THE ABILITY TO SHIFT EMOTIONS IN INDIVIDUALS WITH GENERALIZED ANXIETY DISORDER

A Dissertation in Psychology

by

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ABSTRACT

This thesis investigated the specific nature of emotional deficits in generalized anxiety disorder (GAD). Previous research has suggested that individuals with GAD experience emotional hyperreactivity and emotion dysregulation, which could interfere with their ability to respond appropriately to changing emotional stimuli. To test this notion, participants with GAD and healthy, control participants watched fear- and sadness-eliciting film clips in counter-balanced order, each followed by a recovery period. They rated their emotions before and after every clip and physiological arousal was recorded throughout. At baseline, participants with GAD reported greater fear and sadness, but did not differ from the control group in their physiology. Participants with GAD exhibited hyperreactivity in response to each emotional film clip in terms of their self-reported emotions when fear was elicited first. The physiological findings indicated that participants with GAD demonstrated a blunted emotional response to the first emotion-eliciting film clip and heightened reactivity to the second film clip with no return to baseline levels of physiological arousal during the recovery period, regardless of which emotion was elicited first. The relation between these findings and overall deficits in reactivity and regulation are discussed, including implications for the theoretical underpinnings of GAD.
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Chapter 1
INTRODUCTION

Emotional processes have been established as critical in understanding the etiology and maintenance of many psychological disorders (Berenbaum, Raghavan, Le, Vernon, & Gomez, 2003; Kring & Bachorowski, 1999; American Psychiatric Association, 2000). According to the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV), the vast majority of diagnoses include specific criteria that refer to emotional dysregulation, overabundance of negative affect, lack of positive affect, or other emotion-related problems (e.g., impulsivity).

Accordingly, empirical studies considering the impact of psychopathology on emotional processes, or vice versa, have grown in number over the decades. It has generally been observed that different types of psychopathology result in problems such as an inability to regulate affective states, over- or under-reactivity to emotional stimuli, or pervasive negative affect (Berenbaum et al., 2003). However, there has been less research focusing on the overlap between these deficits and how this overlap may impact individuals experiencing specific forms of psychopathology, such as anxiety disorders, where these deficits are pronounced.

Anxiety disorders involve a predominance of negative affect, particularly fear and anxiety (Barlow, 2003). The repeated experience of fear or other negative emotions in response to certain internal or external experiences leads to a state of anxious apprehension, wherein an individual anticipates that similar, bad events will occur in the future. Individuals with anxiety disorders respond to this apprehension with avoidance of or worry regarding these real or imagined threats. For instance, generalized anxiety disorder (GAD) is characterized by pervasive worry about a range of topics and associated symptoms of tension and anxiety (APA, 2000). It is a prevalent anxiety disorder, with a 12-month prevalence rate of 3.1% and a lifetime
prevalence rate of 5.1%, and is difficult to treat effectively (see Wittchen, 2002 for a review). Because GAD is marked by disturbances in both emotional reactivity and emotion regulation, this disorder is an appropriate subject for studying the confluence of these deficits.

One method for understanding how or why emotions play such a critical role in psychopathology is through the use of a functionalist approach. The functionalist theory proposes that emotions serve the adaptive purpose of providing information about the environment to the individual so that he or she can react as needed (Frijda, 1988; Keltner & Gross, 1999). Thus, dysregulated emotion in psychopathology may disrupt the capacity for an individual to accurately process important information from the environment and react accordingly, which likely perpetuates or exacerbates a state of distress. This disruption appears to be most related to the experience of negative emotion, whereas the chronic experience of positive emotions tends to be associated with enhanced psychological functioning (Cohn, Fredrickson, Brown, Mikels, & Conway, 2009). Thus, emotional processes such as emotional reactivity and regulation may be particularly relevant to understanding the dysfunction experienced in disorders such as GAD that are characterized by uncontrollable negative affect (Barlow, 2003). However, it is not yet clear how deficits in these areas interact with each other to impact the course of an emotional response. This gap in the literature may be attributed to the complexity of separating processes related to emotional reactivity or generation from emotion regulation (Gross & Barrett, 2011; Gross, Sheppes, & Urry, 2011), making it difficult to determine precisely where deficits occur. The focus of this thesis is to consider the role of deficits in emotional reactivity and deficits in emotion regulation among individuals with GAD and how these deficits may decrease the capacity for individuals to adaptively shift between emotional states.
**Emotions and Emotion Functions**

An emotion is a relatively short-term, subjective experience that occurs in response to an internal or external stimulus and typically invokes changes in physiological state and behavioral outcomes (Frijda 1988; Gross, 2007; Keltner & Gross, 1999). According to many current theoretical approaches, most of which are based on William James’s (1884, 1894) early conceptualizations of emotion, emotions are essentially response tendencies (Frijda, 1986; Gross, 1998; Scherer, 1984). This means that emotions arise in response to personally relevant events in the environment that then trigger specific reactions (i.e., behavioral, experiential, biological) that can occur if the individual, either consciously or subconsciously, does not modulate the response in some way (Gross, 1998). The emotional generation phase has been termed “emotional reactivity” while the capacity to change an emotion is considered “emotion regulation” (Cole, Michel, & Teti, 1994; Gross, 1998). Emotion regulation includes the process of evaluating the environment and the emotions resulting from that evaluation to determine whether some aspect of the emotion (e.g., subjective experience, behavioral outcome) should be changed. Emotion regulation can occur before (e.g., selecting whether to enter a situation), during (e.g., changing the appraisal of an emotion-eliciting stimulus), or after (e.g., suppressing or hiding the behavioral response to an emotion) the emotional reaction, and has been shown to be at times either effortful or automatic (Gross, 1998; Mauss, Bunge, & Gross, 2007).

The modern, functionalist view of emotion differs from proposals by some early theorists, such as behaviorists, who viewed emotion as a useless byproduct of changes in internal states (e.g., Dewey, 1895; Hebb, 1949; Skinner, 1948). Indeed, Skinner (1948) once stated, “We all know that emotions are useless and bad for our peace of mind and our blood pressure” (p. 92). In stark contrast to this view, the functionalist account proposes that emotions serve to prioritize...
and direct behavior in order to most efficiently and effectively respond to the demands of the internal or external environment (Keltner & Gross, 1999; Witherington & Crichton, 2007). To this end, Levenson (1999) specifies that emotions serve to direct attention, activate relevant memory networks, coordinate biological systems (e.g., autonomic nervous system, endocrine system, muscular systems, vocal tone, facial expression), and motivate approach or withdrawal from environmental stimuli. These systems are highly sensitive to feedback, so the emotion unfolds dynamically in response to continuous environmental input, and in turn initiates an automatic and rapid response.

Because of the adaptive function of emotions, the individual is necessarily impacted when a subsystem is not working properly (Berenbaum et al., 2003). Kring and Bachorowski (1999) argue that the function of emotion is the same in individuals with psychopathology, but that components of the emotional process are impaired. Therefore, these individuals may experience an emotion that is not commensurate with the stimulus, which then guides their actions and subsequent emotions similar to how this process occurs in healthy populations. For example, fear typically motivates avoidance of the feared stimulus. However, in anxiety disorders, a fear that may have started adaptively becomes maladaptive when the fear becomes too generalized and begins to interfere with daily functioning (e.g., fear of enclosed spaces due to war trauma leads to avoidance of classes for student veterans). Keltner and Kring (1998) further suggest that the emotional deficits associated with psychological disorders disturb an individual’s capacity to function adaptively in social relationships. In particular, emotions serve to convey information about the people and objects involved in a social interaction, and one’s personal emotional experience in a social interaction provides an assessment of the quality of the social relationship.
Emotional Reactivity Versus Emotion Regulation

Given that disruption in emotional responding, especially emotional reactivity and emotion regulation, can lead to these significant functional impairments, it may be important to consider the individual impacts of deficits in each area. However, most theorists agree that emotional reactivity and emotion regulation are inextricably linked (Gross & Barrett, 2011). When an emotion occurs, it necessarily elicits an almost immediate impulse to modify the emotion to respond to situational demands (Cole et al., 1994; Frijda, 1988; Gross, 1998; Gross & Barrett, 2011; Gross et al., 2011). Gross et al. (2011) outline ways in which a distinction between emotional reactivity and regulation is helpful, particularly in clinical psychology where many models of dysfunction in mood and anxiety disorders highlight a division between the generative and regulatory processes involved in emotion (e.g., Cisler, Olatunji, Feldner, & Forsyth, 2010; Nolen-Hoeksema, 1991). However, they also discuss how issues such as difficulty differentiating between reactivity and regulation during the timeline of the emotional response may render the separation of these processes as less useful. Given the theoretical utility of distinguishing between problems in reactivity versus regulation, it may be important to try to utilize available methods (e.g., psychophysiology) to more closely specify how these processes interact to create the deficits observed in psychopathology.

One such construct that may be related to both emotional reactivity and emotion regulation is emotional inertia (Kuppens, Allen, & Sheeber, 2010; Shuls, Green, & Hillis, 1998). Emotional inertia refers to resistance to emotional change, operationalized as the degree to which one’s current emotional state is related to the state that directly preceded it. Therefore, individuals experiencing emotional inertia display an enduring affective state that continues despite changes in the situation indicating that a different emotion would be more appropriate.
Kuppens et al. (2010) discuss how emotional inertia may underlie a range of psychological disorders, including those that typically involve excessive emotional reactivity (e.g., anxiety disorders) or a lack of reactivity to emotional stimuli (e.g., emotional context insensitivity in depression; Rottenberg, 2005). Psychological flexibility, and particularly the capacity to adapt to fluctuating situational demands, has been identified as a core feature of psychological health (Kashdan & Rottenberg, 2010). This flexibility includes experiencing negative emotion when faced with an upsetting situation, since this emotion helps to motivate the individual to react appropriately, and then being able to recover once this situation has passed. Kashdan and Rottenberg note that anxiety disorders in particular have been associated with rigidity in response style (e.g., experiential avoidance) and reduced autonomic flexibility.

The majority of emotional inertia research has focused on depressed populations, and has revealed conflicting findings. Kuppens et al. (2010) determined that depression was associated with higher rates of inertia in both positive and negative emotions, and Kuppens, Sheeber, Yap, Whittle, Simmons, and Allen (2010) found that greater emotional inertia in adolescents predicted development of depression 2.5 years later. In contrast, Thompson, Mata, Jaeggi, Buschkuehl, Jonides, and Gotlib (2012) found that depressed participants reported greater instability of negative affect and similar levels of inertia and reactivity as compared to their control group. Koval and Kuppens (2012) similarly demonstrated that psychological maladjustment was associated with more inertia in daily life, but also noted less inertia (i.e., more emotional variability) when faced with a laboratory-based social stressor. This has led to some discussion of whether a certain amount of inertia may be “optimal,” but that excessively high or low inertia is problematic (Kashdan & Rottenberg, 2010; Koval & Kuppens, 2012).
A state of emotional inertia may result from simultaneous dysfunction in both emotional reactivity and emotion regulation, which as previously suggested may be important in capturing the range of emotional problems experienced in anxiety disorders. For example, an individual might become overly anxious in a situation (emotional reactivity problem), be unable to down-regulate their anxiety (emotion regulation problem), and therefore continue experiencing their anxious state even when the original stimulus is no longer present (a state of emotional inertia). In the context of anxiety disorders, to the extent that problems with emotional reactivity and regulation lead to enduring or “inert” emotional states, this process may preclude individuals from gathering important information or may provide misinformation regarding their environment (e.g., enduring anxiety suggests that the situation continues to be threatening when in reality it may no longer be).

Emotional Processes in Generalized Anxiety Disorder

Recent theoretical advances regarding the role of worry and emotions in GAD have greatly increased our understanding of the way that individuals with this disorder experience their emotions. Borkovec, Alcaine, and Behar (2004) suggest that worry functions as a way to avoid emotional processing among those with GAD. Research on worry has consistently demonstrated that, after worrying, participants with and without GAD show a muted cardiovascular response to a negative stimulus, while relaxing or neutral thoughts lead to a strong initial response with habituation after multiple presentations of the stimulus (e.g., Borkovec & Hu, 1990; Borkovec & Sides, 1979; Peasley-Miklus & Vrana, 2000; Vrana, Cuthbert, & Lang, 1986). Therefore, Borkovec et al. (2004) suggest that worry is negatively reinforcing because it disrupts or mutes reactivity to a subsequent stimulus. According to Foa & Kozak (1986), the absence of reactivity indicates a failure to process the emotional stimulus.
The Contrast Avoidance Model (Newman & Llera, 2011) extends Borkovec et al.’s model by noting that GAD is not associated with an overall avoidance of activation, but with the avoidance of a shift from a positive or neutral state to a negative state. Indeed, worry, the state most associated with GAD, is emotionally activating in terms of cardiovascular reactivity (Brosschot, van Dijk, & Thayer, 2007; Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Glyn, Christenfeld, & Gerin, 2002; Llera & Newman, 2010; Stapinski, Abbott, & Rapee, 2010) and subjective negative emotion (Borkovec & Inz, 1990; Llera & Newman, 2010; Stapinski et al., 2010). In addition, individuals with GAD report that worry helps to distract from more emotional topics (Borkovec & Roemer, 1995; Freeston, Rheaume, Letarte, & Dugas, 1994).

**Emotional reactivity in GAD.** There is substantial evidence that people with GAD are more reactive to negative stimuli in terms of self-reported emotion than healthy individuals. On a daily basis, individuals with GAD report higher negative emotion (Mennin, Holaway, Fesco, Moore, & Heimberg, 2007; Mennin et al., 2005). Furthermore, in response to a variety of laboratory-based stimuli including film clips, mental math tasks, and startle tasks, participants with GAD report experiencing more intense negative emotions (Llera & Newman, 2010; Mennin et al., 2005; Ray et al., 2009). This pattern of hyperreactivity may be related to a hyperresponsive threat detection system such that these individuals interpret ambiguous stimuli as threatening (Mathews & MacLeod, 2005; Waters, Mogg, Bradley, & Pine, 2008; Wilson, MacLeod, Mathews, & Rutherford, 2006).

In contrast, the evidence regarding physiological reactivity in GAD has less clearly indicated whether hyperreactivity is present. For example, individuals with GAD demonstrate higher average heart rate during the day through ambulatory monitoring of physiology (Hoehn-Saric, McLeod, Funderburk, & Kowalski, 2004). However, in the laboratory, hyperreactivity has
been found in certain conditions but not others. Participants with GAD typically do not show elevated cardiovascular reactivity during a measured baseline and sometimes do not differ from a control group in response to positive and negative emotional inductions (Fisher et al., 2010; Fisher & Newman, in press; Llera & Newman, 2010). In contrast, Ray et al. (2009) demonstrated that a group with GAD reacted with a greater startle magnitude (i.e., via electromyography), particularly during tasks that required more inward (versus outward) attentional focus. Physiological hyperreactivity seems to be evident primarily when comparing to a neutral or relaxation baseline, while comparison to a worry period demonstrates muted cardiovascular responding, which as noted previously is theorized to negatively reinforce worry (Peasley-Miklus & Vrana, 2000; Stapinski et al., 2010; Vrana et al., 1986).

**Emotion regulation in GAD.** Although the emotion regulation deficits associated with GAD have been considered to be a primary difficulty of the disorder (e.g., the Emotion Dysregulation Model; Mennin, Heimberg, Turk, & Fresco, 2002; Mennin et al., 2005), the specific nature of these deficits has received inadequate attention in past research. As indirect evidence, high trait anxiety and a range of anxiety disorders are related to difficulties in the ability to effectively regulate emotions (Baker, Holloway, Thomas, Thomas, & Owens, 2004; Cisler et al., 2010; Cloitre, Scarvalone, & Difede, 1997; Hanley & Soto, under review; Salters-Peneault, Roemer, Tull, Rucker, & Mennin, 2006). Furthermore, the Emotion Dysregulation Model suggests that GAD is characterized by heightened intensity of emotions, poor understanding of emotion, fear of emotions, and ineffective strategies to regulate emotions, all of which interfere with the adaptive use of emotions. In support of this proposal, individuals with GAD report difficulty recovering from negative emotional states (Mennin et al., 2005; Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). Furthermore, worry, theorized to be a primary
emotion regulation strategy in GAD, has been associated with poor emotional outcomes, including dampening of parasympathetic and sympathetic activation following exposure to phobic images (Borkovec & Hu, 1990; Borkovec, Lyonfields, Wiser, & Deihl, 1993) and fearful film clips (Llera & Newman, 2010). However, few studies have considered how self-reported emotion and physiology change over the course of responding to several stimuli and then recovering from these inductions.

Taken together, prevailing theories of GAD (Borkovec et al., 2004; Newman & Llera, 2010) and evidence that worry precludes emotional processing suggest that the emotional deficits in GAD may be characterized as emotional inertia due to the maintenance of a negative affective state. Indeed, Shuls et al. (1998) observed a “lag effect” in negative emotion, or enduring emotional states that remain unchanged despite changes in the environment, for highly neurotic individuals, a trait that closely relates to GAD symptoms. Furthermore, Fisher et al. (2010) and Fisher and Newman (in press) observed that participants with GAD who showed high baseline levels of sympathetic activity did not demonstrate an increase in sympathetic activation in response to an emotional stimulus. These findings could indicate that, when already experiencing a sympathetically-arousing emotion, individuals with GAD are unable to further react to emotional stimuli. Indeed, Brosschot and colleagues (e.g., Brosschot, Pieper, & Thayer, 2005; Brosschot et al., 2007; Pieper & Brosschot, 2005) have suggested that the process of worry, which sustains the negative representation of a stimulus, prolongs the negative emotionality associated with that stimulus.

**Psychophysiology as a Tool for Studying Reactivity and Regulation**

In order to address the difficulty of distinguishing between emotional reactivity and regulation, it is important to go beyond self-report information. This may also be particularly
helpful in studies of individuals with psychopathology (such as the proposed work), whose emotional deficits may interfere with the capacity to accurately report on their emotional experience (e.g., alexithymia; Bagby, Parker, & Taylor, 1994). In this regard, physiological recording of autonomic arousal may serve as an important indicator of momentary changes in emotional processes that cannot be captured by subjective reports.

Similar to the relationship between facial expressions and specific emotions (i.e., evidence for universality of six basic facial expressions), there is growing evidence that distinct physiological profiles may be associated with certain emotions or emotional processes (Levenson, 2003; see Cacioppo, Berntson, Larson, Poehlmann, & Ito, 2000, Tomaka, Blaskovich, Kelsey, & Leitten, 1993, and Barrett, 2006 for alternative views). Levenson (2003) observed that, in general, activation of the sympathetic nervous system (SNS) is related to the experience of negative emotion (e.g., anger, fear, disgust) while the parasympathetic nervous system (PNS) is activated primarily by positive emotions (e.g., calm, relaxation, happiness). Furthermore, the work of Levenson and colleagues (e.g., Levenson, 1992; Levenson, Ekman, & Friesen, 1990; Ekman, Levenson, & Friesen, 1983) has typically supported the hypothesis of “autonomic specificity” (i.e., specific physiological profiles that differ between emotions) for six basic emotions: fear, anger, sadness, disgust, happiness, and surprise.

For the purpose of this project, in which fear and sadness were experimentally induced, highlighting the distinct patterns of physiology associated with each of these emotions is critical. When fear is experimentally induced, heart rate increases, cardiac output increases, diastolic blood pressure increases, respiration rate increases, skin temperature decreases, and total peripheral resistance decreases, indicating an overall increase in sympathetic activity (Boudewyns & Levis, 1975; Ekman, Levenson, & Friesen, 1983; Funkenstein, King, &
Fear is also associated with a decrease in parasympathetic indices, including heart rate variability and vagal tone (Friedman, 2007). In contrast, experimental inductions of sadness have led to increased heart rate, decreased cardiac output, increased diastolic blood pressure, decreased respiration rate, decreased skin temperature, and increased total peripheral resistance, most consistent with an overall decrease in sympathetic activation (Ekman et al., 1983; Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006; Gross & Levenson, 1997; Kreibig et al., 2007; Krumhansl, 1997; Levenson et al., 1990; Sinha, Lovallo, & Parsons, 1992; Tsai, Levenson, & Cartenson, 2000). Parasympathetic activity (i.e., heart rate variability) during sadness inductions typically demonstrates no change from baseline (Ritz, Thöns, Fahrenkrug, & Dahme, 2005; Etzel et al., 2006). Thus, fear is distinguished from sadness by its higher sympathetic and lower parasympathetic activation.

Similar to the idea of autonomic specificity for emotions, many researchers have come to view PNS activity as indicative of successful emotion regulation. Appelhans and Luecken (2006) reviewed studies suggesting that heart rate variability, an indicator of PNS activation, increases when an individual is in a “well-regulated” state. Furthermore, according to Porges’ (1997, 2001) polyvagal theory, the PNS developed with the primary purpose of coordinating social behaviors, which necessarily involves regulating the emotional processes (e.g., intensity, timing) that underlie social behavior. Emotion regulation research has demonstrated that use of certain regulation strategies impacts autonomic activation in different ways (Gross, 2002). For example, expressive suppression is typically associated with increased SNS activation (Gross & Levenson, 1997; Egloff, Schmukle, Burns, & Schwerdtfeger, 2006) while cognitive reappraisal does not typically impact autonomic activity (Egloff et al., 2006). Therefore, physiological
recording offers a useful tool that can be used to help approximate when an individual is experiencing emotion and regulating it successfully.

**The Present Study**

The current study is intended to address how emotional functioning is disrupted in GAD. As reviewed previously, GAD seems to be associated with some degree of hyperreactivity (Llera & Newman, 2010; Mennin et al., 2005; Mennin et al., 2000; Ray et al., 2009) and reported difficulties with regulating emotions (Mennin et al., 2005; Turk et al., 2005). However, it is not yet clear how problems in reactivity and regulation interact to impact the course of the emotional response. In order to understand the interplay between initial emotional reactivity and subsequent conscious or subconscious efforts to regulate, it may be especially important to consider the full timeline of the emotional response, particularly the recovery period following an emotion induction. Few studies have measured emotional processes beyond the initial induction period. Furthermore, the impact of heightened reactivity or deficits in regulation from an initial emotional stimulus on the ability to respond to subsequent stimuli has not been considered in GAD, despite its important functional implications.

The present study has been designed to consider the primary question of whether individuals with GAD are able to adaptively shift their emotions when different emotional stimuli are presented in sequence. A secondary but related question is whether any observed emotional deficits are driven by emotional hyperreactivity or deficits in emotion regulation. In order to answer these questions, participants with and without GAD were presented with two emotion-inducing film clips, one that induces fear and another that induces sadness. Self-reported emotion and physiology were assessed during a pre-film baseline, films, and post-film
recovery periods in order to comprehensively assess the nature of the emotional response (i.e., reactivity, regulation, ability to shift) in these populations.

Hypotheses

**Baseline emotional reactivity.** As has been observed in previous studies (e.g., Fisher et al., 2010; Llera & Newman, 2010), I hypothesized that participants with GAD would demonstrate higher levels of self-reported negative emotions, but not physiological activity, at the start of the study protocol (i.e., baseline) than control participants (Hypothesis 1).

**Emotional response profiles.** Predicted patterns of responding to the two emotional stimuli lead to one specific hypothesis and one test of competing hypotheses. Three possible patterns for responding during the emotion inductions and recovery periods are depicted in Figure 1. I proposed that the *adaptive responding pattern* (see solid line in Figure 1) would be shown by the group of healthy, control participants (Hypothesis 2). To support this hypothesis, changes in self-reported emotions and psychophysiology commensurate with the emotion being elicited (i.e., fear response for fear film and sad response for sad film) with a return to baseline functioning during the recovery periods must be observed. In terms of physiology, participants would demonstrate an adaptive or relaxed physiological profile at baseline and during the recovery periods (i.e., low sympathetic arousal and high parasympathetic arousal), a fear profile during the fear film clip (i.e., increased sympathetic activation and decreased parasympathetic activation), and a sadness profile during the sad film clip (i.e., decreased sympathetic activation and no change from the previous baseline period in parasympathetic activation). This pattern implies that participants were able to respond appropriately to each emotion that is induced, could regulate their responding back to baseline during the recovery period, and experienced a change in emotional responding from the first emotion-eliciting stimulus to the second.
Participants with GAD were expected to demonstrate an emotional response consistent with either the hyperreactivity pattern or the emotional inertia pattern (Hypothesis 3). The hyperreactivity pattern indicates that participants diagnosed with GAD would demonstrate heightened self-reported and physiological reactivity to each emotion induction followed by a return to baseline responding during the recovery period (see small dotted line in Figure 1). They would report significantly less of the first emotion elicited during the second film clip (e.g., if fear film is first, they would not report significant amounts of fear during the sad film clip). This pattern indicates that the primary issue in emotional responding is one of heightened emotional reactivity, but that individuals are able to subsequently recover and then respond to the next stimulus.

The emotional inertia pattern indicates that participants with GAD would demonstrate deficits in both emotional reactivity and regulation. Specifically, they would experience a sustained response from the first stimulus that does not decrease during the recovery period and precludes a change in reactivity to the second emotion induction (see large dotted line in Figure 1). These participants would show an increase in self-reported and physiological profiles of fear or sadness (depending on counterbalanced order) from baseline to the first film, though this increase may not be larger than that observed in the control group. This degree of fear or sadness would not decrease during the recovery period or the second film, so that the first emotion elicited endures and the second emotion-eliciting film would not lead to a significant increase in the new emotion. Therefore, if this pattern is supported, participants with GAD would demonstrate an inability to regulate the initial emotional response, which then persists throughout the rest of the procedure despite the introduction of a new emotional stimulus. Although I hypothesized that either the hyperreactivity or emotional inertia pattern would be
supported regardless of the order of film presentation, it is possible that the order of emotional stimulus presentation may impact the pattern, so this will also be tested.

![Figure 1](image.png)

**Figure 1.** A presentation of an approximate depiction of the findings that would be expected for each pattern of emotional responding. Scores along the y-axis represent an increase in general reactivity, meaning an increase in indices of each emotion (i.e., self-reported sadness or fear and physiological indices of sadness or fear).

### Chapter 2

**METHOD**

**Participants**

Participants for this project were 40 individuals diagnosed with GAD and 40 control participants with no psychological diagnoses. Of those with GAD, there were 6 males and 34 females, and the average age was 28.58 years old ($SD = 11.59$, minimum = 18, maximum = 58); among participants without GAD, there were 9 males and 31 females, and the average age was
26.30 years old ($SD = 9.13$, minimum = 18, maximum = 57). There were no significant mean differences between the GAD and control participants on these variables. Participants were evenly divided across the two study conditions (i.e., fear film presented first and sad film presented first) in terms of GAD status, age, and gender, and $t$-tests confirmed that the two study conditions did not differ significantly on these variables. The overall sample was predominantly Caucasian (72.5%; 11.3% Asian, 7.5% African American, 5.0% Multiethnic, 2.5% Latino/Hispanic, and 1.3% Other). Exclusion criteria for all participants included being under age 18 or over age 65, a history of psychosis, current severe major depression, and intellectual disability.

Participants were recruited from the community surrounding the Pennsylvania State University. Advertisements were placed in the local newspaper (Centre Daily Times) and online (Craigslist) to recruit GAD and control participants from the local community. These advertisements either sought participants “in good mental health” or participants who were “anxious” and “worry excessively most days”. Participants with GAD were also recruited through local psychological clinics, which were contacted and asked to provide the study information to their participants being treated for GAD. Finally, some participants were recruited from the Penn State psychology subject pool based on their responses to two prescreening measures administered as a part of their course requirements. A research assistant then administered these same two questionnaires over the phone to all interested participants to determine whether they would qualify for the GAD or control groups. A total of 164 individuals contacted the study and, of those, 129 completed the prescreening procedure. Of those who did not complete the screening, 31 could not be reached and 4 indicated that they were no longer interested. Of those who completed the screening, 40 were not eligible and 89 were eligible. Six
of the eligible participants either canceled or did not show up for their scheduled appointments. Three participants completed the study protocol but were excluded because they did not fit into either the control or GAD group after completing the diagnostic interview. The final sample was composed largely of individuals from the community \((n = 64)\), with some coming from local psychological clinics \((n = 4)\), and the remainder coming from the psychology subject pool \((n = 12)\). Participants were compensated at a rate of $17 per hour for their laboratory participation.

**Diagnostic Screening**

Participants in the control group \((n = 40)\) did not meet criteria for any DSM-IV-TR diagnoses, with the exception of 6 participants that met criteria for a specific phobia. Because the reported phobias were unrelated to the content of the current study, their data are included in all analyses. Participants in the GAD group \((n = 40)\) met criteria for many comorbid diagnoses, including panic disorder without agoraphobia (10.0%), panic disorder with agoraphobia (12.5%), social phobia (27.5%), posttraumatic stress disorder (20.0%), obsessive-compulsive disorder (12.5%), specific phobia (32.5%), major depressive disorder (15% current, 52.5% in remission), bipolar I disorder (2.5%; not currently depressed or manic), bulimia nervosa (2.5%), undifferentiated somatoform disorder (5.0%), hypochondriasis (2.5%), and body dysmorphic disorder (10.0%). The clinical severity rating (CSR) of GAD symptoms was higher among the GAD group \((M = 5.40, SD = 1.08)\) than the control group \((M = .38, SD = .77)\), \(t(78) = 23.90, p < .01\).

**Procedure**

Once in the laboratory, participants provided written informed consent to participate. They completed questionnaires aimed at understanding individual emotion tendencies and in-the-moment emotional experiences. A gender-matched research assistant adhered physiology
sensors to the ribcage, back, and neck of participants. Participants then sat in front of a computer monitor. They first rated their current experience of emotions using the emotion rating questionnaire (self-report emotion baseline) and then sat quietly for four minutes, which allowed for obtaining a physiological baseline. Then, they were instructed to sit again for four minutes, this time while relaxing (i.e., “…close your eyes and allow your mind and body to relax. Breathe from your diaphragm or belly rather than from your chest, and slow the rate of your breathing down to a comfortable pace. Focus your attention on each inhalation and exhalation. With the inhale, gather your tension in. With the exhale, let it all go.”). After rating their emotions, the computer monitor presented the fear and sad films, in counterbalanced order. Immediately after the first film was presented, participants rated their current emotions and then sat for a three minute physiology recovery period. Following the recovery period, participants rated their emotions again and rated the general valence of their thoughts during the recovery period (0 = very negative, 4 = neutral, 9 = very positive). Participants then watched the second film, followed by another emotion rating and three minute recovery period. They completed another emotion rating and the question regarding the valence of their thoughts during the recovery period. Then, participants viewed the calm film clip followed by a final three minute recovery period and emotion rating. The research assistant removed the physiology sensors.

A graduate interviewer then completed a diagnostic interview to confirm the presence or absence of Axis I DSM-IV diagnoses. Finally, the graduate interviewer debriefed the participant and assessed for any risk induced by the study. See Figure 2 for a depiction of the protocol.
Figure 2. Timeline of the full study protocol.
Measures

Pre-screening symptom measures. The Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995) and the Generalized Anxiety Disorder Questionnaire (GAD-Q-IV; Newman et al., 2002) were administered over the telephone by a trained undergraduate research assistant to all interested participants recruited from the community and local advertisements and were completed by all eligible psychology subject pool participants via online questionnaire. To be eligible to participate in the control group, scores on the DASS and GAD-Q-IV had to be below clinical cutoffs (i.e., DASS Depression below 10, DASS Anxiety below 7, DASS Stress below 15, and GAD-Q-IV below 5.7). To be eligible to participate in the GAD group, scores on the GAD-Q-IV had to be above the clinical cutoff of 5.7. In addition to the DASS and GAD-Q-IV, potential participants reported whether they had ever experienced psychotic symptoms (i.e., hallucinations or delusions). Potential control participants were asked whether they had any symptoms of psychopathology within the past five years, and were deemed ineligible if they reported any significant symptoms.

The DASS is a 42-item self-report measure of the symptoms of depression (i.e., anhedonia and negative mood), anxiety (i.e., muscle tension and situational anxiety), and stress (i.e., non-specific arousal such as irritability and nervousness). Items are rated on a Likert-type scale from zero (Did not apply to me at all) to three (Applied to me very much, or most of the time) and refer to how the individual has felt “over the past week”. This measure has been well-validated in both clinical and non-clinical samples and demonstrates a very good to superior model fit on the three subscale factors of depression, anxiety, and stress (Brown, Chorpita, Korotitsch, & Barlow, 1997; Crawford & Henry, 2003). It also shows superior convergent validity with other measures of depression and anxiety and adequate discriminant validity from
general positive and negative affect. Finally, the DASS adequately differentiates between diagnoses of depression and anxiety in clinical samples, as well as accounting well for comorbidity (Brown et al., 1997). In the current sample, internal consistency was high (Cronbach’s alphas were .95 for DASS-Depression, .95 for DASS-Anxiety, and .96 for DASS-Stress).

The GAD-Q-IV (Newman et al., 2002) is a 9-item self-report measure of GAD symptoms as defined by the DSM-IV-TR. The first 6 questions inquire about presence or absence of excessive, uncontrollable worry that has occurred more days than not in the past 6 months, including the types of topics people worry about excessively and uncontrollably. Participants who indicate the absence of these symptoms discontinue the questionnaire at this point. The remaining questions assess for the other symptoms associated with GAD (e.g., presence or absence of irritability, muscle tension and soreness, sleep disturbance) and interference and distress of these symptoms as rated on a 0 (not at all) to 8 (very severely) scale. Scores can range from 0 to 13, with a clinical cutoff score of 5.7. Newman et al. (2002) found that this cutoff score resulted in good sensitivity (83%) and specificity (89%), as well as good concurrence with a clinician-administered interview (kappa = .67 with 88% of participants correctly classified). Furthermore, the GAD-Q-IV demonstrates strong convergent and discriminant validity and test-retest reliability (i.e., 92% of participants had the same GAD categorization after 2 weeks).

**Background information.** Participants provided information about their gender identity, age, ethnicity, family background, religion, sexuality identity, income, educational history, and employment status. They also indicated personal health characteristics including height, weight, psychiatric history, current psychiatric medications, current substance use, and medical history.
Symptom assessment. The Anxiety Disorder Interview Schedule for DSM-IV (ADIS-IV; Brown, Di Nardo, & Barlow, 2004), Pennsylvania State University version, was administered to all eligible participants by graduate students in clinical psychology who received advanced training in administration of this diagnostic tool. The ADIS-IV is a semi structured, clinical interview that assesses for the presence of Axis I DSM-IV disorders, including anxiety, mood, substance, eating, somatoform, and psychotic disorders. The psychotic and substance disorder modules were not administered due to screening out of psychotic disorders and assessment of substance use via self-report (see Background Information above). Furthermore, only current diagnoses were assessed. For each disorder that participants reported experiencing related symptoms, interviewers assigned a clinical severity rating (CSR) from zero (“None”) to eight (“Very severely disturbing/disabling”), where ratings greater than or equal to four indicate that the level of symptoms meet DSM-IV criteria for the disorder. For 21 participants (26.3% of the sample), one of the other graduate student assessors coded a video recording of the ADIS for reliability. Reliability was excellent both for categorical assessment of GAD (kappa = 1.0) and the CSR for GAD (linear-weighted kappa = .88).

Emotion tendencies. The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a 36-item measure that assesses for how participants view their emotions (e.g., impulse control, emotional awareness, emotional clarity, access to emotion regulation strategies, acceptability of emotion; “When I’m upset, I feel guilty for feeling that way”). Items are rated on a Likert-type scale ranging from one (“Almost never, 0-10%”) to five (“Almost always, 91-100%”). The DERS demonstrates good test-retest reliability (alpha is .88) and convergent and discriminant validity with measures of experiential avoidance and emotional expressivity. In the current sample, internal consistency (i.e., Cronbach’s alpha) was .95 for the overall scale.
The Positive and Negative Affect Schedule (PANAS-X; Watson & Clark, 1994) is a 60-item measure of emotional states that can be administered to capture a variety of time points. For the purposes of this study, participants completed a trait-level rating of their emotions by focusing on “to what extent you generally feel this way, that is, how you feel on the average”. Participants rated a series of positive (e.g., interested, at ease) and negative (e.g., distressed, sad) emotions on a one (“Very slightly or not at all”) to five (“Extremely”) scale. The chosen emotions represent all four quadrants of an affective circumplex where pleasant-unpleasant and high arousal-low arousal are the axes. Stability in ratings over two months is good, with retest coefficients ranging from .51 (serenity scale) to .71 (general negative affect scale). The internal consistency (Cronbach’s alpha) for the negative affect scale, the only one used for these analyses, was .90.

**Emotion ratings.** In order to capture momentary changes in emotional experience, participants rated their current experience of 13 emotions on a scale ranging from zero (“Not at all”) to eight (“Very much”) at several points throughout the experiment. The emotions were presented as follows: interest, happiness, surprise, amusement, contentment, relief, anxiety, sadness, annoyance, disgust, embarrassment, boredom, fear, anger, contempt, and stress; only the ratings of fear and sadness were used for the purposes of this study. This rating scale has been used effectively in other studies that assess for state emotion (e.g., Soto, Levenson, & Ebling, 2005).

**Apparatus**

**Audiovisual.** An 18-by-24-inch computer monitor presented stimuli to the participants using the ePrime© software program. Participants were video-taped using a remotely controlled video camera.
**Film clips.** To elicit the emotions of interest (i.e., fear and sadness), two film clips were chosen that are associated with increases in the desired emotion but not other emotions. The fear film was a selection from *The Shining* (1980; clip duration is 4 minutes and 15 seconds) in which an apparently deranged man pursues his wife through a deserted hotel with an axe. This film was shown to reliably elicit fear and anxiety but not other emotions (e.g., sadness, amusement, disgust) in a large sample (Schaefer, Nils, Sanchez, & Philippot, 2009). The sadness film was from *City of Angels* (1998; clip duration is 4 minutes and 15 seconds) in which a woman dies in her lover’s arms. Shaefer et al. (2009) reported that this film reliably elicits sadness and not other emotions. Finally, a film clip designed to induce calmness or relaxation was included to aid in returning all participants to a more pleasant state following the fear and sadness films. The calm film clip (duration is 3 minutes and 25 seconds) displays abstract shapes moving across the screen (Gross & Levenson, 1995).

**Psychophysiology.** A Biopac© MP150 device consisting of an eight-channel polygraph and a microcomputer was used to collect data on autonomic nervous system functioning. (1) *Electrocardiography* (ECG) was measured through three Biopac pre-gelled, self-adhering, disposable electrodes placed on the right clavicle at the midclavicular line, just above the last bone of the ribcage at the left midaxillary line, and just below the last bone of the ribcage at the right midaxillary line. (2) *Cardiac impedance* was measured by four of the same Biopac electrodes placed at the front of the neck between the clavicles, over the sternum at the base of the ribs, at the base of the back of the neck, and on the back approximately four centimeters below where the sternum electrode was placed. AcqKnowledge© software was used to collect and compute second-by-second averages for each of the above-described physiological processes. The resulting data was extracted using Mindware© software. Artifacts and
Arrhythmias in the EKG signal were removed via interpolation of missing heart beats where possible to determine or removal of data segments if interpolation was not possible.

Analysis of these cardiac data provided indicators of both sympathetic and parasympathetic nervous system arousal. Impedance cardiography is considered to be a strong indicator of sympathetic activity in the autonomic nervous system (Berntson, Cacioppo, & Quigley, 1994). SNS indices include left ventricular ejection time (LVET), stroke volume (SV), and pre-ejection period (PEP). LVET is a measure of myocardial contractility and corresponds to the time between the opening and closing of the aortic valve or mechanical systole (Brownley, Hurwitz, & Schneiderman, 2000). SV is the volume of blood ejected by the left ventricle of the heart in one beat, which is derived from subtracting the afterload from the preload or the volume of blood in the heart after ejection through the aorta from the total blood volume in the heart before ejection. Finally, PEP is an indicator of sympathetic myocardial drive and indicates the interval between onset of the EKG Q-wave and onset of the LVET. Unlike LVET and SV, PEP decreases as SNS activity increases. Respiratory sinus arrhythmia (RSA) is considered a relatively unbiased indicator of PNS activity and designates the natural variation in heart rate that occurs between inspiration and expiration in the breathing cycle (Berntson et al., 1994). RSA was computed by removing artifacts from the inter-beat interval series and using a Fast Fourier Transformation to determine the spectral distribution (Berntson, Quigley, Jang, & Boysen, 1990). The integral power in the respiratory frequency band (0.12 to 0.40 Hz) was designated as indicating high frequency heart rate variability, or RSA. Respiration was monitored through impedance cardiography (Ernst, Litvack, Lozano, Cacioppo, & Bernston, 1999). For each time segment of interest (i.e., baseline, fear film, fear recovery period, sad film, and sad recovery
period), second-by-second averages in each of these physiological indices were reduced to mean
values.

**Data Analysis Plan**

To test Hypothesis 1, one-way ANOVAs were used to compare self-reported emotion
(i.e., fear and sadness) and physiological arousal (i.e., LVET, SV, PEP, and RSA) at baseline
across the GAD and control groups. For this study, there were two “baseline” (i.e., pre-film)
periods of 4 minutes each; in the first, participants were instructed to sit quietly while in the
second, participants were instructed to relax and breathe deeply. Differences during each of
these periods were tested.

To test Hypotheses 2 and 3, a series of 2 (group status [GAD vs control]) x 5 (time
points) repeated measures ANOVAs were conducted, where group was the between subject
variable and time was the repeated measure. These analyses examined differences across groups
in our primary dependent variables (i.e., self-reported emotion and physiological arousal) during
the various baseline, film, and recovery periods of the study protocol. Separate analyses were
conducted for the fear film first and sad film first conditions, and, thus, the results for each
condition are presented separately. When analyses revealed a different pattern of responding
between the GAD and control groups, post hoc analyses were performed in order to determine
whether basic difficulties in emotional or cognitive processes (e.g., emotion dysregulation, high
trait negative affectivity, negative cognitions during the protocol) accounted for these differences
between groups, or whether having GAD predicted these differences above and beyond these
components. The post-hoc analyses consisted of repeated measures ANCOVAs with the same
structure as those described for the primary analyses. Separate ANCOVAs were run for each
covariate measuring emotional difficulties (i.e., DERS and PANAS-Negative Affect) and cognitive content (i.e., valence of thoughts during the recovery periods).

Chapter 3

RESULTS

Certain individual factors are known to impact physiological outcomes (e.g., height and weight, use of psychiatric medications; Grossman and Taylor, 2006). In the current sample, body mass index was not significantly correlated with the GAD status or the GAD CSR. However, participants in the GAD group did report taking psychiatric medications more frequently than participants in the control group ($F(1, 78) = 9.32, p < .01$). Therefore, all physiology analyses presented below were replicated with psychiatric medications ($0 = \text{does not take medications}, 1 = \text{takes at least one medication}$) as a covariate. Taking psychiatric medications did not alter the pattern of significant findings with the exception of one model with PEP as the outcome, which is not presented since effects driven by psychiatric medications may not be specific to GAD.

To satisfy assumptions for ANOVA analyses, all outliers were removed from the dataset. The physiological dependent variables were normally distributed, but self-reported fear and sadness were not; however, transforming the variables did not result in normality so the original variables have been used in all analyses.

Emotion Tendencies

Table 1 displays the correlations between GAD status, GAD clinical severity rating (CSR), and the measures of emotion tendencies included in the study. All correlations were in the expected directions. GAD status and the GAD CSR were positively associated with
indicators of emotional distress or ineffective emotion regulation (i.e., DASS depression, anxiety, and stress, DERS, and PANAS-Negative Affect).

Table 1.

Correlations among GAD Presence, GAD Severity Rating, and Measures of Emotion Tendencies in the Full Sample

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. GAD Presence</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2. GAD CSR</td>
<td>.94**</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>3. DASS-Anxiety</td>
<td>.67**</td>
<td>.74**</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. DASS-Depression</td>
<td>.62**</td>
<td>.71**</td>
<td>.68**</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. DASS-Stress</td>
<td>.78**</td>
<td>.85**</td>
<td>.89**</td>
<td>.67**</td>
<td>--</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. DERS</td>
<td>.70**</td>
<td>.76**</td>
<td>.64**</td>
<td>.79**</td>
<td>.68**</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td>7. PANAS-NA</td>
<td>.75**</td>
<td>.82**</td>
<td>.85**</td>
<td>.74**</td>
<td>.87**</td>
<td>.74**</td>
<td>--</td>
</tr>
</tbody>
</table>

* p < .05. ** p < .01. GAD = Generalized Anxiety Disorder; CSR = Clinical Severity Rating; DASS = Depression Anxiety Stress Scales; DERS = Difficulties with Emotion Regulation Scale; PANAS = Positive and Negative Affect Schedule

Baseline Differences

One-way ANOVA was used to test for baseline differences in self-reported emotion and physiology (i.e., differences during resting baseline periods before any films were shown) between the GAD and control groups. Partial eta squared ($\eta_p^2$) is reported as an estimate of effect size here and in the repeated measures ANOVA section below, and values of .01, .06, and .14 can be interpreted as small, medium, and large effect sizes, respectively.

Baseline differences between the GAD and control groups emerged both at the start of the protocol and as rated just after the relaxation baseline for self-reported fear (start of protocol,
30

\[ F(1, 78) = 9.12, \ p < .01, \ \eta_p^2 = .11; \text{relaxation baseline, } F(1, 78) = 8.46, \ p < .01, \ \eta_p^2 = .10 \] and sadness (start of protocol, \( F(1, 78) = 24.47, \ p < .01, \ \eta_p^2 = .24; \text{relaxation baseline, } F(1, 78) = 7.44, \ p < .01, \ \eta_p^2 = .09 \)). Participants with GAD reported higher levels of fear (start of protocol, \( M = 1.20, \ SD = 1.79; \text{relaxation baseline, } M = 1.20, \ SD = .71 \) and sadness (start of protocol, \( M = 2.23, \ SD = 1.99; \text{relaxation baseline, } M = 1.78, \ SD = 2.11 \)) than control participants’ reports of fear (start of protocol, \( M = .28, \ SD = .75; \text{relaxation baseline, } M = .23, \ SD = .89 \)) and sadness (start of protocol, \( M = .45, \ SD = 1.09; \text{relaxation baseline, } M = .68, \ SD = 1.44 \)). There were no significant differences between groups in physiology (i.e., LVET, SV, PEP, and RSA).

\section*{GAD Status and Emotional Responding}

To test for differences in mean levels of the physiology and self-reported emotion across the various study time points, a series of 2 (group status [GAD vs control]) x 5 (time points) repeated measures ANOVAs were conducted. For all tests reported herein, the \( F \) statistic has been adjusted using the Greenhouse-Geisser correction due to significant sphericity as indicated by Mauchly’s Test of Sphericity. In cases where significant differences were evident at the omnibus level, post hoc analyses were conducted using a Bonferroni adjustment to correct for multiple comparisons. For ease of understanding, time segments are labeled t1 through t5 (i.e., \textit{time} one through five) for the reporting of all post hoc results.

\textbf{Fear Film First (FFF) Condition.} Repeated measures ANOVA results are presented in Table 2 and means for each dependent variable across the study are depicted in Figure 3. With self-reported fear as the dependent variable, a significant main effect of time emerged, \( F(2.15, 81.52) = 14.56, \ p < .01, \ \eta_p^2 = .28 \). Post hoc analyses showed that participants rated their fear significantly higher during the fear film (t2; \( M = 2.65, \ SE = .38 \)) when compared to baseline (t1; \( M = .90, \ SE = .26; \ p < .01 \)), the fear recovery period (t3; \( M = .98, \ SE = 27; \ p < .01 \)), the sad film
Furthermore, there was a significant main effect of GAD status \( (F(1, 38) = 14.24, p < .01, \eta^2_p = .27) \), indicating that participants with GAD rated their fear as higher across all time points than participants without GAD. The interaction between time and GAD status was not significant. Thus, all participants rated their fear as highest during the fear film with lower fear during other time points, and participants with GAD consistently reported higher levels of fear across all time points.

With self-reported sadness as the dependent variable, the main effect of time was significant, \( F(2.79, 105.88) = 10.56, p < .01, \eta^2_p = .22 \). Sadness was significantly higher during the sad film \( (t4; M = 3.03, SE = .40) \) than at baseline \( (t1; M = 1.23, SE = .31; p < .01) \), the fear film \( (t2; M = 1.63, SE = .30; p < .01) \), the fear recovery period \( (t3; M = 1.10, SE = .31; p < .01) \), and the sad recovery period \( (t5; M = 1.45, SE = .33; p < .01) \). The main effect of GAD status was also significant \( (F(1, 38) = 9.39, p < .01, \eta^2_p = .20) \), indicating that participants with GAD reported higher levels of sadness than control participants across all time points. The interaction between time and GAD status was not significant. Therefore, all participants reported significantly higher sadness in response to the sad film with lower levels throughout the rest of the protocol, and participants with GAD reported consistently higher sadness than control participants across all time points.

With LVET as the dependent variable, there was no main effect of time or GAD status. A trend emerged for the interaction between time and GAD status, \( F(2.54, 91.51) = 2.32, p = .09, \eta^2_p = .06 \). The follow-up post hoc tests indicated no significant differences across time points for participants in the control condition. In contrast, several significant differences were evident in the GAD group. LVET was significantly lower at baseline \( (t1; M = 270.38, SE = \)
11.28) than during the sad film (t4; M = 293.71, SE = 10.06; p < .05) and the sad recovery period (t5; M = 291.04, SE = 10.37; p < .05). LVET was also lower during the fear film (t2; M = 267.80, SE = 11.26) than the sad film (p < .01) and the sad recovery period (p < .01). LVET was marginally lower during baseline than the fear recovery period (t3; M = 284.40, SE = 10.71; p = .07). Thus, while LVET did not change significantly among control participants, participants with GAD experienced significantly higher LVET, indicating higher SNS activation, during the sad film and sad recovery period than they did during other time points. There were no significant main effects or interactions for the other SNS indices, SV and PEP.

With RSA as the dependent variable, there was a significant main effect of time, F (2.90, 107.27) = 2.99, p < .05, ηp² = .08. RSA was significantly higher at baseline (t1; M = 6.56, SE = .21) than during the fear film (t2; M = 6.16, SE = .17; p < .05). Furthermore, there was a trend toward a main effect of GAD status (F (1, 37) = 2.78, p = .10, ηp² = .07), suggesting that RSA, and therefore PNS activation, was lower across all time points among participants with GAD than control participants. The interaction between time and GAD status was not significant.

In summary, in the FFF condition, all participants reported experiencing the corresponding emotion to each film with decreases during the recovery periods; however, participants with GAD consistently reported higher levels of fear and sadness across all time points. In terms of physiology, the control participants experienced little change in SNS activity (i.e., LVET) and decreased PNS activity (i.e., RSA) during the fear film, suggesting little reactivity overall to the film clips with the exception of decreased PNS activity during the fear film, which might indicate being less well-regulated during this period. In contrast, participants with GAD evinced a muted response during the fear film and fear recovery period (i.e., low SNS and PNS activity as indexed by LVET and RSA, respectively) and a stress response, typically
associated with fear (i.e., high SNS and low PNS activity in LVET and RSA, respectively), during the sad film and sad recovery period.
Table 2

Repeated Measures ANOVA and Significant Bonferroni Comparisons for Self-Reported Fear, Self-Reported Sadness, LVET, and RSA during the Fear Film First Condition

<table>
<thead>
<tr>
<th>Effect</th>
<th>Mean Square</th>
<th>df</th>
<th>F</th>
<th>$\eta_p^2$</th>
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<tr>
<td><strong>Self-Reported Fear</strong></td>
<td></td>
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<tr>
<td>Time</td>
<td>47.63</td>
<td>2.15</td>
<td>14.56**</td>
<td>0.28</td>
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<tr>
<td>GAD</td>
<td>109.52</td>
<td>1.00</td>
<td>14.24**</td>
<td>0.27</td>
</tr>
<tr>
<td>Time × GAD</td>
<td>2.25</td>
<td>2.15</td>
<td>0.69</td>
<td>0.02</td>
</tr>
<tr>
<td>Error</td>
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<td>38.00</td>
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<th>Standard Error</th>
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<th>Upper Bound</th>
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<tr>
<td>t2 to t1</td>
<td>1.75**</td>
<td>0.39</td>
<td>0.58</td>
<td>2.92</td>
</tr>
<tr>
<td>t2 to t3</td>
<td>1.68**</td>
<td>0.40</td>
<td>0.48</td>
<td>2.87</td>
</tr>
<tr>
<td>t2 to t4</td>
<td>1.75**</td>
<td>0.38</td>
<td>0.63</td>
<td>2.87</td>
</tr>
<tr>
<td>t2 to t5</td>
<td>1.93**</td>
<td>0.41</td>
<td>0.70</td>
<td>3.15</td>
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<table>
<thead>
<tr>
<th><strong>Self-Reported Sadness</strong></th>
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<tbody>
<tr>
<td>Time</td>
<td>34.57</td>
<td>2.79</td>
<td>10.56**</td>
<td>0.22</td>
</tr>
<tr>
<td>GAD</td>
<td>120.13</td>
<td>1.00</td>
<td>9.39**</td>
<td>0.20</td>
</tr>
<tr>
<td>Time × GAD</td>
<td>0.70</td>
<td>2.79</td>
<td>0.21</td>
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<tr>
<td>Error</td>
<td>9.12</td>
<td>38.00</td>
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<tr>
<td>t4 to t1</td>
<td>1.80**</td>
<td>0.44</td>
<td>0.50</td>
<td>3.10</td>
</tr>
<tr>
<td>t4 to t2</td>
<td>1.40**</td>
<td>0.38</td>
<td>0.27</td>
<td>2.53</td>
</tr>
<tr>
<td>t4 to t3</td>
<td>1.93**</td>
<td>0.39</td>
<td>0.76</td>
<td>3.09</td>
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<tr>
<td>t4 to t5</td>
<td>1.58**</td>
<td>0.36</td>
<td>0.50</td>
<td>2.65</td>
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<table>
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<tbody>
<tr>
<td>Time</td>
<td>2096.57</td>
<td>2.54</td>
<td>1.73</td>
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</tr>
<tr>
<td>GAD</td>
<td>49.93</td>
<td>1.00</td>
<td>0.01</td>
<td>0.00</td>
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<tr>
<td>Time × GAD</td>
<td>2815.63</td>
<td>2.54</td>
<td>2.32†</td>
<td>0.06</td>
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<td>Error</td>
<td>1213.67</td>
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### Respiratory Sinus Arrhythmia

<table>
<thead>
<tr>
<th>Effect</th>
<th>Mean Square</th>
<th>df</th>
<th>F</th>
<th>$\eta_p^2$</th>
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<tbody>
<tr>
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<td>2.90</td>
<td>2.99*</td>
<td>0.08</td>
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<tr>
<td>GAD</td>
<td>14.90</td>
<td>1.00</td>
<td>2.78†</td>
<td>0.07</td>
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<tr>
<td>Time × GAD</td>
<td>0.17</td>
<td>2.90</td>
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<td>107.27</td>
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95% Confidence Interval

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<th>Standard Error</th>
<th>Lower Bound</th>
<th>Upper Bound</th>
</tr>
</thead>
<tbody>
<tr>
<td>t1 to t2</td>
<td>0.40*</td>
<td>0.12</td>
<td>0.05</td>
<td>0.75</td>
</tr>
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</table>

† p < .10, * p < .05, ** p < .01; t1 = Baseline, t2 = Fear Film, t3 = Fear Film Recovery Period, t4 = Sad Film, t5 = Sad Film Recovery Period. Greenhouse-Geisser correction applied to repeated measures ANOVA statistics.
Figure 3. Changes in self-report (fear and sadness) and physiological (LVET, SV, PEP, and RSA) indices throughout the fear film first condition.
Sad Film First (SFF) Condition. Repeated measures ANOVA results are presented in Table 3 and means for each dependent variable across the study are depicted in Figure 4. With self-reported sadness as the dependent variable, there was a trend for a main effect for time, \( F(3.18, 120.76) = 2.28, p = .08, \eta^2_p = .06 \). Sadness was higher during the sad recovery period (t3; \( M = 2.03, SE = .38 \)) than the baseline period (t1; \( M = 1.23, SE = .27; p < .05 \)). There was no main effect of GAD status or interaction between time and GAD status. These findings indicate that sadness was highest during the sad recovery period (rather than the sad film period), as compared to baseline, and otherwise did not change significantly.

With self-reported fear as the dependent variable, there was a significant main effect of time, \( F(2.38, 90.58) = 8.17, p < .01, \eta^2_p = .18 \). Fear was higher during the fear film (t4; \( M = 1.65, SE = .36 \)) than at baseline (t1; \( M = .53, SE = .20; p < .05 \)), the sad recovery period (t3; \( M = .43, SE = .17; p < .01 \)), and the fear recovery period (t5; \( M = .83, SE = .23; p < .05 \)). Fear ratings were also marginally higher during the fear film than during the sad film (t2; \( M = .88, SE = .28; p = .08 \)). There was no main effect of GAD status nor was there a significant interaction between time and GAD status. Overall, participants both with and without GAD rated their fear as highest during the fear film than during any of the other time points.

Mean levels of LVET differed significantly across the five time points, \( F(2.64, 95.12) = 3.33, p < .05, \eta^2_p = .09 \); however, post hoc comparisons among time points indicated no significant contrasts between time points. There was a trend indicating lower LVET at baseline (t1; \( M = 274.06, SE = 4.27 \)) than during the fear recovery period (t5; \( M = 283.11, SE = 4.26; p = .06 \)). There was no main effect of GAD status. A trend emerged for the interaction between time and GAD status, \( F(2.64, 95.12) = 2.41, p = .08, \eta^2_p = .06 \). The post hoc analysis for participants with GAD indicated lower LVET at baseline (t1; \( M = 280.82, SE = 6.04 \)) than the
fear recovery period (t5; $M = 289.71, SE = 6.03; p < .05$) and lower LVET during the sad recovery period (t3; $M = 275.28, SE = 7.00$) than the fear recovery period ($p < .05$). In contrast, LVET differed significantly at several time points for the control group, with lower LVET at baseline (t1; $M = 267.31, SE = 6.04$) than the sad film (t2; $M = 286.09, SE = 7.43; p < .05$), the fear film (t4; $M = 294.50, SE = 7.83; p < .01$), and the fear recovery period (t5; $M = 276.50, SE = 6.03; p < .05$). Among control participants, LVET was also lower during the sad recovery period (t3; $M = 270.92, SE = 7.00$) than the sad film ($p < .05$) and the fear film ($p < .01$). A trend emerged for the difference between LVET during the fear film and the fear recovery period ($p = .06$). Thus, LVET changed less for participants with GAD than for healthy controls, who experienced increased LVET (i.e., increased SNS activation) in response to both films and decreased LVET during the baseline and recovery periods. Participants with GAD showed diminished LVET during the sad recovery period and increased LVET during the fear recovery period. No significant main effects or interactions emerged for SV or PEP, the other indices of SNS activity.

With RSA as the dependent variable, the main effect of time was statistically significant, $F(2.84, 105.05) = 9.75, p < .01, \eta_p^2 = .08$. The post hoc analysis indicated higher RSA at baseline (t1; $M = 6.98, SE = .16$) than during the sad film (t2; $M = 6.43, SE = .16; p < .05$), the sad recovery period (t3; $M = 6.52, SE = .14; p < .01$), the fear film (t4; $M = 6.28, SE = .15; p < .01$), and the fear recovery period (t5; $M = 6.50, SE = .15; p < .05$). This pattern demonstrates that RSA, and therefore PNS activation, was significantly lower across all participants during films and recovery periods as compared to baseline. There was no main effect of GAD status and the interaction between time and GAD status was not significant.
In summary, all participants reported heightened sadness during the sad recovery period and heightened fear during the fear film. Furthermore, participants in the control group evinced a stress response, typically associated with fear (i.e., high SNS and low PNS activity in LVET and RSA, respectively) during both the sad and fear films with decreased SNS activity during both recovery periods. In contrast, participants with GAD experienced blunted reactivity (i.e., lower SNS and PNS activity in LVET and RSA, respectively) during the sad recovery period and a heightened fear response (i.e., higher SNS and lower PNS activity in LVET and RSA, respectively) during the fear recovery period. For participants with GAD, physiology did not change significantly from baseline during the film clips.
Table 3

Repeated Measures ANOVA and Significant Bonferroni Comparisons for Self-Reported Fear, Self-Reported Sadness, LVET, and RSA during the Sad Film First Condition

<table>
<thead>
<tr>
<th>Effect</th>
<th>Mean Square</th>
<th>df</th>
<th>F</th>
<th>η²p</th>
<th>95% Confidence Interval</th>
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<tr>
<td></td>
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<td></td>
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<tr>
<td><strong>Self-Reported Sadness</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>6.96</td>
<td>3.18</td>
<td>2.28†</td>
<td>0.06</td>
<td></td>
</tr>
<tr>
<td>GAD</td>
<td>11.52</td>
<td>1.00</td>
<td>0.91</td>
<td>0.02</td>
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<tr>
<td>Time × GAD</td>
<td>2.42</td>
<td>3.18</td>
<td>0.79</td>
<td>0.02</td>
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<tr>
<td>Error</td>
<td>3.05</td>
<td>120.76</td>
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<td></td>
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<tr>
<td><strong>Comparisons</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>t1 to t3</td>
<td>-0.80*</td>
<td>0.26</td>
<td>-1.58</td>
<td>-0.02</td>
<td></td>
</tr>
</tbody>
</table>

| **Self-Reported Fear** |             |     |      |     |                         |
| Time                 | 15.56       | 2.38| 8.17**| 0.18|                         |
| GAD                  | 0.72        | 1.00| 0.09 | 0.00|                         |
| Time × GAD           | 1.84        | 2.38| 0.97 | 0.03|                         |
| Error                | 1.91        | 90.58|      |     |                         |
| **Comparisons**      |             |     |      |     |                         |
| t4 to t1             | 1.13*       | 0.33| 0.15 | 2.10|                         |
| t4 to t2             | 0.78†       | 0.28| -0.06| 1.61|                         |
| t4 to t3             | 1.23**      | 0.32| 0.27 | 2.18|                         |
| t4 to t5             | 0.83*       | 0.26| 0.06 | 1.59|                         |

| **Left Ventricular Ejection Time** |             |     |      |     |                         |
| Time                 | 2525.01     | 2.64| 3.33*| 0.09|                         |
| GAD                  | 387.79      | 1.00| 0.15 | 0.00|                         |
| Time × GAD           | 1824.13     | 2.64| 2.41†| 0.06|                         |
| Error                | 757.61      | 95.12|      |     |                         |

<table>
<thead>
<tr>
<th></th>
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<th>GAD Group</th>
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<tr>
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<td></td>
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<tr>
<td>95% Confidence Interval</td>
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<td>Comparisons</td>
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<td>Standard Error</td>
</tr>
<tr>
<td>-------------</td>
<td>----------------</td>
<td>----------------</td>
</tr>
<tr>
<td>t1 to t2</td>
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</tr>
<tr>
<td>t1 to t4</td>
<td>-27.19**</td>
<td>7.91</td>
</tr>
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<td>t1 to t5</td>
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<td>t2 to t3</td>
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<td>5.98</td>
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</tr>
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<td>t3 to t5</td>
<td>ns</td>
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</tr>
<tr>
<td>t4 to t5</td>
<td>18.00†</td>
<td>9.24</td>
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**Respiratory Sinus Arrhythmia**

<table>
<thead>
<tr>
<th>Effect</th>
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<th>df</th>
<th>F</th>
<th>(\eta_p^2)</th>
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<tr>
<td>Time</td>
<td>3.74</td>
<td>2.84</td>
<td>9.75**</td>
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<tr>
<td>GAD</td>
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<td>1.21</td>
<td>0.03</td>
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<td>Time (\times) GAD</td>
<td>0.24</td>
<td>2.84</td>
<td>0.61</td>
<td>0.02</td>
</tr>
<tr>
<td>Error</td>
<td>0.38</td>
<td>105.05</td>
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<tr>
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<td>0.55*</td>
<td>0.16</td>
<td>0.09</td>
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<td>t1 to t3</td>
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<td>0.12</td>
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<td>0.83</td>
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<td>t1 to t4</td>
<td>0.70**</td>
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<td>1.13</td>
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<td>0.48*</td>
<td>0.14</td>
<td>0.07</td>
<td>0.88</td>
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† \(p < .10\), * \(p < .05\), ** \(p < .01\); t1 = Baseline, t2 = Sad Film, t3 = Sad Film Recovery Period, t4 = Fear Film, t5 = Fear Film Recovery Period.

\(\hat{p} < .05\).
Figure 4. Changes in self-report (fear and sadness) and physiological (LVET, SV, PEP, and RSA) indices throughout the sad film first condition.
Post Hoc Analyses

The repeated measures ANOVA revealed that the GAD and control groups differed in their trajectories through the protocol in both conditions, though in different ways. Given the pattern of findings, repeated measures ANCOVA was used to determine whether the relation between GAD and emotional responding remained significant when including the influence of general emotion tendencies (i.e., DERS and PANAS-Negative Affect) and cognitions (i.e., positive or negative valence of thoughts were during the recovery periods) in separate models. Based on the pattern of significant findings in the repeated measures ANOVA analyses, ANCOVAs were conducted for fear, sadness, LVET, and RSA in the FFF condition, while these analyses were conducted only for LVET in the SFF condition. This yielded a total of 20 ANCOVAs; to avoid potential inflation of type I error, a Bonferroni correction was applied so that, to be significant, $p$ had to be less than or equal to .0025. Although these emotional and cognitive measures may have been related to general responding throughout the study, only models where inclusion of these measures significantly changed the findings reported previously are presented.

In the FFF condition, the PANAS-Negative Affect scale influenced the pattern of findings such that, when included as a covariate predicting sadness, the effects of both time and GAD status became non-significant and a main effect of PANAS-Negative Affect emerged, $F(1, 33) = 22.68, p < .001, \eta_p^2 = .41$. This main effect indicated that participants with higher levels of general negative affect reported greater sadness across all study time points. There were no other models where including these covariates influenced the relation between GAD status and self-report or physiological outcomes. Furthermore, in the SFF condition, including these measures as covariates did not change the pattern of reported findings with LVET.
Chapter 4

DISCUSSION

The goal of this study was to determine whether individuals with GAD can shift between emotional states and the relation to disruptions in emotional reactivity and regulation. Results indicate that participants with GAD were able to shift between fear and sadness, particularly in terms of their subjective experience. However, GAD was associated with hyperreactivity in self-reported emotions in the FFF condition and a sustained physiological response following the second film clip in both conditions indicative of emotional inertia. These findings suggest that individuals with GAD experience differing degrees of difficulty in both reactivity and regulation of emotions depending on the characteristics of the situation.

Baseline Differences between GAD and Control Groups

The first hypothesis, stating that participants in the GAD group would demonstrate higher self-reported negative emotion (i.e., fear and sadness) at baseline, was supported. Participants with GAD reported that they were experiencing higher levels of fear and sadness both at the start of the study protocol and following the relaxation baseline period. However, similar to previous research on this population (e.g., Fisher et al., 2010; Llera & Newman, 2010), there were no differences in physiological responding at baseline between the groups. This divergence between subjective experience and physiological response could reflect the tendency for individuals with GAD to report chronically high levels of negative affect without necessarily demonstrating the corresponding physiological arousal (Brown, Chorpita, & Barlow, 1998). Furthermore, heightened self-reported negative affect is consistent with models highlighting the role of pervasive, subjective negative affectivity in GAD (Mennin et al., 2005, 2007), while
heightened physiological arousal is often seen in response to emotional stimuli instead of at baseline (Fisher et al., 2010; Peasley-Miklus & Vrana, 2000).

**Emotional Responding among Control Participants**

The second hypothesis, that participants in the control group would experience increases in self-report and physiological indices of each emotion corresponding with the presented film clip and would return to baseline during the recovery periods, was partially supported. In both conditions, self-reported fear and sadness increased in response to the corresponding film clips and decreased during the recovery periods. One exception was with sadness in the SFF condition, where sadness was highest during the sad recovery instead of the sad film, which could reflect the lingering nature of sadness and how it is not necessarily tied to the immediacy of threat, like fear (Power & Dagleish, 1997).

The expected emotion profiles did not clearly emerge in physiology. Specifically, in the FFF condition, SNS activity did not change significantly throughout the study, but PNS activity decreased only during the fear film. Therefore, instead of corroborating the changes in emotion reported by participants, physiology indicated that participants were generally non-reactive throughout the protocol with the exception of being less well-regulated (i.e., lower PNS activity) during the fear film. They also did not show lower PNS activity during the sad film, suggesting that they were able to maintain a more well-regulated state. In contrast, participants in the SFF condition evinced a physiological response consistent with fear (i.e., increased SNS and PNS activation) during both the fear and the sad film and a return to baseline during the recovery periods. Although most evidence indicates that SNS activity decreases in response to sad stimuli, there is also some evidence that, with more intense sadness, SNS activity can increase (Davydov, Zech, & Luminet, 2011). It is therefore possible that the presented sad film clip
contained more intense sadness than what has typically been studied. This pattern in the SFF condition differed from the FFF condition in that those in the SFF condition demonstrated a fear response to both films while those in the FFF condition showed fewer changes and blunted responding to the fear film. Therefore, the order of film presentation influenced the overall intensity of responding to the protocol.

Despite these mixed findings, participants in the control group demonstrated less emotional arousal in self-report and physiology during the baseline and recovery periods across conditions, whereas responding during the film clips, especially in self-report measures, indicated appropriate reactivity to that stimulus. Furthermore, in each condition but especially the SFF condition, control participants responded with similar physiology to the first and second film clips, possibly indicating that the physiological response to the first film was “sensitized” and recurred more easily in response to the following film. Thus, control participants’ subjective experience differentiated between film clips, while their physiology indicated a similar response.

**Emotional Responding among Participants with GAD**

In Hypothesis 3, I tested whether participants with GAD would demonstrate either the *hyperreactivity pattern* (higher fear and sadness to the corresponding films than control participants with return to baseline during recoveries) or *emotional inertia pattern* (reactivity to the first film that lasts through the other film and recovery periods). In both study conditions, the findings were mixed. When the fear film was presented first, participants with GAD exhibited heightened self-reported sadness and fear compared to the control group throughout the study with decreases during the recovery periods, supportive of the hyperreactivity pattern. The decrease in reported negative emotion during the recovery periods indicates some degree of regulation that occurred for this group, at least at the subjective level. Furthermore, participants
with GAD reported increases in the appropriate emotion to each film (i.e., sadness with the sad film, fear with the fear film), clearly not indicating emotional inertia. In the SFF condition, however, participants with GAD reported the same degree of sadness and fear as the control participants, suggesting that hyperreactivity was only evident when fear, the central emotion associated with GAD, was elicited first. This suggests that when individuals with GAD are faced with a non-fear-related stimulus, they do not report heightened negative emotion even when a subsequent fear stimulus is presented; however, when the fear stimulus appears first, it leads to the typically observed heightened fear response and this hyperreactivity continues into the next emotion induction.

The pattern depicted by the physiological measures diverged in important ways from the self-report measures. In the FFF condition, participants with GAD displayed a blunted response (i.e., low SNS and PNS activity) to the fear film and recovery period, indicating potentially less processing of the emotional stimulus (Foa & Kozak, 1986) and a profile often associated with emotional responding after worrying among individuals with GAD (Borkovec et al., 2004; Fisher & Newman, in press; Llera & Newman, 2010). It is certainly possible that participants with GAD worried prior to the fear film (e.g., worrying about what would come next in the study while rating their emotions following the relaxation baseline) or even during the beginning of the fear film, though this was not directly assessed. Participants with GAD also experienced a clear “stress” response typically associated with fear (i.e., high SNS and low PNS activity) in response to the sad film that extended through the sad recovery period. This pattern may indicate that participants with GAD experienced a delay in their physiological fear response that precluded the typical sad physiological profile and extended through the second recovery period. In contrast, it could also be that participants with GAD did not respond as strongly to a fear
stimulus because it is congruent with their typical or preferred mood, consistent with the implications of Newman and Llera’s (2011) theory that individuals with GAD may maintain a negative state through worry to prevent an unexpected change. In this case, it seems possible that these participants became more stressed when exposed to an emotion other than the one they are most accustomed to (i.e., sadness as opposed to fear/anxiety). These findings do not support a pattern of hyperreactivity but instead might reflect an emotional inertia response, with the caveat that the results highlighted were primarily statistical trends and not consistent, and therefore require further replication.

In the SFF condition, participants with GAD did not show significant differences from baseline during the sad or fear films and instead experienced differences in reactivity during the recovery periods. Specifically, they evinced a blunted response (i.e., low SNS and PNS activity) during the sad recovery period and a heightened fear response (i.e., high SNS and low PNS activity) during the fear recovery period, indicative of emotional inertia following the fear film. Participants with GAD had the reverse experience than control participants in that they had increased, instead of decreased, SNS activity during the recovery.

Interestingly, in both study conditions, participants with GAD displayed blunted reactivity to the first emotion induction and/or first recovery period. It was only in response to the second film clip and recovery period, irrespective of which emotion was elicited, that participants with GAD experienced a fear or stress physiological response and sustained responding through the recovery period suggesting that emotional inertia may have occurred. This adds a caveat to models that highlight emotion regulation deficits as central in GAD (e.g., Mennin et al., 2002, 2005); specifically, individuals with GAD may be able to dampen their responding following the first exposure to an emotional situation, potentially by using
avoidance-based strategies that result in blunted physiological reactivity (e.g., worry; Borkovec & Hu, 1990; Peasley-Miklus & Vrana, 2000). However, when that situation is followed by another emotion, difficulties with regulation emerge more strongly.

Although hyperreactivity may be evident, particularly in self-report measures, in response to the initial stimulus, the lack of emotion regulation resources becomes especially evident with ongoing exposure to emotion. This pattern has been observed in the larger field of self-regulation research as “ego depletion”, or the tendency for individuals to have limited self-regulatory resources wherein repeated efforts to regulate emotions, cognitions, volitions, and actions lead to increasing difficulty with effectively completing these acts (Baumeister, Bratslavsky, Muraven, & Tice, 1998). Indeed, high trait-level ego depletion (i.e., low self-control) has been associated with poor psychological functioning and adjustment (Tangney, Baumeister, & Boone, 2004). Therefore, it is possible that participants with GAD were unable to regulate effectively during the second film and recovery because of a limited overall capacity for self-regulation that was exhausted with participants’ efforts to regulate during the first induction.

Unlike the self-report findings, which support the hyperreactivity pattern in the FFF condition and neither pattern in the SFF condition, the physiology findings indicate emotional inertia most frequently in both study conditions. These findings are not consistent with the bulk of emotional inertia studies that show that individuals with emotional difficulties, such as depression and low self-esteem (Koval & Kuppens, 2012; Kuppens et al., 2010), demonstrate emotional inertia in self-report measures, though these studies have not included physiological recording. Furthermore, the present findings indicate that more variability is evident on a shorter time scale than has typically been studied (e.g., across a day). This may also indicate important differences between the emotional experiences of those with major depression and GAD. While
depression may be associated with the subjective experience of chronic emotional inertia (e.g., emotional context insensitivity; Rottenberg et al., 2005), individuals with GAD report strong reactions to emotional stimuli with more variability (i.e., hyperreactivity across multiple emotions with recovery between).

Another important finding was the inconsistency between self-reported emotion and physiological indicators of emotional experience across participants, which has been found in previous studies of healthy participants (see Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005 for a review). As found here, previous research has found that this divergence is particularly strong among participants with GAD (Fisher et al., 2010). In addition to the influence of contrasting subjectively distressed versus not distressed groups, this may also reflect the difficulty that individuals with high anxiety have with accurately identifying the components of their emotional response (Bagby, Parker, & Taylor, 1994; Mennin et al., 2005). Heightened reports of subjective negative emotion, consistent in this study with the proposed hyperreactivity pattern, replicates past research where higher negative emotion has also been found only at the self-report level (Llera & Newman, 2010; Mennin et al., 2005, 2007). Also similar to previous research with GAD (Fisher et al., 2010), emotional inertia was only evident in physiology, suggesting that individuals with GAD may not be directly aware of this sustained emotional response at the level of rating their subjective emotions.

**Post Hoc Analyses**

The post hoc analyses were completed to aid in understanding whether GAD accounted for participants’ responding to the emotional stimuli beyond general difficulties with emotion and negative cognitions. In the vast majority of cases, the pattern of findings observed in the relation between GAD status and emotional responding was significant beyond the contribution
of these measures. This suggests that the hyperreactivity and regulation difficulties observed in individuals with GAD may be best understood as related to the confluence of difficulties in GAD as opposed to any one component of the disorder. However, it could also be that the measures used in this study do not capture the broad range of factors that contribute to the disorder, so future research aimed at elucidating this further could include a wider assessment of factors, such as measures of general cognitive style, use of behavioral and cognitive/emotional avoidance, and more specific aspects of how participants with GAD handle their emotions moment-to-moment.

Despite this overall finding, when chronic high negative affect was included in the model for GAD status and self-reported sadness in the FFF condition, GAD was no longer associated with sadness. This pattern indicates that chronically high levels of negative affect likely persisted through the study and contributed to the heightened sadness reported during the FFF condition, beyond the effect of the emotion induction.

**Broad Implications**

The findings presented herein have important implications for conceptualizing GAD and the role of emotional functioning. Consistent with Mennin and colleagues’ Emotion Dysregulation Model (Mennin et al., 2005), results indicated that individuals with GAD are distinguished from a control group by their differences in emotional reactivity and regulation. However, these findings further suggest that this model could be made more specific by noting the hyperreactivity in subjective experience among those with GAD, particularly when faced with fear-inducing stimuli, as well as the cumulative effects of emotion regulation problems evident when individuals experienced multiple emotional stimuli in succession. In addition, the pattern of responding observed in this study may resemble how individuals with GAD respond when not able to “protect” themselves from emotional contrast through worry, as noted in the
Contrast Avoidance Model (e.g., being unable to prevent unexpected changes to new emotional states; Newman & Llera, 2011). When starting in a relatively neutral state (i.e., comparison to a relaxation baseline), participants with GAD do report stronger negative emotion and demonstrate difficulty regulating back to a relaxed state following multiple emotional stimuli, suggesting that their typical defense of maintaining negativity through worry might be preferable to this emotional “rollercoaster”. Indeed, these findings indicate that avoidance of emotional processing might be relatively more effective at decreasing reactivity in GAD since they tend to experience their emotions as more intense and become potentially overwhelmed in complex situations with multiple emotions activated.

Furthermore, in line with the functionalist approach to emotions, these findings suggest that individuals may miss important information about their environment due to problematic elements of their response (Frijda, 1988; Keltner & Gross, 1999). Heightened reactivity to the stimulus may lead to a stronger reaction to the situation than is called for, while inertia following an emotional event could lead to an extended, inappropriate response. We can observe this in the present study where participants with GAD reported heightened negative emotion at the subjective level and exhibited physiology indicative of heightened fear or distress as the protocol continued, suggesting that these participants received ongoing information that their environment was threatening, potentially leading to continued, unnecessary distress. Determining the impacts of the potential misinformation individuals with GAD receive due to their emotional responding deficits on both intrapersonal (e.g., decision-making, avoidance behavior) and interpersonal systems would be a very important area of future research.

Strengths and Limitations
This study expanded upon past research by addressing the important topic of the experience of emotional reactivity and regulation in GAD, thus addressing an important gap in the understanding of the specific nature of the disorder’s emotional deficits. Furthermore, the study benefited from a view of emotion that was both uniquely large and focused in scope; large in the inclusion of reactivity to and recovery from two subsequent emotional stimuli as opposed to reaction to one stimulus, and focused on moment-to-moment changes in reaction to specific stimuli instead of changes in mood over the course of hours or days (e.g., ecological momentary assessment designs; Koval & Kuppens, 2012; Kuppens et al., 2010; Thompson et al., 2012). Frequent self-report ratings and continuous physiological measurement provided a micro-level assessment of emotional responding across the presentation of multiple emotional stimuli and recovery periods. This study design provided the specificity needed to capture the level of variability involved in the unfolding of an emotional response.

Despite these strengths, a number of limitations are noteworthy. In particular, certain characteristics of the sample may have contributed to the multitude of statistical trends instead of clearly significant findings. The sample size (n = 40 in control group and n = 40 in GAD group) may not have been large enough to provide the statistical power to detect significant differences. Furthermore, the inclusion of a community-based sample of individuals with GAD instead of treatment-seeking individuals could have led to an overrepresentation of individuals with GAD who were functioning well enough overall. However, rates of seeking treatment are relatively low in GAD (Vesga-López et al., 2008), so this sample may be more representative of how GAD functions in the general community. In addition, CSRs for participants with GAD indicated moderate to severe symptom severity, suggesting that these participants were not experiencing subthreshold GAD. Another important limitation to note is the inherent lack of specificity in
physiological measures. Physiological changes occur in response to many factors in the internal and external environment aside from emotions; however, in the current study, care was taken to minimize other confounding variables that could impact physiology (e.g., physical movement, extraneous stimuli in the laboratory). Finally, although the emotional stimuli used were standardized using prior samples, there may nevertheless be natural variability across individual responses to the films, as well as the potential for multiple emotions (i.e., beyond fear and sadness) to be elicited by the stimuli within one individual.

Conclusions

This study expanded on previous research on emotion and GAD by providing a focused view of the specific nature of emotional deficits in this disorder. It illuminated how both emotional hyperreactivity and emotion dysregulation may play differential roles in their impact on emotional functioning in GAD. This type of research is important in broadening our understanding of the nature of problems in GAD, both for understanding why GAD is associated with certain functional deficits (e.g., emotional avoidance, overreacting to situations) and for providing more effective clinical intervention to reduce distress. For example, these findings indicate that the cumulative impact of multiple emotion inductions is important, indicating the need for further study on how individuals experiencing GAD or other psychopathology handle complex emotional situations. Furthermore, clinicians might provide more comprehensive support in building emotion regulation skills, such as by encouraging the use of physiology-focused strategies (e.g., deep breathing, progressive muscle relaxation) throughout the course of an emotional situation (i.e., beyond the first exposure to a stimulus) or using cognitive reappraisal or other effective strategies to decrease the subjective heightened negative emotion.
Future research in this area will be integral in extending these findings to understand the specific nature of the range of emotional deficits in GAD.
REFERENCES


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