

The Role of Anger in Symptoms and Processes of Generalized Anxiety Disorder

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ABSTRACT

The Role of Anger in Symptoms and Processes of Generalized Anxiety Disorder

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Research investigating the associations between anger and symptoms of generalized anxiety disorder (GAD) is limited. The goals of the current program of research were to examine the various forms of anger associated with GAD as well as the cross-over effects of worry and anger on cognitive mechanisms characteristic of GAD and high trait anger. In Study 1, participants ($N = 381$) completed a series of questionnaires assessing various aspects of anger and GAD diagnostic criteria via self-report. Our results indicated that GAD analogues reported higher levels of trait anger, anger suppression, and hostility than less anxious participants. In Study 2, the effects of laboratory-induced anger on negative style, negative beliefs about uncertainty, and worry were examined. Participants were randomized to an anger induction condition ($n = 43$) or a control condition ($n = 34$). An interpretation bias task, questionnaire items assessing beliefs about uncertainty, and a structured worry task were administered following the manipulation. Participants in the anger condition reported greater increases in negative interpretive style and in the belief that uncertainty is unfair and spoils everything than participants in the control condition; however no group differences were found with worry. In Study 3, the effects of anger and worry on interpretive style and hostile attributions were examined. Participants were randomized to a worry induction ($n = 51$), anger induction ($n = 50$), or control condition ($n = 49$). We also examined whether GAD analogues reported greater hostile interpretations of ambiguous intent than less anxious participants, and whether GAD analogue status interacted with the worry and anger experimental conditions in predicting increased hostile

and threatening interpretations of ambiguous information. Although we found no effects of condition or interactions between GAD and condition on interpretive style or hostile attributions, we found that GAD symptoms were associated with greater negative interpretive style and greater hostile attributions. Together, findings from these studies suggest that elevated levels of multiple dimensions of anger, as well as hostile attributions, characterize individuals who meet diagnostic criteria for GAD and provide some, albeit limited, support for the notion that elevated anger contributes to cognitive vulnerabilities underlying GAD.

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Summary of Author Contributions

I was responsible for choosing the overall focus of this program of research as well as the selected topics for each study, in consultation with Dr. Michel Dugas and Dr. Jean-Philippe Gouin. I was principally responsible for the implementation of the studies, including participant recruitment and scheduling, data collection and management, conducting statistical analyses, interpreting findings, and preparing manuscripts for publication. Throughout all stages of this program of research, Dr. Michel Dugas provided ongoing supervision, consultation, and feedback. Dr. Jean-Philippe Gouin provided supervision, consultation, and feedback throughout

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CHAPTER 1:

GENERAL INTRODUCTION

Anxiety and anger are basic, cross-culturally experienced, adaptive human emotions (Darwin, 1872; Ekman & Friesen, 1971). These emotions, however, can become maladaptive when experienced excessively. In accordance, the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (American Psychiatric Association [APA], 2013) includes a number of diagnostic categories for excessive anxiety; yet there is a near absence of diagnostic categories for excessive anger. There is also less empirical research on anger than on anxiety (DiGiuseppe & Tafrate, 2001; Kassinove & Sukhodolsky, 1995), and there is little empirical research examining the co-occurrence of both anger and anxiety (DiGiuseppe & Tafrate, 2007). The broad goal of this program of research is to examine the associations between anger and generalized anxiety disorder (GAD) symptoms, as well as to identify possible pathways linking anger and GAD.

Anxiety and GAD

Whereas fear occurs in the presence of perceived imminent threat, anxiety results from the anticipation of future threat (APA, 2013, p. 189). Anxiety can be broken down into state anxiety and trait anxiety. Although similar features (e.g., muscle tension, increased heart rate) characterize state and trait anxiety, state anxiety is experienced in the moment, whereas trait anxiety refers to the general disposition to experience state anxiety (Spielberger et al., 1983). Fear and anxiety can be adaptive in certain situations, as these emotions can promote quick energy mobilization to enable escape in objectively dangerous situations (Foa & Kozak, 1986; Nesse & Elmsworth, 2009). Fear and anxiety, however, can also be pathological and maladaptive. Pathological fear or anxiety occurs when the fear response to a stimulus is

exaggerated and distorts reality (Barlow, 2002; Foa, Huppert, & Cahill, 2006), interferes with psychosocial functioning, causes substantial distress, and promotes maladaptive behaviours such as avoidance of situations that pose no objective danger (Foa et al., 2006).

Pathological fear and anxiety are the defining features of the anxiety disorders (APA, 2013), which include social anxiety disorder (SAD), specific phobias, panic disorder (PD), and GAD, and until recently also included obsessive compulsive disorder (OCD) and post-traumatic stress disorder (PTSD; APA, 2000)¹. The anxiety disorders share the common characteristics of excessive fear or anxiety, but differ in the nature of the fear or anxiety-provoking stimulus and associated symptoms. For instance, PD is associated with fear of arousal-related bodily sensations, SAD with fear of social situations and negative evaluation, specific phobias with fears about specific animals, objects or situations, OCD with anxiety triggered by intrusive thoughts or images and their respective situational cues, PTSD with anxiety triggered by the memories of a traumatic event, and GAD with anxiety triggered by the possibility of negative future events (Abramowitz, Deacon, & Whiteside, 2011; APA, 2013). Of the anxiety disorders, GAD is one of the least studied (Dugas, Anderson, Deschênes, & Donegan, 2010) despite its relatively high prevalence and considerable personal and societal costs (Wittchen, 2002). Therefore, research efforts to improve our understanding of GAD are warranted.

Generalized anxiety disorder. GAD is defined as excessive anxiety and worry about a number of events or activities on more days than not, for period of at least six months (APA, 2013). Individuals with GAD worry about a number of topics, such as finances, relationships, work or academic performance, and physical danger. The worries are difficult to control,

¹ The DSM-5 now classifies OCD as an obsessive-compulsive and related disorder, and classifies PTSD as a trauma- and stressor-related disorder (APA, 2013). For the purpose of this dissertation, for which writing began prior to the publication of the DSM-5, OCD and PTSD are considered anxiety disorders (APA, 2000).

interfere with psychosocial functioning, and are accompanied by at least three of six somatic symptoms, including restlessness, irritability, fatigue, difficulty concentrating, muscle tension, and sleep disturbance (APA, 2013). Epidemiological studies estimate a 12-month prevalence rate for GAD of 3.1% and a lifetime prevalence rate of 5.7% (Kessler & Wang, 2008). Women are approximately twice more likely than men to be diagnosed with GAD (Vesga-López et al., 2008).

GAD is a debilitating and costly disorder; a diagnosis of GAD has been associated with quality of life impairments and reduced work productivity (Hoffman, Dukes, & Wittchen, 2008), elevated risk for developing cardiovascular diseases (Tully, Cosh, & Baune, 2013), impaired immune system functioning (e.g., Vieira et al., 2010), an elevated number of prescribed medications (Kertz & Woodruff-Borden, 2011), and greater use of health care resources (Hoffman et al., 2008; Wittchen, 2002). In Canada, GAD is associated with elevated costs to the health care system (Koerner et al., 2004). Taken together, GAD leads to poor health and occupational outcomes and has a high societal cost. Although there are a number of efficacious psychological treatments for GAD (e.g., Cuijpers et al., 2014), recovery rates of only 50-60% are nonetheless common (Fisher, 2006). Therefore, a notable number of individuals who undergo treatment do not attain remission. Furthering our understanding of the mechanisms underlying GAD can not only lead to refinements in conceptual models of GAD, but can ultimately lead to improvements in treatment protocols and reductions in the personal burden and societal cost of GAD.

Anger

Many definitions of anger have been proposed (Eckhardt, Norlander, & Deffenbacher, 2004). DiGiuseppe and Tafrate (2007) define anger as a “subjectively experienced emotional

state that is elicited by a perception of threat. It is associated with cognitions focused on other's misdeeds and is communicated by a variety of behaviours influenced by social roles, learning history, and environmental contingencies" (p.31). In addition, anger can range on a continuum from mild annoyance to intense fury and rage (Spielberger, 1988). Similar to anxiety, anger can also be broken down into trait anger or state anger. State anger is characterized by perceptions of injustice, of being attacked or treated unfairly, or of being frustrated by goal-blocking barriers, whereas trait anger is the general disposition to respond to situations with elevations in state anger (Spielberger, 1999). That is, individuals with high trait anger tend to experience more frequent and more intense state anger than individuals with low trait anger.

There are many instances in which anger can be adaptive. For example, feelings of anger can motivate attack in dangerous or threatening situations (Nesse & Elmsworth, 2009). Anger can also help a person recognize a problem, such as interpersonal conflict, and thus motivate corrective actions such as reconciliation (DiGiuseppe & Tafrate, 2001). Yet, anger can also become maladaptive. Excessive or misplaced anger can lead to negative emotional and behavioural consequences such as aggression (DiGiuseppe & Tafrate, 2007) or interpersonal conflict (Scherer & Wallbott, 1994), and can have negative health consequences such as elevated risk of developing cardiovascular diseases (e.g., Martens, Mols, Burg, & Denollet, 2010). Anger is unique as a negative emotion, however, as it is associated with a tendency to approach, rather than avoid, anger-eliciting stimuli (Carver & Harmon-Jones, 2009; Scherer & Wallbott, 1994). It is also unique as a negative emotion in that individuals express little desire to control their experience of anger, similarly to the experience of joy (Scherer & Wallbott, 1994). Although there is currently no diagnostic category for anger as a primary symptom (APA, 2013), researchers have argued that an established classification system for problematic anger would

improve conceptual models of maladaptive anger and treatment protocols (see DiGiuseppe & Tafrate, 2007).

Anger and Anxiety Disorders

Some research demonstrates a link between anxiety disorders and elevated anger. For instance, frequent anger outbursts have been reported in more than half of a sample of individuals with anxiety disorders (Lee & Cameron, 1986). Elevated levels of anger and hostility, the cognitive component of anger, have also been reported in individuals with different anxiety disorders, including PD (Moscovitch et al., 2008), OCD (Moscovitch et al., 2008; Radomsky, Ashbaugh, & Gelfand, 2007), and SAD (Erwin, Heimberg, Schneier, & Liebowitz, 2003; Moscovitch et al., 2008).

Of the anxiety disorders, PTSD and GAD include certain components of anger as part of their diagnostic criteria. Specifically, PTSD includes irritability or outbursts of anger as diagnostic symptoms (APA, 2013); consequently, several studies and meta-analyses suggest that anger is elevated and problematic in individuals with PTSD (e.g., Hawkins & Cougle, 2011; Meffert et al., 2008; Novaco, 2010; Olatunji, Ciesielski, & Tolin, 2010; Orth, Cahill, Foa, & Maercker, 2008; Orth & Wieland 2006). Surprisingly, less is known about co-occurrence of anger and GAD despite irritability, defined as a lowered threshold for responding with anger (DiGiuseppe & Tafrate, 2007, p. 31), being a symptom of GAD. A limited number of studies have examined the association between anger and GAD. One study found that several dimensions of anger, including trait anger, outward anger expression, and anger suppression were more elevated in individuals with GAD than in individuals with low anxiety, and that anger control was greater in the less anxious individuals than in those with GAD (Erdem, Celik, Yetkin, & Ozgen, 2008). In addition, Hawkins and Cougle (2011) found epidemiological

evidence of an association between elevated anger experience over the past 30 days and a diagnosis of GAD. Although this suggests associations between these constructs, the nature of the associations between anger, worry, and GAD requires further exploration.

Cognitive Mechanisms

One possible pathway linking anger and anxiety is a common underlying cognitive-processing style. The importance of cognitive processing in the elicitation of *either* anger or anxiety has been recognized by researchers who have suggested that a sense of mastery and control over the emotion-eliciting situation may differentiate the experience of anxiety/fear and anger. Specifically, perceiving the threatening stimulus as insurmountable can lead to the experience of fear and anxiety, whereas perceiving the threatening stimulus as conquerable can lead to the experience of anger (e.g., Barlow, 2002; Frijda, 1986). However, there have been few empirical investigations of the mechanisms involved in the *co-occurrence* of anxiety and anger.

Given that cognitive-processing styles are involved in the etiology and maintenance of anxiety disorders and high trait anger, shared cognitive processes may explain this relationship. The cognitive theory of anxiety is a central etiological theory of fear and anxiety disorders (see Clark & Beck, 2010). The theory posits that core beliefs (i.e., schemas) affect the way in which information from the environment is processed. In highly anxious individuals, distorted schemas increase the likelihood of processing of information as threatening, which in turn leads to anxiety and related symptoms (e.g., worry, fear). A number of conceptual models of GAD have been developed based on this theory, with cognitive vulnerabilities at the root of symptom expression. For instance, intolerance of uncertainty, defined as “a dispositional characteristic that results from a set of negative beliefs about uncertainty and its implications” (Dugas & Robichaud, 2007, p. 24), has been identified as a core vulnerability involved in the etiology and maintenance of

GAD. Research based on this model demonstrates that individuals with GAD fear the unknown and imagine worst case scenarios when feeling uncertain; they also have difficulty tolerating the distress associated with uncertainty (Dugas & Robichaud, 2007). In addition to content-based cognitive vulnerabilities (i.e., beliefs, thoughts and attitudes), GAD is also characterized by cognitive processing biases towards threat (i.e., information processing). For instance, individuals with high trait anxiety or a diagnosis of GAD report more threatening interpretations of ambiguous, potentially threatening or neutral, situations (e.g., Dagleish, 1994; Davey, Hampton, Farrell, & Davidson, 1992; MacLeod & Cohen, 1993; Mathews, Richards, & Eysenck, 1989; Mogg et al., 1994). In keeping with cognitive theory, greater intolerance of uncertainty is associated with a greater likelihood of interpreting ambiguous events as threatening (Koerner & Dugas, 2008).

Cognitive vulnerabilities and processing biases involved in emotional disorders are, at least to some extent, transdiagnostic (e.g., Carleton et al., 2012; Mahoney & McEvoy, 2012). It is not surprising, therefore, that cognitive processes characteristic of anxiety disorders are also involved in high trait anger (Owen, 2011). These biased cognitive-processing styles include selective attention towards anger-related stimuli (e.g., Putman, Hermans, & van Honk, 2004) or hostile stimuli (Wilkowski, Robinson, Gordon, & Troop-Gordon, 2007), enhanced memory for anger-related concepts (Parrott, Zeichner, & Evces, 2005), and interpretation biases such as appraising ambiguous situations as hostile (e.g., Hazebroek, Howells, & Day, 2001). Thus, a hyper-vigilance for hostility seems to relate to high trait anger.

Current Program of Research

Overall, a number of studies suggest that anger is elevated in individuals with anxiety disorders, and a few studies demonstrate this association in GAD. Little is known, however,

about the various forms of anger experience (e.g., hostility, trait anger) and expressions (e.g., anger suppression, aggression, anger control) that are associated with GAD, or about the cognitive mechanisms (e.g., cognitive vulnerabilities, biased interpretations) underlying this association. The overarching objective of this program of research is to examine the nature of the association between anger and symptoms of GAD with three independent studies. The main goal of study 1 is to examine which facets of anger relate to GAD, with the use of self-report questionnaires. The main goal of study 2 is to examine the effects of state anger on cognitive vulnerabilities and processes underlying GAD, using an ecologically-valid anger induction. Finally, the main goals of study 3 are to examine the effects of worry and anger inductions, compared to a relaxation induction, on cognitive processes associated with high trait anger and GAD, and to examine whether individuals who meet diagnostic criteria for GAD (i.e., GAD analogues) report greater cognitive processing biases.

CHAPTER 2:

THE ROLE OF ANGER IN GENERALIZED ANXIETY DISORDER

Anger and anxiety have historically been linked through shared physiological reactions to stress (e.g., Cannon, 1929). In particular, anger and anxiety may be related through underlying biological vulnerabilities, such that when confronted with threat, individuals react either with anger or anxiety, that is, “fight” or “flight” (Barlow, 2002). According to this theory, the individual’s sense of mastery over the threatening situation predicts the type of reaction, with high perceived mastery predicting anger, and low perceived mastery predicting anxiety. This suggests that appraisals are an important feature of both emotions. Despite this, anger, defined as an emotion elicited by perceptions of threat caused by the misdeeds of others (DiGiuseppe & Tafrate, 2007), has received little empirical attention in the context of anxiety disorders.

However, there is some evidence to suggest that elevated levels of anger are present in individuals with anxiety disorders. In addition to trait and state anger, some dimensions of anger that have been examined include hostility (the cognitive component of anger), aggression (the behavioural component of anger), internalized anger expression (the tendency to suppress angry feelings), externalized anger expression (the tendency to outwardly express angry feelings), and anger control (the ability to regulate anger). Specifically, Moscovitch and colleagues (2008) found elevated levels of hostility in individuals with SAD, OCD, and PD, relative to non-anxious controls. They also found that individuals with PD reported higher levels of aggressive anger, and that individuals with SAD reported lower verbal aggression compared to non-anxious controls. Erwin and colleagues (2003) also found elevated trait anger and internalized anger expression in individuals with SAD, relative to non-anxious individuals. Of the anxiety disorders, anger has mostly been examined in relation to PTSD (Novaco, 2010). For example,

Meffert and colleagues (2008) found that greater levels of anger mediated the relationship between trauma exposure and PTSD symptoms among police officers. In addition, meta-analytical findings suggest that PTSD symptoms are related to various dimensions of anger, particularly internalized anger expression, with large effects (Olatunji et al., 2010; Orth & Wieland 2006). However, a recent study showed that, after controlling for demographic variables, PTSD did not significantly predict anger expression, but did significantly predict anger experience over a 30-day period (Hawkins & Cogle, 2011).

Only a few studies have examined the relation between anger and GAD. This is surprising because irritability, which is characterized by a lowered threshold for anger (DiGiuseppe & Tafrate, 2007), is a symptom of GAD (APA, 2000). Erdem and colleagues (2008) found that individuals with GAD had greater levels of trait anger, externalized anger expression, internalized anger expression, as well as lower anger control (i.e., lower self-regulation of anger), than did non-anxious individuals. More recently, Hawkins and Cogle (2011) found that greater anger expression, as well as anger experience over a 30-day period, was associated with GAD independently of shared associations with other psychiatric conditions. Overall, these findings suggest that anger may be an important emotion associated with GAD. Although the abovementioned studies examined the relations between specific anger dimensions (e.g., trait anger, anger expression) and GAD, the *relative* contributions of each anger dimension to GAD is largely unknown.

The goal of the current study was to examine the relations between specific dimensions of anger and the presence and severity of GAD by: 1) comparing individuals who meet diagnostic criteria for GAD to individuals who do not meet diagnostic criteria for GAD on a combination of anger dimensions; 2) exploring the relative contribution of each anger dimension

to GAD status; and 3) examining the extent to which anger dimensions predict GAD symptom severity, in individuals who meet diagnostic criteria for GAD. The anger dimensions examined were based on the subscales of the questionnaires used in the current study (see below).

It was hypothesized that individuals who meet diagnostic criteria for GAD would differ from individuals who do not meet diagnostic criteria for GAD on a combination of anger dimensions, and that lower scores on externalized and internalized anger control and higher scores on all other anger dimensions would predict greater GAD symptom severity. Although the examination of the relative contribution of anger dimensions to GAD status was largely exploratory, we expected that trait anger and internalized anger (i.e., inwards anger expression, hostility) would contribute more to GAD than would externalized anger (i.e., outwards anger expression, physical aggression, and verbal aggression). This hypothesis was derived from the evidence suggesting that internalized anger (e.g., Olatunji et al., 2010) and hostility (e.g., Moscovitch et al., 2008) are strong predictors of other anxiety disorders.

Method

Participants

Three hundred and eighty-one ($N = 381$) undergraduate students, between the ages of 18 and 57 ($M = 23.49$, $SD = 6.27$), participated in the study in exchange for course credit. The majority of the sample was female (85.79%) and studying in the field of psychology (71.39%). Most participants (38.10%) were in their first year of study, 25.93% were in their second year, 20.63% were in their third year, and 15.34% were in their fourth year, with 87.73% of the sample studying full-time. The majority of the sample (63.47%) reported English as their first language, 14.67% reported French, and 21.87% reported “other” as their first language. The majority of participants reported their ethnicity as Caucasian (65.00%), 7.10% as Asian, 6.84% as Multi-

Racial, 5.53% as Black, 5.00% as Latino, and 5.00% as Middle Eastern, whereas 5.53% reported “other” as their ethnicity.

Measures

Generalized Anxiety Disorder Questionnaire (GAD-Q-IV; Newman et al., 2002). The GAD-Q-IV was developed as a screening tool for the diagnosis of GAD. It is composed of 14 self-reported items that assess the symptoms of GAD based on the DSM-IV (APA, 2000) diagnostic criteria. Eleven of the items are rated dichotomously (i.e., the presence or absence of symptoms), one item requires participants to list worry topics, and two items assess the degree of interference and the degree of distress resulting from worrying on a Likert scale ranging from 0 (*None*) to 8 (*Very severe*). The GAD-Q-IV demonstrates convergent and discriminant validity, a kappa agreement of .67 with a structured diagnostic interview of GAD, and test-retest reliability of 92% over two weeks. The recommended cut-off score for individuals meeting diagnostic criteria is 5.7 out of a total of 13 (Newman et al., 2002), with scores of 5.7 and above indicating the presence of GAD (i.e., GAD analogues), and a score below 5.7 indicating the absence of GAD (i.e., non-GAD).

Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ is a 16-item self-report measure that assesses the tendency to worry. Participants rate the extent to which items are typical of themselves on a Likert scale ranging from 1 (*Not at all typical*) to 5 (*Very typical*), with items such as “*I am always worrying about something*”. The PSWQ has demonstrated convergent and divergent validity, excellent internal consistency ($\alpha = .93$), and test-retest reliability over eight to ten weeks ($r = .92$).

State-Trait Anger Expression Inventory-2 (STAXI-2; Spielberger, 1999). The STAXI-2 is a 57-item self-report measure with scales developed to assess anger as a

dispositional characteristic (trait anger scale), situational anger (state anger scale), and the expression of anger (anger expression scale). Given the goals of the current study, only the trait anger and anger expression scales were included. The 10-item Trait Anger scale (T-ANG) assesses the frequency and intensity of anger experiences, with items such as “*I get angry when I’m slowed down by other’s mistakes*” rated on a Likert scale ranging from 1 (*Almost never*) to 4 (*Almost always*). The 32-item Anger Expression scale is composed of four subscales that assess how people react when they are angry. For each 8-item subscale, the extent to which participants generally react when angry is rated on a Likert scale ranging from 1 (*Almost never*) to 4 (*Almost always*). The Anger Expression-Out (AX-O) subscale measures the expression of anger towards objects or other individuals with the use of physically or verbally aggressive behaviours, and includes items such as “*I strike out at whatever infuriates me*”. The Anger Expression-In (AX-I) subscale measures the extent to which angry feelings are experienced yet suppressed (i.e., lack of expression), and includes items such as “*I boil inside, but I don’t show it*”. The Anger Control-Out (AC-O) subscale assesses the extent to which a person controls his or her anger by preventing the externalized expression of anger, and includes items such as “*I keep my cool*”. Finally, the Anger Control-In (AC-I) subscale assesses the extent to which a person controls angry feelings by attempts to calm down and cool off, and includes items such as “*I do something relaxing to calm down*”. The STAXI-2 subscales have demonstrated construct validity and adequate internal consistency with α ’s ranging from .70 to .85.

Aggression Questionnaire (AQ; Buss & Perry, 1992). The AQ is a 29-item self-report measure that assesses the disposition of aggression, and is composed of four subscales. For each subscale, the extent to which each statement is characteristic or uncharacteristic of participants is rated on a Likert scale ranging from 1 (*Extremely uncharacteristic of me*) to 5 (*Extremely*

characteristic of me). The Physical Aggression (AQ-PA) subscale is composed of 8 items and assesses the motor component of aggressive behaviour, which involves the desire to harm others, with items such as “*Once in a while, I can’t control the urge to strike another person*”. The Verbal Aggression (AQ-VA) subscale is composed of 5 items and assesses instrumental aggression with items such as “*When people annoy me, I may tell them what I think of them*”. The Anger (AQ-ANG) subscale is composed of 7 items and assesses the affective component of aggression, including physiological arousal and preparation for aggression, with items such as “*I have trouble controlling my temper*”. Finally, the Hostility (AQ-HOST) subscale is composed of 8 items and measures the cognitive component of aggression, including feelings of injustice, with items such as “*When people are especially nice, I wonder what they want*”. The AQ has good internal consistency ($\alpha = .89$) and test-retest reliability over nine weeks ($r = .80$).

Procedure

Participants were recruited in psychology undergraduate classes or through the Department of Psychology’s Participant Pool at Concordia University. They were invited to complete a series of questionnaires on anger and anxiety, administered in a quasi-counterbalanced order, either individually or in groups of up to ten participants. The experimenter (the first author) obtained informed consent (see Appendix A) and debriefed all participants.

Results

Preliminary Analyses

Data were normally distributed (all skewness values < 3.0 and kurtosis values < 10.0 ; Kline, 2009), and therefore no outliers were removed. The correlations between the GAD-Q-IV and all STAXI-2 and AQ subscales were statistically significant (r s ranging from .10 to .46, $ps <$

.05). Male sex was significantly correlated with greater externalized anger control and greater physical aggression, whereas female sex was significantly correlated with higher scores on the GAD-Q-IV. Age was negatively related to hostility. Because the strengths of the correlations were weak ($r_s < .17$), we did not statistically control for age and sex in subsequent analyses. See Table 1 for a correlation matrix.

Next, we used the recommended cut-off score (5.7; Newman et al., 2002) on the GAD-Q-IV to create the GAD-analogue ($n = 131$) and non-GAD ($n = 250$) groups. Given that worry is the primary feature of GAD, we examined the validity of the GAD-Q-IV in our sample by conducting an independent-samples t -test between the groups on PSWQ scores. We found that, as expected, the GAD-analogue group had significantly higher scores ($M = 63.00$, $SD = 10.64$) than did the non-GAD group ($M = 46.00$, $SD = 12.44$), $t(378) = -13.26$, $p < .001$. These means and standard deviations are comparable to those of clinical samples of individuals with GAD (e.g., $M = 65.27$, $SD = 8.50$; Ladouceur et al., 2000) and samples of non-anxious individuals (e.g., $M = 47.08$, $SD = 13.24$; Behar, Alcaine, Zuellig, & Borkovec, 2003).

Anger and GAD Group Membership

To examine the difference between the GAD-analogue group and the non-GAD group on the STAXI-2 subscales, a multivariate analysis of variance (MANOVA) was conducted. GAD group membership served as the independent variable, and the STAXI-2 subscales served as the dependent variables. As expected, there was a statistically significant difference between the GAD-analogue group and the non-GAD group on the combined STAXI-2 anger subscales, $\Lambda = 0.90$, $F(5, 374) = 8.09$, $p < .001$, $\eta_p^2 = .098$. See Table 2 for means and standard deviations of the STAXI-2 subscales by GAD group membership.

Table 1

Correlations between the GAD-Q-IV, the STAXI-2, and the AQ (N = 381)

	1	2	3	4	5	6	7	8	9	10	11	12
1. GAD-Q-IV	1.00	.34**	.25**	.33**	-.16**	-.15**	.17**	.10*	.34**	.46**	-.033	-.16**
2. T-ANG ¹		1.00	.70**	.38**	-.57**	-.41	.63**	.52**	.73**	.55**	-.09	-.07
3. AX-O			1.00	.20**	-.59**	-.40**	.57**	.61**	.63**	.34*	-.10	-.04
4. AX-I				1.00	-.06	-.14**	.16**	.07	.28**	.55**	-.04	.03
5. AC-O					1.00	.66**	-.43**	-.45**	-.62**	-.26**	.02	.12*
6. AC-I						1.00	-.29**	-.29**	-.44**	-.27**	.06	.04
7. AQ-PA							1.00	.49**	.60**	.37**	-.06	.13*
8. AQ-VA								1.00	.54**	.36**	-.06	.05
9. AQ-ANG									1.00	.50**	-.01	-.08
10. AQ-HOST										1.00	-.12*	-.08
11. Age											1.00	.02
12. Sex ^a												1.00

* $p < .05$, ** $p < .01$

Note. GAD-Q-IV = Generalized Anxiety Disorder Questionnaire IV; STAXI-2 = State-Trait Anger Expression Inventory, second edition; AQ = Aggression Questionnaire; T-ANG = State-Trait Anger Expression Inventory II – Trait Scale; AX-O = State-Trait Anger Expression Inventory II – Anger Expression-Out subscale; AX-I = State-Trait Anger Expression Inventory II – Anger Expression-In subscale; AC-O = State-Trait Anger Expression Inventory II – Anger Control-Out subscale; AC-I = State-Trait Anger Expression Inventory II – Anger Control-In subscale; AQ-PA = Aggression Questionnaire – Physical Aggression Subscale; AQ-VA = Aggression Questionnaire – Verbal Aggression Subscale; AQ-ANG = Aggression Questionnaire – Anger Subscale; AQ-HOST = Aggression Questionnaire – Hostility Subscale.

¹Data missing for one participant ($n = 380$)

^aPoint-biserial correlation, 0 = female, 1 = male.

Table 2

Means and Standard Deviations for the STAXI-2 by GAD Group Membership

STAXI-2 subscales	GAD-analogue (<i>n</i> = 131)		Non-GAD (<i>n</i> = 250)	
	Mean	<i>SD</i>	Mean	<i>SD</i>
T-ANG ¹	21.50 ^a	6.19	18.52 ^b	4.73
AX-O	16.35 ^a	4.91	14.74 ^b	3.59
AX-I	19.42 ^a	4.62	16.86 ^b	4.83
AC-O	22.18 ^a	5.22	23.39 ^b	4.51
AC-I	20.86 ^a	5.06	22.38 ^b	4.71

Note. Means with differing superscripts are significantly different ($p < .05$). STAXI-2 = State-Trait Anger Expression Inventory, second edition; GAD = generalized anxiety disorder; T-ANG = State-Trait Anger Expression Inventory II – Trait Scale; AX-O = State-Trait Anger Expression Inventory II – Anger Expression-Out subscale; AX-I = State-Trait Anger Expression Inventory II – Anger Expression-In subscale; AC-O = State-Trait Anger Expression Inventory II – Anger Control-Out subscale; AC-I = State-Trait Anger Expression Inventory II – Anger Control-In subscale.

¹Data missing for one participant (Non-GAD: $n = 249$)

A discriminant function analysis was conducted to examine the relative contribution of each STAXI-2 subscale to GAD group membership. The correlations between the predictors and the discriminant function (i.e., the structure matrix) suggest that elevated T-ANG (Trait Anger) and AX-I (Anger Expression-In) accounted for the most variance in GAD group membership (loadings less than .50 are not interpreted; Tabachnick & Fidell, 2007). See Table 3 for canonical coefficients and the structure matrix. Using Jackknife classification, a method used to classify each case by the functions derived from all other cases, the discriminant function could be used to correctly classify 63.95% ($n = 243$) of individuals into their respective groups, with 57.25% ($n = 75$) correctly classified as GAD-analogue and 67.47% ($n = 168$) correctly classified as non-GAD. The difference in the proportions of correct classification was significant, $\chi^2 = 3.89$, $p = .049$, suggesting that the STAXI-2 subscales can better identify individuals who do not meet diagnostic criteria for GAD than those who do.

To assess the effect of anger on GAD symptom severity, a multiple regression analysis predicting GAD-Q-IV continuous scores was conducted within the GAD-analogue group ($n = 131$), with the STAXI-2 subscales entered as predictors. As expected, the regression model was statistically significant, $F(5, 125) = 3.54$, $R^2 = .124$, $p = .005$. Of the predictor variables, only AX-I significantly predicted GAD symptom severity ($\beta = .22$, $p = .017$). See Table 4 for detailed results of the multiple regression analysis.

Aggression and GAD Group Membership

To examine the difference between the GAD-analogue group and the non-GAD group on the AQ subscales, a MANOVA was conducted. GAD group membership served as the independent variable, whereas AQ subscales served as the dependent variables.

Table 3

*Standardized Canonical Coefficients and Structure Matrix for the STAXI-2 Predicting GAD**Group Status (N = 380¹)*

STAXI-2 Subscales	Standardized Canonical Coefficients	Structure Matrix
T-ANG	.570	.815
AX-O	.090	.567
AX-I	.553	.780
AC-O	.190	-.372
AC-I	-.266	-.462

Note. STAXI-2 = State-Trait Anger Expression Inventory, second edition; GAD = generalized anxiety disorder; T-ANG = State-Trait Anger Expression Inventory II – Trait Scale; AX-O = State-Trait Anger Expression Inventory II – Anger Expression-Out subscale; AX-I = State-Trait Anger Expression Inventory II – Anger Expression-In subscale; AC-O = State-Trait Anger Expression Inventory II – Anger Control-Out subscale; AC-I = State-Trait Anger Expression Inventory II – Anger Control-In subscale.

¹Data missing for one participant

Table 4

Multiple Regression for the STAXI-2 Predicting GAD Symptom Severity in GAD-Analogues (n = 131)

STAXI-2 Subscales	R^2	B	SE	β	[Lower, Upper]
					95% Confidence Interval for B
STEP 1	.124				
T-ANG		.067	.045	.211	[-.022, .155]
AX-O		.019	.057	.048	[-.094, .133]
AX-I		.091*	.038	.215	[.016, .165]
AC-O		.011	.055	.029	[-.097, .119]
AC-I		.014	.044	.036	[-.074, .101]

* $p < .05$

Note. STAXI-2 = State-Trait Anger Expression Inventory, second edition; GAD = generalized anxiety disorder; T-ANG = State-Trait Anger Expression Inventory II – Trait Scale; AX-O = State-Trait Anger Expression Inventory II – Anger Expression-Out subscale; AX-I = State-Trait Anger Expression Inventory II – Anger Expression-In subscale; AC-O = State-Trait Anger Expression Inventory II – Anger Control-Out subscale; AC-I = State-Trait Anger Expression Inventory II – Anger Control-In subscale.

As expected, there was a statistically significant difference between the GAD-analogue group and the non-GAD group on the combined AQ subscales, $\Lambda = 0.84$, $F(4, 376) = 17.34$, $p < .001$, $\eta_p^2 = .156$. See Table 5 for means and standard deviations of AQ subscales by GAD group membership.

A discriminant function analysis was conducted to examine the relative contribution of each AQ subscale to GAD group membership. The correlations between the predictors and the discriminant function suggest that elevated AQ-HOST (Hostility) and AQ-ANG (Anger) accounted for the most variance in GAD group membership. See Table 6 for canonical coefficients and the structure matrix. Using Jackknife classification, the discriminant function could be used to correctly classify 66.93% ($n = 255$) of individuals into their respective groups, with 64.89% ($n = 85$) correctly classified as GAD-analogue and 68.00% ($n = 170$) correctly classified as non-GAD. The difference in the proportions of correct classification was not statistically significant, $\chi^2 = .38$, $p = .54$.

To assess the association of aggression to GAD symptom severity, a multiple regression analysis with AQ subscales predicting GAD-Q-IV continuous scores was conducted within the GAD-analogue group. As expected, the regression model was statistically significant, $F(4, 126) = 7.80$, $R^2 = .198$, $p < .001$. Of the predictor variables, only hostility (AQ-HOST) significantly predicted GAD symptom severity ($\beta = .39$, $p < .001$). See Table 7 for detailed results of the multiple regression analysis.

Discussion

The goal of the current study was to examine the relations between specific dimensions of anger and the presence and severity of GAD. Overall, our results suggest that heightened levels of anger, in particular trait anger, internalized anger expression, anger as the affective component

Table 5

Means and Standard Deviations for the AQ by GAD Group Membership

AQ subscales	GAD-analogue (<i>n</i> = 131)		Non-GAD (<i>n</i> = 250)	
	Mean	<i>SD</i>	Mean	<i>SD</i>
AQ-PA	19.42 ^a	7.97	17.70 ^b	6.63
AQ-VA	14.80 ^a	4.96	14.38 ^a	4.04
AQ-ANG	19.05 ^a	6.60	15.64 ^b	5.44
AQ-HOST	23.95 ^a	5.74	19.15 ^b	6.39

Note. Means with differing superscripts are significantly different ($p < .05$). AQ = Aggression Questionnaire; GAD = generalized anxiety disorder; AQ-PA = Aggression Questionnaire – Physical Aggression Subscale; AQ-VA = Aggression Questionnaire – Verbal Aggression Subscale; AQ-ANG = Aggression Questionnaire – Anger Subscale; AQ-HOST = Aggression Questionnaire – Hostility Subscale.

Table 6

*Standardized Canonical Coefficients and Structure Matrix for the AQ Predicting GAD Group**Membership (N = 380¹)*

AQ Subscales	Standardized Canonical Coefficients	Structure Matrix
AQ-PA	-.163	.268
AQ-VA	-.444	.108
AQ-ANG	.624	.645
AQ-HOST	.799	.862

Note. AQ = Aggression Questionnaire; GAD = generalized anxiety disorder; AQ-PA =

Aggression Questionnaire – Physical Aggression Subscale; AQ-VA = Aggression Questionnaire

– Verbal Aggression Subscale; AQ-ANG = Aggression Questionnaire – Anger Subscale; AQ-

HOST = Aggression Questionnaire – Hostility Subscale.

¹Data missing for one participant

Table 7

Multiple Regression for the AQ Predicting GAD Symptom Severity in GAD-Analogues (n = 131)

AQ Subscales	R^2	B	SE	β	[Lower, Upper]
					95% Confidence Interval for B
STEP 1	.198				
AQ-PA		.036	.028	.149	[-.019, .091]
AQ-VA		.002	.043	.006	[-.083, .087]
AQ-ANG		-.011	.034	-.037	[-.078, .057]
AQ-HOST		.131**	.030	.386	[.072, .190]

** $p < .01$

Note. AQ = Aggression Questionnaire; GAD = generalized anxiety disorder; AQ-PA = Aggression Questionnaire – Physical Aggression Subscale; AQ-VA = Aggression Questionnaire – Verbal Aggression Subscale; AQ-ANG = Aggression Questionnaire – Anger Subscale; AQ-HOST = Aggression Questionnaire – Hostility Subscale.

of aggression, and hostility, are uniquely related to GAD status. Our results also suggest that, when controlling for shared variance between the subscales, only internalized anger expression from the STAXI and hostility from the AQ uniquely contribute to the severity of GAD symptoms within individuals who meet diagnostic criteria. These findings are broadly consistent with our hypotheses.

The current findings are also in keeping with previous research on anger and anxiety disorders demonstrating that elevated anger levels, particularly internalized anger expression (e.g., Bridewell & Chang, 1997; Orth & Wieland, 2006) and hostility (e.g., Moscovitch et al., 2008), are present in anxious individuals. Also in accordance with our findings, Erdem and colleagues (2008) found that individuals with GAD have elevated levels of trait anger and anger expression. Although Hawkins and Cogle (2011) showed that a diagnosis of GAD was related to elevated anger experience and a greater tendency to express anger *externally*, they did not assess the tendency to express anger *internally*. Our results suggest that when the shared variance between internal and external anger expression is controlled, internalized anger expression is a stronger predictor of GAD.

Although our results do not address the question of *why* anger and GAD tend to co-occur, one possibility is that they are functionally related due to shared information processing biases. For example, Barrazone and Davey (2009) found that both angry and anxious mood inductions led to increased threat interpretations of ambiguous homophones (e.g., slay/sleigh). Relatedly, Owen (2011) concluded based on a review of the published literature that high trait anger is characterized by similar transdiagnostic cognitive processes (e.g., selective attention) as other emotional disorders such as depression and anxiety disorders. In addition, anger and GAD may share underlying cognitive vulnerabilities such as intolerance of uncertainty. Intolerance of

uncertainty arises from a set of negative beliefs, including the belief that uncertainty is unfair (Sexton & Dugas, 2009). Similarly, anger has been associated with perceived unfairness (e.g., Barclay, Skarlicki, & Pugh, 2005). One possibility is that perceiving a state of uncertainty as unfair can lead to anger, anxiety, or both, in individuals who do not cope well with uncertainty. Future studies should aim to examine the role of intolerance of uncertainty in anger. Overall, it seems possible that similar cognitive processes contribute to both anger and anxiety. Another possibility relates to a model of GAD that posits that the heightened intensity of many emotions contribute to GAD (Mennin, Heimberg, Turk, & Fresco, 2005). Thus, individuals with GAD may find anger and other emotions overwhelming, and these individuals may therefore worry about the consequences of losing control over their anger.

Anger may be particularly important to examine in the context of anxiety disorders as it can interfere with cognitive-behavioural treatment (CBT). For instance, one study found that pre-treatment anger predicted poorer response to CBT for individuals with SAD (Erwin et al., 2003). Although the mechanisms by which anger leads to poor CBT responses are unknown, one possibility is that anger interferes with common therapy factors in the treatment of anxiety disorders. For example, anger may interfere with the development of a strong therapeutic alliance, as suggested by DiGiuseppe, Tafrate, and Eckhardt (1994). In addition, anger may lead to lower motivation in treatment or resistance to change, or a less collaborative approach to goal setting, all of which are known to affect treatment response (Hubble, Duncan, & Miller, 2004).

It is currently unknown whether anger leads to poor responses in CBT for GAD. We can postulate, however, that anger may interfere with some components of empirically-supported CBT protocols for GAD. For example, Roemer and Orsillo (2007) developed a treatment protocol that targets experiential avoidance, which is characterized by attempts to reduce the

intensity and frequency of negative internal experiences. Anger may interfere with clients' ability to focus awareness on the present moment and accept internal experiences. Another empirically-supported CBT protocol for GAD includes problem-solving training as a component of treatment (Dugas & Robichaud, 2007). Given that high levels of anger and hostility have been found to predict poor social problem-solving skills (D'Zurilla, Chang, & Sanna, 2003), individuals with GAD who have elevated anger may be faced with particular challenges when attempting to solve their day-to-day problems. The effect of anger on specific components of treatment, however, requires further exploration.

The finding that scores on measures of anger and aggression correctly classified individuals meeting diagnostic criteria for GAD at a greater than chance level (57.3% and 64.9%, respectively) is noteworthy. These findings suggest that it may be valuable for clinicians to inquire about anger difficulties in clients with GAD to obtain a more complete understanding of potential emotional problems, particularly given that difficulties with anger management are not screened for in common diagnostic assessments, with the exception of borderline personality disorder (e.g., The Structured Clinical Interview for DSM-IV Axis-II Disorders (SCID-II); First et al., 1997).

Limitations

A possible caveat to empirically investigating anger is the lack of a consistent definition of anger and its related constructs (Eckhardt et al., 2004). There is currently little agreement on definitions for the dimensions of anger, and this likely affects the development of self-report anger assessments. Thus, the reliance on such self-report measures in the present study is a limitation. Future studies could improve on this by using multi-method assessments of anger. In addition, our study is limited by the use of an analogue sample of GAD composed of university

students enrolled in at least one psychology course. Although analogue samples have been shown to be similar to clinical samples of individuals with GAD on measures of worry and anxiety (Roemer, Borkovec, Posa, & Borkovec, 1995), we cannot be certain that the anger levels reported by our GAD-analogue group would be comparable to individuals with GAD who were recruited from a clinical setting.

Arguably, another limitation is that our statistical analyses did not control for depression. Elevated anger levels have been found in individuals with major depression (e.g., Riley, Treiber, & Woods, 1989), and GAD and major depression are highly comorbid (e.g., Brown et al., 2001). It is therefore possible that our results were in part due to shared variance between anger and depression. However, the decision to exclude depression as a covariate was made to increase the ecological validity of our results. Specifically, there are a number of symptoms of GAD and depression that overlap, such as difficulty concentrating, fatigue, and sleep disturbance (APA, 2000), and these criteria were included in our measure of GAD. In addition, negative affect is common to both anxiety and depression, as suggested by the tripartite model of depression and anxiety (Clark & Watson, 1991). Relatedly, depressive symptoms are important features of the clinical presentation of GAD, and controlling for these would “exclude” a number of symptoms that make up the diagnostic criteria for GAD, thereby limiting the generalizability of our results. Furthermore, Miller and Chapman (2001) suggested that statistically “removing” shared variance between two conceptually similar constructs (e.g., anxiety and depression) leads to poor construct validity of the target construct. In summary, we chose not to control for depression, given the overlapping nature of GAD and depression.

Conclusions

The potential link between anger and GAD in cognitive-behavioural contexts has not been given much attention. This is reflected in the scarce literature on anger and GAD, and the lack of recommendations for addressing anger-related symptoms in evidence-based treatments for GAD. The current findings highlight the importance of examining the co-occurrence of anger and GAD. Overall, our results suggest that multiple facets of anger are related to GAD symptoms; although further research is needed to identify the mechanisms by which high trait anger, internalized anger expression, and hostility are related to GAD.

CHAPTER 3:

BRIDGE

Results from study 1 demonstrate that GAD is positively related to multiple components of anger, including trait anger, anger suppression, and hostility. It is unclear, however, *why* GAD and anger are related. Possibly, elevated anger perpetuates symptoms of GAD as well as underlying cognitive vulnerabilities and processes. One way to test this hypothesis is by inducing anger in one group to observe its effects on cognitive mechanisms and symptoms of GAD relative to a control group. In study 2, anger was elicited in one group of participants via a hostile interaction with a research assistant. A neutral interaction with a research assistant served as a control condition. We examined the impact of induced anger on worry, a primary diagnostic symptom of GAD (APA, 2013), as well as on negative beliefs about uncertainty and threatening interpretations of ambiguous information, which are cognitive mechanisms involved in the etiology and maintenance of GAD (e.g., Dugas & Robichaud, 2007; Eysenck et al., 1991).

CHAPTER 4:
THE EFFECTS OF ANGER ON WORRY AND COGNITIVE VULNERABILITIES
UNDERLYING GENERALIZED ANXIETY DISORDER

Generalized anxiety disorder (GAD), characterized by excessive worry and anxiety (American Psychiatric Association [APA], 2000; 2013), is a debilitating disorder that is costly to society (Wittchen, 2002) and affects approximately 5.7% of the general population (Kessler et al., 2005). Despite its prevalence and economic burden, GAD remains an understudied disorder (Dugas, Anderson, et al., 2010). Although irritability is a criterion for the diagnosis of GAD, the association between anger, a construct closely related to irritability, and GAD is particularly understudied. Cross-sectional studies suggest associations between anger and symptoms of GAD; however the nature of these associations is not fully understood. Erdem and colleagues (2008) demonstrated that individuals diagnosed with GAD endorsed greater levels of trait anger, internalized anger expression (i.e., anger suppression), and externalized anger expression, than non-anxious individuals. Similarly, Deschênes, Dugas, Fracalanza, and Koerner (2012) showed that various facets of anger, particularly anger suppression and hostility, differentiated individuals who endorsed symptoms of GAD from those who did not. Although the abovementioned studies suggest associations between anger and symptoms of GAD, little research has focused on *why* anger and GAD are associated. Cognitive vulnerabilities such as core beliefs and information processing styles contribute to the development and maintenance of psychopathology (Clark & Beck, 2010), and conceivably, anger may perpetuate the cognitive vulnerabilities that maintain GAD symptomology.

A dominant theory of emotion posits that cognitions, such as interpretations of events, influence emotional responses (Lazarus, 1991). In accordance with this theory, Clark and Beck

(2010) propose that biases in interpretive processing (i.e., consistently negative regardless of the event) are involved in the development and maintenance of anxiety disorders, including GAD. Individuals with GAD tend to experience a hypervigilance for threat and danger, which is evidenced by threat-related interpretations of ambiguous (potentially negative) information (Anderson et al., 2012; Clark & Beck, 2010; Davey et al., 1992). As such, a negative interpretive style is associated with increased risk of developing GAD symptoms. Another cognitive vulnerability to GAD is intolerance of uncertainty. A cognitive model of GAD (Dugas & Robichaud, 2007) postulates that intolerance of uncertainty, which results from negative beliefs about uncertainty, contributes to the development and maintenance of GAD symptoms. These negative beliefs about uncertainty include the beliefs that uncertainty has negative behavioural and self-referent implications, and that uncertainty is unfair and spoils everything (Sexton & Dugas, 2009). This cognitive model has been validated in multiple cross-sectional (e.g., Buhr & Dugas, 2006) and experimental studies (e.g., Ladouceur, Gosselin, & Dugas, 2000), and has led to a successful empirically-supported cognitive-behavioural intervention that targets beliefs about uncertainty in individuals with GAD (e.g., Dugas, Brillon, et al., 2010; Ladouceur, Dugas, et al., 2000).

Bidirectional relationships between cognitive processes and affective states have also been proposed, postulating that emotion can precede cognitive processing (Schwarz & Clore, 2007; Zajonc, 1984). Indeed, there is evidence that anger affects interpretive style by increasing the likelihood of interpreting ambiguous stimuli in a threatening manner. Specifically, Barazzone and Davey (2009) found that laboratory-induced anger led to greater threat-related spellings of ambiguous homophone words (e.g., slay/sleigh; pain/pane), relative to a control condition. Although this suggests that anger increases the tendency to interpret ambiguous situations as

threatening, an investigation of a broader range of ambiguous situations is necessary to draw conclusions regarding interpretive style in GAD. That is, individuals with GAD worry about various topics such as finances, health, relationships, and harm, and it is therefore important to examine how anger might affect interpretations of various ambiguous situations.

In addition, it is unclear whether the experience of anger directly affects GAD symptoms or beliefs about uncertainty, given that the available studies assessing these relationships are correlational. Specifically, high trait anger and anger expression were associated with a greater tendency to endorse negative beliefs about uncertainty in a group of individuals with OCD (Radomsky et al., 2007). Further, negative beliefs about uncertainty partially mediated the association between GAD symptom severity and various forms of anger (i.e., anger suppression, hostility, trait anger) in university students (Fracalanza, Koerner, Deschênes, & Dugas, 2014). High levels of trait anger were also associated with high levels of trait worry (e.g., Stewart et al., 2010), and more broadly, with GAD symptom severity (Deschênes et al., 2012; Erdem et al., 2008). One method to assess the direct effect of anger on features of GAD is by the use of experimental manipulations; however to date, no studies have examined the impact of experimentally inducing anger on GAD symptoms and associated cognitive vulnerabilities.

To further understand the nature of the association between GAD and anger, the goals of the study were to examine the effects of experimentally induced anger on worry, the primary diagnostic feature of GAD, and cognitive vulnerabilities underlying GAD. Anger was elicited in participants using a well-validated anger-induction procedure. Following the induction, the extent to which participants interpreted ambiguous (possibly neutral or threatening) information as negative, endorsed negative beliefs about uncertainty, and worried was measured. We hypothesized that participants in an anger-eliciting condition would demonstrate greater negative

interpretive style, endorse greater negative beliefs about uncertainty, and engage in worry to a greater extent than participants in a neutral condition.

Method

Participants

Our sample consisted of 77 undergraduate students who participated in the study in exchange for course credit or financial compensation. The average age of the sample was 22.88 ($SD = 4.96$) years. The majority of participants were Caucasians (55.8%), 15.6% were Asian, 7.8% were Black, 6.5% were Middle Eastern, 6.5% were multi-racial, 3.9% were Hispanic, and 3.9% of our sample reported “other” as their ethnic origin. Most participants were female (81.80%), studied full-time (92.40%), and majored in psychology (56.10%). More than half of our sample was recruited from Concordia University (62.10%), whereas 37.9% were recruited from other universities in Montreal, Québec.

Measures and Materials

The Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1971) was used to assess current levels of emotions, including anger, sadness, anxiety, fear, well-being, calmness, vigor, and fatigue. We used an abbreviated version containing 25 items based on a factor analysis by Usala and Hertzog (1989). Scores on the anger, sadness, anxiety, fear, well-being, calmness, and vigor subscales ranged from 0 – 15, and scores on the Fatigue subscale ranged from 0 – 20.

The State-Trait Inventory for Cognitive and Somatic Anxiety – trait version (STICSA; Ree, French, MacLeod, & Locke, 2008) is a 21-item self-report questionnaire that assesses cognitive and somatic symptoms of anxiety. Items are assessed on a 4-point Likert scale ranging from 1 (*Not at all*) to 4 (*Very much so*). Higher scores on the STICSA reflect greater symptoms

of anxiety. The STICSA has demonstrated construct, convergent, and discriminant validity (Grös, Antony, Simms, & McCabe, 2007). Internal consistency in the current sample was $\alpha = .88$.

The Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) is a 16-item self-report measure that assesses the tendency to worry. Items are rated on a Likert scale ranging from 1 (*Not at all typical*) to 5 (*Very typical*). Higher scores on the PSWQ reflect a greater tendency to worry. The PSWQ has demonstrated convergent and divergent validity, and test-retest reliability between eight to 10 weeks ($r = .92$). Internal consistency in the current sample was $\alpha = .94$.

The Trait Anger subscale (T-ANG) of the State-Trait Anger Expression Inventory-2 (STAXI-2; Spielberger, 1999) is a 10-item scale that assesses the frequency and intensity of anger experiences. Items are rated on a Likert scale ranging from 1 (*Almost never*) to 4 (*Almost always*). Internal consistency in the current sample was $\alpha = .84$.

The Hostility subscale of the Aggression Questionnaire (AQ; Buss & Perry, 1992) is an 8-item scale measuring the cognitive component of aggression, including feelings of injustice. Participants rate the extent to which each statement is characteristic or uncharacteristic of them on a Likert scale ranging from 1 (*Extremely uncharacteristic of me*) to 5 (*Extremely characteristic of me*). The AQ has good test-retest reliability over nine weeks ($r = .80$). Internal consistency for the hostility subscale (AQ-HOST) in the current sample was $\alpha = .72$.

Interpretive style. A scrambled sentence task (SST) was developed to examine interpretation styles associated with GAD (Donegan & Dugas, in prep; see Appendix B), based on the SST developed for social anxiety (Standage, Ashwin, & Fox, 2010). The task requires participants to rearrange scrambled sentences to form meaningful statements using only 5 of the 6 words presented. These sentences can be disambiguated to form either a positive sentence (e.g.,

“my date will be pleased”) or a negative sentence (e.g., “my date will be disappointed”). To increase cognitive load and thus reduce social desirability effects, participants begin by memorizing a 6-digit number over 10 seconds. After 6 minutes of unscrambling sentences, participants are instructed to recall the 6-digit number shown at the start of the task. This SST was developed to directly target the interpretive processing style underlying GAD, by including all major worry themes (academic, work, finances, social, health of self and others, and safety of self and others) and targeting both cost (inability to cope with threat) and likelihood (heightened perceived probability of negative events) estimates of common worry themes. For our analyses, we calculated the ratio of the number of negative sentences formed to the total number (positive and negative) of sentences formed. A score greater than .5 reflects more negative interpretations whereas a score of less than .5 reflects more positive interpretations. There were 40 sentences in total; 20 sentences were completed at baseline and the remaining 20 sentences were completed following the manipulation. The order of the two versions of the SST was counterbalanced.

Beliefs about uncertainty. Negative beliefs about uncertainty were assessed using items from the Intolerance of Uncertainty Scale (IUS; Buhr & Dugas, 2002). The IUS is a 27-item questionnaire that assesses individuals’ intolerance of uncertainty on a 5-point Likert scale, ranging from 1 (*Not at all true of me*) to 5 (*Very much true of me*). A two-factor structure for the IUS has been reported (Sexton & Dugas, 2009), with Factor 1 reflecting the belief that uncertainty has negative behavioural and self-referent implications (IU-1) and Factor 2 reflecting the belief that uncertainty is unfair and spoils everything (IU-2). Higher scores on the IUS reflect greater negative beliefs about uncertainty. The IUS has been shown to have good test-retest reliability over five weeks ($r = .74$), and has demonstrated convergent and divergent validity (Buhr & Dugas, 2002). Two versions of the IUS were used in this study, with each version

consisting of 13 items. Counterbalanced across participants, one version was administered at baseline, and the other was administered following the manipulation. Items were selected for each version based on Factor 1 and Factor 2 item loadings, with an equal representation of strong loadings in both versions. One weak-loading item was omitted so that total scores on each version would be comparable.

Worry. The Catastrophizing Interview is a well-validated structured worry task (Provencher, Freeston, Dugas, & Ladouceur, 2000; Vasey & Borkovec, 1992) that assesses various aspects of worrying and provides idiosyncratic content by using participant-generated worry themes. In the first phase of this interview, participants are asked to disclose their most severe worry theme. The experimenter then proceeds to the catastrophizing phase of the interview, which begins by asking “*What is it about (worry) that worries you?*” Once a response is provided, the experimenter asks “*If (worry) were to happen, what are you afraid would happen next?*” This question is repeated in response to the previous answer until the participant cannot generate any more feared consequences. Participants then rate the likelihood of each catastrophizing step on a scale from 1 (*Not at all likely*) to 100 (*Extremely likely*), and the severity of each catastrophizing step on a scale from 1 (*Not at all severe*) to 8 (*Extremely severe*). Three variables are thus derived from this task: the number of catastrophizing steps, the average likelihood of steps, and the average severity of steps.

Manipulation check. Heart rate (beats per minute) was measured continuously throughout the study using a Polar Heart Rate monitor (Polar RS800CX) to assess physiological reactivity to the manipulation. The POMS was used to assess current anger as well as other emotional states. The POMS was administered at baseline and immediately following the manipulation.

Procedure

Participants were recruited from Concordia University's Psychology Department Participant Pool or from advertisements placed at a nearby university. They were informed that the goal of the study was to examine how personality variables affect reactions to various mental tasks as well as cardiac activity. After providing written consent to participate (see Appendix C), participants completed a series of questionnaires assessing anger (T-ANG), hostility (AQ-HOST), negative beliefs about uncertainty (IUS), and worry (PSWQ). They also completed the SST, and were then randomly assigned to an anger induction ($n = 43$) or a control condition ($n = 34$). We randomized a greater number of participants to the anger induction condition to offset for the possibility that some participants might not become angry in response to the hostile interaction and would therefore be excluded prior to analyses (see below), and to compensate for the possibility that participants may refuse to continue the study following the hostile interaction (which did not occur). The dependent measures (i.e., the SST, the IUS, and the Catastrophizing Interview) were administered following the manipulation by the first experimenter, who was blind to participant condition.

For our anger manipulation, we used an ecologically-valid anger-induction paradigm involving a hostile interaction with a second experimenter (e.g., Neumann et al., 2011; Suarez, Harlan, Peoples, & Williams, 1993; Suarez & Williams, 1989). Following the completion of baseline measures, a heart rate monitor was attached to participants. Participants were informed there would be a 5-minute rest period to obtain resting heart rate. In each condition, at the end of the 5 minute rest period, the experimenter walked down the hallway past the participant's room and asked a research assistant to complete the next task as she had to step out for a few moments. The door to the participant's room was left slightly ajar so that participants could overhear the

conversation. The research assistant then entered the participant's room to announce that she would be taking over until the experimenter returned from a brief impromptu meeting. In the hostile condition, the research assistant announced her displeasure about having to do this (e.g., "Hi I'm (Name), the experimenter told me to continue testing for her since she had to step out for a moment, so it looks like I'm stuck having to do this now. Hopefully she won't be too long, but whatever, let's just get started with the first task"). In the control condition, she simply announced her presence.

In both conditions, the participants completed a task consisting of five-letter solvable anagrams presented on Microsoft PowerPoint and participants were asked to verbalize their answers. Participants solved as many anagrams as possible for 6 minutes, and were informed that the top 40% of those who solved the most correct anagrams would be entered in a draw to win \$50 (all participants were entered in this draw). In the anger condition, the research assistant delivered 8 standardized harassing statements at 30-second intervals during the 6-minute task (e.g., "You have 4 minutes remaining. You will need to try harder if you want to be entered in the draw"). In the neutral condition, the research assistant simply stated the remaining time in the task at 30-second intervals. As a manipulation check, participants completed the POMS immediately following the task. The experimenter then returned, apologized for her absence, and administered the tasks assessing the study's dependent variables. The tasks included another SST, IUS items, and the Catastrophizing Interview. The SST and the IUS were administered in a counterbalanced order across participants. The Catastrophizing Interview was always the final task administered. The length of the interview differs across participants, and if administered prior to other tasks, the duration of the interview may have confounded the results. Also, given that emotional consequences of the interview can differ across participants, administering the

interview as the final task prevented any possible affective changes taking place during the interview from interfering with the other tasks. Finally participants were debriefed and for participants in the anger induction condition (see Appendix D for post-study consent form), the “hostile” research assistant returned to introduce herself and thank them for their participation.

Results

The anger induction led to greater self-reported anger and physiological arousal relative to the control condition, suggesting that our manipulation was successful. Specifically, those in the anger condition reported significantly greater state anger on the POMS ($M = 2.67$, $SD = 2.84$) following the anagram task than those in the control group ($M = .53$, $SD = .93$), $t(52.88^2) = -4.64$, $p < .001$, and had a higher mean heart rate immediately following the anagram task ($M = 85.91$, $SD = 14.65$) than those in the control group ($M = 78.00$, $SD = 11.26$), $t(58) = -2.32$, $p = .02^3$.

To examine the specificity of our anger manipulation relative to other emotions, we conducted additional t -tests between conditions on the different subscales of the POMS. There were no significant between-group differences following the manipulation on depression, anxiety, fear, well-being, calmness, or vigor ($ps > .12$). There was, however, a between-group difference on level of fatigue following the manipulation, such that the neutral condition reported higher levels of fatigue than the anger condition, $t(75) = 2.33$, $p = .02$. This finding was not surprising however, as state anger is an emotion associated with elevated physiological arousal (e.g., Lobbestael, Arntz, & Wiers, 2008).

² Degrees of freedom were corrected to account for the violated assumption of homogeneity of variance (assessed by Levine’s test of equality of variance, $p < .001$).

³ Hardware malfunction led to inaccurate heart rate recordings for 17 participants. The excluded participants did not differ from the included participants on socio-demographic variables or baseline psychological measures (all $ps > .26$).

Given that our hypotheses were that state anger would affect subsequent worry and cognitive processes, only participants who responded to the anger induction were included in the main analyses. That is, participants in the anger condition who did not experience an increase in anger following the hostile interaction were excluded. To be included, participants in the anger induction group were required to report an increase of at least 1-point on the POMS from baseline to post-induction (11 participants were excluded from the anger induction group). A difference of 1-point on the POMS is greater than the standard deviation of the POMS-anger subscale at baseline. Our final sample included 66 participants ($n = 32$ in the anger condition, $n = 34$ in the control condition). With our final sample, participants in the anger condition did not differ from participants in the control condition on socio-demographic features (i.e., age, sex, or education year), recruitment type, or trait psychological variables measured at baseline (i.e., negative beliefs about uncertainty, trait anxiety, worry, trait anger, or hostility) prior to the manipulation ($ps > .10$). See Table 8 for correlations between trait psychological variables measured at baseline.

To test our hypothesis that the anger induction would lead to greater negative interpretive style, we conducted a repeated measures analysis of variance (ANOVA) with condition as the between-subjects factor and scores on the SST task before and after the manipulation as the within-subjects factor. There was no main effect of group or main effect of time. However in support of our hypotheses, we found a significant interaction between group and time such that greater increases in negative interpretations of the scramble sentences from baseline to post-manipulation were observed in the anger condition relative to the control condition, $F(1, 64) = 5.60, p = .02, \eta_p^2 = .08$.

Table 8

Correlations between Trait-Level Psychological Variables Measured At Baseline (N = 77)

	1	2	3	4	5	6	7
1. PSWQ	1.00	.51**	.65**	.55**	.52**	.53**	.31**
2. IU-1 – baseline		1.00	.63**	.64**	.39**	.53**	.27*
3. IU-2 – baseline			1.00	.43**	.46**	.48**	.30**
4. STICSA-Trait				1.00	.52**	.60**	.34**
5. STAXI-2-Trait					1.00	.60**	.25*
6. AQ-Hostility						1.00	.30**
7. SST- baseline							1.00

Notes: PSWQ = Penn State Worry Questionnaire; IU-1 = Factor-1 Intolerance of Uncertainty Scale (belief that uncertainty has negative behavioural and self-referent implications); IU-2 = Factor-2 Intolerance of Uncertainty Scale (belief that uncertainty is unfair and spoils everything); STICSA = State-Trait Inventory for Cognitive and Somatic Anxiety; STAXI-2 = State-Trait Anger Expression Inventory, second edition; AQ-HOST = Aggression Questionnaire – Hostility Subscale; SST = Scrambled Sentence Task, assessed at baseline; HR-baseline = heart rate (beats per minute) assessed at baseline.

* $p < .05$, ** $p < .01$

Table 9

Means and Standard Deviations per Condition before and after the Manipulation

	Neutral		Anger	
	Pre	Post	Pre	Post
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>
POMS-Anger	.47 (1.35)	.53 (0.93)	.38 (0.94)	3.59 (2.75)
HR	76.54 (11.69)	78.00 (11.26)	79.81 (9.82)	89.39 (14.30)
IU-1	16.62 (5.42)	16.76 (6.25)	15.97 (4.28)	15.19 (4.97)
IU-2	18.15 (5.00)	16.97 (5.19)	16.69 (4.49)	17.56 (5.02)
SST	.25 (.15)	.21 (.15)	.19 (.17)	.23 (.16)
CI-Steps	--	6.21 (2.63)	--	7.00 (2.81)
CI-Likelihood	--	59.32 (18.75)	--	55.65 (15.80)
CI-Severity	--	5.86 (1.10)	--	6.11 (1.07)

Notes: POMS-Anger = Profile of Mood States, Anger subscale; HR = heart rate, measured in beats per minute; IU-1 = Intolerance of Uncertainty Scale-Factor-1 (belief that uncertainty has negative behavioural and self-referent implications); IU-2 = Intolerance of Uncertainty Scale-Factor-2 (belief that uncertainty is unfair and spoils everything); SST = Scrambled Sentence Task (measures interpretation style); CI = Catastrophizing Interview.

To test our hypothesis that the anger induction would lead to greater negative beliefs about uncertainty, we conducted two repeated measures ANOVAs with condition as the between-subjects factor and scores on IU-1 and IU-2 from baseline and post-manipulation assessments as the within-subject factors. There was no main effect of group or main effect of time, and we found no group by time interaction on changes in IU-1 from baseline to post-manipulation, $F(1, 64) = 1.51, p = .22, \eta_p^2 = .02$; however we found a significant interaction with IU-2, such that greater increases in IU-2 from baseline to post-manipulation were found in the anger condition relative to the control condition, $F(1, 64) = 6.16, p = .02, \eta_p^2 = .09$.

Finally, to test our hypothesis that anger would lead to greater worry relative to a neutral condition, we conducted three separate one-way ANOVAs with condition as our between-subject factor and the Catastrophizing Interview variables as the dependent measures (i.e., number of steps, and perceived likelihood and severity of steps). However, we found no between-group differences on the Catastrophizing Interview variables. Specifically, there were no between-group differences on the number of steps, $F(1, 64) = 1.41, p = .24, \eta_p^2 = .02$, the average likelihood of the steps, $F(1, 64) = 0.73, p = .40, \eta_p^2 = .01$, or the average perceived severity of the steps, $F(1, 64) = 0.86, p = .36, \eta_p^2 = .01$. Table 9 presents means and standard deviations within our final sample for baseline and post-manipulation measures of state anger, heart rate, IU-1, IU-2, and the SST, as well as the Catastrophizing Interview variables.

Discussion

The goal of the study was to examine the effects of induced anger on worry and associated cognitive vulnerabilities, including negative interpretive style and negative beliefs about uncertainty. Overall, our hypotheses were partially supported. We found that participants in the anger condition experienced a greater increase in negative interpretive style and in the

belief that uncertainty is unfair and spoils everything, relative to participants in the control condition. However, no effects were found with the belief that uncertainty has negative behavioural and self-referent implications or with worry. These findings provide partial support for the notion that state anger perpetuates some of the cognitive vulnerability factors underlying GAD, and therefore may partially account for the association between anger and GAD.

Our finding that induced anger increased threatening interpretations of ambiguous stimuli is consistent with previous literature (e.g., Barazzone & Davey, 2009; Wenzel & Lystad, 2005), and suggests that when individuals are angry, their interpretive style resembles that of anxious individuals. Barazzone and Davey (2009) found that manipulating state anger using anger-eliciting music and vignettes led to a greater likelihood of spelling ambiguous homophone words in a negative manner compared to a control condition. In contrast to their study, we used an anger-induction paradigm that reduces social desirability effects and increases ecological validity, and used an interpretation task that was tailored for GAD. Therefore, these findings extend the current literature on the effects of anger on interpretive style by replicating recent findings using a “naturalistic” anger induction and providing a GAD framework.

In addition, although this was the first study to examine the direct impact of state anger on beliefs about uncertainty, our findings are in line with previous reports of associations between trait anger and negative beliefs about uncertainty (Fracalanza et al., 2014; Radomsky et al., 2007). We found that participants in the anger condition endorsed the belief that uncertainty is unfair and spoils everything to a greater extent than participants in the control condition. Similarly, Fracalanza and colleagues (2014) found that this belief uniquely mediated the association between externalized anger and GAD symptoms, as well as hostility and GAD symptoms. Our experimental results extend these correlational findings. One possibility as to

why the belief that uncertainty is unfair and spoils everything was endorsed to a greater extent in the experimental condition than the control condition relates to the nature of the manipulation. Specifically, the hostile interaction may have been perceived by participants as unfair. Given that a determinant of anger is the attribution of unfairness (e.g., Smith & Ellsworth, 1985), beliefs about unfairness, which can include the belief that uncertainty is unfair, may have been primed to a greater extent than the belief that uncertainty has negative behavioural and self-referent implications.

We expected that the anger induction would lead to increased worry; however we did not find any effects of the manipulation on the worry process assessed by the Catastrophizing Interview. One explanation for these null findings is that any effects of the anger induction dissipated prior to the administration of the Catastrophizing Interview, given that the interview was the final dependent measure administered. Possibly, significant results may have emerged if worry was assessed immediately after the manipulation. Therefore, it is necessary to explore this possibility further using a study design that assesses worry immediately following an anger induction. At the moment, it is unclear how state anger directly affects worry.

Despite the strengths of our study, which include the use of an ecologically-valid anger induction procedure, certain limitations should be noted. First, our findings are limited by the use of a non-clinical sample. To better understand the mechanisms underlying the association between anger and GAD, the direct effect of state anger should be examined in a clinically anxious population. Second, it is unclear from this study whether anger directly leads to greater negative interpretive style and beliefs that uncertainty is unfair and spoils everything, or whether general negative affect produced by the hostile interaction triggered these effects. To clarify these findings, future research should compare the effects of an anger induction to that of a

depressive mood induction on subsequent cognitive vulnerabilities. Finally, our effect sizes regarding the impact of state anger on GAD-related cognitive processes were relatively small, which highlights the need for replication.

To conclude, this study provides partial support for the notion that anger contributes to cognitive vulnerabilities underlying GAD, by intensifying negative interpretive style and specific negative beliefs about uncertainty. From a broader perspective, our findings are also consistent with the notion that cognitive vulnerabilities can be affected by emotional states. Although cognitive accounts of emotion (Clark & Beck, 2010; Lazarus, 1991) propose that cognitions are necessary for the experience of emotions, our findings are in line with the theories proposing that cognition and emotion are independent interrelated systems (Zajonc, 1984), and that affective states can subsequently affect judgments and thought processes (Schwarz & Clore, 2007). From a clinical perspective, our findings offer preliminary support for the idea that anger may obstruct attempts at generating alternative appraisals for potentially threatening ambiguous events or attempts to decrease negative beliefs about uncertainty, both of which are components of cognitive-behavioural treatments for GAD.

CHAPTER 5:

BRIDGE

Results from study 2 demonstrate that induced state anger potentiates, at least to some extent, cognitive factors associated with GAD. The cognitive factors impacted were the belief that uncertainty is unfair and spoils everything and threatening interpretations of ambiguous stimuli. An important step in furthering our understanding of the nature of the association between anger and GAD is to examine whether worry impacts anger-related cognitive processing style. For instance, individuals with high trait anger tend to interpret ambiguously hostile behaviours as deliberate and purposeful (e.g., Hazebroek et al., 2001; Owen, 2011). A goal of study 3 was to examine the effect of a worry induction, compared to a relaxation induction, on hostile interpretations of ambiguous intent. The cross-over effects of anger and worry on cognitive processes involved in GAD and high trait anger were also examined in study 3. Therefore, an anger induction was also included in study 3 and the effects of induced anger on threatening interpretations of ambiguous information were compared to a relaxation induction. Finally, we examined whether GAD analogues reported greater hostile interpretations of ambiguous intent and whether GAD analogue status interacted with the worry and anger experimental conditions to predict increased hostile and threatening interpretations of ambiguous information.

CHAPTER 6:
AN INVESTIGATION OF THE EFFECTS OF WORRY AND ANGER ON
THREATENING INTERPRETATIONS AND HOSTILE ATTRIBUTIONS OF
AMBIGUOUS SITUATIONS

A number of studies have demonstrated that anger, an emotion that varies in intensity from mild irritability to intense fury and rage (Spielberger, 1988), is associated with generalized anxiety disorder (GAD), a disorder characterized by excessive worry and anxiety (American Psychiatric Association [APA] 2000, 2013). Erdem and colleagues (2008) found that relative to non-anxious individuals, those with GAD reported higher trait anger, outward anger expression, and anger suppression, as well as lower anger control. In addition, Deschênes and colleagues (2012) found that elevated trait anger, hostility, and anger suppression predicted greater GAD symptom severity.

Two large epidemiological investigations using nationally-representative samples in the United States (Hawkins & Cogle, 2011) and Australia (Barrett, Mills, & Teesson, 2013) have demonstrated that a diagnosis of GAD is associated with elevated anger. Specifically, Hawkins and Cogle (2011) found that GAD was related to greater anger experience over the past 30 days, and this relationship remained after controlling for socio-demographic features and comorbid diagnoses, including other anxiety disorders, bipolar disorder, substance abuse disorders, depression, and borderline personality disorder. Similarly, Barrett and colleagues (2013) found that GAD was related to greater anger severity over the past 30 days, controlling for socio-demographic features, comorbid anxiety disorders, mood and bipolar disorders, and substance use disorders. Together, these studies suggest that elevated anger seems to characterize individuals with GAD, independent of comorbid conditions, such as mood or anxiety disorders.

Research investigating the mechanisms underlying the association between anger and symptoms of GAD is scarce. One possibility is that shared underlying information-processing styles are associated with both anger and anxiety or worry responses. Many studies support the notion that biased information processing (e.g., more negative) is an important factor contributing to the etiology and maintenance of GAD symptomology. For instance, individuals with GAD or elevated trait anxiety are quicker to attend to threatening information (MacLeod, Mathews, & Tata, 1986; Mogg & Bradley, 2005; Williams, Mathews, & Hirsch, 2014) and interpret ambiguous information as more threatening (Eysenck et al., 1991; Eysenck, MacLeod, & Mathews, 1987; Mathews et al., 1989) than non-anxious individuals. Recent studies have demonstrated that modifying information processing style to either increase attention to non-threatening information (Amir, Beard, Burns, & Bomyea, 2009) or to facilitate benign interpretations (Hayes, Hirsch, Krebs, & Mathews, 2010) leads to reductions in GAD symptoms.

Some studies also suggest that anger is related to cognitive vulnerabilities associated with GAD. For instance, Fracalanza and colleagues (2014) found that intolerance of uncertainty, a cognitive vulnerability for GAD (Dugas & Robichaud, 2007), mediated the relationship between GAD symptoms and trait anger. Similarly, Deschênes, Dugas, Anderson, and Gouin (2014) found that inducing state anger using a naturalistic insult paradigm led to increases in intolerance of uncertainty. They also found that the state anger induction led to increases in threatening interpretations of ambiguous sentences, which indicates that anger is also related to information processing styles characteristic of individuals with GAD. Similarly, Barazzone and Davey (2009) found that laboratory-induced state anger led to increases in threatening interpretations of ambiguous homophone words. Together these findings suggest that elevations in anger impact cognitive vulnerabilities and processes that underlie GAD.

Certain information processing styles also contribute to elevated anger. For example, following provocation, individuals with high trait anger attend to anger-related words more rapidly than individuals with low trait anger (Eckardt & Cohen, 1997; Van Honk et al. 2001). High trait anger is also associated with the tendency to perceive the intent of others in situations that lead to negative outcomes as deliberately hostile (Hazebroek et al., 2001). In accordance, a computerized training program aimed at reducing such hostile attributions led to greater decreases in anger in response to an insult than a computerized training program aimed at increasing hostile interpretations (Hawkins & Coughle, 2013). These findings suggest that hostile interpretations of ambiguous social interactions are more pronounced in angry versus non-angry individuals (see Owen, 2011, and Wilkowski & Robinson, 2010, for reviews).

A limited number of studies have examined the associations between trait anxiety and information processing styles associated with high trait anger. These studies generally suggest that elevated trait anxiety is indeed associated with anger-related cognitive processes. For instance, when examining naturally-occurring thought content in reaction to daily emotional experiences, Wickless and Kirsch (1988) found that although the strongest associations were seen between the emotional experience (i.e., anger, anxiety, or sadness) and the emotion-congruent thought content (i.e., transgressions, threat, and loss, respectively), anxiety was associated with increased thoughts of transgressions. In addition, Byrne and Eysenck (1995) found that low trait anxiety was associated with a slower latency to detect angry faces from a set of happy faces. These studies provide evidence suggesting that elevated anxiety is associated with cognitive factors underlying trait anger such as hostile thoughts and biased attention towards anger-related stimuli. However, Van Honk et al. (2001) found that although trait anger was consistently associated with greater attention towards angry faces, their two experiments

failed to find that trait anxiety predicted greater attention to angry faces. Further research is needed on the association between trait anxiety and information processing biases associated with high trait anger.

Taken together, there is some correlational and experimental evidence suggesting that elevated anger is related to information processing styles underlying GAD symptoms, and that GAD symptoms are related to information processing styles underlying high trait anger. It is currently unknown, however, whether worry directly increases information processing styles associated with anger. Therefore, the goal of this study was to experimentally increase worry to examine the effects on hostile attribution bias. In addition, we aimed to replicate previous findings by examining whether induced anger leads to greater threat-related interpretation bias. These effects were compared to a relaxation control condition. We hypothesized that relative to a relaxation condition, both worry and anger would lead to greater threatening interpretations of ambiguous situations and to greater hostile attributions.

We also explored whether GAD analogues (i.e., participants who met diagnostic criteria for GAD by self-report) had a greater likelihood of exhibiting a negative information processing style than less anxious participants, and whether this effect would be enhanced in the worry and anger conditions relative to the relaxation condition. Individuals with GAD tend to have different information processing styles (e.g., more biased towards negative information/interpretations) than non-anxious individuals (e.g., Eysenck, et al., 1991; Mathews et al., 1989). Cognitive theory suggests that this tendency is more pronounced when cognitive vulnerabilities become activated; such as in stressful situations or following a mood or worry induction (see Clark & Beck, 2010). Thus, we hypothesized that GAD analogues would report greater threatening interpretations and

hostile attributions than less anxious individuals, and that these effects would be increased in the worry or anger conditions relative to the relaxation condition.

Method

Participants

One hundred and fifty ($N = 150$) psychology undergraduate students, recruited from the institutional participant pool, participated in exchange for course credits. The average age of the sample was 22.68 ($SD = 4.57$) years and the majority of participants were female ($n = 123$; 82%). Ninety-eight participants (65.3%) were White, 20 (13.3%) were Middle Eastern, 11 (7.3%) were Multi-Racial, 9 (6%) were Asian, 8 (5.3%) were Hispanic, 3 (2%) were Black, and one (0.7%) participant reported “other” as their ethnic origin.

Materials

Current affective state. State levels of anxiety, worry, anger, sadness, happiness, and relaxation were assessed using 100 mm visual analogue scales (VAS). Higher scores represent a greater intensity of each affective state.

Measures of GAD symptoms. The Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990) is a 16-item measure that assesses the general tendency to worry on a 5-point Likert Scale ranging from 1 (*Not at all typical*) to 5 (*Very typical*). Scores can range from 16 to 80, with higher scores reflecting a greater propensity to worry. The PSWQ has demonstrated convergent and divergent validity, test-retest reliability over eight to ten weeks ($r = .92$), and excellent internal consistency in our sample ($\alpha = .93$).

The State-Trait Inventory for Cognitive and Somatic Anxiety – Trait scale (STICSA-T; Ree et al., 2008) is a 21-item self-report measure that assesses the general tendency to experience somatic (e.g., muscle tension) and cognitive (e.g., worry) symptoms of anxiety on a 4-point

Likert scale ranging from 1 (*Not at all*) to 4 (*Very much so*). Scores can range from 21 to 84, with higher scores reflecting greater trait anxiety. The STICSA-T demonstrates construct, convergent, and discriminant validity (Grös, Antony, Simms, & McCabe, 2007), and we found good internal consistency in our sample ($\alpha = .89$).

The Generalized Anxiety Disorder Questionnaire (GAD-Q-IV; Newman et al., 2002) is a 14-item self-report screening tool developed to assess the presence of GAD based on DSM-IV (APA, 2000) diagnostic criteria. No changes were made to the symptoms of GAD in the latest revision of the DSM (i.e., DSM-5; APA, 2013); as such, the GAD-Q-IV assesses current conceptualizations of GAD symptomology. Items are rated either dichotomously (presence or absence of symptoms) or on the degree of interference and distress produced by the symptoms on a Likert scale ranging from 0 (*None*) to 8 (*Very severe*). Scores can range from 0-13, with higher scores reflecting greater GAD symptomology. Scores of 5.7 and above indicate the presence of GAD and scores below 5.7 indicate the absence of GAD, based on recommendations from Newman et al. (2002). With this cut-score, participants in our sample meeting GAD diagnostic criteria (i.e., GAD-analogue group; $n = 41$) reported significantly greater worry assessed by the PSWQ ($M = 62.83$, $SD = 9.03$) than participants not meeting diagnostic criteria for GAD (i.e., non-GAD group; $n = 109$) ($M = 47.43$, $SD = 11.97$), $t(148) = -7.47$, $p < .001$. The GAD-analogue group also reported significantly greater trait anxiety assessed by the STICSA ($M = 44.54$, $SD = 8.19$) than the non-GAD group ($M = 32.35$, $SD = 6.73$), $t(148) = -9.31$, $p < .001$.

Dependent variables. The Ambiguous Unambiguous Situations Diary (AUSD; Davey et al., 1992; Koerner & Dugas, 2008) assesses interpretations of ambiguous situations as threatening or benign. The original version (Davey et al.) consists of positive, negative, and ambiguous scenarios. Given that our hypotheses pertained to the interpretation of ambiguous

events, we omitted the positive and negative scenarios from the study and retained only the ambiguous scenarios (AUSD-A). As such, the modified version of the AUSD (Koerner & Dugas) contains 22 ambiguous passages (i.e., where the outcome of the situation can be positive/neutral, or threatening/negative), resembling diary entries written in the first person (e.g., “Today, I was on the bus when I noticed some of my classmates sitting behind me, talking with each other in a low voice”). The worry domains covered in the scenarios include social relationships, academic and work performance, finances, personal health and health of others, physical danger, the future, and self-concept. Participants rate their perceived level of concern for each scenario on a scale from 1 (*Not at all concerned*) to 5 (*Extremely concerned*). Scores can range from 22 to 110, with greater concern reflecting greater threatening interpretations of the ambiguous scenarios.

The Social Information Processing–Attribution and Emotional Response Questionnaire (SIP–AEQ; Coccato, Noblett, & McCloskey, 2009) assesses attributional and emotional responses to aversive, socially ambiguous situations involving one or more provocateurs. For the purpose of this study, only attributional responses were examined. The SIP-AEQ contains eight vignettes describing an aversive situation (e.g., “You and a group of your co-workers went on a business trip. While at the hotel, waiting to meet a customer, you stop to buy a cup of coffee. Suddenly, one of your co-workers bumps your arm and spills your coffee over your shirt. The coffee is hot and your shirt is wet”). For each vignette, the perceived likelihood of direct hostile intent as the cause of the situation (e.g., “My co-worker wanted to burn me with the hot coffee”) and the perceived likelihood of benign intent (e.g., “My co-worker did it by accident”) are rated on a 4-point Likert scale ranging from 0 (“*Not at all likely*”) to 3 (“*Very likely*”). Scores on each

scale can range from 0 to 24, with higher scores reflecting greater hostile attributions or greater benign attributions, respectively.

Procedure

To mask the true purpose of the study, participants were invited to take part in a study examining how thoughts about past or future events affect interpersonal behaviour. After providing informed consent (see Appendix E), participants completed a series of questionnaires, including a socio-demographic form, PSWQ, STICSA-T, and GAD-Q-IV. They also completed pre- and post-manipulation state affect measures using VASs.

Following the completion of baseline measures, participants were instructed to rest for a 5-minute period and were then randomly assigned to one of three conditions: the worry induction condition ($n = 51$), the anger induction condition ($n = 50$), or the relaxation condition ($n = 49$). The manipulation was used to produce affective changes in worry and anxiety, anger, and positive mood and relaxation, respectively. The manipulation instructions were as follows; for the worry condition, participants were instructed to pick their most worrisome topic and worry about it as intensely as possible for five minutes, focusing on the consequences of the feared outcome if it were to happen (adapted from Fisher & Newman, 2013). For the anger condition, we used an anger rumination task adapted from Waldstein et al. (2000). Participants were instructed to think about an unresolved incident that made them feel very angry, frustrated, or irritated for five minutes, and to try to mentally recreate the incident from beginning to end, focusing on what was said and done, the location, the person or persons involved, and how they felt during the incident. For the relaxation condition, participants were instructed to relax as much as possible and to shift their focus onto their breathing for five minutes, as well as to try to breathe slowly and evenly as they inhaled and exhaled. Instructions were adapted from Fisher

and Newman (2013). Following the manipulation, dependent measures were administered on a computer and consisted of assessing threat interpretation bias using the AUSD-A and assessing hostile attribution bias using the SIP-AEQ, which were administered in a counterbalanced order. Participants were debriefed at the end of the study (see Appendix F for post-study consent form).

Results

Data Screening and Randomization Checks

All data were normally distributed (skewness values < 3.0 and kurtosis values < 10.0 ; Kline, 2009). To examine whether randomization was successful, we compared groups on baseline measures of trait and state psychological characteristics. As expected, the groups did not differ on measures of state affect upon arrival to the laboratory. In addition, there were no main effects of condition on the PSWQ, $F(2, 147) = 0.92, p = .40, \eta_p^2 = .01$, but there was a marginal effect of group on the STICSA-T, $F(2, 147) = 2.34, p = .10, \eta_p^2 = .03$. Pairwise comparisons indicated that participants in the worry induction condition ($M = 33.76, SD = 8.07$) had significantly lower trait anxiety than participants in the relaxation condition ($M = 37.61, SD = 9.46$), $p = .03$. As such, the STICSA-T was entered as a covariate in our primary analyses. See Table 1 for means and standard deviations on the VASs.

Manipulation Checks

To examine the specificity of our manipulation on current psychological states measured by the VASs, we conducted a series of 2X3 mixed factorial analyses of variance (ANOVAs) with VAS before and after the manipulation as the repeated factor and condition as the between-groups factor (see Table 10 for means and standard deviations). As expected, we found a significant condition by time interaction from pre to post manipulation on worry, $F(2,147) =$

Table 10

Means and Standard Deviations per Condition for Visual Analogue Scales Administered Pre and Post Manipulation

	<u>Condition</u>					
	Worry		Anger		Relaxation	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<i>Measure and Time</i>						
Worry*						
Pre	17.41	18.48	28.22	23.57	18.92	20.93
Post	52.41	24.43	36.80	25.18	14.88	20.68
Anger*						
Pre	4.59	6.36	5.28	8.76	4.51	10.12
Post	15.49	19.81	31.96	23.76	3.31	7.58
Anxiety*						
Pre	15.33	16.84	15.26	16.90	18.45a	22.09
Post	42.71	23.57	31.44	22.45	13.49d	19.21
Sadness*						
Pre	12.43	16.11	10.54	16.17	10.27	19.02
Post	27.33	26.43	28.40	27.36	8.71	17.01
Relaxation*						
Pre	67.90	22.52	68.58	19.90	68.14	24.57
Post	34.45	20.87	42.94	24.47	72.98	25.44
Happiness*						
Pre	62.71	22.90	63.90	22.14	60.29	17.82
Post	46.76	22.86	48.54	25.10	62.80	18.12

Notes: Groups did not differ on any state measure baseline ($ps > .27$). All changes from pre to post manipulation within each condition were statistically significant ($ps \leq .05$), with the exception of changes in anger within the relaxation condition ($p = .08$). * indicates a significant group by time interaction, $p < .001$.

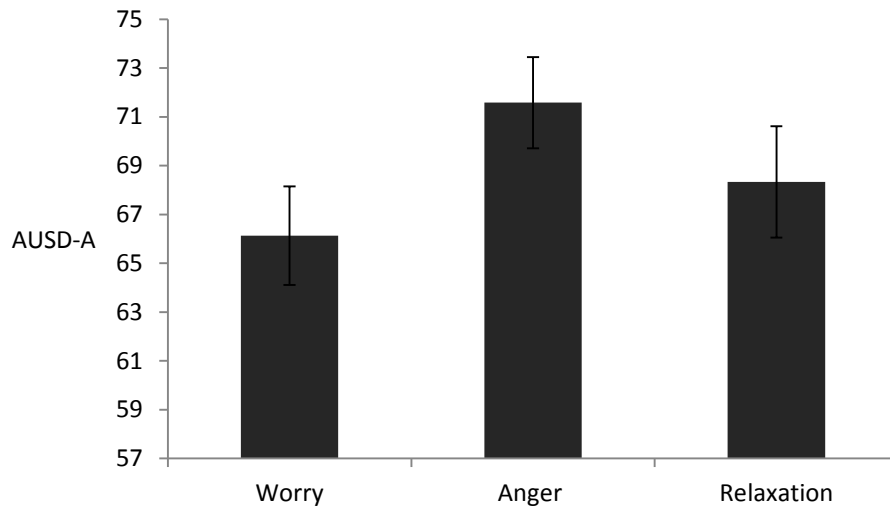
60.75, $p < .001$, $\eta_p^2 = .45$, anger, $F(2,147) = 33.99$, $p < .001$, $\eta_p^2 = .32$, and relaxation, $F(2, 147) = 46.97$, $p < .001$, $\eta_p^2 = .39$.

Follow-up comparisons demonstrated that participants in both the worry and the anger conditions experienced greater increases in worry, anxiety, anger, and sadness, as well as greater decreases in relaxation and happiness (all $ps < .001$), than participants in the relaxation condition. Participants in the worry and anger conditions did not significantly differ from each other on changes in relaxation ($p = .08$), happiness ($p = .86$), or sadness ($p = .47$); however as expected, participants in the worry induction condition experienced greater increases in worry ($p < .001$) and anxiety ($p = .003$) than those in the anger induction condition, whereas participants in the anger condition experienced greater increases in anger ($p < .001$) than those in the worry condition. These results suggest that the manipulation was successful in increasing current levels of worry in the worry induction condition, anger in the anger condition, and relaxation in the relaxation condition.

Effects of the Experimental Manipulation on Information Processing

To examine the impact of the manipulation on threat-related interpretation bias, we conducted a one-way factorial analysis of covariance (ANCOVA) with condition as the between-groups factor, STICSA-T scores as the covariate, and the AUSD-A as the outcome variable. Unexpectedly, there was no significant main effect of condition on the AUSD-A, $F(2, 146) = 2.05$, $p = .13$, $\eta_p^2 = .03$ (see Figure 1, panel A). Next, we examined the effects of the manipulation on attributions of intent towards negative situations using a one-way multivariate analysis of covariance (MANCOVA) with condition as the between-groups factor, STICSA scores as the covariate, and the SIP-AEQ variables (i.e., hostile and benign attributions) as the

A



B

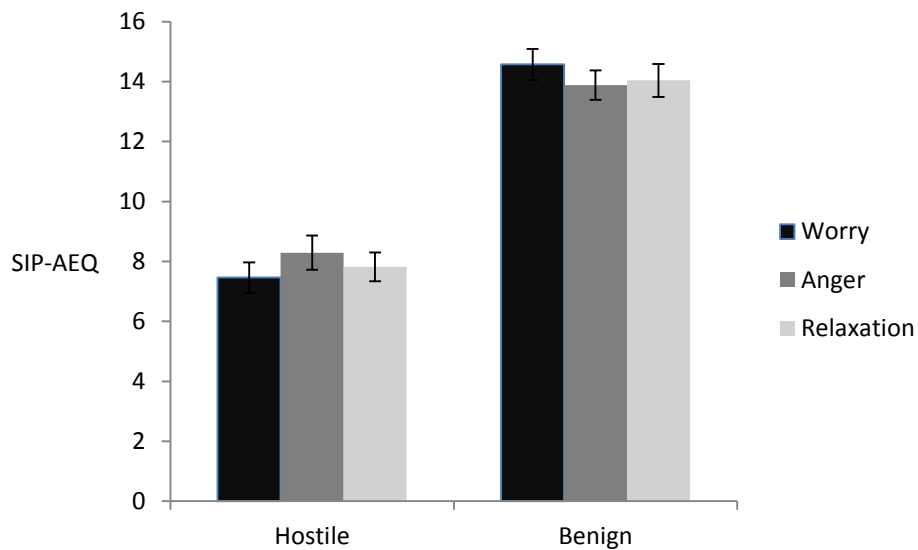


Figure 1. Means (standard errors) per experimental condition on the Ambiguous Unambiguous Situations Diary, Ambiguous subscale (A) and the Social Information Processing-Attribution and Emotional Response Questionnaire, Direct Hostile Intent and Benign Intent subscales (B). Means are adjusted for trait anxiety measured by the State Trait Inventory for Cognitive and Somatic Anxiety (STICSA-T).

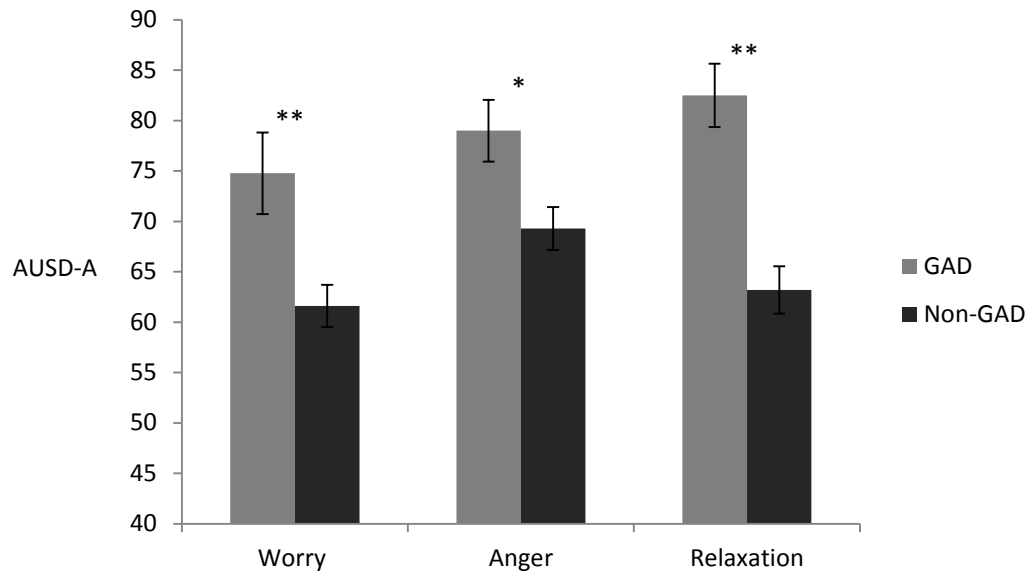
outcome variables. There was no main effect of condition on the combined SIP-AEQ variables, $\lambda = 0.98$, $F(4, 292) = .76$, $p = .55$, $\eta_p^2 = .01$ (see Figure 1, panel B). These results indicate that our hypotheses that anger and worry would increase threatening interpretations of ambiguous information and hostile attributions relative to relaxation were unsupported.

GAD and Information Processing

To examine the effects of GAD status and the interaction between GAD status and condition on threat-related interpretation bias, we conducted a 2X3 two-way ANOVA with GAD status (GAD-analogue or non-GAD) and condition (worry, anger, or relaxation) as between-groups factors, and the AUSD-A as the outcome variable. There was a main effect of GAD status on the AUSD-A, $F(1, 144) = 34.04$, $p < .001$, $\eta_p^2 = .19$, suggesting that the GAD-analogue group had greater negative interpretations of ambiguous situations ($M = 79.02$, $SD = 12.84$) than the non-GAD group ($M = 64.77$, $SD = 13.50$). However, there was no interaction between GAD status and condition on the AUSD-A, $F(2, 144) = 1.38$, $p = .25$, $\eta_p^2 = .02$ (see Figure 2, panel A). To examine the effects of the manipulation on a composite of hostile attributions of intent, we conducted a two-way multivariate analysis of variance (MANOVA) with condition and GAD status as the between-groups factors and the SIP-AEQ variables as the outcome variables. There was a main effect of GAD status on the combined SIP-AEQ variables, $\lambda = 0.85$, $F(2, 143) = 12.69$, $p < .001$, $\eta_p^2 = .15$. However, there was no interaction between GAD status and condition on the combined SIP-AEQ variables, $\lambda = 0.99$, $F(4, 286) = .52$, $p = .73$, $\eta_p^2 = .01$ (see Figure 2, panel B).

A discriminant function analysis was conducted to examine the relative contribution of each SIP-AEQ variable to GAD group status. The correlations between the predictors and the discriminant function suggest that elevated hostile attributions accounted for more variance ($r =$

A



B

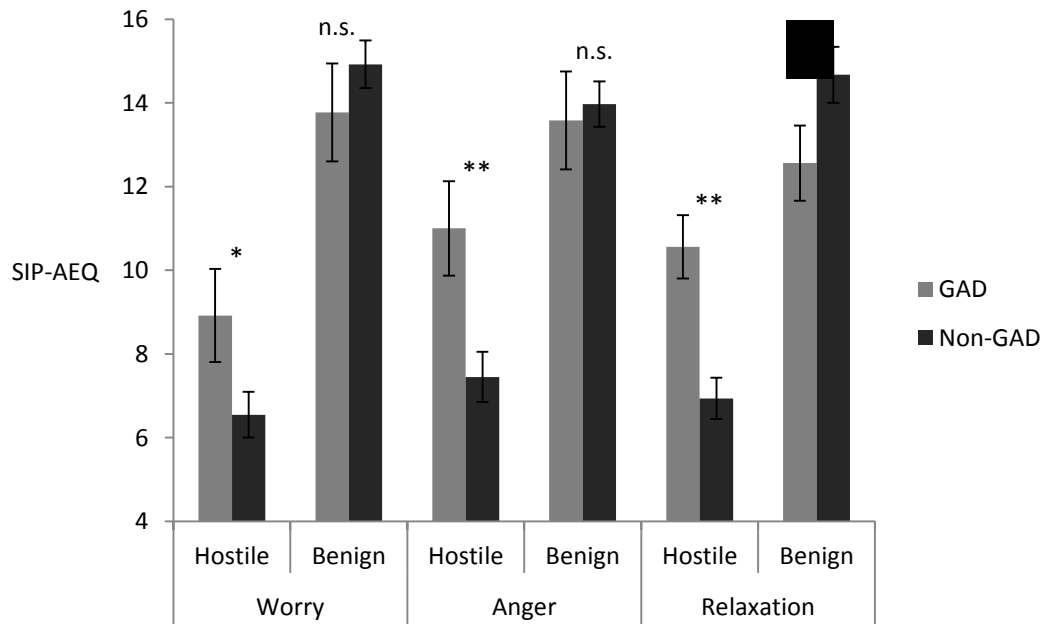


Figure 2. Means (standard errors) per GAD group and experimental condition on the Ambiguous Unambiguous Situations Diary, Ambiguous subscale (A) and the Social Information Processing-

Attribution and Emotional Response Questionnaire, Direct Hostile Intent and Benign Intent subscales (B). Sample sizes per condition for the GAD-analogue group are $n = 13$ for worry, $n = 12$ for anger, and $n = 16$ for relaxation; sample sizes per condition for the non-GAD group are $n = 38$ for worry, $n = 38$ for anger, and $n = 33$ for relaxation. * $p < .05$, ** $p < .01$, † $p < .10$.

.99) in GAD group status than lower benign attributions ($r = -.37$). Using Jackknife classification, the discriminant function correctly classified 68% ($n = 102$) of individuals into their respective GAD groups, with 70.70% ($n = 29$) correctly classified as GAD-analogue and 67% ($n = 73$) correctly classified as non-GAD. Collectively, these results suggest that relative to the non-GAD group, the GAD-analogue group reported greater interpretations of threat regarding ambiguous situations and attributed greater hostile intent to the provocateurs of the aversive situations, independent of the emotional induction.

Discussion

The goals of the study were to compare the effects of induced worry, anger, and relaxation on threatening interpretations of ambiguous situations and hostile attributions of ambiguous intent in aversive situations. Contrary to our hypotheses, there were no effects of the experimental conditions on interpretive or attributional styles. To further explore the association between GAD and anger, we also examined the information processing style of GAD analogues. The GAD-analogue group interpreted ambiguous situations as more threatening and attributed greater hostile intent to the ambiguous aversive situations than the non-GAD group. However, in contrast to our hypothesis, there were no interactions between GAD status and condition on the information processing tasks.

Many studies have reported that GAD symptoms are associated with a hyper-vigilance for threat in ambiguous situations (e.g., Eysenck et al., 1987; Mathews et al., 1989); thus our finding that the GAD group reported more concern over ambiguous diary passages is consistent with previous literature. This is the first study that we are aware of, however, to examine the effects of GAD symptoms on hostile attributions. We found that GAD analogues attributed greater hostile intent to provocateurs of aversive situations where *intent* of the provocateur was

ambiguous. Therefore, the tendency for individuals with GAD to resolve ambiguity in a negative manner extends to hostile attributions. This may be due to greater rigidity in negative information processing styles in GAD analogues than in less anxious individuals. Importantly, this is a stable, intransient effect that is largely unaffected by current affective state, given that information processing style was unaffected by the emotional induction.

Methodological constraints may partially explain the null findings regarding the effect of the manipulation on information processing. One possibility is that participants may not have wanted to report hostile attributions due to social desirability concerns. Another possibility is that the effects of the worry and anger inductions dissipated over the course of the administration of our information processing tasks. Participants completed a set of VASs prior to these tasks, and the tasks together took between 15 to 20 minutes to complete. As such, it is possible that the effects of our relatively mild emotion inductions were reduced over time. Perhaps more powerful emotion inductions are necessary to have a longer lasting influence on information processing (e.g., Barazzone & Davey, 2009; Deschênes et al., 2014).

Several limitations to our study are noteworthy. First, we used a self-report measure to assess GAD diagnostic criteria. Although our GAD group had greater worry and trait anxiety levels than our non-GAD group, these individuals may differ from a clinical sample in the amount of distress resulting from their GAD symptoms. Future research should aim to replicate this finding with a clinician-diagnosed sample of individuals with GAD. Second, although the manipulation checks suggested that the worry, anger, and relaxation inductions were successful, it is possible that the effects of the manipulation may have been inflated due to demand characteristics. Stated differently, participants may have felt compelled to report greater increases in the respective states following explicit instructions to do so. Despite potentially

confounding impact of demand characteristics, experimental research has suggested that effects of mood inductions using explicit instructions (i.e., not masking the true purpose) are not solely artifacts of demand characteristics and can indeed lead to changes in affect (e.g., Polivy & Doyle, 1980). Third, although our manipulation checks demonstrated specificity of the manipulation on subsequent psychological states measured by the VAS, it is important to note that other psychological states (e.g., sadness and happiness) not directly targeted were also affected by the manipulations (albeit to a lesser extent).

To conclude, although we did not find an effect of induced worry, anger, or relaxation on interpretations of ambiguous situations or on attributions of intent to aversive situations, we found that GAD analogues reported greater threatening interpretations of ambiguous situations and attributed greater hostile intent to aversive situations than less anxious individuals. These findings suggest that individuals with GAD not only interpret ambiguous situations as threatening, but also interpret ambiguous intent as hostile. Treatments aiming to reduce threatening interpretations of ambiguous information have been developed and seem like promising techniques to reduce symptoms of anxiety (see Beard, 2011, for a review); our results suggest that attempts to reduce hostile attributions may also lead to GAD symptom reduction.

CHAPTER 7:

GENERAL DISCUSSION

The goals of this program of research were to further our understanding of the associations between anger and GAD by 1) examining the relative contributions of various forms of anger to symptoms of GAD, 2) examining the effects of induced state anger on cognitive vulnerabilities and processes associated with GAD, 3) examining the effects of induced worry and anger on cognitive processes associated with GAD and high trait anger, and 4) examining whether GAD analogues report greater hostile attributions of intent than do less anxious individuals.

Summary of Findings

Overall, we found that anger is positively associated with symptoms of GAD cognitive vulnerabilities and processes, including intolerance of uncertainty and threatening interpretations of ambiguous information. We also found that GAD is associated with hostile attributions of ambiguous intent. These cognitive processes may provide a potential pathway linking GAD and elevated anger. Results from study 1 demonstrated that compared to less anxious participants, GAD analogues reported high levels of numerous forms of anger. Specifically, elevated trait anger, anger suppression, and hostility differentiated GAD-analogue participants from less anxious participants. Within the GAD analogues, anger suppression and hostility uniquely predicted greater GAD symptom severity. These findings are consistent with the broader literature on anger and anxiety disorders (e.g., Bridewell & Chang 1997; Erdem et al., 2008; Erwin et al., 2003; Hawkins & Cogle, 2011; Meffert et al., 2008; Moscovitch et al., 2008; Orth & Wieland 2006), and together suggest that many forms of anger can be problematic for anxious individuals.

Results from study 2 indicated that state anger predicted increases in intolerance of uncertainty, a cognitive vulnerability for GAD. Of the two higher order beliefs in the construct of intolerance of uncertainty (Sexton & Dugas, 2009), only the belief that uncertainty is unfair and spoils everything (and not the belief that uncertainty has negative self-referent and behavioural implications) was impacted by increases in state anger. In addition, state anger predicted increases in negative interpretations of ambiguous information, a cognitive process underlying GAD. These findings are in line with previous research demonstrating that intolerance of uncertainty mediates the relationship between anger and GAD symptoms (Fracalanza et al., 2014), and that state anger increases the likelihood of reporting threatening interpretations of ambiguous information (Barazzone & Davey, 2009). Together, these findings provide preliminary support for the notion that cognitive factors partially explain the association between anger and symptoms of GAD.

Results from study 3 failed to support the hypothesis that state worry and state anger, compared to relaxation, would increase threatening interpretations of ambiguous situations and would lead to greater hostile attributions of ambiguous intent. However, results showed that GAD analogues reported greater threatening interpretations and greater hostile attributions than less anxious participants. These associations were independent of induced affective state. Results from this study are consistent with previous research demonstrating interpretation biases for threat in GAD (e.g., Eysenck et al., 1987; Mathews et al., 1989) and contribute to this literature with the novel finding that GAD symptoms are also associated with hostile attributions, a cognitive process associated with high trait anger (Owen, 2011). Therefore, it seems that GAD symptoms relate to information processing biases across anxiety- and anger-provoking ambiguous situations, independently from affective state.

A noteworthy difference emerged from the results of studies 2 and 3. Study 2 demonstrated that induced state anger impacted the likelihood of interpreting ambiguous information as threatening, whereas study 3 found no effect of induced state anger on interpretation bias. Methodological differences in state anger inductions may account for this difference. The manipulation in study 2 involved anger about a present situation (i.e., a hostile experimenter), whereas the manipulation in study 3 consisted of an anger recall task. It is possible that the anger recall task elicited anger rumination to a greater extent than the emotion of anger and was therefore not powerful enough to impact cognitive processes. Although additional research is needed to address this inconsistency, the evidence from study 2 as well as from a previous study suggests that state anger does potentiate threatening interpretations of ambiguous information. Specifically, Barazzone and Davey (2009) paired anger-eliciting music with anger-inducing vignettes and found an increase in threatening interpretations of ambiguous homophone words compared to a control condition. This type of manipulation may have been similar in intensity to the manipulation used in study 2.

Future Directions

Findings from the current program of research suggest that GAD and anger comorbidity warrant further investigation. We examined the associations between anger and GAD with a series of correlational and experimental studies in non-clinical samples (i.e., no clinician-rated measures of GAD symptomology were included); an important future direction would be to examine these effects in clinician-diagnosed samples of individuals with GAD, as well as in samples of individuals seeking help for elevated anger. In addition, previous studies have demonstrated that elevated anger interferes with the successful psychological treatment of SAD (Erwin et al., 2003) and PTSD (Forbes et al., 2008). Elevated anger may also interfere with the

treatment of GAD; future research should examine the effects of anger on outcomes of psychological treatments for GAD. Anger may interfere with the therapist-client alliance, homework compliance, or treatment credibility, all of which contribute to treatment progress (DiGiuseppe, Tafrate & Eckhardt, 1994). Similarly, elevated anger can lead to the experience of interpersonal problems (e.g., Scherer & Wallbott, 1994), and interpersonal problems, in turn, have been shown to predict poor response to cognitive-behavioural treatment for GAD (Borkovec, Newman, Pincus, & Lytle, 2002). Future research should aim to examine whether addressing anger during the treatment of GAD decreases interpersonal problems, thus promoting positive treatment outcomes.

Further experimental research on the comorbidity between GAD and problematic anger would also be beneficial. For instance, it is currently unclear whether anger triggers worry and anxiety, whether worry and anxiety trigger anger, or some combination of both. Our results show that induced state anger directly increased negative beliefs about uncertainty and negative interpretations of ambiguous information; however whether these cognitive factors in turn produced greater worry is unclear. It is also unknown whether state worry or anxiety directly increases hostile cognitive processes, and whether this leads to greater state anger. Therefore, future studies should continue to investigate these associations with mediational study designs and experimental research. Experimental research in clinical psychology provides the ability to study clinical phenomena in highly controlled settings (Davey, 2003), and therefore notably contributes to our understanding of the nature of psychological distress.

This program of research examined specific pathways linking anger and GAD, although a number of other possible associations may exist and warrant future investigation. One possible avenue for identifying putative pathways is the examination of previous research on GAD

comorbidity. GAD is often comorbid with other psychological disorders, with comorbidity rates of up to approximately 90% (Blanco et al., 2014). Explanations have been provided for the comorbidity between GAD and depressive disorders, and include GAD and depression reflecting different expressions of a common underlying vulnerability (e.g., Clark, Waltson, & Mineka, 1994) or the presence of a higher order construct such as negative affect (Clark & Watson, 1991). Similar processes could also explain GAD and elevated anger comorbidity. For instance, a higher order negative affectivity factor (Clark & Watson, 1991) or shared biological vulnerabilities for hypervigilance and excessive responses to stressors (Barlow, 2002) may also function as risk factors for both excessive anxiety and anger. However, although the propensity to experience negative affect contributes to the experience of both emotional states, it is unlikely that negative affect explains all variability due to the distinct behavioural, motivational, and physiological profiles associated with anger and anxiety.

Conceptual models of GAD provide another avenue for examining potential pathways linking GAD and anger. Indeed, one potential explanation for this association relates to the contrast avoidance model of GAD proposed by Newman and Llera (2011). The model suggests that individuals with GAD are averse to large shifts in emotional experiences, and they therefore tend to worry about worst possible outcomes to various situations to maintain a state of constant negativity. By sustaining a negative affective state, the distress associated with large changes in mood is reduced. Thus, having a lowered threshold for becoming angry may also serve to maintain negativity and avoid large shifts in emotionally contrasting states. Similarly, another conceptual model suggests that individuals with GAD tend to experience heightened emotional reactivity and poor emotional regulation (Macatee & Cougle, 2013; Mennin et al., 2005), and therefore elevated anger in GAD may be a consequence of emotional hyperactivity. Taken

together, a number of mechanisms could explain the association between anger and GAD symptoms, in addition to the cognitive factors examined in the current series of studies.

Implications

Although many theories suggest that cognitive processes, such as interpretations and appraisals of events, elicit emotions (Clark & Beck, 2010; Lazarus, 1991), our findings are consistent with theories proposing that emotions also affect cognitive processes (Schwarz & Clore, 2007; Zajonc, 1984). Conceivably, intricate combinations of these theoretical perspectives likely explain the associations between anger and GAD symptoms. For instance, increased anger may potentiate perceptions of threat and danger in the environment, which in turn increases anxiety. Likewise, anxiety may potentiate perceptions of hostile intent in others, which in turn increases anger. Together, cognitive processes seem to be involved in the elicitation of worry and anxiety as well as anger, and the elicitation of these emotions may consequently feed back into biased cognitive processing. Therapeutically targeting the underlying cognitive processes may alleviate problematic anger as well as excessive worry and anxiety in individuals with GAD. The current series of studies also demonstrated that covert types of anger (i.e., anger suppression, hostility, hostile attributions) are associated with GAD symptom severity. Given the lack of overt behavioural manifestations of anger, these findings suggest that clinicians treating clients with GAD should not only assess overt anger, but also assess covert anger that may otherwise go unreported yet interfere with treatment success.

To conclude, individuals with GAD seem to be prone to experience elevated anger, hostility, and anger suppression. Cognitive vulnerabilities, such as negative beliefs about uncertainty, and information-processing biases, such as increased perceptions of threat and hostility in the environment, may increase the risk of developing or maintaining symptoms of

GAD and high trait anger. Although further research is needed, assessing and addressing anger-related difficulties in individuals with GAD could present an important avenue for refining treatment protocols for GAD.

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Appendix A

Consent Form for Study 1

CONSENT TO PARTICIPATE IN RESEARCH

This is to state that I agree to participate in a program of research being conducted by Sonya Deschenes, under the supervision of Dr. Michel Dugas in the Department of Psychology at Concordia University. Sonya Deschenes may be reached at 514-848-2424 ext. 2229 or by email at so_desch@live.concordia.ca.

A. PURPOSE

I have been informed that the purpose of the research is to investigate the relationship between anger, beliefs about perfection and uncertainty, and various symptoms of anxiety.

B. PROCEDURES

I have been informed that I will first be asked to read and sign this consent form. Next, I will be asked to fill out a general information form and 9 questionnaires designed to assess different dimensions of anger, beliefs about perfection and uncertainty, and various symptoms of anxiety. These will be completed in a room with up to eight people. The completion of this study will take approximately 45 minutes. I will receive 1 participant pool credit as compensation for my participation. Identifying information, which consists of my consent form and the lab copy of my participation receipt, will be stored separately from my data in the Anxiety Disorders Laboratory and each will be kept under lock and key. Code numbers alone will be used to identify the questionnaires. I understand that my participation in the study, and the information I provide, are strictly confidential. I understand that I am free to discontinue my participation in the study at any time without negative consequences.

If I am asked to return for a retesting session held approximately 4 weeks later, I will complete three of the questionnaires which I completed during the original testing session. Participation in the retesting session will take approximately 15 minutes and I will receive another participant pool credit for attending the retesting session.

C. RISKS AND BENEFITS

There is minimal risk associated with this study, however, it is possible that some of the questions I am about to answer may temporarily cause slight uneasiness (possibly, by causing me to reflect on my

difficulties). The questionnaires in this study have been used in previous research and discomfort is rare.

If, for some reason, I experience uneasiness or discomfort during testing, I should discuss it with the experimenter.

I will benefit from my participation in this study in that I will receive credit for the Department of Psychology participant pool and will contribute to our understanding of the relationship between anger, beliefs about perfection and uncertainty, and anxiety.

D. CONDITIONS OF PARTICIPATION

- I understand that I am free to withdraw my consent and discontinue my participation at anytime without negative consequences.
- I understand that my participation in this study is ANONYMOUS (i.e., my participation will be tracked to provide course credit, but it will be impossible for my data to be linked to my identity).
- I understand that the data from this study may be published.

I HAVE CAREFULLY STUDIED THE ABOVE AND UNDERSTAND THIS AGREEMENT. I
FREELY CONSENT AND VOLUNTARILY AGREE TO PARTICIPATE IN THIS STUDY.

NAME (please print) _____

SIGNATURE _____

If at any time you have questions about the proposed research, please contact the study's Principal Investigator, Dr. Michel Dugas of the Department of Psychology at Concordia University at 514-848-2424 ext. 2215 or by email at Michel.Dugas@concordia.ca.

If at any time you have questions about your rights as a research participant, please contact the Research Ethics and Compliance unit, Concordia University, Kyla Wiscombe, at (514) 848-2424 x2425 or by email at kwiscomb@alcor.concordia.ca.

Appendix B

Scrambled Sentence Task

(Study 2)

Please read the following instructions carefully:

Each scrambled sentence below contains 6 words. Your task is to rearrange the words in each scrambled sentence to form a meaningful statement. Use only 5 of the 6 words in each scrambled sentence to form each new statement. Indicate the proper order of the words in your new statement by placing a number over each of the five words indicating the proper order.

For example, if you see the following 6 words:

1 2 3 4 5 6
man the wearing glasses is shoes

You may choose to rearrange these words into the following meaningful 5-word statement:

2 1 5 3 6
the man is wearing shoes

Each sentence can be rearranged into more than one statement, but you should form only **one** statement from each scrambled sentence. The new statements you form should be statements and not questions. Note that contractions (e.g., I'll, I'm, can't) are considered one word. You have 4 minutes to unscramble as many sentences as possible. **Work as quickly and as accurately as you can.**

Version A

1. disease always to rarely vulnerable I'm
2. be likely I'll unlikely attacked it's
3. are superiors my rarely usually impressed
4. changes rarely disease suggest bodily usually
5. succeed unlikely to academically I'm likely
6. vulnerable I'm less others than more
7. partner's improve worsen health will my
8. will me my won't colleagues respect
9. will my be harmed won't partner
10. very serious most are injuries minor

11. social make I can mistakes can't
12. children me my will won't respect
13. stability ruin cause decisions my financial
14. my rated well is poorly work
15. schoolwork my strengths weaknesses reveals challenging
16. me my won't leave partner will
17. catastrophic be arguments resolved family can
18. will I enough have won't money
19. be mild my symptoms will severe
20. will date disappointed my be pleased

Version B

1. learning mistakes failure lead can to
2. like won't people probably me will
3. will friend sick won't get my
4. danger rarely in often children are
5. relationship won't romantic will last my
6. serious my usually are injuries minor
7. get illnesses better will most worse
8. rarely possible stability is financial always
9. can financial can't fixed mistakes be
10. families stay won't will most together
11. it's I'll promoted unlikely be likely
12. vulnerable very family is my safe

13. my me family boring finds exciting
14. strengthen marriage end a can disagreements
15. rarely usually think teachers I'm intelligent
16. strengths my notice others will faults
17. risk am often I at rarely
18. unlikely it's sick likely I'll get
19. serious my will minor be illnesses
20. notice colleagues faults my skills my

Appendix C

Consent Form for Study 2 (pre-study)

CONSENT FORM TO PARTICIPATE IN RESEARCH

This is to state that I agree to participate in a program of research being conducted by Sonya Deschenes, under the supervision of Dr. Michel Dugas of the Department of Psychology at Concordia University.

A. PURPOSE

I have been informed that the purpose of this research is to examine personality traits and how they relate to physiology during the performance of various tasks, including mental challenges and worry-related tasks.

B. PROCEDURES

After reading and signing this consent form, you will first be asked to complete a series of questionnaires. Following this, a heart rate monitor will be attached to you, and you will complete a series of tasks. These tasks include an anagram task, a task where you will unscramble words to make a sentence, a worry-related interview, as well as complete other questionnaires.

C. RISKS, SECONDARY EFFECTS AND DISADVANTAGES

It may be possible that certain measures or tasks temporarily cause slight uneasiness (possibly, by causing you to reflect on your difficulties). However, these measures and tasks have been used in previous research and discomfort is rare. If, for some reason, you should experience uneasiness or discomfort during testing, please discuss it with the experimenter.

D. COMPENSATION

You will receive a compensation of 2 Participant Pool points, as well as an entry in a draw for a \$50 cash prize, for your participation in this study.

E. CONFIDENTIALITY

All information collected from you throughout the course of this study will remain confidential, within the limits defined by law, and you will be identified solely by a numeric code. No publication or presentation resulting from this study will contain any identifying information.

F. RIGHTS AS A PARTICIPANT

If you accept to participate in this study, you are not giving up any of your rights or liberties to the researchers, funding organizations (Canadian Institutes of Health Research), nor are those involved released of their legal and professional responsibilities.

G. VOLUNTARY PARTICIPATION AND WITHDRAWAL FROM THE STUDY

Your participation in this study is voluntary. Therefore you are free to refuse to participate. You can also withdraw from the study at any moment without negative consequences (i.e., you will still be compensated for your time).

H. CONDITIONS OF PARTICIPATION

- I understand that I am free to withdraw my consent and discontinue my participation at any time without negative consequences.
- I understand that my participation in this study is anonymous.
- I understand that the data from this study may be published.

I HAVE CAREFULLY STUDIED THE ABOVE AND UNDERSTAND THIS AGREEMENT.

I FREELY CONSENT AND VOLUNTARILY AGREE TO PARTICIPATE IN THIS STUDY

NAME (please print) _____

SIGNATURE _____

Date _____

If at any time you have questions about the proposed research, please contact the study's Principal Investigator Dr. Michel Dugas, Department of Psychology, (514)848-2424 ext. 2215, Michel.dugas@concordia.ca.

If at any time you have questions about your rights as a research participant, please contact the Research Ethics and Compliance Advisor, Concordia University, 514.848.2424 ex. 7481 ethics@alcor.concordia.ca

Appendix D

Consent Form for Study 2 (post-study)

CONSENT FORM TO PARTICIPATE IN RESEARCH

Post-Study Consent

This is to state that I have been made aware of the true nature of this study, and that I agree to have my data included in the final sample of the study (conducted by Sonya Deschenes and Dr. Michel Dugas of the Department of Psychology at Concordia University).

A. PURPOSE

I have been informed that the true purpose of this study is to examine the impact of trait and state anger on heart rate, beliefs about uncertainty, interpretations of ambiguous situations, and worry level.

I have been informed that I was either in the anger induction condition or the neutral condition. The anger induction consisted of having the research assistant interact with me in a negative way prior to and during the anagram task, and her interaction with me was by no means related to my actual performance on the task. Finally, if I was in the anger induction condition, I have been told that the researchers aimed to increase my state anger in order to determine whether anger has a direct effect on heart rate, intolerance of uncertainty, interpretations of ambiguous situations, and levels of worry. It was important to conceal the true purpose of the study (by claiming that the goal of the study was related to personality and physiology during different tasks) to ensure the success of the manipulation.

I have been informed that should I wish that my data not be retained for this study, I may indicate so by informing the experimenter.

C. RISKS, SECONDARY EFFECTS AND DISADVANTAGES

It may be possible that certain measures may have caused slight uneasiness temporarily (possibly, by causing you to reflect on your difficulties). However, these measures have been used previously many times and discomfort is rare. If you should, for some reason, experience uneasiness or discomfort following the study, please discuss it with the evaluator/experimenter.

D. COMPENSATION

You will receive a compensation of 2 Participant Pool points, as well as an entry in a draw for a \$50 cash prize, for your participation in this study.

E. CONFIDENTIALITY

All information collected from you throughout the course of this study will remain confidential, within the limits defined by law, and you will be identified solely by a numeric code. No publication or presentation resulting from this study will contain any identifying information.

F. RIGHTS AS A PARTICIPANT

If you accept to participate in this study, you are not giving up any of your rights or liberties to the researchers, funding organizations (Canadian Institutes of Health Research), nor are those involved released of their legal and professional responsibilities.

G. VOLUNTARY PARTICIPATION AND WITHDRAWAL FROM THE STUDY

Your participating in this study is voluntary. Therefore you are free to refuse to participate. You can also withdraw from the study at any moment, without having to give an explanation when you make your decision known to the evaluator/experimenter.

H. CONDITIONS OF PARTICIPATION

- I understand that I am free to withdraw my consent and discontinue my participation at anytime without negative consequences.
- I understand that my participation in this study is confidential.
- I understand that the data from this study may be published.

I HAVE CAREFULLY STUDIED THE ABOVE AND UNDERSTAND THIS AGREEMENT. I FREELY CONSENT AND VOLUNTARILY AGREE TO PARTICIPATE IN THIS STUDY.

NAME (please print) _____

SIGNATURE _____

Date _____

If at any time you have questions about the proposed research, please contact the study's Principal Investigator

Dr. Michel Dugas, Department of Psychology, (514)848-2424 ext. 2215, Michel.dugas@concordia.ca.

If at any time you have questions about your rights as a research participant, please contact the Research Ethics and Compliance Advisor, Concordia University, 514.848.2424 ex. 7481 ethics@alcor.concordia.ca

Appendix E

Consent Form for Study 3 (pre-study)

CONSENT FORM TO PARTICIPATE IN RESEARCH

“Thoughts and Interpersonal Behaviour”

I understand that I have been asked to participate in a research project being conducted by Sonya Deschênes of the Psychology department of Concordia University (so_desch@live.concordia.ca; (514)848-2424 ext.2246), under the supervision of Jean-Philippe Gouin of the Psychology department of Concordia University (jp.gouin@concordia.ca, (514) 848-2424 ext. 7538).

A. PURPOSE

I have been informed that the purpose of this research is to examine the relations between thoughts, interpersonal behaviour, and cardiac activity.

B. PROCEDURES

I understand that I will be asked to participate in a 90-minute laboratory visit. I understand that during this visit, after reading and signing this consent form, I will be asked to complete a series of questionnaires about various personal characteristics such as my mood, my worries, and various thoughts about myself. Next, I will be asked to wear a heart rate monitor that will record my heart rate during different activities. I understand that I will first be asked to sit quietly for about 5 minutes. Following this, I will be asked to complete different tasks that will require me to either think about a recent or potential future event, or to relax, for approximately 5 minutes. I understand that I will next complete computerized interpersonal behaviour tasks, and will finally be asked to rest for an additional 5 minutes.

C. RISKS, SECONDARY EFFECTS AND DISADVANTAGES

I understand that it may be possible that certain tasks temporarily cause slight uneasiness (possibly, by causing you to reflect on past or potential future difficulties). However, these measures and tasks have been used in previous research and discomfort is rare. I understand that if

I experience strong anxiety in response to the task, I should discuss this with the experimenter. I understand that I may also experience some mild skin irritation from wearing the heart monitor. I understand that I will receive participation credits for the Department of Psychology's Participant Pool program after completing this research study.

D. CONDITIONS OF PARTICIPATION

- I understand that I am free to withdraw my consent and discontinue my participation at anytime without negative consequences.
- I understand that my participation in this study is CONFIDENTIAL (i.e., the researcher will know, but will not disclose my identity)
- I understand that the data from this study may be published.

I HAVE CAREFULLY STUDIED THE ABOVE AND UNDERSTAND THIS AGREEMENT. I FREELY CONSENT AND VOLUNTARILY AGREE TO PARTICIPATE IN THIS STUDY.

NAME (please print) _____

SIGNATURE _____

If at any time you have questions about the proposed research, please contact the study's Principal Investigator, Sonya Deschênes, M.A., Department of Psychology of Concordia University, (514)848-2424 ext. 2246, so_desch@live.concordia.ca, or contact Jean-Philippe Guoin, Ph.D., Department of Psychology of Concordia University, (514)848-2424 ext. 7538, jp.gouin@concordia.ca.

If at any time you have questions about your rights as a research participant, please contact the Research Ethics and Compliance Advisor, Concordia University, 514.848.2424 ex. 7481
ethics@alcor.concordia.ca

Appendix F

Consent Form for Study 3 (post-study)

CONSENT FORM TO PARTICIPATE IN RESEARCH

“EXAMINING THE IMPACT OF WORRY ON HOSTILE COGNITIONS”

Post-Study Consent

This is to state that I have been made aware of the true nature of this study, and that I agree to have my data included in the final sample of the study (conducted by Sonya Deschenes of the Department of Psychology at Concordia University).

A. PURPOSE

I have been informed that the true purpose of this study is to examine the impact of worry and state anger on heart rate and hostile attribution bias, a cognition associated with anger.

B. PROCEDURES

I have been informed that I was either in the worry induction condition, the anger induction condition, or the relaxation (control) condition. It was important to conceal the true purpose of the study (by claiming that the goal of the study was related to thoughts and interpersonal behaviour) to ensure the success of the manipulation and dependent measures.

I have been informed that should I wish that my data not be retained for this study, I may indicate so by informing the experimenter.

C. RISKS, SECONDARY EFFECTS AND DISADVANTAGES

I understand that it may be possible that certain tasks temporarily cause slight uneasiness (possibly, by causing you to reflect on past or potential future difficulties). However, these measures and tasks have been used in previous research and discomfort is rare. I understand that if I experience strong anxiety in response to the task, I should discuss this with the experimenter. I understand that I may also experience some mild skin irritation from wearing the heart monitor. I

understand that I will receive participation credits for the Department of Psychology's Participant Pool program after completing this research study.

D. CONDITIONS OF PARTICIPATION

- I understand that I am free to withdraw my consent and discontinue my participation at any time without negative consequences.
- I understand that my participation in this study is CONFIDENTIAL (i.e., the researcher will know, but will not disclose my identity)
- I understand that the data from this study may be published.

I HAVE CAREFULLY STUDIED THE ABOVE AND UNDERSTAND THIS AGREEMENT. I FREELY CONSENT AND VOLUNTARILY AGREE TO PARTICIPATE IN THIS STUDY.

NAME (please print) _____

SIGNATURE _____

If at any time you have questions about the proposed research, please contact the study's Principal Investigator, Sonya Deschênes, M.A., Department of Psychology of Concordia University, (514)848-2424 ext. 2246, so_desch@live.concordia.ca, or contact Jean-Philippe Gouin, Ph.D., Department of Psychology of Concordia University, (514)848-2424 ext. 7538, jp.gouin@concordia.ca.

If at any time you have questions about your rights as a research participant, please contact the Research Ethics and Compliance Advisor, Concordia University, 514.848.2424 ex. 7481
ethics@alcor.concordia.ca