were not found to be significantly related, testing Hypothesis 4 was unnecessary.

Chapter 4: Discussion

This study sought to better understand the relationship between anxiety sensitivity and emotion regulation, and their relative roles in relation to anxiety (research question 1). Such an understanding of the relative role of anxiety sensitivity and other risk factors is important, because it impacts decisions as to the most viable means of preventative care and anxiety management training of at risk populations. Further, the scientific activity on anxiety sensitivity has been focused principally on *main effect* types of questions (Kashdan, Zvolensky, & McLeish, 2007), and how anxiety sensitivity may interplay with other processes relevant to anxiety psychopathology is less well documented. Various researchers have argued that anxiety sensitivity is unlikely to be a proximal risk factor for psychopathology, speculating that the relation is probably moderated (Kashdan et al., 2008; Cisler et al., 2010) or mediated by other constructs (e.g., Zinbarg, Brown, Barlow, & Rapee, 2001; Zvolensky & Forsyth, 2002). Following this train of thought, this study found that emotion regulation was a better predictor of anxiety than anxiety sensitivity in our non-clinical sample, and that emotion regulation was predominantly the mechanism (mediator) through which anxiety sensitivity impacted anxiety.

First, the predictive value of emotion regulation, alexithymia, and anxiety sensitivity on anxiety was compared. Regression analyses demonstrated that emotion regulation was more closely associated with anxiety than anxiety sensitivity or alexithymia. Anxiety sensitivity accounted for 22% of the variance in anxiety in our sample when it was the sole predictor in a regression equation, while emotion regulation accounted for 48% under the same procedure. When both variables were included in the

model, with emotion regulation entered first and anxiety sensitivity second, anxiety sensitivity accounted for only a further 1% of the variance in anxiety, pointing to the possibility that some of the predictive value of anxiety sensitivity is shared with emotion regulation.

Interestingly, alexithymia functioned similarly to anxiety sensitivity. When it was included in a model with the DERS subscale, it explained only a further 2% of the variance in anxiety. This is unsurprising since alexithymia is conceptually similar to the emotion regulation variables of lack of emotional clarity (Clarity) and lack of emotional awareness (Awareness). Alexithymia also outperformed anxiety sensitivity as a predictor in that it remained a significant predictor, even after controlling for emotion regulation difficulties.

Following this, a series of analyses were carried out with the subscales of the three variables of interest. When anxiety sensitivity subscales were entered into the regression model, only the cognitive concerns subscale was significantly associated with anxiety. Of the six emotion regulation subscales, four (impulse control difficulties, limited access to emotion regulation strategies, lack of emotional awareness, and lack of emotional clarity) were significant predictors of anxiety. These subscales were far more closely associated with anxiety in our sample than any of the anxiety sensitivity subscales.

These findings provide a sound rationale for Hypotheses 1 and 2, since they demonstrate that emotion regulation and anxiety sensitivity are related in some manner, even though the precise nature of the relationship between the two variables is still unclear in the literature. Hypotheses 1 and 2 posited that emotion regulation respectively

moderated or mediated the relationship between anxiety sensitivity and anxiety. The results showed that emotion regulation did not moderate the relationship between anxiety sensitivity and anxiety, that is, having emotion regulation difficulties did not increase or decrease the strength of the relationship between anxiety sensitivity and anxiety, except for a very small negative effect that was found for the aspect of emotion regulation called lack of emotional clarity. Having greater clarity about one's emotions slightly increased the association between anxiety sensitivity and anxiety, such that presumably, an individual high in anxiety sensitivity is likely to suffer greater levels of anxiety if she is clearer about her emotions. This result was small and unexpected, and not in line with theory or previous research, making it difficult to explain why it was found in this study. In general, hypothesis 1 was not supported: emotion regulation variables did not increase or decrease the strength of the relationship between anxiety sensitivity and anxiety.

Mediation analysis was then conducted to test whether emotion regulation is the mechanism by which anxiety sensitivity impacts anxiety (Hypothesis 2). The results indicated that emotion regulation difficulties do in fact partially mediate the relationship between anxiety sensitivity and anxiety. In the current sample, 77.8% of the effect of anxiety sensitivity on anxiety was found to be mediated by emotion regulation difficulties.

The cross-sectional design of the study limits our ability to make causal statements: in this case, that emotion regulation difficulties *cause* anxiety in individuals with high anxiety sensitivity. Thus, an alternative model was tested in which anxiety sensitivity was the mechanism through which emotion regulation difficulties impact anxiety. Anxiety sensitivity was not a significant mediator in this model, explaining only

7% of the effect of emotion regulation on anxiety. This alternative model was therefore unsupported.

Since emotion regulation difficulties were found to be the mechanism (mediator) through which anxiety sensitivity asserts its impact on anxiety, the next set of analyses proceeded to examine which particular aspects of emotion regulation difficulty accounted for this impact. The analyses indicated that the limited access to emotion regulation strategies was the most important mediator, followed by impulse control difficulties, lack of emotional clarity, and difficulties in engaging in goal-directed behavior when distressed (in descending order).

The same emotion regulation skills of emotional clarity, impulse control, and access to emotion regulation strategies had been found in the earlier analysis to have the highest correlation with anxiety (along with emotional awareness, which was not a significant mediator). Interestingly, although difficulty engaging in goal-directed behavior was not a significant predictor of anxiety, it was a significant mediator of the relationship between anxiety sensitivity and anxiety. These subscales were far more closely associated with anxiety in our sample than any of the anxiety sensitivity subscales. Thus, all aspects of emotion regulation, except for the lack of emotional acceptance, are directly predictive of anxiety and/or are also largely responsible for the association between anxiety sensitivity and anxiety.

These findings are interesting in that they seem to contradict the theoretical view of anxiety sensitivity as a latent personality trait (Naragon-Gainy, 2010). Emotion regulation is conceptualized as a series of skills that can presumably be taught through mechanisms such as ACT and Dialectic Behavior Therapy, and the present findings

indicate that the effect of anxiety sensitivity can be mitigated by the inculcation of emotion regulation skills. Although the effect of anxiety sensitivity on anxiety is not totally removed by these skills, the residual effect is minimal after they are taken into account.

Further, although the literature examining the role of impulse control and goal-directed behavior on anxiety disorders is sparse and most studies do not dismantle the effects of these elements from other elements of ACT and the DBT protocols, our results indicate that these are important strategies. Presumably, when acting in a goal-directed manner, the individual would be forced to confront much of the same stimuli that would have to be tolerated under traditional behavioral interventions (e.g., staying in a social situation to further one's goal of increasing friendships, despite the intense social anxiety one feels) and this may be the mechanism by which impulse control and goal directed behaviors have their impact.

Thus, teaching students who are a conveniently accessible through the school system, for example, how to become aware of the feelings they are experiencing, identify the specific emotions, motivating them and giving them ways in which to be aware of their impulses, identify more strategically beneficial goals, and to continue to engage in goal directed behaviors may be effective ways of preventing the development of high anxiety levels and reducing the risk and/or impact of anxiety sensitivity.

While this study found emotional awareness, emotional clarity, impulse control, and goal-directed behavior aspects of emotion regulation to be important, lack of access to emotion regulation strategies was the most significant mediator of the effect of anxiety sensitivity on anxiety, and a key direct predictor of anxiety. The lack of access to such

strategies was seen by endorsements of items such as *When I'm upset, I believe that I'll end up feeling very depressed*, *When I'm upset, I believe that I will remain that way for a long time*, *When I'm upset, I believe that wallowing in it is all I can do*, and *When I'm upset, my emotions feel overwhelming*. It is therefore possible that providing a set of strategies that individuals can practice and internalize beforehand may help improve self-efficacy, enable them to feel more in control of emotions when upset, and reduce levels of both anxiety sensitivity and anxiety.

Although the findings indicate that anxiety sensitivity may in fact be partially addressed by the provision of such emotion regulation skills, one factor of emotion regulation, specifically emotional acceptance, did not factor as a direct predictor of anxiety or as a large mediator of the relationship between anxiety sensitivity and anxiety. Interestingly, the insignificance of emotional acceptance is contrary to the theoretical and therapeutic premises of ACT and the many empirical studies that have shown it to be a factor in depression (DeGenova, Patton, Jurich, & MacDermid, 1994), substance abuse (Ireland, McMahon, Malow, & Kouzekanani, 1994), and the sequelae of child sexual abuse (Leitenberg, Greenwald, & Cado, 1992). However, findings mimic the meta-analytic findings of Aldao, Nolen-Hoeksema, and Schweizer (2010) on the impact of emotion regulation strategies across psychopathology, which demonstrated that the effect size of acceptance was medium and non-significant in both anxiety and other disorders. The reason for the poor empirical support for the clinically widely targeted aspect of emotional acceptance is unclear from this and other cross-sectional, self-report dependent studies, and more longitudinal, dismantling studies of its role in established protocols may be necessary.

The second research question pertains to the role of culture and alexithymia (and the related emotion regulation subfactors) in the relationship between anxiety sensitivity and anxiety. The hypothesis that anxiety sensitivity would vary between the cultural groups, with Chinese participants having the highest levels, was not supported in this study. Although alexithymia is thought to contribute to anxiety sensitivity and the construct has been demonstrated to be elevated in Chinese individuals relative to individuals of other cultures (Dion, 1996), this was not the case in the current sample in comparison to the Indian and Chinese groups. Because anxiety sensitivity did not vary across cultures, Hypothesis 4, which proposed that alexithymia mediated the relationship between culture, anxiety sensitivity, and anxiety, could not be tested. However, the data allowed for a number of interesting observations and several analyses that further the literature in un-hypothesized ways.

First, Malay and Chinese participants appeared to have generally higher levels of anxiety than Indian participants. The same trend was observed in alexithymia, emotional awareness, and clarity. The reasons for Indian Singaporeans' lower levels of difficulty in these areas is not clear, and aspects of the Singaporean Indian culture would have to be better understood in order to make sense of this finding. One possibility that has to be addressed in cross-cultural samples is less willingness to report difficulties. A second possibility is that Indians who historically (from the British occupation of India) have greater familiarity and comfort with the English language have developed lower levels of alexithymia that is reducing their susceptibility to high levels of anxiety seen in other Singapore groups. Although the current study had insufficient Indian participants to conduct this analysis, it would be beneficial if future studies could explore the role of

emotion regulation subfactors in predicting anxiety amongst this group versus the other ethnic groups. Such an analysis would help tailor prevention protocols more appropriately to each ethnic group.

Second, anxiety seemed to vary across language groups, with the individuals who were most comfortable only in Mandarin having greater trait and somatic anxiety scores than those who spoke only English. This variation was not accompanied by similar differences in anxiety sensitivity or emotion regulation difficulties. Thus, although the reason for the difference in anxiety scores is not clear from the study, one possibility is that a lack of comfort with the English language may be heightening the general sense of anxiety experienced by this group, as English is the most commonly used language in Singapore and is the medium of instruction in schools and colleges. A second possible reason for the heightened anxiety amongst this group is that there has been a recent wave of immigration to Singapore from China and India, and Chinese students in particular tend to struggle also as a fact of their recent immigration experience. Again, more information is necessary to confirm this and to tailor appropriate interventions so that this group may be effectively targeted for preventative measures and greater psychosocial support.

Third, there appeared to be significant differences in anxiety and anxiety sensitivity scores in Singapore compared to international samples. Although our current sample did not demonstrate differences in anxiety sensitivity across Chinese, Indian, and Malay participants, the overall anxiety sensitivity and anxiety scores by the Singapore sample were in marked contrast to those previously reported for American and international participants. For example, Taylor et al. (2007) found in their assessment of

the validity and reliability of the ASI-3 that non-clinical participants from France (*M* = 16.4), Mexico (*M* = 15.2), and Spain (*M* = 14.2) displayed moderately higher levels of anxiety sensitivity than participants from the U.S. and Canada (*M* = 12.8). In contrast, the current Singapore sample had a mean anxiety sensitivity score of 37.58, which is far above these scores. While Rodriguez et al. (2007) demonstrated that ASI-3 scores of individuals with panic, generalized anxiety disorder, social phobia, and mood disorder tend to be above 44, Weems et al. (2002) found an anxiety sensitivity score of above 30 indicated panic disorder. In Smit et al.'s (2008) treatment outcome meta-analysis, it was found that studies including at-risk samples stipulated a minimum ASI-3 score of between 16 and 25, while treatment-seeking samples had anxiety sensitivity scores starting as low as 20. The scores of the Singapore participants were more akin to those of panic patients in the U.S. (Taylor et al., 1992) than any non-clinical group. Although all three subscales of anxiety sensitivity were elevated, in particular, Singaporeans have greater cognitive than physical or social concerns, indicating that they are most sensitive to concentration difficulties arising from anxiety. Statements they tended to endorse to a high degree included *When I cannot keep my mind on a task, I worry that I might be going crazy*, *It scares me when I am unable to keep my mind on a task*, *When I feel "spacey" or spaced out I worry that I may be mentally ill*. Interestingly, although panic disorder is a close correlate of the cognitive concerns subscale, the limited number of published studies have not demonstrated elevated rates of panic disorder or generalized anxiety disorder in Singapore (Lim et al., 2005).

In the absence of more similar studies in Singapore and the Southeast Asian region, it is difficult to speculate why these scores are greatly elevated. Not only were

scores higher across the whole group, the scores of ethnic Chinese, Malay, and Indian participants did not differ significantly in this regard. Although racially diverse, the sampled group as a whole identified closely or very closely with their Singapore identity (59%), and the majority were most comfortable speaking the nationally unifying language of English (75%). It is possible then that although anxiety sensitivity did not vary significantly across culture in our sample, our hypothesis that anxiety sensitivity varies across culture stands, with the caveat being that anxiety sensitivity varies between the Singaporean culture and others previously studied.

This study also examined the general anxiety levels of Singapore participants. Although statistical comparisons were not possible, the students as a whole had slightly higher Trait Anxiety Scores (M = 44.4, SD = 9.96) than Canadian students (M = 37.89, SD = 4.46; Lai & Linden, 1993) and Spanish students at the University of Barcelona (M = 38.79, SD = 10.33; Bados, Gómez-Benito & Balaguer, 2010). Scores were more akin to those of non-clinical Japanese college students (M = 47.0, SD = 10.2; Iwata & Higuchi, 2000) and Western clinical samples. For example, patients in orthopedic and psychosomatic wards of a hospital sampled recently in a study by Muschalla, Linden, and Olbrich (2010) found an average trait anxiety score of 47.7 (SD = 13.1). While these differences are specifically in regard to trait anxiety scores internationally (outside of Japan), it does not appear that the differences in anxiety translate to higher levels of pathological anxiety. There were also differences within the group with Chinese and Malay participants having higher levels of anxiety than Indian participants.

Noting the generally higher anxiety and anxiety sensitivity scores in the Singapore sample, the emotion regulation scale (DERS) scores of our sample were

examined and compared to those in recently published studies. The current sample has slightly higher DERS Total scores (M = 85.06, SD = 20.12) than similar non-clinical college samples in the U.S. (Lavender & Anderson, 2009; M = 76.72, SD = 19.22, Salters-Pedneault et al., 2006; M = 82.66, SD = 23.41), but not dramatically so. The difference could not be attributed to any particular subscale/s but appears spread out across emotion regulation subscales.

Alexithymia scores have also been reported in the literature to vary between the cultural groups (Le, Berenbaum, & Raghavan, 2002; Dion, 1996). The current participants did not appear to have distinctly different alexithymia scores in the areas of difficulty identifying and communicating feelings (ID: M = 17.65, SD = 5.52; Com: M = 13.45, SD = 3.63) from European Americans (ID: M = 15.9, SD = 5.5, COM: M = 13.4, SD = 5.2), Asian Americans (ID: M = 17.7, SD = 5.7, COM:M = 15.7, SD = 4.9), and Malaysian students (ID:M = 18.5, SD = 5.7, COM:M = 15.7, SD = 3.6) sampled by Le, Barenbaum, and Raghavan (2002). Singapore students scored slightly higher on the difficulty identifying emotions and communicating/describing emotions than European Americans, similar to Asian Americans, and appeared to have slightly lower scores than the Malaysian students sampled in the study.

Although not statistically confirmed, and more formal analysis is necessary to more comprehensively compare anxiety levels in Singapore to those found internationally, the findings generally indicate that Singapore students have greater anxiety sensitivity and are more anxious than their North American and other international peers. However, these elevated rates have not yet appeared to translate to higher international rates of psychopathology, and the limited number of published

studies does not demonstrate elevated rates of anxiety disorders (Lim et al. 2005). The reason for this is unclear. Iwata and Higuchi (2000) found that the Japanese students whose scores were dramatically higher than their American counterparts were simply less likely to report positive feelings that composed part of the state and trait anxiety subscales, rather than being more likely to report negative feelings. The authors speculated that this reluctance to endorse positive feelings is partially the result of the Japanese collectivist tendency that tends to foster "self-criticism" and less expression of positive affect. While it is possible that similar influences are at work in Singapore, greater study is required.

Findings indicate that cultural and societal factors unique to Singapore may be playing a major role in fostering the greater levels of anxiety and anxiety sensitivity. Commonly cited stressors in Singapore include the high levels of academic stress, high levels of competition in business and work, the achievement-oriented culture that values material success, the lack of guaranteed pensions or social safety nets for those unable to work due to old age or incapacity, and high levels of income inequality and status anxiety. As a result, it is possible that what is considered *normal* levels of anxiety in Singapore are in fact unusually high when compared with Western norms, and that Singaporeans possess an array of coping mechanisms that prevent the translation of high anxiety sensitivity and anxiety into psychopathology. The impact of these and other stressors must be more thoroughly explored, along with the role of coping mechanisms that may be employed to manage them

Whatever the reason for the elevated anxiety and anxiety sensitivity, the logistic regression analyses in the previous section indicate that their slightly elevated (relative to

international levels) emotion regulation difficulties appear to play a bigger role than their greatly elevated anxiety sensitivity levels. These findings point to the value of teaching emotion regulation skills to this population as part of preventative measures as outlined in the previous section.

While the study demonstrates that efforts to teach emotion regulation should focus on the biggest mediators and direct predictors, such efforts are likely to be most effective if they appropriately invoke local expressions and idioms of distress (Hinton and Fernandez, 2010). Similarly, efforts to teach impulse control, goal-directed behavior, and general emotion regulation strategies are likely to be effective if they invoke the experience of Singaporeans by using examples drawn, for instance, from their experience of mandatory national service or living in high rise governmental flats where 80% of the population lives. Similarly, the acceptance of the teaching is likely to be increased by the Use of "Singlish" terms. Singlish is a widely used local pidgin-dialect of "Singlish" that blends together words from Chinese, Malay, and Indian languages.

Limitations. The findings of the present study must be considered in light of the limitations present. First, it employs a non-clinical sample, rendering it impossible to determine whether the mediating relationship found here extends to individuals with actual anxiety disorders, and whether emotion regulation differentially mediates symptoms of the varying anxiety disorders (e.g., is emotion regulation as relevant for understanding obsessive-compulsive disorders as it is for understanding generalized anxiety disorder?) However, this preliminary investigation is consistent with other studies that have identified and clarified the role of clinically relevant phenomena in analogue samples prior to validating their importance in clinical samples (e.g., Mennin et al., 2005;

Roemer, Salters, Raffa, & Orsillo, 2005; Salters-Pedneault et al., 2006; Tull & Roemer, 2007).

The second main limitation is the cross-sectional nature of the data. It is possible that the precise nature of the interrelationships between the variables of interest differ from what was predicted, and although theory and previous research support the choice of statistical analysis, the design does not allow complete certainty of whether emotion regulation difficulties cause greater anxiety sensitivity, or if greater anxiety in fact elevates either or both these variables. A third possibility that cannot be ruled out from this design is that anxiety sensitivity causes greater emotion regulation, but this was tested and statistically unsupported. Alternative models described above should be examined using designs that allow one to determine the exact progression of these variables (i.e., longitudinal and prospective designs).

The third main limitation is common to the majority of studies in the areas of anxiety sensitivity and emotion regulation, specifically that it relies on self-report measures. Participants may not be able to accurately or truthfully report their anxiety sensitivity, emotion regulation difficulties, and anxiety symptoms, and these may be better assessed using more behavioral or physiological measures. Fourth, although the majority of studies investigating anxiety sensitivity to date have examined the construct as a continuous variable, recent evidence suggests that the latent structure of anxiety sensitivity may be taxonic (see Bernstein et al., 2006), and, therefore, the examination of anxiety sensitivity as continuously distributed may not provide the best representation of this variable. It will be important for future studies to explore the relationship between anxiety sensitivity and other variables using a taxonic conceptualization.

Conclusion

Despite the limitations, the findings from the current study provide evidence that it is more important to target emotion regulation than anxiety sensitivity during anxiety prevention efforts with individuals from Singapore. Poorer emotion regulation skills present a greater direct association with anxiety than anxiety sensitivity, and emotion regulation difficulties were found to be the mechanism by which anxiety sensitivity impacts anxiety. Given the high incidence of anxiety disorders and the high levels of comorbidity that exists among them, preventative efforts targeting the strongest predictors of anxiety disorders would go far to reducing the personal and public costs associated with anxiety. This study demonstrates that the widespread teaching of emotion regulation skills would be more effective than targeting anxiety sensitivity, as these skills impact anxiety both directly and through anxiety sensitivity, at least in this population.

Second, although anxiety sensitivity did not appear to vary across cultures in Singapore, Singapore students in general appear to suffer far greater anxiety sensitivity than their peers internationally, as well as slightly higher levels of anxiety. The reason for the difference is unclear, as neither alexithymia nor emotion regulation difficulties differed as dramatically across international lines, although more specific studies are necessary to confirm this. Cultural and societal factors may play a significant role in explaining the difference in anxiety sensitivity and anxiety, and the higher rates of anxiety may be an important area of concern for Singapore's public health system. Looking more closely at differences across cultural groups in Singapore, the Chinese and Malays have far higher levels of anxiety and greater emotion regulation difficulties than Indians. Again, the specific reasons for these differences is unclear, but Malay and

Chinese interest groups may find that efforts to disseminate emotion regulation strategies may go far in reducing the personal and societal burden of high levels of anxiety and anxiety disorders.

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Appendix A

Informed Consent

The purpose of this study is to collect information on anxiety and anxiety management practices amongst Singaporean students. Participation will take approximately 15 minutes of your time and we ask that you complete the survey as honestly as you can.

The risks of participating in this study are minimal, and no personally identifying information is being collected that can link your responses to you individually. Confidentiality will be maintained at all times. Your name and your email address will not be linked to the surveys in any way. The answers you provide will be combined with other participants' answers in order to conduct group analyses. Any publications or presentations resulting from this study will refer only to the grouped results.

If you begin to feel uncomfortable at any point during the survey, you have the right to stop and exit. Your participation is voluntary; there is no penalty for not participating and you can choose to withdraw at any time. Although there is no direct benefit to you for participating in this study, you will be providing valuable information that may be beneficial to the understanding of anxiety and related disorders amongst Singaporean youth.

I agree _____ (Proceed to Survey)

I do not agree _____ (Survey ends)

1. Demographics

Gender: Male____ Female_____

Age:

Ethnicity: Chinese_____ Indian_____ Malay ____ Others (Please Specify) _____

Yearly household income:

0 - $30,000

$31,000 - $60,000

$61,000 - $100,000

$100,000 - $150,000

> $150,000

How closely do you identify with your ethnic group?

Not at all Somewhat Closely Very Closely

What language are you most comfortable Using?

English ____

Chinese or Chinese dialect (Mandarin/Hokkien/Teochew/ Cantonese/Hainanese/etc) ____

Indian language (Hindi/Punjabi/Tamil/Urdu etc) ____

Malay ____

Others (Please Specify) ____