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**AN EXPERIMENTAL EXAMINATION OF EMOTIONAL AVOIDANCE IN  
GENERALIZED ANXIETY DISORDER:  
SUPPORTING A NEW THEORY OF EMOTIONAL CONTRAST AVOIDANCE**

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by

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## ABSTRACT

An important emphasis of the recent literature on generalized anxiety disorder (GAD) is improved understanding of the function of emotion dysregulation in the etiology and maintenance of this disorder. The purpose of the present study is to propose and test a new model for conceptualizing emotional sequelae in GAD, which is defined as the Contrast Avoidance Model of Worry. The model states that individuals with GAD are more sensitive to unexpected negative events, and that worry (the key pathological feature of GAD) functions to prolong and maintain a negative emotional state, thereby avoiding an unexpected, sharp shift (or contrast) in negative emotions. To test this model, the present study examined the effect of worry on physiological and subjective negative emotionality, both during worry inductions and in response to negative and positive emotional stimuli. Participants with GAD and nonanxious controls were randomly assigned to engage in worry, relaxation, or neutral inductions prior to sequential exposure to each of three emotion-inducing film clips designed to elicit fear, sadness, and happiness. Self reported emotionality was assessed at baseline and following each induction and exposure, and physiological arousal (both sympathetic and parasympathetic activity) was measured throughout. Results demonstrate that worry led to an increase in negative emotionality that was sustained across negative exposures. Also, participants with GAD described this experience as more helpful in emotional coping than did controls, providing partial support for the Contrast Avoidance model. A contextual understanding of this model in relation to extant models of emotional functioning in GAD is provided.

## TABLE OF CONTENTS

List of Tables .....	vii
List of Figures .....	viii
INTRODUCTION .....	1
Worry and Avoidance of Fearful Reactivity in GAD.....	2
Experiential Avoidance?.....	4
Worry Creates and Sustains Negative Emotionality .....	6
An Experimental Investigation of Worry and Emotional Avoidance .....	11
Avoidance of Negative Emotional Contrast: the Contrast Avoidance Model .....	15
Goals of the Present Study .....	18
Hypotheses .....	21
<u>Hypothesis one:</u> Worry leads to a negative emotional state .....	21
<u>Hypothesis two:</u> Following worry, negative emotionality will be sustained through negative emotional exposures.....	21
<u>Hypothesis three:</u> Individuals with GAD would have more difficulty than Controls in coping with negative emotional contrasts.....	22
RESEARCH DESIGN AND METHODS .....	23
Overall Design .....	23
Participants .....	23
Selection criteria .....	25
Emotion-Eliciting Stimuli.....	26
Self-report Measures.....	27
Measures of emotional coping .....	27
Physiological Measures .....	28
Procedure .....	30
RESULTS .....	32
Participant Parameters .....	32
Baseline .....	33
Manipulation Check.....	35
Hypothesis 1: Worry Leads to Heightened Negative Emotional State.....	36
Subjective Measures .....	36
Emotion adjectives .....	37
PANAS scores .....	37
Physiological measures.....	38
SCL scores .....	39

NS-SCR scores.....	40
Amplitude average scores .....	42
Heart rate scores.....	44
RSA scores .....	47
Hypothesis 2: Following Worry, Heightened Negative Emotional State is Sustained Through Negative Exposures, and Alleviated During Positive Exposure ....	49
Fear exposure .....	49
Subjective Measures .....	49
Physiological Measures .....	52
Sad exposure .....	55
Subjective Measures .....	55
Physiological Measures .....	57
Happy exposure .....	59
Subjective Measures .....	59
Physiological Measures .....	61
Hypothesis Three: Effects of Inductions in Emotional Coping Differ Between GAD and Nonanxious Control Participants .....	64
DISCUSSION .....	65
Hypothesis One: Worry Leads to Heightened Negative Emotionality.....	66
Hypothesis Two: Worry Leads to Sustained Negative Emotion Across Negative Exposures .....	69
Hypothesis Three: GAD Participants Are More Averse to Negative Emotional Contrast.....	73
Comparison Between the Contrast Avoidance Model and Extant Theories of GAD.....	76
Conclusion .....	80
Limitations .....	81
REFERENCES .....	83
APPENDIX A: TABLES.....	101
Table 1: Means and Standard Deviations of Manipulation Check Scores by Group and Group and Induction Type.....	101
Table 2: Absolute levels of emotion adjective ratings during inductions .....	102
APPENDIX B: FIGURES .....	103
Figure 1: Emotional Coping Scale.....	103
Figure 2. Change in amplitude average scores from baseline to the 1st minute of the pre-fear induction .....	105

Figure 3. Change in RSA scores from baseline to both minutes of the pre-fear induction .....	106
Figure 4. Change in reported Fear from the baseline to induction to fearful exposure for both groups .....	107
Figure 5. Change in negative affect (NA) from the baseline to induction to fearful exposure for both groups .....	108
Figure 6. Change in amplitude average scores from the induction period to the fearful exposure .....	109
Figure 7. Change in sadness from the baseline to induction to sad exposure for both groups .....	110
Figure 8. Change in negative affect (NA) from the baseline to induction to sad exposure for both groups .....	111
Figure 9. Change in amplitude average scores from the induction period to the sad exposure.....	112
Figure 10. Change in RSA from the induction period to the sad exposure .....	113
Figure 11. Change in happiness from the baseline to induction to happy exposure for both groups .....	114
Figure 12. Change in negative affect (NA) from the baseline to induction to happy exposure for both groups .....	115
Figure 13. Change in RSA from the induction period to the happy exposure.....	116
Figure 14. Emotion coping scores by group and induction type .....	117

**LIST OF TABLES**

Table 1: Means and Standard Deviations of Manipulation Check Scores by Group and Group and Induction Type .....	101
Table 2: Absolute levels of emotion adjective ratings during inductions .....	102

## LIST OF FIGURES

Figure 1: Emotional Coping Scale.....	103
Figure 2. Change in amplitude average scores from baseline to the 1st minute of the pre-fear induction .....	105
Figure 3. Change in RSA scores from baseline to both minutes of the pre-fear induction .....	106
Figure 4. Change in reported Fear from the baseline to induction to fearful exposure for both groups .....	107
Figure 5. Change in negative affect (NA) from the baseline to induction to fearful exposure for both groups .....	108
Figure 6. Change in amplitude average scores from the induction period to the fearful exposure .....	109
Figure 7. Change in sadness from the baseline to induction to sad exposure for both groups .....	110
Figure 8. Change in negative affect (NA) from the baseline to induction to sad exposure for both groups .....	111
Figure 9. Change in amplitude average scores from the induction period to the sad exposure.....	112
Figure 10. Change in RSA from the induction period to the sad exposure .....	113
Figure 11. Change in happiness from the baseline to induction to happy exposure for both groups .....	114
Figure 12. Change in negative affect (NA) from the baseline to induction to happy exposure for both groups .....	115
Figure 13. Change in RSA from the induction period to the happy exposure.....	116
Figure 14. Emotion coping scores by group and induction type .....	117

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## Introduction

An expansive literature links dysregulated emotionality with generalized anxiety disorder (GAD) (e.g., Llera & Newman, 2010; Mennin, Heimberg, Turk, & Fresco, 2005; Roemer, Salters, Raffa, & Orsillo, 2005; Salters-Pedneault, Tull, & Roemer, 2004; Turk, Heimberg, Luterek, Mennin, & Fresco, 2005), and much research has been devoted to understanding the cyclical relationship between worry, the cardinal feature of GAD, and emotion regulation problems. A recent theory states that worry may facilitate avoidance of somatic aspects of emotional responding (Borkovec, Alcaine, & Behar, 2004). Later models have suggested that worry may facilitate experiential avoidance of negative emotions altogether. Contrary to later models, however, is the fact that worry itself is defined as part of a negative emotional process, and GAD is considered to be a disorder strongly associated with negative affect. To reconcile the conceptual dissonance in current models of emotional sequelae in GAD, a greater understanding of the relationship between worry and emotional dysregulation is needed.

The aim of the present study is to examine the causal relationship between worry and negative emotionality, both directly (through observation of emotional experiencing *during* worry) and indirectly (through observation of the effects of worry on *subsequent* emotional stimuli). In this study, we first outline and critique the current perspective that worry leads to emotional avoidance and review extant literature on the relationship between worry and negative emotion. Next, we present and test a new theory – the Emotional Contrast Avoidance Model of worry (Newman & Llera, 2011) – that proposes to reconcile the disparities in current perspectives on worry, GAD, and negative emotionality. Finally, we provide a comparison between this new model and current leading models of worry and emotion in GAD.

## **Worry and Avoidance of Fearful Reactivity in GAD**

Borkovec and colleagues' Cognitive Avoidance Theory of Worry (Borkovec, 1994; Borkovec et al., 2004) has played a pivotal role in understanding the relationship between worry and emotional dysfunction. This model suggests that worry functions as a cognitive avoidance response to perceived future threats and may facilitate the avoidance of some aspects of fearful responding. How is it that the process of worry, so closely linked to anxiety, may function as avoidance of fearful responding? Borkovec and colleagues provide a comprehensive argument for how this avoidance may be achieved.

The first mechanism proposed by the authors is that worry facilitates avoidance of potential future threats. If a threat is anticipated but not currently present in the external environment, it remains an abstract concept in the mind and thus cannot be behaviorally avoided. Therefore, cognitive problem solving may function as a primary avoidance response. As such, the Cognitive Avoidance theory posits that worry is employed as an attempt to avoid future threats via problem solving and planning. Indeed, a survey on the perceived functions of worry indicates that individuals both with and without GAD rank the avoidance of bad events as among their highest motivators for worry (Borkovec & Roemer, 1995; Freeston, Rheume, Letarte, Dugas, & Ladouceur, 1994). Moreover, worry is shown to be correlated with problem-focused coping, and content analysis reveals that nearly half of worrisome thoughts represent engagement in problem-solving (Davey, 1994; Szabó & Lovibond, 2006). Interestingly, evidence suggests that the vast majority of feared outcomes that people worry about will never come to pass (Borkovec, Hazlett-Stevens, & Diaz, 1999), which the worrier may superstitiously attribute to the process of worry itself.

However, it is inevitable that some negative life events are bound to happen. Therefore, a second avoidance function in the Cognitive Avoidance model is that worry acts as a *coping mechanism* in response to unavoidable threat. Specifically, this model proposes that when faced with an unavoidable negative event, individuals may employ worry as means to cognitively avoid somatic aspects of fearful responding. The second type of avoidance in this theory is based on data showing that speech anxious participants who worry just prior to imaginal exposure to repeated public speaking images show reduced cardiovascular reactivity to those images, whereas those who think relaxing or neutral thoughts before imaginal exposure show strong cardiac response with habituation across images (Borkovec & Hu, 1990; Borkovec & Sides, 1979; Peasley-Miklus & Vrana, 2000; Vrana, Cuthbert, & Lang, 1986; Vrana, Cuthbert, & Lang, 1989). Moreover, amount of time spent in worrisome thought (as opposed to imagery) during the worrying is negatively correlated with cardiac reactivity to subsequent fearful images, whereas the amount of time spent in relaxed thinking during relaxation is positively correlated with cardiac reactivity to the images (Borkovec, Lyonfields, Wiser, & Deihl, 1993). Taken together, these data suggest that worrisome thoughts lead to inhibited reactivity to fearful stimuli.

In sum, according to the Cognitive Avoidance Theory, worry may be employed to a) strategically avoid future threats via problem solving, and/or b) reduce somatic activation associated with threat perception (Borkovec, 1994; Borkovec et al., 2004). Given that worry can be perceived as facilitating avoidance of threat and/or threat-response, this theory also suggests that worry will be negatively reinforced. However, according to Foa and Kozak (1986), a failure to respond to threatening material on either subjective or physiological levels precludes emotional processing necessary for extinction. Thus, Borkovec and colleagues suggest that

worry precludes emotional processing that would otherwise generate reductions in fearful associations.

### **Experiential Avoidance?**

Given that worry has been proposed to preclude processing of fearful stimuli, other theories have extended the Cognitive Avoidance model to suggest that worrisome thinking may be associated with avoidance of emotional experience or arousal more generally (Mennin, Heimberg, Turk, & Fresco, 2002; Newman, Castonguay, Borkovec, & Molnar, 2004; Roemer et al., 2005). Indeed, it has been purported that individuals who demonstrate discomfort with and avoidance of fear processing are likely to be bothered by a range of emotions as well as other internal experiences. Here it would be useful to review the concept of *experiential avoidance* as discussed by Hayes and colleagues (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996).

Hayes and colleagues (1996) describe the concept of experiential avoidance as an unwillingness to tolerate certain private experiences (e.g., thoughts, emotions, physical sensations, memories). The authors emphasize that many important internal experiences are difficult to endure (e.g., fright, grief, painful memories), but also serve such crucial functions as facilitating emotional learning (e.g., reducing anxious associations) or providing psychological impetus necessary for change. Though a certain amount of avoidance of negative internal experiences is healthy (e.g., extreme physical pain), Hayes et al. argue that pervasive avoidance of negative internal experience can itself become a major problem leading to psychopathological conditions. For example, engaging in drug/alcohol abuse to avoid grief, remaining isolated to avoid anxiety-provoking social situations, and self-harming (e.g., cutting) to distract from overwhelming emotions may all be signs of psychopathological functioning with avoidance of

painful internal experience as a common source. Furthermore, Williams and colleagues (Williams, Chambless, & Ahrens, 1997) suggest that fear and avoidance of subjective internal experiences may be associated with a fear of *loss of control* over these experiences. As such, the experience of emotions, including those in the positive realm, may become categorized as threatening if it is perceived as being out of the subject's control.

As stated above, the concept of experiential avoidance has been theoretically linked to individuals with GAD. Mennin and colleagues (Mennin et al., 2002) proposed that individuals with GAD find their own emotional responses overwhelming which leads to attempts at suppression via cognitive attention shifting (i.e., worry). They note that this leads to inflexible problem solving and paradoxically inflated subjective emotional experiences. Newman and colleagues (Newman et al., 2004) posit that worrisome thinking as a way to avoid painful emotions may represent a major underlying mechanism maintaining GAD symptomatology, and stress that this can interfere therapeutically with accessing emotions necessary for change. Roemer and colleagues (Roemer & Orsillo, 2002; Roemer et al., 2005) combine several theories to propose that GAD is a disorder of experiential avoidance resulting from the development of threatening associations to emotional responding.

In support of these theories, there is evidence to suggest that individuals with GAD do experience subjective discomfort with a range of emotions beyond fear. For example, studies have found that persons with GAD report higher perceived threat of emotions (i.e., fear/anxiety, anger, sadness/depression, guilt, positive emotions, and strong emotions in general), greater fear of the negative consequences of these emotions, less emotional acceptance, and less perceived control over their emotions than do non-anxious controls (Llera & Newman, 2010; Mennin et al.,

2005; Roemer et al., 2005). Individuals with GAD also report greater fear of depression than those with another anxiety disorder (Turk et al., 2005).

Research also shows that individuals with GAD report difficulty with emotion management, and demonstrate a tendency to under-regulate their emotions. For example, participants with GAD report experiencing more difficulty recovering from a negative mood state than nonanxious controls (Turk et al., 2005). In an experimental design, people with GAD reported greater difficulty than nonanxious controls in managing negative mood in response to both anxious and sad mood inductions (Mennin et al., 2005). In another study, GAD analogues were objectively rated as demonstrating more anxiety and sadness than control participants in response to sad and conflictual emotional disclosures by a confederate (Erickson & Newman, 2007). Moreover, when asked why they worry, patients with GAD differed from both nonworried anxious and control participants by indicating that “worry helps distract me from more emotional topics,” (Borkovec & Roemer, 1995; Freeston et al., 1994), supporting theories that individuals with GAD may use their worry to try to control emotional arousal. Together, these studies suggest that persons with GAD experience greater subjective distress associated with a range of emotions than do non-anxious controls.

### **Worry Creates and Sustains Negative Emotionality**

Contrary to later theoretical models of emotion and GAD, Newman and Llera (2011) argue that worry creates and sustains negative emotionality. An important part of this argument lies in the fact that the experimental studies which support the emotion avoidance perspective have focused primarily on the capacity for worry to reduce *subsequent* emotional reactivity during negative emotional exposures, and not on the direct experience of emotionality *during*

worry. Moreover, worry has been defined as a mental process that is negatively affect-laden (Borkovec, Robinson, Pruzinsky, & DePree, 1983), and GAD is shown to be strongly associated with negative affect (Brown, Chorpita, & Barlow, 1998). In the following section, data are reviewed as they pertain to the experiential emotional impact of the worry process.

Overwhelming evidence suggests that individuals experience significant negative emotion caused by worry on both subjective and physiological levels. For instance, laboratory worry inductions lead to elevated subjective ratings of negative emotion (Andor et al., 2008; Borkovec et al., 1993; Borkovec & Inz, 1990; Borkovec et al., 1983; Hofmann, Moscovitch, Litz, Kim, Davis, & Pizzagalli, 2005; Llera & Newman, 2010; Stapinski et al., 2010), including both anxious and depressive responses (Andrews & Borkovec, 1988).

In terms of physiological data, both state and trait worry are associated with changes in autonomic nervous system (ANS) activity. This has been measured in terms of both increased sympathetic nervous system (SNS) activity and decreased parasympathetic nervous system (PNS) activity. Specifically, data show that worry is associated with increased SNS activity including cardiovascular activity, higher skin conductance levels (SCLs), and increased non-specific skin conductance responses (NS-SCRs) (Brosschot, Van Dijk, & Thayer, 2007; Dua & King, 1987; Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Glynn, Christenfeld, & Gerin, 2002; Pieper, Brosschot, van der Leeden, & Thayer, 2007, 2010; Roger & Jamieson, 1988; Segerstrom, Glover, Craske, & Fahey, 1999; Suchday, Carter, Ewart, Larkin, & Desiderato, 2004) and decreased PNS activity as evidenced by reduced heart rate variability (HRV) (Brosschot et al., 2007; Pieper et al., 2007, 2010). Experimentally, laboratory induced worry episodes result in increased SNS activity (Andor et al., 2008; Davis, Montgomery, &

Wilson, 2002; Hammel et al., 2011; Hofmann et al., 2005; Peasley-Miklus & Vrana, 2000; Stapinski et al., 2010; Vrana & Lang, 1990; York, Borkovec, Vasey, & Stern, 1987) and decreased PNS activity (Hammel et al., 2011; Hofmann et al., 2005; Llera & Newman, 2010; Lyonfields, Borkovec, & Thayer, 1995; Thayer, Friedman, & Borkovec, 1996) when compared to neutral tasks, relaxation, or baseline levels.

Based on extant data, Brosschot and colleagues (Brosschot, Gerin, & Thayer, 2006; Brosschot, Pieper, & Thayer, 2005; Brosschot et al., 2007; Pieper et al., 2007) have proposed that worry may lead to sustained negative emotionality, in which the cognitive representation of a stressor is prolonged. Data show that daily worry predicts later autonomic functioning, including high heart rate and low HRV during both sleep and wake (Brosschot et al., 2007). Further, in a recent ambulatory study combining momentary assessment of worry, stress, and mood, whereas stressful events were not associated with heart rate or HRV, worry episodes heightened both concurrent and succeeding cardiac activity for 2 hours following each worry episode (Pieper et al., 2010). The impact of worry on cardiac activity in this study was independent of emotions, physical activity, posture, gender, age, body mass index, smoking, alcohol, coffee consumption, and time of day. In sum, extant data strongly suggest that worry leads to and sustains physiological activation, which supports the concept of an emotionally activating model of worry more so than one of emotional avoidance.

Indeed, Borkovec (1985) has also theorized that the functional effect of worry may be the maintenance of anxiety. Worrying involves repeated rehearsal of catastrophic outcomes (Borkovec, 1985; Davey & Matchett, 1994). Such repetitious catastrophizing can worsen anxiety (Grayson & Borkovec, 1978). Similarly, studies show that worrying immediately after

watching a gruesome film (as opposed to image rehearsal or calming down) led to increased cognitive intrusions over a subsequent three day period (Butler, Wells, & Dewick, 1995; Wells & Papageorgiou, 1995). Thus, although worry may lead to a temporary respite from some aspects of anxious responding, in the long-term it may paradoxically prolong the experience of negative emotions. In sum, worry in response to perceived threats reduces the ability to fully process these experiences, leading to the maintenance of threatening associations, and potentially increasing the prevalence of threatening material.

Considering that experimental induction of worry can impact physiological functioning, it is possible that chronic worry may have lasting ramifications on ANS functioning. Indeed, people with GAD have been shown to demonstrate reduced tonic PNS activity (Brosschot et al., 2003; Lyonfields et al., 1995; Thayer et al., 1996), shorter SCL declines during sleep and wake (Roth et al., 2008), and reduced SNS activity range (Hoehn-Saric, McLeod, Funderburk, & Kowalski, 2004; Hoehn-Saric, McLeod, & Zimmerli, 1989) as compared to controls, indicating possible physiological rigidity and reduced potential to respond to shifting environmental stimuli. As suggested by Hoehn-Saric and colleagues (Hoehn-Saric et al., 2004), diminished physiological flexibility may represent a partial but insufficient effort of the body to adapt to the physiological changes induced by chronic anxiety. This lack of physiological flexibility in GAD has been argued to indicate overall reduced autonomic arousal symptoms in this disorder (Brown et al., 1998).

However, it should be noted that the ANS is a dynamic system made up of both sympathetic and parasympathetic branches, which can function reciprocally, independently, or nonreciprocally of each other (e.g., coactivation/coinhibition of both systems) (Berntson,

Cacioppo, & Quigley, 1991; 1993). This phenomenon may help to illustrate some inconsistencies in the literature describing autonomic functioning in GAD. As reviewed by Fisher, Granger, and Newman (2010), in describing the physiological inflexibility observed in GAD, whereas some authors have argued that this is driven by inhibition of SNS activity, others suggest it results from reduced PNS activity. However, in order to represent a more complete picture of ANS functioning in GAD, it is necessary to measure indices of both SNS and PNS activity together to understand overall psychophysiological functioning. As such, although it is important to note that some studies do not show worry inductions leading to physiological activation relative to relaxation or neutral activities (Borkovec & Hu, 1990; Borkovec et al., 1983), this may be due to a failure to measure indices that reflect both SNS and PNS activity (see Thayer et al., 1996).

Furthermore, GAD and worry also impact information processing in ways that interfere with learning from experience and that contribute to the maintenance of anxious meanings attributed to various stimuli. For example, even in the absence of specific negative life events, anxious individuals have a hyper-responsive threat detection system and a biased tendency to interpret ambiguous threat cues as negative (Albu, 2008; Bradley, Mogg, Millar, & White, 1995; Bradley, Mogg, White, Groom, & de Bono, 1999; Eysenck, Mogg, May, Richards, & Mathews, 1991; Hazlett-Stevens & Borkovec, 2004; MacLeod, Mathews, & Tata, 1986; Mathews & MacLeod, 2005; Mogg, Millar, & Bradley, 2000; Waters, Mogg, Bradley, & Pine, 2008; Wilson, MacLeod, Mathews, & Rutherford, 2006). Therefore, whether or not a specific negative event is happening, anxious individuals may be perpetually scanning their environments for potential threats. However, once such a threat is detected, studies show that anxious individuals also

demonstrate subsequent difficulty processing this material, such as by attempting to inhibit or forget emotional material (Albu, 2008; Mogg, Mathews, & Weinman, 1987; Verkuil, Brosschot, de Beurs, & Thayer, 2009). Together, this suggests a failed vigilance-inhibition pattern in response to threatening material. Such cognitive biases are likely to prolong negative emotionality by reducing the capacity for emotional learning. Thus, rather than via avoidance of emotionality, another mechanism by which worry may prevent emotional processing may be via sustained negative emotionality, and a dampening of emotional learning.

### **An Experimental Investigation of Worry and Emotional Avoidance**

Recently, a study by Llera and Newman (2010) tested the emotional avoidance model against the perspective that worry leads to greater negative emotionality. Results from this study further illustrate the proposition that worry does not enable avoidance of emotion, on both subjective and physiological levels. This study examined the causal effect of worry on physiological and subjective emotionality, both during worry periods as well as during subsequent responding to a range of emotional stimuli. Participants included both GAD analogues and non-anxious controls who were randomly assigned to engage in worry, relaxation, or neutral inductions, and were subsequently exposed to standardized film clips representing fearful, sad, calm, and happy emotions. Physiological and subjective responding was continuously measured using indices of vagal activity (a marker of PNS activity) and self-reported positive and negative affect. During worry, participants with GAD had lower vagal activity than they did during relaxation, with a neutral induction falling nonsignificantly in between. Those in the worry induction also reported higher levels of negative affect than both relaxation and neutral inductions, which were statistically similar.

When observing changes from induction to emotion exposures, results showed that worry led to less physiological and subjective responding to the fearful exposure than relaxation, and less subjective responding than the neutral induction. Worry also led to less subjective responding than both relaxation and neutral to the sad exposure, but no difference in responding to the positive exposures. The finding that both relaxation and neutral inductions led to similarly greater subjective reactivity than worry suggests that these effects were driven by worry precluding further reactivity (as opposed to relaxation facilitating further reactivity) to both fearful and sad emotions but not positive emotions.

If negative emotions were successfully avoided via worry, then these findings would lend support to an avoidance theory of worry. However, to obtain a comprehensive picture of the effects of worry on emotions, Llera and Newman (2010) examined absolute levels of emotional states throughout the experiment. Despite worry leading to greater negative emotionality prior to the fear exposure, results demonstrated no differences between prior worry, relaxation, or neutral activity in absolute levels of negative emotionality *during* the fear exposure. This finding is similar to a study by Peasley-Miklus and Vrana (2000), demonstrating that worrisome thinking led to greater physiological and subjective responding as compared to baseline or relaxed thinking periods, and did not facilitate further increase during an aversive imagery task. On the other hand, whereas prior relaxation and neutral inductions led to increased sad affect, prior worry led to a *decrease* in reported sadness during the sad clip from previously heightened levels experienced during worry. Nonetheless, as with the fearful film, absolute levels of negative affect during exposure to the sad clip were not actually lower in participants who had worried

previously than in participants in the neutral or relaxation inductions. Therefore, these individuals also did not avoid negative affect associated with sadness.

In sum, data from this study suggest that worry led to *heightened* negative emotionality and simply precluded further increases in emotional reactivity in response to negative exposures. Thus, despite the avoidance of *increased* negative emotionality via prior worry, in both Llera and Newman (2010) and Peasley-Miklus and Vrana (2000), evidence suggests that the adverse emotional state was not avoided but rather was *sustained*. Therefore, the previous conclusion that worry helps to avoid negative emotions requires further exploration.

Here, it is important to revisit extant studies demonstrating that worry reduces somatic responding to negative stimuli. Although studies have evidenced a muting effect of prior worry on subsequent reactivity to fearful stimuli, it should be noted that responses to stimuli were in fact compared to the prior worry induction levels as opposed to a pre-worry baseline level (e.g., Borkovec & Hu, 1990; Llera & Newman, 2010). This suggests that reactivity may have been muted due to already heightened levels of negative arousal experienced during the worry period, and similar to Llera and Newman (2010), prior worry only prevented a *further increase* in arousal. Several studies show that when comparing post-worry fear reactivity to a pre-worry baseline, there is no muting effect on subsequent reactivity (Peasley-Miklus & Vrana, 2000; Stapinski et al., 2010; Vrana et al., 1986; Vrana et al., 1989). Moreover, in a recent study, Fisher, Granger and Newman (2010) showed that only GAD participants who exhibited sympathetic arousal at baseline (as measured by salivary alpha-amylase) showed muted reactivity to a stressor. Those GAD participants without sympathetic arousal at baseline (perhaps individuals who were not in the midst of worrying about something) showed reactivity

comparable to nonanxious control participants. In sum, these findings indicate that the only avoidance facilitated by worry may be that of a further increase in negative arousal following the worry period.

Although these data do not support the perspective that worry enables avoidance of emotion, they do suggest that worry precludes *processing* of negative emotions, as suggested by Borkovec and colleagues (2004). It is therefore critical to emphasize a distinction between emotional avoidance and avoidance of emotional processing. By emotional avoidance, we are referring to the absence or reduction of a negative emotional state despite the presence of a stressor. According to Foa and Kozak (1986), emotional processing requires subjective and physiological *reactivity* upon exposure to a stressor. In the absence of subjective and/or physiological reactivity to an emotional stimulus, the authors posit that the full fear structure has not been accessed and processing is unlikely to have occurred. Data from Llera & Newman (2010) show that worry, relaxation, and neutral inductions prior to negative film clips led to equivalence in absolute levels of negative affect during the clips, suggesting that worry does not facilitate avoidance of negative emotion. Evidence that prior worry precludes further reactivity to fearful and sadness-inducing stimuli (Borkovec & Hu, 1990; Llera & Newman, 2010; Peasley-Miklus & Vrana, 2000; Vrana et al., 1986; Vrana et al., 1989) suggests that a central aspect of emotional processing of negative affect is eliminated by chronic worry. Additional evidence is exhibited with respect to a failure of those with GAD to demonstrate inhibitory learning (Craske et al., 2008) despite chronic exposure to negative emotional states. Taken together, these findings suggest that people with GAD neither avoid nor process negative emotions.

### **The Avoidance of Negative Emotional Contrast Model**

So why would someone who dislikes negative emotions initiate a process that leads to perpetual negative emotionality? We propose an explanation of this phenomenon through the following argument: *we believe that individuals with GAD engage in worry because they prefer to feel chronically distressed in order to prepare for the worst outcome rather than to experience a sharp shift from positive or neutral emotions to negative emotions.* This argument will be referred to as the Contrast Avoidance Model (Newman & Llera, 2011).

Literature suggests that individuals with high worry tend to overestimate the likelihood of negative outcomes to uncertain situations (MacLeod, Williams, & Bekerian, 1991) as well as their own personal risk for experiencing threatening events (Vasey & Borkovec, 1992), even if the probability of a negative event occurring is low (Borkovec et al., 1999). Patients with GAD also distrust their problem-solving skills more so than nonanxious controls (Dugas, Gagnon, Ladouceur, & Freeston, 1998), and catastrophic worry has been associated with low problem-solving confidence (Davey, Jubb, & Cameron, 1996). Furthermore, individuals with GAD believe they are ill equipped to handle their emotional responses to negative events (e.g., Roemer et al., 2005; Turk et al., 2005).

We propose that the process of worry itself provides a sense of protection from the effects of these anticipated negative life experiences, and indeed findings show that worry is utilized as an attempt to prepare for negative events, e.g., *if I expect the worst, then I won't be disappointed/surprised/sad*, and so forth. (Borkovec & Roemer, 1995; Davey, Tallis, & Capuzzo, 1996; Freeston et al., 1994). We posit that individuals with GAD are utilizing worry specifically as an attempt to avoid experiencing an unexpected *negative emotional contrast*, or

sharp increase in their negative emotions. This model is also consistent with the viewpoint of Borkovec and colleagues (2004) that people may create distress for themselves as a means to preclude even greater subsequent pain.

This new theory is partly informed by the theory of affective contrast, which has roots in cognitive psychology. Affective Contrast Theory states that the impact of an emotional experience is contingent on its degree of contrast with a preceding emotional state (Bacon, Rood, & Washburn, 1914). It was first used to explain how an affective contrast can heighten the experience of discrepancy between two discordant states. For example, the experience of unpleasantness can be heightened (or even created) through contrast with a preceding pleasant affective state (Bacon et al., 1914). This theory was then extended to suggest that a negative contrast experience is created by the subjective discrepancy between the aggregate pleasantness of a group of preceding stimuli, rather than just the most recent prior experience (Beebe-Center, 1929). These theories have received empirical support (Harris, 1929; Williams, 1942), and have been expanded to indicate that amount of affective discrepancy between stimuli is critical to create a negative contrast experience (Dermer, Cohen, Jacobsen, & Anderson, 1979). These theories apply a variety of pleasant or noxious stimuli (e.g., smells, colors, music, etc.), and also to the ranking of emotions themselves. Studies show that people judge others' emotional expressions as more negative when preceded with strongly contrasting expressions (i.e., sad faces preceded by happy faces) (Thayer, 1980). This effect is also true when ranking one's own emotional state. Another study found that negative emotional reactions are perceived as stronger when preceded by equally strong positive stimuli (Manstead, Wagner, & MacDonald, 1983).

Based on this cognitive psychology literature as well as recent findings (e.g., Llera & Newman, 2010), we have theorized that people with GAD have developed a stronger aversive reaction and are even more sensitive to a negative emotional contrast than are nonanxious individuals, and that it is negative emotional contrast avoidance that motivates their worry. Specifically, given that individuals with GAD describe their emotional experiences as noxious and difficult to manage, the experience of negative emotional contrasts and resulting subjectively heightened emotional responding would likely feel even more threatening. Therefore, as a way to avoid such experiences, it is proposed that individuals with GAD may find it preferable – and even safer – to maintain a feeling of negativity at all times. Moreover, maintaining a perpetual negative state allows for a positive emotional contrast should a good event occur.

This proposition is similar to Gray's (1982) neurophysiological theory of anxiety, which states that one trigger for anxious responding is the detection of a mismatch between expected and encountered stimuli in the environment. As such, if individuals with GAD use worry to prepare for the worst on an emotional level, then they are reducing the likelihood of an increase in negative emotion if they do experience a negative event (see Borkovec, 1994). Overall, this is consistent with what we tell clients who feel that worry helps them to prepare for the worst. When they worry, their mind and body reacts as though the worst thing has already happened. This is the case even though the vast majority of events they have worried about will never happen (Borkovec et al., 1999). Thus, in terms of the impact of worry, rather than preventing negative outcomes, it actually creates an unnecessary negative impact on the body.

## Goals of the Present Study

The purpose of the current study was to explore the Contrast Avoidance Model of GAD as it relates to worry and emotion. Overall, extant theories of GAD and emotion have failed to discriminate: a) use of a resting baseline from use of a worry baseline, b) somatic response to worry from somatic response to stress following worry, c) initial reactivity to a feared stimulus from absolute levels of emotionality *during* a feared stimulus, and consequently, d) emotional avoidance from emotional processing avoidance. It is likely that absence of an exploration of these differences has led to the inconsistencies in the literature on emotion in GAD. This study attempted to address each of these points, and in so doing, tested the hypothesis that worry facilitates avoidance of emotional contrast, and that individuals with GAD are more averse to a negative emotional contrast than are nonanxious controls.

The primary goals of this study were two-fold. The first goal was to test the hypothesis that worry leads to a heightened negative emotional state (as compared to relaxation and neutral inductions) relative to a resting baseline, and that this negative state is sustained through negative emotional exposures. This was measured 1) by determining absolute levels of, and change in, negative emotion from resting baseline to worry, relaxation, and neutral inductions, and 2) by measuring absolute levels of, and change in, negative emotion from inductions to emotional exposures. (Specific hypotheses for each measured variable are described in the following section.) Such findings may help to differentiate responses to worry from responses to emotional exposures. Also, through observation of absolute levels of emotionality throughout the experiment, we hoped to discriminate between initial reactivity versus absolute levels of

emotionality during an emotional stimulus. Overall, this provided a sense of whether worry prior to emotional exposures resulted in emotional avoidance versus emotional processing avoidance.

The second goal was to test the hypothesis that individuals with GAD are more averse to the experience of a negative emotional contrast than are non-anxious controls, and that those with GAD find worry subjectively helpful in functioning to avoid such a contrast. This was tested via exploration of subjective appraisals of the usefulness of worry in helping to cope with emotions during the emotional exposures for both individuals with GAD and nonanxious controls. In so doing, beyond merely assessing for increased or decreased emotionality during the emotion exposures, this study attempted to ascertain a holistic appraisal of the overall effect of worry versus relaxation or neutral inductions on ability to cope with the impact of the various emotion-inducing film clips.

The present study also examined reactivity to a range of emotional stimuli, including fearful, sad, and happy-valenced exposures, as a way to replicate and extend Newman & Llera (2010). Also, a neutral induction, not typically included in extant studies of worry and emotional reactivity, was deemed important in order to distinguish between effects that were driven uniquely by either the worry or relaxation inductions, and not just the difference between the two. Subjective emotion measures included broad indices of positive and negative affect, as well as specific emotion adjectives to further distinguish the nature of emotional responding.

Also, in order to observe a more complete picture of physiological responding, both PNS and SNS activity were measured. Parasympathetic activity levels were monitored using the construct of vagal activity. Levels of vagal activity are considered to reflect efferent communication between the brain and various bodily organs, including the heart, for the purpose

of physiological regulation. The vagus helps to maintain physiological homeostasis in these areas by delivering feedback to the peripheral autonomic nervous system associated with parasympathetic activity (see Beauchaine, 2001; Porges, 2007 for reviews). Final effects of vagal modulation on heart rhythm at the sinoatrial (S-A) node have been measured using respiratory sinus arrhythmia (RSA), or heart rate variability (HRV) as a function of respiration (Berntson et al., 1997). RSA represents power in the high frequency (HF) band of HRV (0.15-0.4 Hz), the typical breathing rate in healthy adults at rest. Parasympathetic activity can be suppressed (vagal withdrawal) to facilitate arousal in response to challenging environmental situations, including fearful arousal (Rainville, Bechara, Naqvi, & Damasio, 2006; Kreibig et al., 2007).

As opposed to Llera and Newman (2010), in the current study measures of sympathetic activity were also measured via electrodermal activity (EDA) and heart rate. Human sweat glands are innervated predominantly by sympathetic cholinergic mechanisms, providing a fairly robust measure of sympathetic activity (see Dawson, Schell, & Filion, 2007 for a review). In addition, these glands are considered especially sensitive to psychological/emotional responding (Nikula, 1991; Winton, Putnam, & Krauss, 1984) and they can be measured using a non-invasive procedure. As such, this was considered the most appropriate measure of sympathetic activity for this study. In terms of specific EDA indices, skin conductance level (SCL) is often used as a measure of tonic physiological arousal. Researchers have also recently looked at nonspecific skin conductance responses (NS-SCRs) as a measure of phasic sympathetic arousal in anxiety disorders, including GAD (e.g., Andor et al., 2008; Fisher et al., 2010; Upatel & Gerlach, 2008), and NS-SCRs have been linked to thoughts associated with anxiety and negative emotions

(Nikula, 1991; Nikula, Klinger, & Larson-Gutman, 1993). Moreover, individuals with GAD described the thoughts temporally linked to NS-SCRs as being more anxiety provoking and less controllable (Upatel & Gerlach, 2008). Also, Dawson and colleagues (2007) recommend measuring both frequency and amplitude of responses (e.g., average response size) in order to more fully represent electrodermal activity. As such, an electrodermal response with a larger amplitude represents greater sympathetic activity than one with a smaller amplitude. In sum, SCL, frequency of NS-SCRs, average response amplitude of NS-SCRs, and heart rate represented appropriate measurements for phasic changes in levels of arousal in response to both inductions and emotional exposures.

### **Hypotheses**

**Hypothesis one: Worry leads to a negative emotional state.** We predicted that relative to a baseline period, worry inductions would lead to greater increase in and higher absolute levels of subjective negative emotionality, SNS activity (as represented by increased SCL, more frequent NS-SCRs with higher average response amplitude, and higher heart rate), and reduced PNS activity (lower RSA), as compared to relaxation and neutral inductions.

**Hypothesis two: Following worry, negative emotionality will be sustained through negative emotional exposures.** We expected that for those receiving the worry induction, levels of negative emotionality would be sustained across the negative emotional exposures and somewhat alleviated during the positive emotional exposure.

Of note, positive emotions have been shown to produce a shift towards higher sympathetic as well as parasympathetic activity (Llera & Newman, 2010; McCraty, Atkinson, Tiller, Rein, & Watkins, 1995; Rainville et al., 2006). Sadness has recently been shown to

produce an increase in parasympathetic activity (Llera & Newman, 2010), as well as markers that suggest increased sympathetic activity (heart rate, SCL, NS-SCRs) (see Kreibig et al., 2007 for a review). Conversely, the fear response is associated with increased SNS and decreased PNS activity (Rainville et al., 2006; Kreibig et al., 2007). Thus, we predicted that prior worry would preclude changes in both physiological arousal (e.g., vagal withdrawal, increased SCL, NS-SCRs, average amplitude of responses, and heart rate) and subjective negative emotionality in response to fearful stimuli. We also predicted that prior worry would preclude further increase in subjective emotionality in response to the sad exposure, but not the happy exposure. However, considering that Llera and Newman (2010) did not show worry to preclude physiological responding to sad or happy exposures, we predicted that worry prior to these exposures would not interfere with shifts towards higher autonomic activity. In sum, we predicted that prior worry would preclude SNS, PNS, and subjective reactivity in both GAD analogues and control participants to the fear exposure, the preclusion of subjective reactivity to the sad exposure, but not the preclusion of emotional reactivity to the happy exposure.

For those receiving the relaxation and neutral inductions, we did not expect to see negative reactivity in response to the inductions and we did not expect prior relaxation or neutral activity to inhibit reactivity to the negative or positive emotional exposures.

**Hypothesis three: Individuals with GAD would have more difficulty than Controls in coping with negative emotional contrasts.** We predicted that participants with GAD and nonanxious controls would demonstrate divergent appraisals of the worry inductions. We predicted that those with GAD would subjectively experience the worry induction as helping to cope with emotional experiences, whereas nonanxious participants would not. We also predicted

that the GAD group would report more difficulty coping with emotions following the relaxation and neutral inductions than nonanxious control participants.

## **Research Design and Method**

### **Overall Design**

A 2 (group: GAD vs. non-anxious) X 3 (induction type: worry, relax, or neutral) block design was used to explore the differential effects of worry, relaxation, and neutral inductions on reactivity to 3 different emotional stimuli (fear, sadness, and happiness) in both individuals with GAD and non-anxious controls.

### **Participants**

Ninety-five participants (68 females;  $M$  age = 19.03 years,  $SD$  = 1.71 years) were recruited for this study from introductory psychology courses at a rural state university. Students were given class credit as compensation for their participation in this research. The ethnic distribution of participants was 85.3% Caucasian, 6.3% African American, 6.3% Asian, 1.1% Latino(a), and 1.1% other (“mixed race”).

Participants were selected based on their scores on the Generalized Anxiety Disorder Questionnaire-IV (GAD-Q-IV; Newman et al., 2002) and the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). Scores from these measures were used to assign participants to either the analogue GAD ( $n = 48$ ) or non-anxious control group ( $n = 47$ ). Individuals were included in the GAD group if they scored above the cutoff of 5.7 on the GAD-Q-IV ( $M = 13.27$ ,  $SD = 1.59$ ), endorsed symptoms for at least 6 months, and answered affirmatively to each of the first 4 questions on the GAD-Q-IV representing the major diagnostic criteria for GAD (i.e., Do you experience excessive worry? Is your worry excessive in intensity,

frequency, or amount of distress it causes? Do you find it difficult to control your worry [or stop worrying] once it starts? Do you worry excessively and uncontrollably about minor things such as being late for an appointment, minor repairs, homework, etc.?). Participants were also required to score at least one standard deviation above the overall mean ( $M = 43.35$ ,  $SD = 17.11$ ) on the PSWQ (GAD group:  $M = 68.29$ ,  $SD = 6.22$ ). Mean PSWQ scores for this GAD group were comparable to mean levels of those with clinically diagnosed GAD (see Startup & Erikson, 2006).

Individuals were included in the control group if they answered negatively to the first 4 questions and scored below the 5.7 cutoff on the GAD-Q-IV ( $M = 1.64$ ,  $SD = 1.74$ ), and within a standard deviation of the mean on the PSWQ ( $M = 35.09$ ,  $SD = 4.94$ ). Groups did not differ in terms of gender, ethnicity, or age, and these factors were also balanced across conditions.

In order to maximize the likelihood that results would generalize to others with GAD, comorbid disorders were not restricted as participants with GAD tend to have high rates of concurrent comorbidity (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Sanderson, Beck, & Beck, 1990). Nonetheless, because depression is associated with a diminished capacity to habituate to anxious states and may hinder emotional processing (Foa & Kozak, 1986), all participants were tested for comorbid depressed mood using the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979) and completed this measure just prior to the experiment. Measurement of depression allowed for determination of whether differential levels of depressed mood between induction conditions might explain any differences found between these groups.

**Selection criteria.** The GAD-Q-IV (Newman et al., 2002) is a 9-item self-report questionnaire reflecting the criteria for GAD as delineated in the *Diagnostic and Statistical Manual for Mental Disorders, 4<sup>th</sup> Edition* (DSM-IV) (American Psychiatric Association, 2000). Internal consistency (Cronbach's alpha = .94) and 2-week retest reliability (92% of the sample) are strong. In addition, the measure has demonstrated convergent and discriminant validity, and kappa agreement of .67 with a structured interview. A cutoff of 6.7 leads to sensitivity of 83% and specificity of 89%. Students diagnosed with GAD by the GAD-Q-IV were not significantly different on this measure than a GAD community sample, but both groups had significantly higher scores than students identified as not meeting criteria for GAD, demonstrating clinical validity of the GAD-Q-IV (Newman et al., 2002).

The PSWQ (Meyer et al., 1990) is a 16-item self-report inventory designed to assess trait worry and to measure the generality, excessiveness, and uncontrollability characteristics of pathological worry. Items are scored on a 5-point Likert-type scale. Factor analysis indicates that the PSWQ assesses a uni-dimensional construct with an internal consistency coefficient of .91 (Meyer et al.). High retest reliability (ranging from .74-.93) was also demonstrated across periods ranging from 2-10 weeks (Molina & Borkovec, 1994). The PSWQ has also been shown to distinguish individuals with GAD from each of the other anxiety disorder groups (Brown, Antony, & Barlow, 1992). Correlations between the PSWQ and measures of anxiety, depression, and emotional control supported the convergent and discriminant validity of the measure (Brown et al., 1992).

The BDI (Beck et al., 1979) assesses the presence and severity of affective, cognitive, motivational, vegetative, and psychomotor components of depression. Items include statements

such as “I feel sad” and “I feel discouraged about the future” and are ranked on a scale of severity from 0 to 3. Retest reliabilities have been from good to very good, ranging from .48 for psychiatric patients after 3 weeks to .74 for undergraduate students after 3 months (Beck, Steer, & Garbin, 1988). The BDI has also been shown to have high concurrent validity with other measures of depression and there is evidence that it discriminates psychiatric from non-psychiatric patients (Beck et al., 1988).

### **Emotion-Eliciting Stimuli**

Film clips were thought to be the most appropriate stimuli for this study to ensure consistency of emotional exposure across participants. Also, compared with static stimuli such as photographs, film clips allow emotions to be experienced in a more natural, gradually increasing manner that may be more characteristic of emotional events experienced in participants’ lives (Gross & Levenson, 1995; Sloan, 2004).

Participants viewed three brief film clips (ranging in length from 120 to 165 seconds) representing negative (fearful, sad) and positive (happy) emotions. The film clips used in this study have been successful at eliciting the desired emotions in previous studies (Frederickson & Levenson, 1998; Gross & Levenson, 1995; Llera & Newman, 2010; Rottenberg, Kasch, Gross, & Gotlib, 2002; Rottenberg, Salomon, Gross, & Gotlib, 2005; Sloan, 2004). These clips include scenes of a plane crash (fear), a son grieving over his dying father (sadness), and slapstick comedy (happiness). The clips were presented in counterbalanced order to control for sequencing effects.

### **Self-Report Emotion Measures**

One set of emotion measures was adapted from a self-report emotional response inventory first used by Gross and Levenson (1993). The inventory consists of 14 emotion terms including *amusement, anger, arousal, confusion, contempt, contentment, embarrassment, fear, happiness, interest, relief, sadness, surprise, and tension*. Subjects were asked to rate the greatest amount of each emotion they felt during baseline, inductions, and exposures using an anchored 9-point Likert scale (0 = *none* and 8 = *the most in my life*).

The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) is an index of self-report emotional reactivity designed to assess the extent of emotion experienced. Recently used to assess level of emotional reactivity to film clips (e.g., Llera & Newman, 2010; Sloan, 2004), the PANAS is a 20-item mood adjective checklist designed to measure both Positive Affect (PA) and Negative Affect (NA). Participants rate the extent to which their current emotional experience matches with each adjective on a five-point Likert scale ranging from “very slightly or not at all” to “extremely”. Internal consistency reliabilities for the PANAS scales are high, with the PA scale ranging from .86 to .90, and the NA scale ranging from .84 to .87. Retest reliability is variable, with the PA scale ranging from .47 to .68, and the NA scale ranging from .39 to .71. Convergent validity of both scales ranges from .89 to .95, whereas discriminant correlations are low, ranging from -.02 to -.18 (Watson et al., 1988).

**Measures of emotional coping.** In order to determine the extent to which worry, relaxation, and neutral inductions helped participants cope with the subjective experience of emotional exposures, a questionnaire was created using questions adapted from Why Worry Scale - II (Gosselin et al., 2003) (see Figure 1). This questionnaire asked participants to use a 5-

point Likert scale ranging from “not at all true” to “absolutely true” to rate the extent to which the worry, relaxation, or neutral inductions prior to the emotion-inducing film clips helped them cope with their emotions during the film clips. Three items aimed to determine the extent to which prior inductions helped to facilitate emotional coping during the films (e.g., feeling less upset by the negative film clips). Two reverse-scored items aimed to determine the extent to which prior exposures prevented coping with a negative exposure (e.g., leading to feeling *more* upset by the events in the film clips). Depending upon induction condition, participants were asked questions based on the effect of their assigned induction type prior to emotional exposures (e.g., “*Because I was in a relaxed state...*” versus “*Because I was already worrying...*”). Internal consistency reliability for the emotional coping scale total is high (.73). Each item also demonstrated high item-total correlations, ranging from .39 - .64.

### **Physiological Measures**

Vagal activity, respiration rate, and electrodermal activity were monitored throughout the experiment using a Biopac Isolated Amplifier (MP30; Biopac Systems Inc., Santa Barbara, CA) at a sampling rate of 500Hz. In order to measure vagal activity, heart rate variability (HRV), a measure of physiological rhythms in the beat-to-beat interval of each cardiac signal, was utilized. Cardiac activity was measured using wrist- and ankle-mounted electrodes. HRV was analyzed with *AcqKnowledge* 4.0 software using a template-matching approach in the frequency domain method. First, a band pass filter was applied on the ECG data between 0.5 – 35Hz, using 1600 coefficients. Next, R-R intervals were extracted using a modified Pan-Tomkins QRS detector. The R-R intervals were then re-sampled to a continuous sampling rate in order to extract frequency information. Considering the relatively low sampling rate of the MP30 system

(500Hz), a cubic-spline interpolation was used on the ECG signals to decrease error (Electrophysiology, 1996). HRV levels were then calculated on the R-R intervals using power spectrum analyses of the interpolated R-R tachograms, analyzing the power at the high frequency (HF: 0.15-0.4Hz) bands of the power spectrum density, the power band typically associated with respiration. Power in the HF bands of the HRV output was chosen because research considers this to be a good estimate of vagal efferent effects on the heart. Power spectral density values in the HF band are reported in milliseconds<sup>2</sup>. To control for the influence of respirator activity on this variable, HRV data is reported while controlling for respiration rate. Respiration rate was measured using a respiration belt attached around the diaphragm.

Electrodermal activity was measured using two Ag–AgCl electrodes filled with isotonic NaCl electrode gel placed on the distal phalanges of the index and middle finger of the non-dominant hand. Prior to electrode placement, participants washed their hands with a non-abrasive soap. The electrodermal level of skin conductance (skin conductance level, SCL) was measured continuously as a mean level using a scale of microSiemens ( $\mu\text{S}$ ). Electrodermal reactivity was measured using nonspecific skin conductance responses (NS-SCRs). The presence of NS-SCRs was determined using a minimal amplitude value of 0.05  $\mu\text{S}$ . Once responses were identified, the skin conductance response amplitude was quantified by measuring the difference between the zero-slope onset and the peak of the wave in  $\mu\text{S}$ . Amplitude was averaged for all responses within a given time period. Considering that SCL data is frequently found to be positively skewed and leptokurtic (Venables & Christie, 1980), a log transformation will be performed on this data.

For the purposes of this study, we used short-term recordings (both 60 second periods within each 120 second period) to observe SCL, NS-SCRs, amplitude average, and the HF components of the HRV output while controlling for respiration. Recordings were taken during the baseline periods, induction periods, emotional exposures, and recovery periods. In order to control for individual differences on physiological measures, change scores were created for the induction period by subtracting baseline levels for all psychophysiological measures. Change scores were also calculated for the exposure period by subtracting induction period scores and for recovery periods by subtracting exposure period scores.

### **Procedure**

Research assistants followed a standardized protocol for all participants. Participants were informed that they would be tested individually in a study of how people respond to movie scenes. Participants were then randomly assigned to receive a worry ( $n = 31$ ), relaxation ( $n = 32$ ), or neutral ( $n = 32$ ) induction. After completing the consent form, participants washed their hands with a non-abrasive soap and water. Wrists and ankles were then cleaned by the research assistant with an exfoliating scrub. Participants were fitted with the psychophysiology-monitoring equipment and seated in a comfortable chair facing a computer monitor. They were provided a low table to rest their arm that was monitored with electrodermal electrodes, and instructed to try to keep this arm still during periods of recording.

Participants then completed a demographic information sheet and the BDI followed by a 5-minute resting period to acclimate to the experimental situation. The last two minutes of this resting period were used as a psychophysiological baseline. Participants were then asked to complete baseline emotion rating scales. Next, participants were trained in their assigned

induction task (worry, relaxation, or neutral thought activity), and practiced this task for one minute. Instructions were given for worrying (“think about your most worrisome topic and worry about it as intensely as you can”), relaxing (instructions on slowed diaphragmatic breathing), or neutral activity (reading a series of neutral informational passages), depending upon condition. Participants were informed that if at any point their mind wanders off task, they were to simply refocus their thoughts on the task. Our worry, relaxation, and neutral inductions were congruent with those used in previous studies (e.g., Borkovec & Inz, 1990; Behar, Zullig, & Borkovec, 2005; Llera & Newman, 2010; Oathes et al., 2008; Ray et al., 2009). In order to ensure that inductions were successful, participants were given a manipulation check following each induction period consisting of three 5-point Likert scales assessing levels of anxiety, worry, and relaxation.

Following training, participants began with a two-minute self-administered induction, followed by a manipulation check and emotion ratings. They then viewed the first emotional film clip, after which they were instructed to sit quietly for two minutes in order to record the recovery period, and then they completed the post-clip emotion ratings which asked them to identify how they felt *during the film clip*. This was followed by a one-minute distracter task, which was a neutral segment excerpted from the same film as the emotion exposure (e.g., a calm conversation between two characters). All film clips were presented in counterbalanced order.

To begin the next segment, participants sat quietly for 2 minutes in order to obtain another baseline period. They were then asked to re-engage in the induction task (2 minutes) and repeated the same procedure until all three emotion exposures were played. Following exposures, participants were asked to complete contrast questionnaires. At the end of the

session, psychophysiological monitors were removed and participants were fully debriefed. Care was taken that participants were not experiencing lingering and distressing negative emotions, and counselors were available if necessary.

## **Results**

Based on an a priori power analysis, it was determined that a sample size of at least 95 participants was required to observe a medium between-groups effect size (Cohen's  $f = .30$ ) with an  $\alpha$ -level of .05 and power of .80 (Faul, Erdfelder, Lang, & Buchner, 2007). In order to avoid chance effects, Bonferroni adjustments were used when comparing more than two groups in the same analysis. In order to control for participant variables that could have influenced physiological variables, these analyses were run controlling for age, gender, ethnicity, current anti-anxiety or anti-depression medication use, order of exposure type, and comorbid depression. Also, findings were tested by observing all participants together, as well as observing each group (GAD versus controls) individually.

### **Participant Parameters**

Preliminary analyses showed no significant differences between GAD (worry = 16, relax = 16, neutral = 16) and non-anxious control participants (worry = 15, relax = 16, neutral = 16) in age,  $F(1, 93) = .58, p = .45, \eta_p^2 = .007$ , gender,  $F(1, 93) = 2.85, p = .10, \eta_p^2 = .03$ , or ethnicity,  $F(1, 93) = .32, p = .57, \eta_p^2 = .004$ . Similarly, there were no demographic differences between those who were randomly assigned to the worry ( $n = 31$ ), relaxation ( $n = 32$ ), or neutral ( $n = 32$ ) conditions in age,  $F(1, 93) = .79, p = .46, \eta_p^2 = .017$ , gender,  $F(1, 93) = .54, p = .59, \eta_p^2 = .01$ , or ethnicity,  $F(1, 93) = 1.05, p = .36, \eta_p^2 = .02$ .

**Baseline.** There were no differences between groups or conditions on resting baseline levels of reported anxiety. However, there was a significant group difference in reported levels of relaxation,  $F(1, 92) = 4.07, p = .047, \eta_p^2 = .04$ , in that the control group reported greater levels of relaxation ( $M = 3.64, SD = .79$ ) than the GAD group ( $M = 3.23, SD = 1.09$ ). Additionally, there was a trending group difference in reported levels of worry,  $F(1, 92) = 2.94, p = .09, \eta_p^2 = .03$ , in that the GAD group reported marginally higher levels of worry ( $M = 1.46, SD = .65$ ) than the control group ( $M = 1.24, SD = .57$ ).

There was a significant effect of group,  $F(1, 94) = 3.83, p = .004, \eta_p^2 = .19$ , on baseline ratings of emotion adjectives. Specifically, the GAD group reported lower levels than controls on *contentment* (GAD:  $M = 3.24, SD = 2.3$ ; Control:  $M = 4.33, SD = 1.82$ ), *happiness* (GAD:  $M = 2.83, SD = 1.91$ ; Control:  $M = 3.78, SD = 1.8$ ), and *interest* (GAD:  $M = 3.22, SD = 1.94$ ; Control:  $M = 4.35, SD = 2.02$ ). The GAD group also reported higher levels than controls on *embarrassment* (GAD:  $M = 1.52, SD = 0.86$ ; Control:  $M = 1.2, SD = 0.52$ ) and *sadness* (GAD:  $M = 1.46, SD = 0.84$ ; Control:  $M = 1.05, SD = 0.32$ ), though these latter ratings were overall on the low end of the rating scale (between “very slightly” and “a small amount”).

There was also an effect of group on baseline PANAS scores, showing that participants with GAD scored lower on reported PA levels ( $M = 15.35, SD = 6.07$ ) than did controls ( $M = 18.55, SD = 6.56$ ),  $F(1, 93) = 6.04, p = .016, \eta_p^2 = .06$ . There was a trending group effect on baseline NA levels,  $F(1, 93) = 3.41, p = .068, \eta_p^2 = .04$ , in that participants with GAD reported marginally higher levels ( $M = 12.94, SD = 4.04$ ) than did controls ( $M = 11.70, SD = 2.38$ ).

There were no baseline differences on SCL between groups,  $F(1, 88) = .06, p = .81, \eta_p^2 = .001$ , induction types,  $F(2, 87) = .15, p = .86, \eta_p^2 = .003$ , or an interaction,  $F(3, 85) = .80, p =$

.45,  $\eta_p^2 = .02$ . There were no baseline differences on NS-SCRs between groups,  $F(1, 88) = .51$ ,  $p = .48$ ,  $\eta_p^2 = .006$ , induction types,  $F(2, 87) = .21$ ,  $p = .81$ ,  $\eta_p^2 = .005$ , or an interaction,  $F(3, 85) = 1.96$ ,  $p = .15$ ,  $\eta_p^2 = .045$ . These findings also held when controlling for age, gender, ethnicity, current anti-anxiety or anti-depression medication use, order of exposure type, and comorbid depression.

There were no baseline differences on amplitude average between induction types,  $F(2, 87) = .05$ ,  $p = .95$ ,  $\eta_p^2 = .001$ , or an interaction,  $F(3, 85) = 2.33$ ,  $p = .10$ ,  $\eta_p^2 = .05$ . However, a significant effect of group was found on amplitude average during the first minute of the baseline period,  $F(1, 88) = 3.95$ ,  $p = .05$ ,  $\eta_p^2 = .05$ , in that individuals with GAD actually demonstrated lower average response amplitudes ( $M = .002$ ,  $SD = .52$ , in log-transformed values) than nonanxious controls ( $M = .23$ ,  $SD = .53$ ). This effect held when controlling for age, gender, ethnicity, current anti-anxiety or anti-depression medication use, and order of exposure type. However, when controlling for comorbid depression, this effect was no longer significant,  $F(1, 88) = .79$ ,  $p = .38$ ,  $\eta_p^2 = .009$ .

When comparing heart rate at baseline, there was no effect of group,  $F(1, 87) = 1.30$ ,  $p = .28$ ,  $\eta_p^2 = .046$ , or induction types,  $F(2, 86) = .97$ ,  $p = .45$ ,  $\eta_p^2 = .034$ . However, there was a marginally significant effect of the interaction,  $F(3, 84) = 2.00$ ,  $p = .069$ ,  $\eta_p^2 = .07$ . When divided by group, the GAD group demonstrated a significant effect of induction type,  $F(2, 40) = 3.38$ ,  $p = .04$ ,  $\eta_p^2 = .14$ , in that those in the neutral condition demonstrated significantly higher baseline heart rate ( $M = 90.97$ ,  $SD = 16.19$ ) than those in the relaxation condition ( $M = 71.96$ ,  $SD = 25.58$ ,  $p = .04$ ), but not those in the worry condition ( $M = 78.79$ ,  $SD = 14.20$ ,  $p = .32$ ). Worry and relaxation conditions did not differ significantly from each other ( $p = 1.0$ ). There was no

effect of induction type for the control group,  $F(2, 43) = 1.0, p = .41, \eta_p^2 = .04$ . This was true when controlling for age, gender, ethnicity, order of exposure type, and comorbid depression.

When controlling for respiration, there were no baseline differences on RSA levels between groups,  $F(1, 84) = .01, p = .94, \eta_p^2 < .001$ , induction types,  $F(2, 83) = 1.08, p = .34, \eta_p^2 = .027$ , or an interaction,  $F(3, 81) = 2.11, p = .13, \eta_p^2 = .05$ . This was true when controlling for age, gender, ethnicity, current anti-anxiety or anti-depression medication use, order of exposure type, and comorbid depression.

As expected, individuals with GAD had significantly higher BDI scores at baseline ( $M = 10.23, SD = 7.94$ ) than did non-anxious participants ( $M = 3.27, SD = 3.88$ ),  $F(1, 93) = 30.97, p < .001, \eta_p^2 = .26$ . Although the means for each group were within the normal to low range of depressed mood, it should be noted that 18.75% of individuals in the GAD group had scores reflecting moderate to severe levels of depression ( $M = 22.78, SD = 4.94$ ). Importantly, there were no significant differences in BDI scores across worry, relaxed and neutral induction conditions,  $F(2, 92) = 1.40, p = .25, \eta_p^2 = .03$ , and this was true within both the GAD,  $F(2, 45) = 2.78, p = .073, \eta_p^2 = .11$ , and control groups,  $F(2, 44) = 0.50, p = .61, \eta_p^2 = .02$ .

### **Manipulation Check**

Because some manipulation check scores were significantly non-normal, a log transformation was performed on all manipulation check data. In order to assess the effectiveness of worry, relaxation, and neutral inductions, a MANOVA was run using results of the manipulation check (collapsed across the three inductions) as the dependent variable, and group and induction type as the independent variables. Results indicated a main effect of group,  $F(1, 84) = 4.04, p = .01, \eta_p^2 = .13$ , and induction type,  $F(2, 83) = 15.71, p < .001, \eta_p^2 = .37$ , but

as expected, no group by induction interaction,  $F(3, 79) = 1.13, p = .35, \eta_p^2 = .04$ . There were no significant changes in manipulation check scores from the first to the last induction, suggesting that induction effects did not diminish over time.

Overall, participants with GAD reported greater levels of worry,  $F(1, 84) = 3.98, p = .049, \eta_p^2 = .05$ , and anxiety,  $F(1, 84) = 8.53, p = .005, \eta_p^2 = .10$ , and less relaxation,  $F(1, 84) = 10.35, p = .002, \eta_p^2 = .12$ , than controls regardless of induction type. For both individuals with GAD and nonanxious controls, those in the worry condition reported significantly more worry and anxiety than did those in the relaxation and neutral conditions,  $F(1, 84) = 4.13, p = .045, \eta_p^2 = .05$  (Bonferroni post-hoc tests,  $p < .001$  for both), whose scores were statistically similar ( $p = 1.0$ ). As for the relaxation condition, all participants in this condition reported significantly higher levels of relaxation than those in the worry condition,  $F(1, 90) = 9.01, p = .004, \eta_p^2 = .09$  (Bonferroni post-hoc tests,  $p < .001$ ), but only marginally higher levels than those in the neutral condition ( $p = .09$ ). (See Table 1 for mean scores and standard deviation values.)

### **Hypothesis 1: Worry Leads to Heightened Negative Emotional State**

**Subjective measures.** Because some subjective rating scores were not normally distributed, a log-transformation was performed on all subjective rating data. A series of MANOVAs were run using group and induction type as the independent variables and absolute levels of subjective measures as the dependent variables. Next, to determine change from the baseline to induction levels, change scores were created by subtracting baseline levels from induction levels. When measuring subjective variables, scores across the three induction periods were averaged together. However, given that physiological levels might have shifted across the

experimental procedure, induction scores were not averaged across induction periods but were measured separately. As such, order effects were controlled for in these analyses.

**Emotion adjectives.** Absolute levels of emotion adjective ratings during inductions were averaged across the experiment. There was a significant effect of induction type,  $F(2, 70) = 3.43, p < .001, \eta_p^2 = .51$ , but no group by induction interaction,  $F(3, 68) = 1.45, p = .08, \eta_p^2 = .30$ . It was found that worry led to significantly greater reported levels of *anger, contempt, embarrassment, fear, sadness, and tension*, and significantly lower levels of *contentment* than did relaxation and neutral inductions, which were statistically similar (See Table 2). This was true when controlling for order of exposure and comorbid depression.

When comparing change scores, there was a significant effect of induction type,  $F(2, 70) = 2.82, p < .001, \eta_p^2 = .30$ , but no effect of group,  $F(1, 71) = .79, p = .63, \eta_p^2 = .11$ , or an interaction,  $F(3, 68) = 1.55, p = .09, \eta_p^2 = .18$ . Worry led to greater increase from baseline in reported levels of *anger, fear, sadness, and tension* than relaxation and neutral, which were statistically similar. Worry also led to a greater decrease in levels of *contentment* than did relaxation and neutral inductions, which were statistically similar. This was true when controlling for order of exposure and comorbid depression.

**PANAS scores.** Comparing post-induction PANAS levels averaged across trials, there was a significant effect of group,  $F(1, 91) = 6.79, p = .002, \eta_p^2 = .14$ , and induction type,  $F(2, 90) = 16.52, p < .001, \eta_p^2 = .28$ , but no group by induction type interaction,  $F(3, 88) = 1.99, p = .10, \eta_p^2 = .04$ . It was found that on average, individuals with GAD reported higher NA levels ( $M = 14.87, SD = 5.97$ ) than controls ( $M = 12.83, SD = 4.59$ ) regardless of induction type,  $F(1, 91) = 7.13, p = .009, \eta_p^2 = .08$ . Furthermore, all participants endorsed greater NA levels during the

worry versus relaxation inductions,  $F(1, 61) = 52.59, p < .001, \eta_p^2 = .46$  ( $M = 19.05, SD = 5.92$ ;  $M = 11.47, SD = 2.49$ , respectively), as well as versus neutral inductions,  $F(1, 59) = 56.59, p < .001, \eta_p^2 = .49$  ( $M = 11.11, SD = 2.70$ ), but relaxation and neutral inductions did not differ significantly from each other. When comparing PA levels, however, all inductions led to statistically similar levels,  $F(2, 90) = 1.38, p = .26, \eta_p^2 = .03$ , though there was a marginal group difference,  $F(1, 91) = 3.80, p = .054, \eta_p^2 = .042$ , in that controls reported higher PA levels ( $M = 15.44, SD = 4.96$ ) than those with GAD ( $M = 13.72, SD = 3.99$ ) regardless of induction type. PANAS effects still held when controlling for order effects and comorbid depression. (All PANAS scores are reported in non-transformed values.)

When comparing NA change scores, there was a significant effect of induction type,  $F(2, 90) = 32.71, p < .001, \eta_p^2 = .43$ , but no effect of group,  $F(1, 91) = .75, p = .39, \eta_p^2 = .009$ , or an interaction,  $F(3, 88) = .52, p = .60, \eta_p^2 = .01$ . Worry led to greater increase from baseline in reported levels of NA ( $M = 5.92, SD = 5.52$ ) than both relaxation ( $M = -.84, SD = 2.7$ ) and neutral ( $M = -.36, SD = 2.0$ ;  $p < .001$  for both), which were statistically similar ( $p = 1.0$ ). This was also true when controlling for order effects and comorbid depression. For change in PA scores, there was no effect of group,  $F(1, 91) = 2.04, p = .16, \eta_p^2 = .02$ , induction type,  $F(1, 91) = 2.13, p = .13, \eta_p^2 = .05$ , or an interaction,  $F(1, 91) = 1.19, p = .31, \eta_p^2 = .03$ .

**Physiological measures.** Because some physiological scores were not normally distributed, a log-transformation was performed on all physiological measurement data, unless otherwise noted. Change scores were also created in order to control for individual differences, and were compared across group and induction type. A series of MANOVAs were run using

group and induction type as the independent variables and physiological scores, or physiological change scores, as the dependent variables.

**SCL scores.** For the effects of the pre-fear induction on SCL, there was no main effect of group,  $F(1, 89) = .43, p = .55, \eta_p^2 = .005$ , induction type,  $F(2, 88) = 1.50, p = .23, \eta_p^2 = .035$ , or an interaction,  $F(3, 86) = .83, p = .44, \eta_p^2 = .02$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. As for change scores for the pre-fear induction, there was no main effect of group,  $F(1, 87) = .51, p = .60, \eta_p^2 = .01$ , induction type,  $F(2, 86) = 2.19, p = .07, \eta_p^2 = .05$ , or an interaction,  $F(3, 84) = 1.25, p = .29, \eta_p^2 = .029$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When looking at each group separately, these effects were still not significant.

For the pre-sadness induction, there was also no main effect of group on SCL,  $F(1, 88) = .77, p = .38, \eta_p^2 = .009$ , induction type,  $F(2, 87) = .22, p = .80, \eta_p^2 = .005$ , or an interaction,  $F(3, 85) = .79, p = .46, \eta_p^2 = .02$ , even when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. For the pre-sad induction on change in SCL, there was also no main effect of group,  $F(1, 87) = .06, p = .94, \eta_p^2 = .002$ , induction type,  $F(2, 86) = .67, p = .61, \eta_p^2 = .02$ , or an interaction,  $F(3, 84) = .53, p = .71, \eta_p^2 = .01$ , even when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When looking at each group separately, these effects were still not significant.

For the pre-happy induction, there was no main effect of group on SCL,  $F(1, 89) = .11$ ,  $p = .75$ ,  $\eta_p^2 = .001$ , induction type,  $F(2, 88) = 1.07$ ,  $p = .35$ ,  $\eta_p^2 = .025$ , or an interaction,  $F(3, 86) = .83$ ,  $p = .44$ ,  $\eta_p^2 = .02$ , which was also true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. Similarly, for the pre-happy induction on change in SCL, there was no main effect of group,  $F(1, 88) = .15$ ,  $p = .86$ ,  $\eta_p^2 = .004$ , induction type,  $F(2, 87) = 1.83$ ,  $p = .13$ ,  $\eta_p^2 = .04$ , or an interaction,  $F(3, 85) = .75$ ,  $p = .56$ ,  $\eta_p^2 = .02$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When looking at each group separately, these effects were still not significant.

**NS-SCR scores.** Because NS-SCRs were normally distributed, log transformations were not performed. For the pre-fear induction on NS-SCRs, there was no main effect of group,  $F(1, 88) = .25$ ,  $p = .78$ ,  $\eta_p^2 = .006$ , induction type,  $F(2, 87) = 2.13$ ,  $p = .08$ ,  $\eta_p^2 = .05$ , or an interaction,  $F(3, 85) = 2.62$ ,  $p = .08$ ,  $\eta_p^2 = .059$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When divided by group, there was an effect of induction type for the first minute of the pre-fear induction for the GAD group only,  $F(2, 42) = 4.45$ ,  $p = .018$ ,  $\eta_p^2 = .18$ , in that worry actually led to fewer NS-SCRs ( $M = 10$ ,  $SD = 4.21$ ) than relaxation ( $M = 15.81$ ,  $SD = 5.94$ ,  $p = .015$ ), with the neutral induction falling nonsignificantly in between ( $M = 12.36$ ,  $SD = 6.06$ ). When observing change in NS-SCRs for the pre-fear induction, there was no main effect of group,  $F(1, 86) = .30$ ,  $p = .74$ ,  $\eta_p^2 = .007$ , induction type,  $F(2, 85) = 1.09$ ,  $p = .37$ ,  $\eta_p^2 = .026$ , or an interaction,  $F(3, 83) = 1.10$ ,  $p = .36$ ,  $\eta_p^2 = .026$ , which was true when

controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. There was also no effect of induction when divided by group.

For the pre-sad induction on NS-SCRs, there was also no main effect of group,  $F(1, 88) = .34, p = .72, \eta_p^2 = .008$ , induction type,  $F(2, 87) = 1.63, p = .17, \eta_p^2 = .037$ , or an interaction,  $F(3, 85) = 2.35, p = .10, \eta_p^2 = .05$ , even when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When divided by group, there was an effect of induction type for the first minute of the pre-sad induction for the GAD group only,  $F(2, 41) = 5.43, p = .008, \eta_p^2 = .21$ , in that again relaxation led to more NS-SCRs ( $M = 16.63, SD = 6.31$ ) than worry ( $M = 11.79, SD = 4.32, p = .042$ ), and the neutral induction ( $M = 12.36, SD = 6.06, p = .013$ ), whereas worry and neutral were not significantly different. When observing change in NS-SCRs for the pre-sad induction, there was no main effect of group,  $F(1, 87) = .03, p = .97, \eta_p^2 = .001$ , induction type,  $F(2, 86) = 1.13, p = .34, \eta_p^2 = .027$ , or an interaction,  $F(3, 84) = .08, p = .99, \eta_p^2 = .002$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. There was also no effect of induction when divided by group.

For the pre-happy induction on NS-SCRs, there was no main effect of group,  $F(1, 89) = .54, p = .58, \eta_p^2 = .01$ , induction type,  $F(2, 88) = 2.10, p = .08, \eta_p^2 = .047$ , or an interaction,  $F(3, 86) = .60, p = .66, \eta_p^2 = .01$ , which was also true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When observing change in NS-SCRs for the pre-happy induction, there was no main

effect of group,  $F(1, 89) = .16, p = .86, \eta_p^2 = .004$ , induction type,  $F(2, 88) = 1.24, p = .30, \eta_p^2 = .03$ , or an interaction,  $F(3, 86) = .49, p = .74, \eta_p^2 = .01$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When divided by group, there were no differences in induction types for either absolute levels or change scores for the pre-happy induction.

***Amplitude average scores.*** For the pre-fear induction on average amplitude of responding, there was a main effect of group,  $F(1, 87) = 4.28, p = .04, \eta_p^2 = .05$ , in that participants in the GAD group evidenced lower average amplitudes ( $M = .14, SD = .54$ , in log-transformed values) than control participants ( $M = .38, SD = .57$ ) across both minutes of the induction period, regardless of induction type, which was true even when controlling for medication use, order of exposure, ethnicity, age, and gender. However, when controlling for comorbid depression, this effect was no longer significant,  $F(1, 87) = 1.51, p = .22, \eta_p^2 = .02$ . There was also an effect of induction type,  $F(2, 86) = 3.47, p = .036, \eta_p^2 = .08$ , in that the relaxation condition led to lower average amplitudes ( $M = -.01, SD = .58$ ) than worry ( $M = .12, SD = .67$ ) and neutral conditions ( $M = .40, SD = .56$ ), when controlling for medication use, order of exposure, ethnicity, age, gender, and comorbid depression. However, Bonferonni post-hoc tests indicate that only the neutral condition was statistically different from the relaxation condition ( $p = .04$ ). There was no effect of the interaction,  $F(3, 84) = .20, p = .82, \eta_p^2 = .005$ . When divided by group, this effect was not evident for the control group, but was still significant for the GAD group,  $F(2, 41) = 4.24, p = .02, \eta_p^2 = .17$ .

When exploring change in amplitude average for the pre-fear induction, there was no effect of group,  $F(1, 85) = .49, p = .69, \eta_p^2 = .02$ , or an interaction,  $F(3, 82) = .40, p = .88, \eta_p^2 =$

.02. However, there was a significant effect of induction type for change from baseline to the first minute of the pre-fear induction period,  $F(2, 84) = 5.31, p = .007, \eta_p^2 = .11$ . Relaxation led to a significantly greater decrease in average amplitude from baseline than did worry ( $p = .012$ ) and neutral inductions ( $p = .032$ ), which were statistically similar ( $p = 1.0$ ), using Bonferonni post-hoc tests (see Figure 2). However, the neutral condition was nonsignificantly in between the worry and relaxation conditions when subject #48 (a GAD participant with the highest score in the neutral condition, and who may have represented an outlier) was removed. This effect remained significant when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression.

For the pre-sad induction on amplitude average, there was also a main effect of group,  $F(1, 87) = 3.96, p = .05, \eta_p^2 = .05$ , in that participants in the GAD group evidenced lower average amplitudes ( $M = .17, SD = .60$ , in non-transformed values) than control participants ( $M = .43, SD = .62$ ) across the first minute of the induction period, regardless of induction type, which was true when controlling for ethnicity, age, and gender, but not when controlling for medication, order of exposure, or comorbid depression. However, there was no main effect of induction type,  $F(2, 86) = .96, p = .39, \eta_p^2 = .02$ , or an interaction,  $F(3, 84) = .89, p = .50, \eta_p^2 = .03$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When exploring change from baseline to the pre-sad inductions, there were no significant effects of group,  $F(1, 87) = 1.11, p = .35, \eta_p^2 = .04$ , induction type,  $F(2, 86) = 1.55, p = .17, \eta_p^2 = .05$ , or an interaction,  $F(3, 84) = .94, p = .47, \eta_p^2 = .03$ . When looking at each group separately, these effects were still not significant.

For the effect of the pre-happy induction on amplitude average, there was also a main effect of group,  $F(1, 88) = 5.71, p = .02, \eta_p^2 = .06$ , in that participants in the GAD group evidenced lower average amplitudes ( $M = .00, SD = .61$ , in log-transformed values) than control participants ( $M = .30, SD = .60$ ) across the second minute of the induction period, regardless of induction type, which was true when controlling for medication, order of exposure, ethnicity, age, and gender, but not when controlling for comorbid depression. However, there was no main effect of induction type,  $F(2, 87) = .70, p = .65, \eta_p^2 = .02$ , or an interaction,  $F(3, 85) = .64, p = .70, \eta_p^2 = .02$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When exploring change from baseline to the pre-happy inductions, there were also no significant effects of group,  $F(1, 89) = 1.82, p = .18, \eta_p^2 = .02$ , induction type,  $F(2, 88) = 1.55, p = .16, \eta_p^2 = .05$ , or an interaction,  $F(3, 86) = .34, p = .91, \eta_p^2 = .01$ . This was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When looking at each group separately, these effects were still not significant.

Given that the GAD group evidenced smaller amplitude averages than controls, but this was not significant when controlling for depression, a correlation was run between depression scores and amplitude average scores for each induction period. It was found that depression had a negative correlation with amplitude average for the pre-fear induction,  $r(89) = -.22, p = .036$ , the pre-sad induction,  $r(87) = -.24, p = .023$ , and the pre-happy induction,  $r(89) = -.27, p = .009$ .

**HR scores.** For the pre-fear induction on absolute HR scores, there was no effect of group,  $F(1, 87) = .24, p = .62, \eta_p^2 = .003$ , induction type,  $F(2, 86) = 2.23, p = .11, \eta_p^2 = .05$ , or an

interaction,  $F(3, 84) = 1.34, p = .27, \eta_p^2 = .03$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When looking at each group separately, there was still no effect of induction type. When observing change in HR from baseline to the pre-fear induction, there was no effect of group,  $F(1, 85) = .79, p = .51, \eta_p^2 = .03$ , induction type,  $F(2, 82) = .91, p = .49, \eta_p^2 = .03$ , or an interaction,  $F(3, 80) = .73, p = .63, \eta_p^2 = .03$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. Looking at each group separately, the GAD group did not evidence an effect of induction type,  $F(2, 39) = .67, p = .52, \eta_p^2 = .03$ , whereas the control group demonstrated a marginally significant effect of induction type for change in HR during the first minute of the induction,  $F(2, 42) = 2.60, p = .086, \eta_p^2 = .11$ . When using LSD post-hoc tests, the relaxation induction led to marginally lower HR ( $M = -3.67, SD = 5.24$ ) than the worry induction ( $M = -.18, SD = 5.0, p = .069$ ) and lower HR than the neutral induction ( $M = .20, SD = 5.3, p = .048$ ), which were statistically similar ( $p = .84$ ). However, when using Bonferroni post-hoc tests, these effects were nonsignificant. This was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression.

For the pre-sad induction on absolute HR scores, there was no effect of group,  $F(1, 88) = .47, p = .70, \eta_p^2 = .02$ , or an interaction,  $F(3, 85) = 1.17, p = .33, \eta_p^2 = .04$ ; however, there was a significant effect of induction type,  $F(2, 87) = 3.70, p = .03, \eta_p^2 = .08$ , across both minutes of the induction period, in that relaxation led to lower HR than neutral activity ( $p = .03$ ) but not worry ( $p = .54$ ), and worry and neutral were statistically similar ( $p = .65$ ). This was true when

controlling for order of exposure type, age, ethnicity, gender, and comorbid depression. When looking at each group separately, the control group did not evidence an effect of induction type,  $F(2, 44) = .71, p = .50, \eta_p^2 = .03$ , whereas the GAD group did,  $F(2, 40) = 3.29, p = .048, \eta_p^2 = .14$ . For the GAD group, those in the relaxation induction evidenced lower HR ( $M = 71.20, SD = 24.26$ ) than those in the neutral induction ( $M = 90.16, SD = 19.27, p = .045$ ), but not those in worry ( $M = 77.68, SD = 14.28, p = 1.0$ ), who were statistically similar to those in neutral ( $p = .31$ ). This was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When observing change in HR from baseline to the pre-sad induction, there was no effect of group,  $F(1, 87) = .44, p = .73, \eta_p^2 = .016$ , induction type,  $F(2, 86) = 1.49, p = .19, \eta_p^2 = .05$ , or an interaction,  $F(3, 84) = 1.03, p = .41, \eta_p^2 = .036$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When observing groups separately, there was still no effect of induction type.

For the pre-happy induction on absolute HR scores, there was no effect of group,  $F(1, 88) = .04, p = .96, \eta_p^2 = .001$ , or an interaction,  $F(3, 85) = 1.09, p = .36, \eta_p^2 = .025$ ; however, there was a significant effect of induction type,  $F(2, 87) = 3.22, p = .045, \eta_p^2 = .07$ , across both minutes of the induction period, in that relaxation led to lower HR than neutral activity ( $p = .05$ ) but not worry ( $p = .36$ ), and worry and neutral were statistically similar ( $p = 1.0$ ). This was true when controlling for order of exposure type, age, ethnicity, gender, and comorbid depression. When looking at each group separately, the control group did not evidence an effect of induction type,  $F(2, 44) = .40, p = .81, \eta_p^2 = .018$ , whereas the GAD group did,  $F(2, 41) = 3.25, p = .049, \eta_p^2 = .14$ . For the GAD group, those in the relaxation induction evidenced lower HR ( $M = 71.20,$

$SD = 23.77$ ) than those in the neutral induction ( $M = 88.32$ ,  $SD = 14.58$ ,  $p = .044$ ), but not those in worry ( $M = 79.49$ ,  $SD = 13.94$ ,  $p = .65$ ), who were statistically similar to those in neutral ( $p = .59$ ). This was true when controlling for medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When observing change in HR from baseline to the pre-happy induction, there was no effect of group,  $F(1, 87) = 1.14$ ,  $p = .34$ ,  $\eta_p^2 = .04$ , induction type,  $F(2, 86) = .78$ ,  $p = .59$ ,  $\eta_p^2 = .028$ , or an interaction,  $F(3, 84) = .74$ ,  $p = .62$ ,  $\eta_p^2 = .026$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When observing groups separately, there was still no effect of induction type.

**RSA scores.** A series of univariate ANOVAs were run using the absolute level induction scores, with group and induction type as the independent variables, and log-transformed RSA as the dependent variable, controlling for respiration rate. For the pre-fear induction on absolute RSA levels, there was no main effect of group,  $F(1, 87) = .98$ ,  $p = .41$ ,  $\eta_p^2 = .035$ , induction type,  $F(2, 86) = 1.27$ ,  $p = .27$ ,  $\eta_p^2 = .045$ , or an interaction,  $F(3, 84) = 1.42$ ,  $p = .21$ ,  $\eta_p^2 = .049$ . This was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When looking at each group separately, there was still no effect of induction type.

A series of univariate ANOVAs were also run using the induction change scores, with group and induction type as the independent variables, and RSA as the dependent variable, controlling for parallel changes in respiration rate. For the pre-fear induction, there was no main effect of group,  $F(1, 83) = .00$ ,  $p = .99$ ,  $\eta_p^2 = .00$ , or induction type,  $F(2, 82) = .59$ ,  $p = .56$ ,  $\eta_p^2 = .015$  on change in RSA. However, a significant group by induction type interaction was found

for the full two minutes of the pre-fear induction,  $F(3, 80) = 4.24, p = .018, \eta_p^2 = .10$ . When divided by group, the GAD group did not evidence significant induction type differences,  $F(2, 39) = .96, p = .39, \eta_p^2 = .05$ , whereas the control group did,  $F(2, 40) = 5.01, p = .01, \eta_p^2 = .20$ , which was true when controlling for order of exposure type, age, ethnicity, gender, and comorbid depression. For controls, when using LSD post-hoc tests, there was a significant differences between the worry and relaxation inductions ( $p = .025$ ), and relaxation and neutral inductions ( $p = .005$ ), but no difference between worry and neutral ( $p = .48$ ). When using Bonferroni post-hoc tests, the relaxation induction led to significantly greater change in RSA levels than neutral ( $p = .015$ ) and to marginally greater change than worry ( $p = .074$ ) (see Figure 3). When controlling for parallel changes in respiration, the control group no longer evidenced a significant change by induction type,  $F(2, 40) = 2.73, p = .078, \eta_p^2 = .12$ ; however, there were no condition differences in respiration rate for the control group,  $F(2, 40) = .85, p = .54, \eta_p^2 = .06$ .

For the pre-sad induction on absolute RSA levels, there was also no main effect of group,  $F(1, 87) = 2.19, p = .10, \eta_p^2 = .075$ , induction type,  $F(2, 86) = 1.29, p = .27, \eta_p^2 = .045$ , or an interaction,  $F(3, 84) = 1.55, p = .16, \eta_p^2 = .05$ , even when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When looking at each group separately, there was still no effect of induction type. When observing change in RSA from baseline to the pre-sad induction, there was a significant main effect of group for both minutes of the induction,  $F(1, 84) = 5.51, p = .02, \eta_p^2 = .07$ , in that the GAD group evidenced less change in RSA than the control group ( $M = 1.08, SD = 117.5, M = 62.04, SD = 119.8$ , respectively), regardless of induction type. This was also true when controlling for order of exposure type, age, ethnicity, gender, and comorbid depression, but not

when controlling for medication use. There was no effect of induction type,  $F(2, 83) = .72, p = .49, \eta_p^2 = .018$ , or an interaction,  $F(3, 81) = 1.07, p = .35, \eta_p^2 = .026$ . Again, when looking at each group separately, there was still no effect of change by induction type.

Similarly, for the pre-happy induction on absolute RSA levels, there was no main effect of group,  $F(1, 87) = .84, p = .48, \eta_p^2 = .03$ , induction type,  $F(2, 86) = 1.01, p = .42, \eta_p^2 = .036$ , or an interaction,  $F(3, 84) = 1.12, p = .35, \eta_p^2 = .039$ , which was also true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. There was still no effect of induction type when observing each group separately. When observing change in RSA from baseline to the pre-happy induction, there was a significant main effect of group for both minutes of the induction,  $F(1, 85) = 4.88, p = .03, \eta_p^2 = .06$ , in that the GAD group again evidenced less change in RSA than the control group ( $M = -9.61, SD = 94.74, M = 45.56, SD = 124.83$ , respectively), regardless of induction type. This was true when controlling for order of exposure type, age, ethnicity, and gender, but not when controlling for medication use or comorbid depression. There was no effect of induction type,  $F(2, 84) = 1.16, p = .32, \eta_p^2 = .028$ , or an interaction,  $F(3, 82) = .60, p = .55, \eta_p^2 = .015$ . Again, when looking at each group separately, there was still no effect of induction type.

**Hypothesis 2: Following Worry, Heightened Negative Emotional State is Sustained Through Negative Exposures, and Alleviated During Positive Exposure**

**Fear exposure.**

*Subjective measures.* First, in order to determine whether there were significant changes from induction periods to emotion exposures, repeated measures ANOVAs were run with group

and induction type as between subject variables, and time and subjective variable levels as within subject variables, including emotion adjectives, PANAS scores, and subjective physiological responding levels. Next, a series of MANOVAs were run using group and induction type as the independent variables and absolute levels of subjective measures as the dependent variables.

For the fear exposure, there were several significant findings from repeated measures analyses of emotion adjectives, including *fear*, *anger*, *sadness*, and *tension*. For reported *fear* levels, the repeated measures ANOVA was significant by time,  $F(1, 85) = 56.22, p < .001, \eta_p^2 = .40$ , and time by induction type,  $F(3, 82) = 12.09, p < .001, \eta_p^2 = .22$ . From the induction to the fear exposure, those in the worry condition reported no significant change in *fear* in response to the exposure,  $F(1, 29) = .17, p = .68, \eta_p^2 = .006$ , whereas those in the relax and neutral inductions reported a significant increase,  $F(1, 54) = 87.25, p < .001, \eta_p^2 = .63$ . However, in terms of absolute levels of reported *fear* during the fear exposure, there was no effect of group,  $F(1, 90) = 1.06, p = .31, \eta_p^2 = .01$ , induction,  $F(2, 89) = .85, p = .43, \eta_p^2 = .019$ , or an interaction,  $F(3, 87) = 1.07, p = .35, \eta_p^2 = .024$  (see Figure 4 for change from baseline to induction to fear exposure).

For reported *anger*, the repeated measures ANOVA was significant by time,  $F(1, 88) = 8.41, p = .005, \eta_p^2 = .09$ , and time by induction type,  $F(3, 85) = 10.16, p < .001, \eta_p^2 = .20$ . From the induction to the fear exposure, those in the worry condition reported a significant decrease,  $F(1, 28) = 11.97, p = .002, \eta_p^2 = .29$ , and those in the relax and neutral inductions reporting no change,  $F(1, 58) = .18, p = .67, \eta_p^2 = .003$ . However, in terms of absolute levels of reported *anger* during the fear exposure, there was no effect of group,  $F(1, 90) = 3.27, p = .07$ ,

$\eta_p^2 = .037$ , induction,  $F(2, 89) = 1.95$ ,  $p = .15$ ,  $\eta_p^2 = .04$ , or an interaction,  $F(3, 87) = .53$ ,  $p = .59$ ,  $\eta_p^2 = .01$ .

For reported *sadness* levels from the induction to the fear exposure, the repeated measures ANOVA was significant by time,  $F(1, 88) = 12.88$ ,  $p < .001$ ,  $\eta_p^2 = .13$ , time by induction,  $F(3, 85) = 8.58$ ,  $p < .001$ ,  $\eta_p^2 = .17$ , and a time by group by induction interaction,  $F(4, 83) = 6.44$ ,  $p = .002$ ,  $\eta_p^2 = .13$ . For participants with GAD in the worry induction, there was a significant decrease in reported *sadness* from induction to fear exposure,  $F(1, 13) = 6.02$ ,  $p = .03$ ,  $\eta_p^2 = .30$ , whereas participants with GAD in the relax and neutral inductions experienced a significant increase,  $F(1, 28) = 26.46$ ,  $p < .001$ ,  $\eta_p^2 = .49$ . On the other hand, controls in the worry condition reported no change from induction to exposure,  $F(1, 13) = .68$ ,  $p = .43$ ,  $\eta_p^2 = .046$ , whereas controls in relaxation and neutral inductions reported an increase,  $F(1, 28) = 7.05$ ,  $p = .014$ ,  $\eta_p^2 = .20$ . In terms of absolute levels of reported *sadness* during the fear exposure, there was no effect of group,  $F(1, 90) = 2.54$ ,  $p = .11$ ,  $\eta_p^2 = .029$ , induction,  $F(2, 89) = 1.52$ ,  $p = .22$ ,  $\eta_p^2 = .03$ , or an interaction,  $F(3, 87) = 1.67$ ,  $p = .19$ ,  $\eta_p^2 = .037$ .

For reported *tension* levels, the repeated measures ANOVA was significant by time,  $F(1, 89) = 13.82$ ,  $p < .001$ ,  $\eta_p^2 = .14$ , and time by induction type,  $F(3, 86) = 10.82$ ,  $p < .001$ ,  $\eta_p^2 = .20$ . From the induction to the fear exposure, those in the worry condition reported no significant change in levels of *tension*,  $F(1, 29) = 3.38$ ,  $p = .076$ ,  $\eta_p^2 = .10$ , whereas those in the relax and neutral inductions reported a significant increase,  $F(1, 58) = 30.30$ ,  $p < .001$ ,  $\eta_p^2 = .35$ . In terms of absolute levels of reported *tension* during the fear exposure, there was no effect of group,  $F(1, 90) = 1.69$ ,  $p = .20$ ,  $\eta_p^2 = .019$ , or induction,  $F(2, 89) = 1.24$ ,  $p = .29$ ,  $\eta_p^2 = .028$ .

For reported NA levels, the repeated measures ANOVA was significant by time,  $F(1, 91) = 15.93, p < .001, \eta_p^2 = .16$ , and time by induction type,  $F(3, 88) = 19.09, p < .001, \eta_p^2 = .31$ . From the induction to the fear exposure, those in the worry condition reported a significant decrease in NA levels,  $F(1, 29) = 6.93, p = .01, \eta_p^2 = .19$ , whereas those in the relax and neutral inductions reported a significant increase,  $F(1, 60) = 47.28, p < .001, \eta_p^2 = .45$ . However, in terms of absolute levels of reported NA during the fear exposure, there was no effect of induction,  $F(2, 90) = 2.27, p = .11, \eta_p^2 = .05$ , or a group by induction interaction,  $F(3, 88) = 1.45, p = .24, \eta_p^2 = .03$ . However, there was an effect of group,  $F(1, 91) = 7.16, p = .009, \eta_p^2 = .076$ , in that participants with GAD reported higher overall NA than nonanxious controls following the fear clip regardless of induction type ( $M = 16.54, SD = 4.97; M = 14.09, SD = 4.94$ , respectively) (See Figure 5 for change from baseline to induction to fear exposure).

**Physiological measures.** When looking at change in SCL from induction period to the fear exposure, there was no effect of group,  $F(1, 88) = .24, p = .78, \eta_p^2 = .006$ , induction type,  $F(2, 87) = 1.63, p = .17, \eta_p^2 = .037$ , or an interaction,  $F(3, 85) = .16, p = .96, \eta_p^2 = .004$ . When observing each group separately, there was still no effect of induction. In measuring differences in absolute SCL during the fear exposure, there was also no effect of group,  $F(1, 88) = .48, p = .70, \eta_p^2 = .017$ , induction type,  $F(2, 87) = .87, p = .52, \eta_p^2 = .03$ , or an interaction,  $F(3, 85) = 1.11, p = .36, \eta_p^2 = .038$ . Again, there was still no effect of induction when looking at each group separately.

When observing change in NS-SCRs from induction to the fear exposure, there was no effect of group,  $F(1, 88) = .03, p = .86, \eta_p^2 < .001$ , or an interaction,  $F(3, 85) = 1.66, p = .20, \eta_p^2 = .038$ . There was an effect of induction type,  $F(2, 87) = 10.85, p < .001, \eta_p^2 = .21$ , in that

relaxation actually led to a greater decrease in NS-SCRs in response to the first minute of the fear exposure ( $M = -4.88, SD = 5.93$ ) than did worry ( $M = 1.28, SD = 5.35; p < .001$ ) and neutral inductions ( $M = -1.03, SD = 4.0; p = .015$ ), which were statistically similar ( $p = .28$ ). This was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression. When divided by group, the control group no longer demonstrated significant differences between induction types, whereas the GAD group did maintain these effects,  $F(2, 41) = 7.87, p < .001, \eta_p^2 = .28$ . In measuring differences in absolute levels of NS-SCRs during the fear exposure, there was no effect of group,  $F(1, 88) = .27, p = .60, \eta_p^2 = .003$ , or an interaction,  $F(3, 85) = .21, p = .81, \eta_p^2 = .005$ . However, there was an effect of induction type,  $F(2, 87) = 3.56, p = .03, \eta_p^2 = .08$ . When using Bonferroni post-hoc tests, worry led to a marginally greater number of NS-SCRs ( $M = 12.93, SD = 6.86$ ) than relaxation ( $M = 9.88, SD = 3.65; p = .052$ ) and neutral inductions ( $M = 10.10, SD = 3.32; p = .09$ ) which were statistically similar ( $p = 1.0$ ). This effect remained when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression.

When comparing change in amplitude average from induction to the fear exposure, there was a significant effect of induction type,  $F(2, 85) = 3.24, p = .04, \eta_p^2 = .07$ , but no effect of group,  $F(1, 86) = .47, p = .70, \eta_p^2 = .018$ , or an interaction,  $F(3, 83) = .45, p = .84, \eta_p^2 = .017$ . When observing Bonferroni post-hoc tests, relaxation led to a significantly greater change in average amplitude in response to the fear exposure than did worry ( $p = .04$ ) with the neutral induction falling non-significantly in between ( $p = 1.0; p = .33$ , respectively). This effect remained when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression (see Figure 6). However, for absolute levels of amplitude average during

the fearful exposure, there was no effect of group,  $F(1, 87) = 1.19, p = .28, \eta_p^2 = .01$ , induction type,  $F(2, 86) = 2.17, p = .12, \eta_p^2 = .05$ , or an interaction,  $F(3, 84) = .55, p = .58, \eta_p^2 = .01$ , which was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression.

When observing change in HR from induction to the fear exposure, there was no effect of group,  $F(1, 87) = .02, p = .98, \eta_p^2 = .001$ , induction type,  $F(2, 86) = .85, p = .49, \eta_p^2 = .02$ , or an interaction,  $F(3, 84) = 1.58, p = .18, \eta_p^2 = .037$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. Looking at each group separately, there was still no effect of induction type. For absolute HR scores during the fear exposure, there was no effect of group,  $F(1, 87) = .25, p = .78, \eta_p^2 = .006$ , induction type,  $F(2, 86) = 1.28, p = .28, \eta_p^2 = .03$ , or an interaction,  $F(3, 84) = 1.81, p = .13, \eta_p^2 = .04$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. When looking at each group separately, there was still no effect of induction type.

When comparing change in RSA from induction to the fear exposure, there was no effect of group,  $F(1, 87) = 1.39, p = .25, \eta_p^2 = .049$ , induction type,  $F(2, 86) = 1.24, p = .29, \eta_p^2 = .043$ , or an interaction,  $F(3, 84) = 1.14, p = .34, \eta_p^2 = .04$ , which was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression. When observing each group separately, there was still no effect of induction type. When comparing absolute levels of RSA during the fear exposure, there was no effect of group,  $F(1, 87) = .36, p = .78, \eta_p^2 = .01$ , induction type,  $F(2, 86) = .55, p = .77, \eta_p^2 = .02$ , or an interaction,  $F(3, 84) = .85, p = .53, \eta_p^2 = .03$ , which was true when controlling for medication use, exposure order, ethnicity,

age, gender, and comorbid depression. Again, when observing each group separately, there was still no effect of induction type.

### **Sad exposure.**

**Subjective measures.** For the sad exposure, there were a number of significant findings from repeated measures analyses for emotion adjectives, including *sadness*, *anger*, *fear*, and *tension*. For reported *sadness* levels, the repeated measures ANOVA was significant by time,  $F(1, 88) = 274.35, p < .001, \eta_p^2 = .77$ , and time by induction type,  $F(3, 85) = 19.46, p < .001, \eta_p^2 = .32$ . From the induction to the sad exposure, all conditions experienced an increase in reported levels of *sadness*, but those in the worry condition increased less than both those in relaxation,  $F(2, 56) = 32.81, p < .001, \eta_p^2 = .36$ , and neutral conditions,  $F(2, 53) = 17.81, p < .001, \eta_p^2 = .24$ , who did not differ significantly from each other. In terms of absolute levels of reported *sadness* during the sad exposure, there was no effect of group,  $F(1, 91) = .00, p = 1.0, \eta_p^2 < .001$ , induction,  $F(2, 90) = 1.08, p = .35, \eta_p^2 = .02$ , or an interaction,  $F(3, 88) = .53, p = .59, \eta_p^2 = .01$  (see Figure 7).

For reported *anger* with group and induction type as between subject variables, and time and *anger* levels as within subject variables, the repeated measures ANOVA was significant by time by induction type,  $F(3, 85) = 10.09, p < .001, \eta_p^2 = .19$ . From the induction to the sad exposure, those in the worry condition reported a significant decrease in *anger*,  $F(1, 28) = 7.80, p = .009, \eta_p^2 = .21$ , with those in the relax and neutral inductions reporting a significant increase,  $F(1, 58) = 11.97, p = .001, \eta_p^2 = .17$ , in anger levels. In terms of absolute levels of reported *anger* during the sad exposure, there was no effect of group,  $F(1, 91) = 2.18, p = .14, \eta_p^2 = .02$ , induction,  $F(2, 90) = .16, p = .85, \eta_p^2 = .004$ , or an interaction,  $F(3, 88) = .76, p = .47, \eta_p^2 = .02$ .

For reported *fear* levels, the repeated measures ANOVA was significant for time by induction type,  $F(3, 83) = 13.30, p < .001, \eta_p^2 = .25$ . From the induction to the sad exposure, those in the worry condition reported a significant decrease in levels of *fear*,  $F(1, 29) = 16.83, p < .001, \eta_p^2 = .36$ , whereas those in the relax and neutral inductions reported a significant increase,  $F(1, 55) = 10.60, p = .002, \eta_p^2 = .16$ . In terms of absolute levels of reported *fear* during the sad exposure, there was no effect of group,  $F(1, 92) = 2.39, p = .13, \eta_p^2 = .026$ , induction,  $F(2, 91) = 2.34, p = .10, \eta_p^2 = .05$ , or an interaction,  $F(3, 89) = .42, p = .66, \eta_p^2 = .009$ .

For reported *tension* levels, the repeated measures ANOVA was significant only in the time by induction type interaction,  $F(3, 87) = 12.58, p < .001, \eta_p^2 = .23$ . From the induction to the sad exposure, those in the worry condition reported a significant decrease in levels of *tension*,  $F(1, 29) = 12.84, p = .001, \eta_p^2 = .30$ , whereas those in the relax and neutral inductions reported a significant increase,  $F(1, 59) = 13.41, p = .001, \eta_p^2 = .18$ . In terms of absolute levels of reported *tension* during the sad exposure, there was no effect of group,  $F(1, 92) = .00, p = .99, \eta_p^2 < .001$ , induction,  $F(2, 91) = 1.13, p = .33, \eta_p^2 = .025$ , or an interaction,  $F(3, 89) = .49, p = .61, \eta_p^2 = .01$ .

For reported NA levels, the repeated measures ANOVA was significant by time,  $F(1, 90) = 9.13, p = .003, \eta_p^2 = .10$ , and time by induction type,  $F(3, 87) = 26.10, p < .001, \eta_p^2 = .38$ . From the induction to the sad exposure, those in the worry condition reported a significant decrease in NA levels,  $F(1, 29) = 11.78, p = .002, \eta_p^2 = .28$ , whereas those in the relax and neutral inductions reported a significant increase,  $F(1, 59) = 55.84, p < .001, \eta_p^2 = .48$ . In terms of absolute levels of reported NA during the sad exposure, there was no effect of group,  $F(1, 91) = 2.19, p = .14, \eta_p^2 = .025$ , induction,  $F(2, 90) = 2.70, p = .07, \eta_p^2 = .058$ , or an interaction,  $F(3, 88) = .68, p = .51, \eta_p^2 = .015$  (see Figure 8).

**Physiological measures.** When looking at change in SCL from induction period to the sad exposure, there was no effect of group,  $F(1, 88) = .33, p = .72, \eta_p^2 = .008$ , induction type,  $F(2, 87) = .93, p = .45, \eta_p^2 = .02$ , or an interaction,  $F(3, 85) = 1.66, p = .16, \eta_p^2 = .038$ . In measuring differences in absolute SCL during the sad exposure, there was also no effect of group,  $F(1, 88) = .19, p = .91, \eta_p^2 = .007$ , induction type,  $F(2, 87) = 1.00, p = .43, \eta_p^2 = .035$ , or an interaction,  $F(3, 85) = .57, p = .76, \eta_p^2 = .02$ . This held true when observing each group separately.

When observing change in NS-SCRs from induction to the sad exposure, there was no effect of group,  $F(1, 88) = .84, p = .44, \eta_p^2 = .02$ , or an interaction,  $F(3, 85) = 1.22, p = .31, \eta_p^2 = .028$ . There was an effect of induction type for the first minute of the sad exposure,  $F(2, 87) = 4.01, p = .022, \eta_p^2 = .09$ , in that relaxation actually led to a greater decrease in NS-SCRs ( $M = -2.66, SD = 5.42$ ) than did worry ( $M = 1.0, SD = 5.56; p = .037$ ), with the neutral induction falling nonsignificantly in between ( $M = .50, SD = 5.8; p = .08; p = 1.0$ , respectively). This was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression. When divided by group, the controls no longer demonstrated this effect, whereas the GAD group did,  $F(2, 41) = 3.55, p = .038, \eta_p^2 = .15$ . In measuring differences in absolute levels of NS-SCRs during the sad exposure, there was no effect of group,  $F(1, 88) = .26, p = .78, \eta_p^2 = .006$ , induction type,  $F(2, 87) = .19, p = .94, \eta_p^2 = .004$ , or an interaction,  $F(3, 85) = 1.88, p = .12, \eta_p^2 = .04$ . This was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression, and was also true when observing each group separately.

When comparing change in amplitude average from induction to the sad exposure, there was no main effect of group,  $F(1, 87) = .90, p = .45, \eta_p^2 = .03$ , induction type,  $F(2, 86) = .70, p =$

.65,  $\eta_p^2 = .025$ , or an interaction,  $F(3, 84) = 1.26, p = .28, \eta_p^2 = .04$ . This was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression. In a repeated measures ANOVA with time and amplitude average as within subject variables and group and induction type as between subject variables, there was a significant effect of time,  $F(1, 87) = 22.14, p < .001, \eta_p^2 = .21$ , in that all groups and induction types experienced a significant increase in amplitude averages in response to the second minute of the sad exposure (see Figure 9). For absolute levels of amplitude average during the sad exposure, there was no effect of group,  $F(1, 88) = 1.35, p = .27, \eta_p^2 = .047$ , induction type,  $F(2, 87) = .73, p = .63, \eta_p^2 = .026$ , or an interaction,  $F(3, 85) = 1.63, p = .14, \eta_p^2 = .056$ , which was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression, and was also true when observing each group separately.

When observing change in HR from induction to the sad exposure, there was no effect of group,  $F(1, 88) = .25, p = .86, \eta_p^2 = .009$ , induction type,  $F(2, 87) = .85, p = .53, \eta_p^2 = .03$ , or an interaction,  $F(3, 85) = 1.54, p = .17, \eta_p^2 = .053$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. Looking at each group separately, there was still no effect of induction type. For absolute HR scores during the sad exposure, there was no effect of group,  $F(1, 88) = .03, p = 1.0, \eta_p^2 = .001$ , or induction type,  $F(2, 87) = 1.50, p = .18, \eta_p^2 = .05$ , but there was an effect of the interaction,  $F(3, 85) = 2.37, p = .032, \eta_p^2 = .08$ , which was true when controlling for order of exposure type, age, ethnicity, gender, and comorbid depression, but not when controlling for medication. There was no effect of induction for controls,  $F(2, 44) = 1.02, p = .42, \eta_p^2 = .067$ . However, there was an effect for the GAD group,  $F(2, 44) = 4.46, p = .018, \eta_p^2 =$

.18, in that those in the relaxation condition had lower HR during the sad exposure ( $M = 66.80$ ,  $SD = 22.49$ ) than the neutral condition ( $M = 86.69$ ,  $SD = 14.08$ ,  $p = .015$ ), but not the worry condition ( $M = 74.13$ ,  $SD = 14.80$ ,  $p = .79$ ), with worry and neutral being statistically similar ( $p = .21$ ).

When comparing change in RSA from induction to the sad exposure, there was no effect of group,  $F(1, 87) = .91$ ,  $p = .44$ ,  $\eta_p^2 = .03$ , induction type,  $F(2, 86) = .24$ ,  $p = .96$ ,  $\eta_p^2 = .01$ , or an interaction,  $F(3, 84) = 1.61$ ,  $p = .15$ ,  $\eta_p^2 = .056$ , which was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression. In a repeated measures ANOVA with time and RSA level as within subject variables and group and induction type as between subject variables, there was a significant effect of time,  $F(1, 87) = 6.16$ ,  $p = .015$ ,  $\eta_p^2 = .07$ , in that all groups and induction types experienced a significant increase in RSA in response to the sad exposure (see Figure 10). Also, when comparing absolute levels of RSA during the sad exposure, there was no effect of group,  $F(1, 87) = .57$ ,  $p = .63$ ,  $\eta_p^2 = .02$ , induction type,  $F(2, 86) = 1.23$ ,  $p = .29$ ,  $\eta_p^2 = .04$ , or an interaction,  $F(3, 84) = 1.40$ ,  $p = .22$ ,  $\eta_p^2 = .049$ , which was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression, as well as when observing each group separately.

### **Happy exposure.**

**Subjective measures.** For the happy exposure, there were several significant findings from repeated measures analyses for positive emotion adjectives, including *happiness*, *amusement*, and *relief*. For reported *happiness*, the repeated measures ANOVA was significant by time only,  $F(1, 88) = 144.82$ ,  $p < .001$ ,  $\eta_p^2 = .063$ . From the induction to the happy exposure, those in all conditions reported a significant increase in *happiness* levels. In terms of absolute

levels of reported *happiness* during the happy exposure, there was no effect of group,  $F(1, 91) = 3.27, p = .074, \eta_p^2 = .036$ , induction,  $F(2, 90) = 1.05, p = .36, \eta_p^2 = .02$ , or an interaction,  $F(3, 88) = 1.40, p = .25, \eta_p^2 = .03$  (see Figure 11).

For reported *amusement* with group and induction type as between subject variables, and time and *amusement* levels as within subject variables, the repeated measures ANOVA was significant by time,  $F(1, 89) = 383.57, p < .001, \eta_p^2 = .82$ , and time by induction type,  $F(3, 86) = 3.40, p = .038, \eta_p^2 = .074$ . From the induction to the happy exposure, those in the worry condition reported a slightly greater increase in *amusement* levels than did those in the neutral condition ( $p = .043$ ) but not those in relaxation ( $p = 1.0$ ), with no difference between relax and neutral ( $p = .20$ ). In terms of absolute levels of reported *amusement* during the happy exposure, there was no effect of group,  $F(1, 91) = .78, p = .38, \eta_p^2 = .009$ , induction,  $F(2, 90) = .65, p = .52, \eta_p^2 = .015$ , or an interaction,  $F(3, 88) = 1.83, p = .16, \eta_p^2 = .04$ .

For reported *relief*, the repeated measures ANOVA had a significant by time by induction type interaction only,  $F(3, 84) = 4.33, p = .016, \eta_p^2 = .094$ . From the induction to the happy exposure, whereas those in the relaxation and neutral conditions reported no change in levels of relief,  $F(2, 55) = .32, p = .58, \eta_p^2 = .005$ , those in the worry condition reported significant increases,  $F(1, 28) = 5.99, p = .021, \eta_p^2 = .17$ . In terms of absolute levels of reported *relief* during the happy exposure, there was no effect of group,  $F(1, 90) = 2.47, p = .12, \eta_p^2 = .028$ , induction,  $F(2, 89) = .07, p = .93, \eta_p^2 = .002$ , or an interaction,  $F(3, 87) = .03, p = .97, \eta_p^2 = .001$ .

For reported PA levels, the repeated measures ANOVA was significant by time only,  $F(1, 90) = 90.97, p < .001, \eta_p^2 = .51$ . From the induction to the happy exposure, all inductions led to significant increases in PA in response to the happy exposure. In terms of absolute levels

of reported PA during the happy exposure, there was no effect of group,  $F(1, 91) = 2.09, p = .15, \eta_p^2 = .02$ , induction,  $F(2, 90) = .63, p = .54, \eta_p^2 = .01$ , or an interaction,  $F(3, 88) = .80, p = .45, \eta_p^2 = .018$ .

For reported NA levels, there was a significant effect of time,  $F(1, 90) = 76.36, p < .001, \eta_p^2 = .47$ , and time by induction type,  $F(3, 87) = 40.05, p < .001, \eta_p^2 = .48$ . From the induction to the happy exposure, those in the worry condition reported a larger decrease in NA levels,  $F(1, 28) = 74.07, p < .001, \eta_p^2 = .72$ , than did those in the relax and neutral conditions, who reported a small decrease,  $F(1, 60) = 7.66, p = .007, \eta_p^2 = .11$ . In terms of absolute levels of reported NA during the happy exposure, there was no effect of group,  $F(1, 91) = .51, p = .15, \eta_p^2 = .005$ , induction,  $F(2, 90) = 2.46, p = .09, \eta_p^2 = .05$ , or an interaction,  $F(3, 88) = .84, p = .44, \eta_p^2 = .02$  (see Figure 12).

**Physiological measures.** When looking at change in SCL from induction period to the happy exposure, there was no effect of group,  $F(1, 87) = 2.02, p = .14, \eta_p^2 = .047$ , induction type,  $F(2, 86) = .26, p = .90, \eta_p^2 = .006$ , or an interaction,  $F(3, 84) = .36, p = .84, \eta_p^2 = .01$ , which was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression. In measuring differences in absolute SCL during the happy exposure, there was also no effect of group,  $F(1, 87) = .042, p = .84, \eta_p^2 = .001$ , induction type,  $F(2, 86) = .97, p = .38, \eta_p^2 = .02$ , or an interaction,  $F(3, 84) = 1.20, p = .31, \eta_p^2 = .03$ . This was also true when observing each group separately.

When observing change in NS-SCRs from induction to the happy exposure, there was no effect of group,  $F(1, 87) = .08, p = .93, \eta_p^2 = .002$ , induction type,  $F(2, 86) = .55, p = .70, \eta_p^2 = .01$ , or an interaction,  $F(3, 84) = .43, p = .79, \eta_p^2 = .01$ . This was true when controlling for

medication use, exposure order, ethnicity, age, gender, and comorbid depression, as well as when observing each group separately. In measuring differences in absolute levels of NS-SCRs during the happy exposure, there was no effect of group,  $F(1, 87) = 1.88, p = .16, \eta_p^2 = .04$ , or an interaction,  $F(3, 84) = .66, p = .62, \eta_p^2 = .02$ . However, there was an effect of induction type across both minutes of the happy exposure,  $F(2, 86) = 4.76, p = .01, \eta_p^2 = .10$ , in that worry led to greater NS-SCRs ( $M = 26.93, SD = 7.4$ ) than neutral ( $M = 20.90, SD = 7.42; p = .008$ ), but not relaxation ( $M = 23.94, SD = 6.88; p = .37$ ), with relaxation and neutral being statistically similar ( $p = .32$ ). This was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression.

When comparing change in amplitude average from induction to the happy exposure, there was a main effect of group,  $F(1, 87) = 3.36, p = .023, \eta_p^2 = .11$ , but no effect of induction type,  $F(2, 86) = .42, p = .87, \eta_p^2 = .015$ , or an interaction,  $F(3, 84) = .41, p = .87, \eta_p^2 = .015$ . Although both groups experienced increases in response to the happy exposure, individuals with GAD evidenced greater increase in amplitude average ( $M = .51, SD = .70$ ) than did controls ( $M = .18, SD = .76$ ), regardless of prior induction type. This was true when controlling for medication use, exposure order, ethnicity, age, and gender, but not when controlling for comorbid depression. For absolute levels of amplitude average during the happy exposure, there was no effect of group,  $F(1, 87) = 2.20, p = .14, \eta_p^2 = .026$ , induction type,  $F(2, 86) = .11, p = .89, \eta_p^2 = .003$ , or an interaction,  $F(3, 84) = .60, p = .55, \eta_p^2 = .01$ , which was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression.

When observing change in HR from induction to the happy exposure, there was no effect of group,  $F(1, 86) = .56, p = .64, \eta_p^2 = .02$ , induction type,  $F(2, 85) = .62, p = .71, \eta_p^2 = .02$ , or

an interaction,  $F(3, 83) = .88, p = .51, \eta_p^2 = .03$ , which was true when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. Looking at each group separately, there was still no effect of induction type. For absolute HR scores during the happy exposure, there was no effect of group,  $F(1, 86) = .54, p = .66, \eta_p^2 = .02$ , induction type,  $F(2, 85) = 1.08, p = .38, \eta_p^2 = .038$ , or an interaction,  $F(3, 83) = .96, p = .45, \eta_p^2 = .03$ , which was true when controlling for medication, order of exposure type, age, ethnicity, gender, and comorbid depression. There was no effect of induction when observing each group separately.

When comparing change in RSA from induction to the happy exposure, there was no effect of group,  $F(1, 85) = .76, p = .52, \eta_p^2 = .028$ , induction type,  $F(2, 84) = .44, p = .85, \eta_p^2 = .016$ , or an interaction,  $F(3, 82) = 1.29, p = .27, \eta_p^2 = .046$ , which was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression. In a repeated measures ANOVA with time and RSA level as within subject variables and group and induction type as between subject variables, there was a significant effect of time only,  $F(1, 85) = 23.27, p < .001, \eta_p^2 = .22$ , in that all participants experienced a significant increase in RSA from the induction to the happy exposure, regardless of group or prior induction type (see Figure 13). Also, when comparing absolute levels of RSA during the happy exposure, there was no effect of group,  $F(1, 85) = 1.23, p = .27, \eta_p^2 = .015$ , induction type,  $F(2, 84) = .79, p = .46, \eta_p^2 = .019$ , or an interaction,  $F(3, 83) = 1.40, p = .25, \eta_p^2 = .03$ , which was true when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression, and was also true when observing each group separately.

### **Hypothesis Three: Effects of Inductions in Emotional Coping Differ Between GAD and Nonanxious Control Participants**

The responses to the emotional coping questions were summed to create a total emotion coping score. Higher scores suggested that individuals found inductions to be more helpful in coping with negative emotional exposures, whereas lower scores suggest individuals did not find inductions helpful in coping with exposures. An ANOVA was run using group and induction type as independent variables and coping total scores as the dependent variable. There was a significant effect of group,  $F(1, 85) = 7.45, p = .008, \eta_p^2 = .084$ , and a group by induction type interaction,  $F(2, 84) = 12.05, p < .001, \eta_p^2 = .23$ .

For individuals in the GAD group, there was a significant effect of induction type,  $F(2, 42) = 5.32, p = .009, \eta_p^2 = .20$ . When using least significant difference (LSD) post-hoc tests to compare induction types, it was found that worry led to significantly higher emotional coping scores than both relaxation ( $p = .036$ ) and neutral ( $p = .003$ ) inductions, which were statistically similar ( $p = .27$ ). For non-anxious control participants, the opposite effect was found. There was also a significant effect of induction type,  $F(2, 39) = 7.61, p = .002, \eta_p^2 = .28$ , but LSD post-hoc tests for this group indicated that worry led to significantly lower emotional coping scores than both relaxation ( $p = .001$ ) and neutral ( $p = .004$ ) inductions, which were statistically similar ( $p = .61$ ) (see Figure 14).

When comparing induction types between groups, it was found that for the worry induction, those in the GAD group were more likely than nonanxious controls to indicate that prior worry had helped them feel more able to cope during the emotional film clips,  $F(1, 26) = 5.09, p = .033, \eta_p^2 = .16$ . The opposite pattern was found for the relaxation and neutral

inductions, in that participants with GAD rated relaxation,  $F(1, 28) = 12.31, p = .002, \eta_p^2 = .31$ , and neutral inductions,  $F(1, 27) = 15.87, p < .001, \eta_p^2 = .37$ , as having made them feel less able to cope during the film clip than did nonanxious controls.

### Discussion

The Contrast Avoidance Model of Worry (Newman & Llera, 2011) states that worry leads to a heightened negative emotional state that is sustained across negative emotional exposures, and somewhat alleviated in response to positive emotional exposures. Therefore, we had hypothesized that the worry induction (versus relaxation and neutral inductions) would lead to greater negative emotionality relative to resting baseline, as well as to the preclusion of autonomic and subjective reactivity to the fearful stimulus, preclusion of subjective reactivity to the sad stimulus, and a decrease in negative emotionality in response to the happy stimulus in both GAD and control participants. We had also hypothesized that absolute levels of emotionality *during* the emotional stimuli would not differ by prior induction type, suggesting that although worry may preclude *reactivity* to emotional stimuli (and thereby, emotional processing), it does not lead to emotional avoidance. Finally, we had hypothesized that individuals with GAD would be more sensitive to a negative emotional contrast, and would therefore report more difficulty in coping with their emotions following a negative emotional contrast experience. The results partially supported these hypotheses.

Initially there were some group differences at resting baseline, in that the GAD group reported higher resting levels of negative emotionality than nonanxious controls. Specifically, the GAD group reported lower levels of relaxation and marginally higher levels of worry, lower levels of positive emotional adjectives (i.e., *contentment, happiness, and interest*) and higher

levels of negative adjectives (i.e., *embarrassment* and *sadness*), lower PA, and marginally higher NA. These findings are similar to Brown, Chorpita, and Barlow (1998), who characterize GAD as a disorder of high negative and low positive affect. As predicted, the GAD group also reported higher levels of depression than the control group.

Physiologically, the only difference between groups at baseline was that the GAD group evidenced smaller average amplitude of NS-SCR responses, which will be discussed in more detail in the following section. In terms of heart rate, the GAD and control groups did not differ from each other; however, the GAD group evidenced a significant within group difference between those randomly assigned to the relaxation and neutral conditions. Those in the neutral condition had on average a higher resting heart rate than those in relaxation, with those in the worry condition nonsignificantly in between. It is possible that this baseline difference could have influenced effects throughout the experiment.

Data from the manipulation check indicated that worry and relaxation manipulations had the intended subjective impact such that all participants reported feeling more worried and anxious during the worry induction than the relax and neutral inductions, and more relaxed during the relaxation induction as compared to the worry induction, and marginally more than the neutral induction. Furthermore, the inductions had the same impact throughout the experiment and did not diminish over time. However, participants with GAD tended to report higher levels of worry and anxiety and lower levels of relaxation, regardless of induction type.

### **Hypothesis One: Worry Leads to Heightened Negative Emotionality**

Consistent with the Contrast Avoidance Model, subjective indices clearly demonstrated that worry led to both a greater increase in negative emotionality from baseline as well as greater

absolute levels of negative emotionality during the induction period than both relaxation and neutral activity. This was true for a number of emotion adjectives as well as NA levels, even when controlling for order effects and comorbid depression. Therefore, subjective emotionality measures support the hypothesis that worry leads to heightened negative emotionality. Also, on average, individuals with GAD reported higher NA and lower PA than controls regardless of induction type, which was similar to higher levels of NA, worry, and anxiety and lower relaxation that were reported at baseline.

As for physiological measures, there were some group differences in parasympathetic and sympathetic measures across induction types. On average, the GAD group evidenced lower response amplitudes during induction periods than controls, regardless of condition, similar to scores at baseline. However, this was no longer significant when controlling for comorbid depression.<sup>1</sup> Also, data indicate that the GAD group demonstrated less change in RSA than controls in response to inductions regardless of condition. Together, these findings are similar to studies showing that GAD is associated with reduced autonomic range (Hoehn-Saric et al., 2004; Hoehn-Saric et al., 1989) as compared to controls, which suggests the possibility of physiological rigidity in this group. However, the results of change in SNS activity, described below, are counter-indicative to the hypothesis that the GAD group demonstrated physiological rigidity.

When observing changes from baseline in SNS activity it was found that, as predicted, worry led to a greater increase in average amplitude during the induction period whereas

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<sup>1</sup> Data from this study show a negative correlation between comorbid depression and amplitude average scores, which is at odds with most studies showing depression to be associated with increased SNS activity (e.g., Guinjoan, Bemabo, & Cardinali, 1995; Udupa et al., 2007; Veith et al., 1994).

relaxation led to a greater decrease, with the neutral induction falling nonsignificantly in between. This effect was significant for both groups, and remained when controlling for current anti-anxiety or anti-depression medication use, order of exposure type, age, ethnicity, gender, and comorbid depression. However, this was true only for the induction period that preceded the fear exposure, and not the other two induction periods. Interestingly, for the GAD group (but not for controls), those in the relaxation condition evidenced more NS-SCRs than those in worry (with neutral nonsignificantly in between), which is contrary to findings that increased NS-SCRs are associated with anxiety and worry (e.g., Nikula, Klinger, & Larson-Gutman, 1993; Upatel & Gerlach, 2008). However, given that change from baseline in average amplitude of these responses matched predictions, this suggests that despite relaxation leading to more responses, those responses decreased in amplitude from baseline as compared to the worry condition, suggesting less sympathetic activity in this condition. In terms of heart rate, for both groups those in relaxation experienced lower heart rate than worry and neutral inductions, though only the difference between relaxation and neutral was significant. Given that there was a difference in the GAD group with those in the neutral condition evidencing higher heart rates at baseline, these findings may be influenced by the trait physiology of participants in neutral condition as opposed to the neutral induction itself.

As for parasympathetic activity (RSA), there were no absolute level differences between induction types for either the GAD or control groups. However, the control group (but not the GAD group) did evidence change in RSA from baseline to the induction period prior to the fear exposure. For these participants, relaxation led to a significantly greater increase in parasympathetic activity than worry and neutral activity, which were statistically similar. This

effect was no longer significant when controlling for respiration; however, research evidence shows that respiration rate has little to no effect on RSA within a range of normal breathing or when there are no substantial breathing differences between groups (see Grossman & Taylor, 2007). We therefore inspected the data for outlying respiration cases as well as condition differences on breathing rate. Given that there were no respiration outliers in this sample nor were there group differences in respiration rate, we can cautiously interpret this effect of RSA as valid. If so, data indicate that for controls the impact of inductions was limited to relaxation increasing, rather than worry decreasing, parasympathetic activity. Also, the fact that no changes by induction type were evident in the GAD group could result from this group evidencing fewer RSA changes than controls overall during induction periods.

In sum, physiological data partially support the hypothesis that worry leads to heightened negative emotionality, although the physiological evidence to support this hypothesis was limited.

### **Hypothesis Two: Worry Leads to Sustained Negative Emotion Across Negative Exposures**

The second hypothesis stated that following the initially higher negative emotionality, those in the worry condition would experience sustained negative emotionality through negative exposures, whereas those in relaxation and neutral conditions would experience a significant increase in negative emotionality, or a negative emotional contrast, in response to negative exposures. Also, this hypothesis stated that absolute levels of emotionality would be equivalent during the exposure regardless of prior induction, suggesting that whereas emotional processing would be avoided via prior worry, negative emotional experiencing would not. Again, results were partially supportive of this hypothesis and differed across emotional exposures.

Consistent with the Contrast Avoidance Model, higher levels of subjective negative emotionality following worry were either sustained across the fear exposure or slightly decreased, whereas lower levels of negative emotionality following relaxation and neutral inductions were significantly increased in response to the fear exposure. The fact that worry differentiated from both relaxation and neutral conditions suggests that these differences were more strongly driven by worry than relaxation. Importantly, absolute levels of negative emotionality during the fear exposure were similar regardless of prior induction type, suggesting that worry may have led to avoidance of a negative emotional contrast (and diminished emotional processing) in response to the fearful exposure, but not to avoidance of subjective negative emotionality. In terms of negative affect, the GAD group reported overall higher levels following the fear clip regardless of prior induction type, which was similar to higher levels across both baseline and induction periods.

As for physiological responding, again data were partially supportive of the Contrast Avoidance Model. Despite prior relaxation leading to a decrease in NS-SCRs during the fear exposure for the GAD group compared to worry and neutral inductions, and prior worry leading to a greater number of responses for both groups than relaxation and neutral, the increase in amplitude of these responses (and therefore sympathetic activity) was greater for those in the relaxation condition than the worry condition (which demonstrated no increase), with the neutral induction falling nonsignificantly in between. This effect was evident for both groups, and held when controlling for medication use, exposure order, ethnicity, age, gender, and comorbid depression. Importantly, absolute levels of amplitude responses were similar during the fearful exposure regardless of prior induction. However, prior worry did not lead to differences in

parasympathetic activity in response to the fear exposure. Therefore, these data partially support the hypothesis that for the fear exposure, worry facilitated avoidance of a negative emotional contrast whereas relaxation did not avoid a contrast, and neither condition avoided negative emotional experiencing.

For the sad exposure, we had predicted that prior worry would preclude subjective reactivity, but not physiological reactivity, and that all conditions would reach similar absolute levels of emotionality. Indeed, for those in the worry condition, higher levels of subjective negative emotionality on most variables were either sustained across the sad exposure or slightly decreased, whereas those in the relaxation and neutral inductions reported a significant increase. However, for reported levels of sadness, prior worry did not fully avoid but may have mitigated a negative emotional contrast. Specifically, subsequent to the worry induction, reported sadness levels did increase in response to the sad exposure, but they increased *less* for those who worried prior to exposure than for those in relaxation and neutral conditions. Again, absolute levels of negative emotionality during the sad exposure were similar regardless of prior induction type, suggesting that whereas emotional processing might have been mitigated for those in the prior worry condition, subjective negative emotionality was not.

We had also hypothesized that similar to Llera & Newman (2010), prior worry would not preclude physiological responding to the sad exposure, predicting that both parasympathetic and sympathetic activity would increase. For GADs, prior relaxation led to a decrease in NS-SCRs during the sad exposure, whereas worry led to a slight increase and neutral was nonsignificantly in between. However, all participants experienced an increase in the average amplitude of these responses to the sad exposure, with all groups and induction types reaching similar levels during

the film clip, which does support hypotheses. In terms of heart rate, the GAD group experienced lower absolute levels of heart rate in the relaxation condition than worry and neutral conditions during the film clip. However, there were no significant changes in heart rate in response to the sad exposure. It is therefore possible that this effect could have been due to initially lower heart rate in the relaxation condition versus neutral and worry conditions at baseline. For parasympathetic activity, as predicted, all induction types led to significant increases in RSA in response to the sad exposure. In sum, data suggest that as hypothesized, worry did preclude and/or mitigate subjective responding to the sad exposure, but did not preclude physiological responding to the sad exposure.

Finally, for the happy exposure, we had predicted that prior worry would not preclude either subjective or physiological responding to this exposure, but would lead to a positive emotional contrast. For positive emotional adjectives and PA scores, all individuals experienced an increase in response to the happy exposure, regardless of prior induction type, which is consistent with findings from Llera and Newman (2010). For NA scores, participants in the worry condition experienced a significantly greater *decrease* in NA than did those in relax and neutral conditions, suggesting a positive emotional contrast. As for NS-SCRs, similar to the sad exposure the prior worry induction led to greater NS-SCRs than the other two conditions, though change scores did not vary by induction. However, when divided by group, this effect disappeared. Finally, as predicted, all participants experienced a significant increase in both sympathetic (amplitude average – though the GAD group increased more than controls, possibly due to initially lower levels) and parasympathetic (RSA) activity in response to the happy exposure, regardless of prior induction. Importantly, all groups and induction types reached

similar levels during the happy clip. Overall, this suggests that worry led to a positive emotional contrast in response to the happy exposure.

In sum, worry led to increased subjective negative affect, and some increases in physiological arousal of the sympathetic system, as compared to relaxation and neutral inductions, relative to a resting baseline. Following induction periods, for the fear exposure worry precluded further increases in subjective negative affect as well as some sympathetic reactivity, whereas relaxation and neutral activity led to a sharp negative emotional contrast in terms of subjective emotionality and some sympathetic reactivity. For the sad exposure, prior worry mitigated or avoided a negative emotional contrast for subjective variables, but did not interfere with physiological responding (which is similar to findings from Llera & Newman, 2010). For the happy exposure, prior worry led to a positive emotional exposure, in that it did not preclude positive emotional responding, and decreased negative affect relative to the prior worry induction period.

### **Hypothesis Three: GAD Participants Are More Averse to Negative Emotional Contrast**

The Contrast Avoidance Model also states that individuals with GAD have developed a stronger aversive reaction and are even more sensitive to a negative emotional contrast than are nonanxious individuals, and that it is the avoidance of this contrast that motivates their worry. In order to test this aspect of the model, we had included a questionnaire designed to ascertain a holistic appraisal of the overall effect of the worry, relaxation, or neutral induction on ability to cope with their emotionality *during* the various emotion-inducing film clips. These preliminary results support this hypothesis.

Results indicate a significant difference between participants with GAD and nonanxious controls on experience of their emotional responses to the film clips following the different inductions. Those in the GAD group reported feeling that prior worry made it *easier* to cope with their emotions to the film clips than both relaxation and neutral inductions. However, controls reported the opposite pattern, in that prior worry made them feel *less* able to cope with subsequent emotions than either relaxation or neutral inductions. Similarly, GAD participants rated relaxation and neutral inductions as having made them feel *less* able to cope during the film clip than did nonanxious controls, whereas nonanxious controls were more likely to indicate that relaxation and neutral inductions helped them feel *more* able to cope compared to those with GAD.

This effect is particularly interesting, in that despite reporting that worry helped them cope, the GAD group actually reported their absolute level of negative affect as being significantly higher than did controls across both worry and subsequent negative clips. Similarly, nonanxious controls, despite experiencing less negative affect than the GAD group, actually found worry as a coping strategy to be less helpful than those with GAD. This finding that participants with GAD experience worry as both leading to greater sustained negative emotion and helping them cope, while far from being a contradiction, in fact highlights that what they found so helpful was to avoid the contrast experience itself, rather than negative emotion *per se*.

These findings may suggest that people with GAD neither avoid nor process negative emotions because, rather than feel relaxed or euthymic, they may prefer to experience the chronically negative emotion associated with worry in order to avoid being vulnerable to a

negative emotional contrast. This suggests that the experience of a negative contrast (i.e., as opposed to worry, feeling relaxed or neutral created a sharp increase in subjective and some physiological negative emotionality in response to the fearful stimulus, and an increase in subjective negative affect in response to the sad stimulus) was more upsetting for the GAD participants than nonanxious controls. Clearly, this discomfort with a negative emotional contrast is different from simply having a fear of negative emotions. Also consistent with a reluctance to let down their guard, people with GAD seem to be especially prone to experiencing relaxation induced anxiety (Heide & Borkovec, 1983, 1984). Perhaps this is due to the fact that relaxation leaves them vulnerable to experiencing a negative contrast in their emotions.

Again, these findings are consistent with Gray's (1982) neurophysiological theory of anxiety, which states that one trigger for anxious responding is the detection of a mismatch between expected and encountered stimuli in the environment. As such, if individuals use worry to prepare for the worst on an emotional level, then they are reducing the likelihood of a negative emotional contrast experience (similar to a mismatch) if they do experience a negative event. Importantly, this argument suggests that the use of worry as a means of emotional contrast avoidance could interfere with the ability to terminate the worry process, as any reduction in worry (including parallel decreases in negative affect) may be an internal anxiety trigger. Because the cycle has been negatively reinforced via relief when the feared event does not occur and/or avoidance of an emotional contrast if it does occur, this anxiety signal will most likely lead to a reactivation of the worry state. Additionally, the likelihood of experiencing a positive contrast (going from a bad to a good state) becomes that much greater if one is always prepared for the worst outcome. Therefore, these individuals may greatly prefer to feel emotions

associated with being pleasantly surprised (i.e., a positive emotional contrast) over unexpectedly disappointed and upset (i.e., a negative emotional contrast), thus also leading to positive reinforcement of the worry process.

### **Comparison Between the Contrast Avoidance Model and Extant Theories of GAD**

A comparison can be made between the Contrast Avoidance Model of GAD and current theories of GAD, including the Intolerance of Uncertainty (IU) model, the Metacognitive (MC) model, the Acceptance-Based (AB) model, and the Emotion Dysregulation (ED) model. In the following section, each model will be reviewed. Models will not be presented in their entirety, but specifically in the ways that each addresses difficulties in emotion regulation as part of GAD maintenance. Then this aspect of each model will be compared to the Contrast Avoidance model.

The IU model (Dugas, Buhr, & Ladouceur, 2004; Dugas et al., 1998; Dugas, Letarte, Rheume, Freeston, & Ladouceur, 1995; Freeston et al., 1994) suggests that IU, or the tendency to experience distress in the face of an ambiguous situation, is a central pathogenic mechanism in the development and maintenance of GAD. Dugas and colleagues purport that individuals with GAD possess positive beliefs about worry, such as the ability for worry to protect against emotions associated with potential negative outcomes of such uncertain situations. According to the IU model, worry facilitates avoidance of aversive imagery and negative emotional arousal in the face of negative emotional stimuli. The authors cite studies indicating that worry facilitates avoidance of mental imagery and its associated physiological responding.

Overall, the IU model represents an important foundation for the Contrast Avoidance model. Specifically, if individuals with GAD experience greater discomfort in uncertain

situations (especially if the anticipated outcome is negative and/or they have poor confidence in their problem-solving skills), this could be in part because they fear the emotional ramifications of this negative experience. However, although the Contrast Avoidance Model states that individuals with GAD view worry as a positive coping strategy for dealing with negative emotions, it does not support the idea that negative images and arousal are avoided. Rather, we argue that an alternative coping strategy is achieved via worry, such that reactions to feared negative outcomes are in fact rehearsed through chronic worry. The Contrast Avoidance model purports instead that chronic worry would lead to feeling badly all the time, which in turn would prevent being taken by surprise. This modification is consistent with but provides further elaboration on the IU model.

The MC model (Wells, 1995, 1999, 2004), suggests that positive beliefs about worry lead to a maladaptive cycle of failed coping attempts in the face of a stressor, interfering with emotional processing and in turn fueling more ineffectual attempts at coping through worry. According to the MC model, positive beliefs about worry (similar to the IU model) include beliefs that worry leads to reduced chances of being overwhelmed by unexpected negative events, protection from surprise, and reduced negative arousal in response to fearful associations. Specifically, worry is purported to facilitate temporary avoidance of somatic activation to feared material, which in fact maintains anxious associations and thus leads to more worry.

Similar to the MC model, the Contrast Avoidance model also purports that positive beliefs about worry increase use of worry as a maladaptive emotion coping strategy, and that this ultimately leads to a failure to process emotions appropriately. The theories are also similar with respect to the idea that the process of worrying engages individuals in a cycle that is ultimately

negatively reinforcing. The two models differ with respect to the concept that worry reduces somatic activation. As reviewed in the first section of this paper and replicated in the results of this study, data suggest that worry is associated with sustained negative emotionality and therefore mutes further reactivity to fearful stimuli. The Contrast Avoidance model states that fear of contrast and the patterns of contrast avoidance provide a crucial link in the emergence and maintenance of worry.

The AB model (Roemer & Orsillo, 2002; Roemer et al., 2005) posits a way to understand the development, maintenance, and treatment of GAD with the main tenet being a focus on acceptance and mindfulness patterns underlying this disorder. According to the AB model, individuals with GAD believe that worry assists in avoidance of noxious internal, emotional experiences (i.e., distraction from distressing emotional topics, avoidance of somatic activation). As such, individuals with GAD may maintain a sense of control (in the short-term) via predicting, and even superstitiously avoiding, potential negative outcomes and resulting distressing internal experiences. However, such avoidance maintains anxious associations, leading to an unproductive cycle of increased worry and avoidance. In sum, the model proposes that a tendency to focus on future events and attempts at distraction from internal experiences underscore a lack of acceptance and mindfulness as maintaining pathological factors in individuals with GAD.

For the most part, the AB model is based on a similar foundation as the Contrast Avoidance model, and has many overlapping and synchronous features. For example, similar to the AB Model, the Contrast Avoidance Model maintains that people with GAD hold positive beliefs about worry, such as a mechanism to avoid uncomfortable, threatening experiences. The

models also hold that worry is maintained via negative reinforcement, and that worry does provide the illusion of control over noxious emotional experiences. Moreover, our data also show that worry enables avoidance of emotional processing. However, whereas the AB Model suggests that worry is used to avoid negative internal experiences, the Contrast Avoidance model specifies that the only internal experience avoided is that of a negative emotional contrast, rather than negative emotion *per se*, which leads to negative reinforcement.

The ED model (Mennin et al., 2002, 2005) also states that worry leads to emotional avoidance, but stresses four underlying emotion dysregulation processes in GAD that might precede the adoption of such an avoidance strategy. These include 1) heightened emotional intensity, 2) poor emotional understanding, 3) negative reactivity to emotions (e.g., fear of emotions), and 4) maladaptive management attempts, such as attempts to control, avoid, or blunt emotional experiences. The model then purports that worry is used as an attempt to avoid such aversive emotional experiences.

There are many conceptual similarities between the Contrast Avoidance model and the ED Model. For example, we also suggest that based on a proclivity to worry as a means of emotional coping, individuals with GAD fail to regulate their emotions appropriately and that such dysregulation interferes with emotional processing. Further, our data also show that people with GAD do experience heightened emotional intensity relative to controls, both during worry and during negative emotional exposures. However, the Contrast Avoidance model proposes that it is the perceived threat and avoidance of a negative emotional contrast that leads to difficulties of emotion regulation, and that such difficulties can interfere with emotional processing on many levels, including subjective intensity as well as understanding.

There are important treatment implications that can be gleaned from these results. For instance, knowing that individuals with GAD may fear negative emotional contrasts and may use worry to avoid these experiences, this provides a specific focus for exposure-based interventions. If patients with GAD are able to experience an emotional exposure following a relaxed state, this could target the exact emotional experience that worry serves to avoid. As such, this focus of treatment could lead to habituation of fear and facilitation of emotional learning.

### **Conclusion**

The goal of this study was to provide preliminary support for the Contrast Avoidance theory of GAD. Results demonstrated partial support for this model when observing both subjective and sympathetic arousal as markers of negative emotionality. Although in need of further support, this model may help to explain the inconsistencies within the current literature on GAD and emotional regulation. By specifically observing the pattern of emotionality caused by worry, this study was able to tease apart the negative emotionality caused by worry versus the emotional response to subsequent stimuli. By measuring absolute levels of negative emotionality throughout the experiment, this study was able to discriminate emotional avoidance from emotional processing avoidance, and to provide support for a causal relationship between worry and processing avoidance. Moreover, by ascertaining a holistic appraisal of worry versus relaxation and neutral inductions on emotional coping, this study was able to provide preliminary support for hypothesis that the avoidance of negative emotional contrast is a factor that discriminates GAD from nonanxious controls. This study also provides support for the hypothesis that individuals with GAD fear negative emotional contrast experiences and use worry in order to avoid feeling vulnerable to having such an experience. Further, we established

the Contrast Avoidance Model within the framework of extant theories and models of dysregulated emotional processes within GAD. Finally, we discussed important implications of this new theory for the treatment of emotion dysregulation, including a potential specific focus for systematic exposure techniques.

### **Limitations**

A number of limitations of this study should be addressed. First, individuals in the GAD group demonstrated physiological differences between those that were randomly assigned to different induction conditions, which may have influenced further responding throughout the experiment. Furthermore, the worry induction did not consistently discriminate physiological effects from the neutral induction. Although this may have been due to higher baseline arousal levels in the neutral condition, it may also have been due to the neutral condition itself. During this induction, participants were given a sheet of paper to hold and read from, whereas in other conditions participants had their arms resting at their sides. However, subjective data show that the neutral induction did have the intended effects, and that these effects supported the Contrast Avoidance model.

In addition, the predicted physiological effects of worry, while present in sympathetic activity during the pre-fear induction, were not consistent across all induction periods. Furthermore, the predicted parasympathetic effects were not found either during inductions or during the fearful exposure. This suggests that more research is needed to determine the extent to which physiological data support the Contrast Avoidance model.

In addition, the results from this study are based on an analogue GAD population and did not include a treatment-seeking group. It is therefore possible that effects specific to the GAD

group may not generalize to a treatment-seeking population. Another limitation of our sample is that it was not very ethnically diverse and was taken from a college population. As such, results should be replicated with a more diverse sample, in terms of both age and ethnicity, before they can be generalized to the population at large. Also, because the current study was not designed to determine whether those with both GAD and comorbid depression had differential responses to emotion inductions compared to those with GAD without comorbid depression, depression levels were balanced across the different inductions. Future research could usefully explore the effects of comorbid depression on emotional responding for individuals with GAD.

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## APPENDIX A

*Table 1*

*Means and Standard Deviations of Manipulation Check Scores by Group and Group and Induction Type*

Manipulation Scale	Induction Type			Group Totals	
	Worry	Neutral	Relax	GAD	Control
Anxiety	2.74 (.76)	1.56 (.95)	1.43 (.53)	2.16 (1.01)	1.72 (.88)
Worry	3.0 (.90)	1.20 (.60)	1.22 (.39)	1.98 (1.14)	1.73 (1.04)
Relaxation	1.98 (.66)	3.14 (1.16)	3.58 (.95)	2.55 (1.14)	3.18 (1.09)

*Note.* Manipulation check scores are reported in non-transformed values.

Table 2

*Absolute levels of emotion adjective ratings during inductions.*

Emotion Adjective	Induction Type			F-value	p-value	$\eta_p^2$
	Worry	Relax	Neutral			
<i>Anger</i>	2.33(1.6)**	1.11(.26)	1.52(1.13)	15.47	< .001	.29
<i>Contempt</i>	2.13(1.47)**	1.23(.39)	1.03(.14)	11.36	< .001	.23
<i>Embarrassment</i>	1.60(.91)*	1.13(.28)	1.04(.15)	7.35	< .001	.16
<i>Fear</i>	2.92(1.6)**	1.24(.56)	1.14(.49)	25.17	< .001	.40
<i>Sadness</i>	2.68(1.6)**	1.10(.36)	1.04(.11)	24.91	< .001	.39
<i>Tension</i>	3.10(1.6)**	1.63(.93)	1.24(.66)	16.34	< .001	.30
<i>Contentment</i>	1.58(.91)**	3.43(1.77)	2.68(1.8)	10.68	< .001	.22

*Note.* Emotion adjective scores are reported in non-transformed values.

\*\* worry vs relax and neutral,  $p < .001$ ; relax vs neutral,  $p = ns$

\* worry vs relax and neutral,  $p < .01$ ; relax vs neutral,  $p = ns$

## APPENDIX B

*Emotional Coping Scale*

Directions: Below are a series of statements that can be related to worry / relaxation / neutral activity. Please think back to times in this study when you worried / relaxed / engaged in neutral activity prior to watching the video clips, and indicate to what extent these statements were true for you.

1	2	3	4	5
not at all true	slightly true	somewhat true	very true	absolutely true

Worry:

1. Because I already felt bad from worrying, it was less of a shock to feel the negative effects of some of the film clips.
2. Because I was already worried, I wasn't caught off-guard by unexpected negative events in the film clips.
3. Because I was already worried, I was less upset when a negative event occurred in the film clips.
4. Because I was already worried, I was actually more disturbed by unexpected negative events in the film clips.
5. Because I was already worried, I was even more upset when a negative event occurred in the film clips.

Relax:

1. Because I already felt relaxed, it was less of a shock to feel the negative effects of some of the film clips.
2. Because I was already relaxed, I wasn't caught off-guard by unexpected negative events in the film clips.
3. Because I was already relaxed, I was less upset when a negative event occurred in the film clips.

4. Because I was already relaxed, I was actually more disturbed by unexpected negative events in the film clips.

5. Because I was already relaxed, I was even more upset when a negative event occurred in the film clips.

Neutral:

1. Because I was in a neutral state, it was less of a shock to feel the negative effects of some of the film clips.

2. Because I was in a neutral state, I wasn't caught off-guard by unexpected negative events in the film clips.

3. Because I was in a neutral state, I was less upset when a negative event occurred in the film clips.

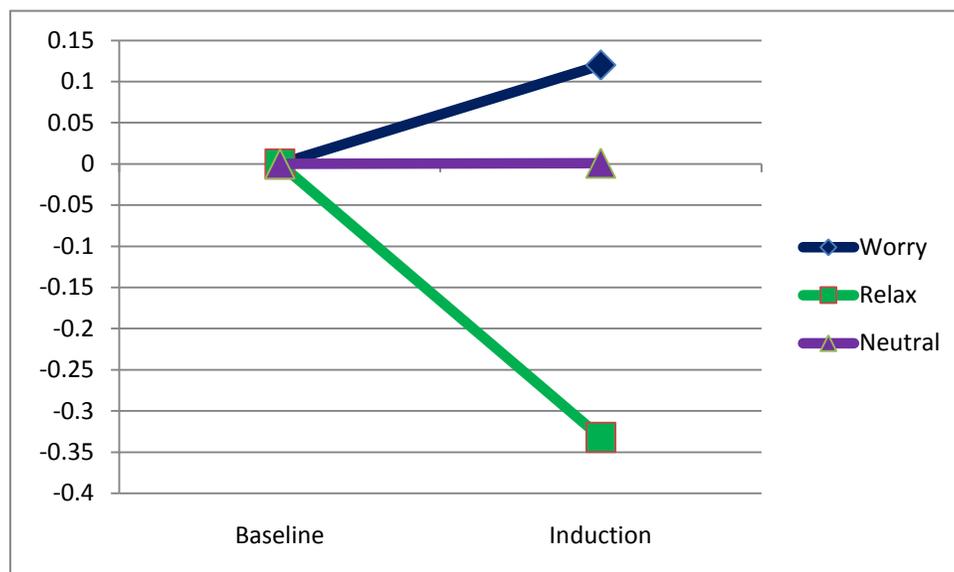
4. Because I was in a neutral state, I was actually more disturbed by unexpected negative events in the film clips.

5. Because I was in a neutral state, I was even more upset when a negative event occurred in the film clips.

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Adapted from: Gosselin et al., 2003.

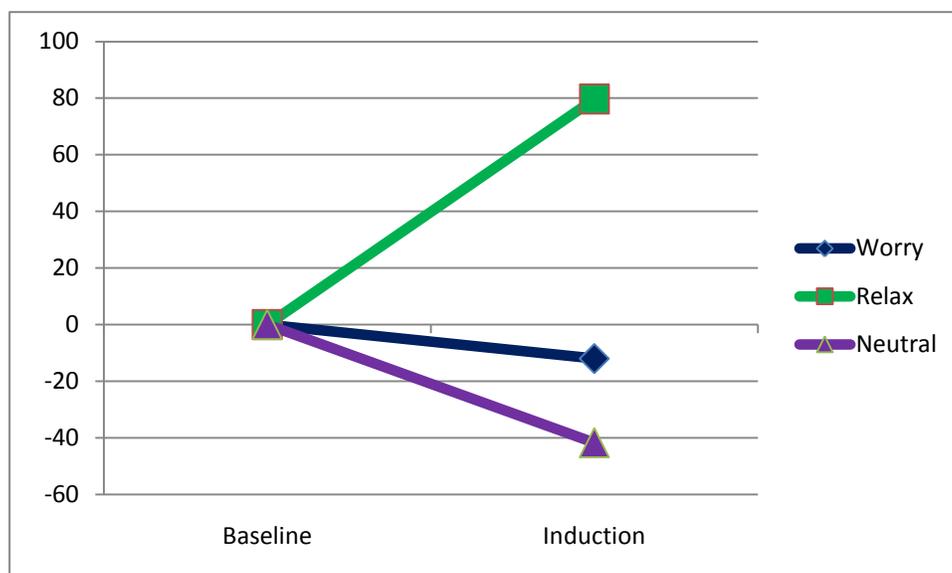
*Figure 1. The emotional coping questionnaire*



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*Note.* Values represent change in log-transformed amplitude average scores in microSiemens.

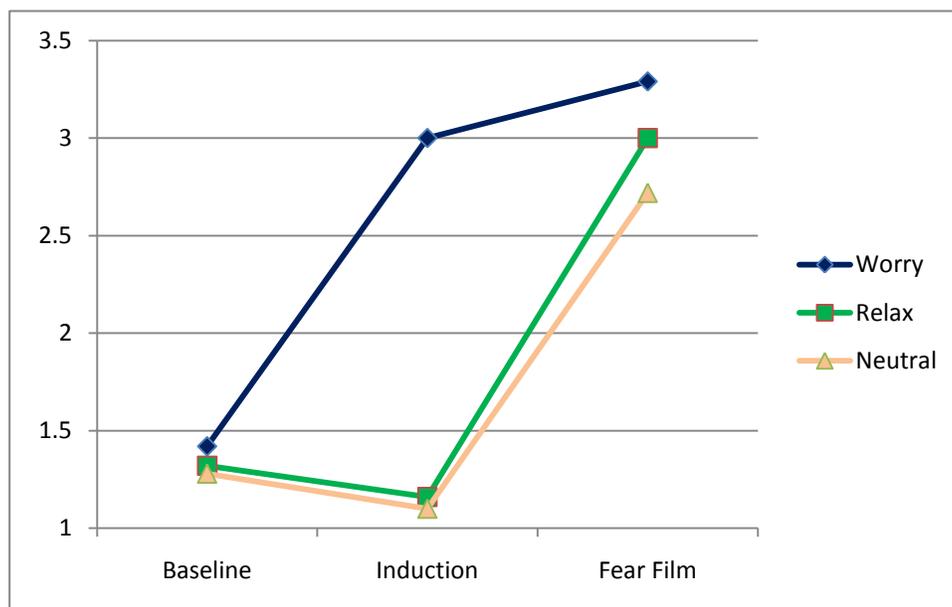
*Figure 2. Change in amplitude average scores from baseline to the 1<sup>st</sup> minute of the pre-fear induction.*



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*Note.* Values represent change in non-transformed RSA scores in ms<sup>2</sup>.

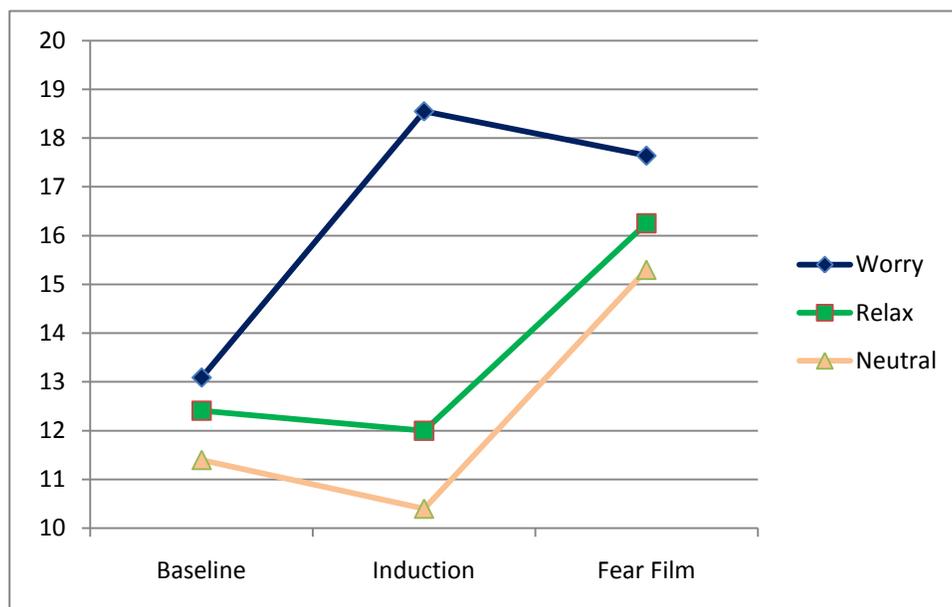
*Figure 3.* Change in RSA scores from baseline to both minutes of the pre-fear induction for the nonanxious control group.



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*Note.* Scores are reported as change in non-transformed values.

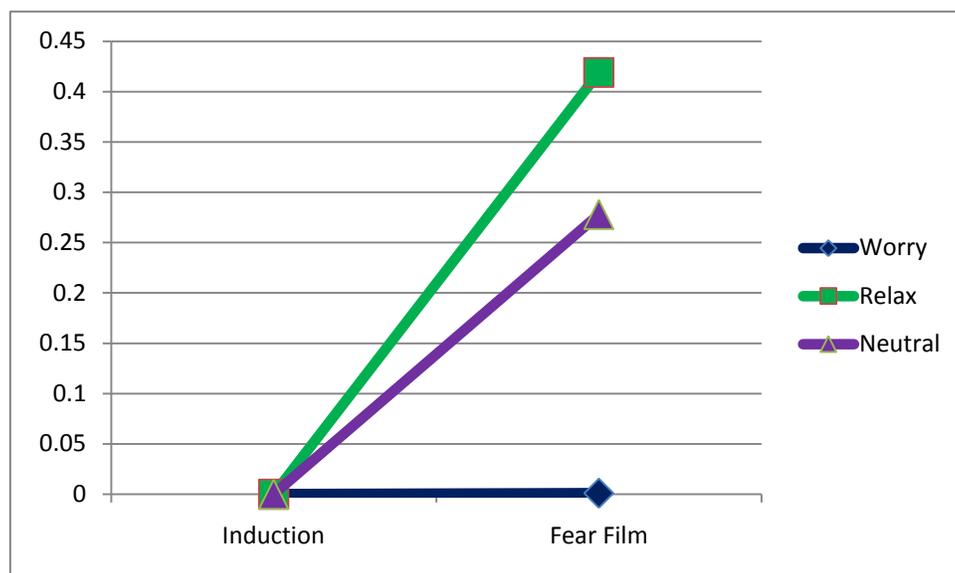
*Figure 4.* Change in reported Fear from the baseline to induction to fearful exposure for both groups.



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*Note.* Scores are reported as change in non-transformed values.

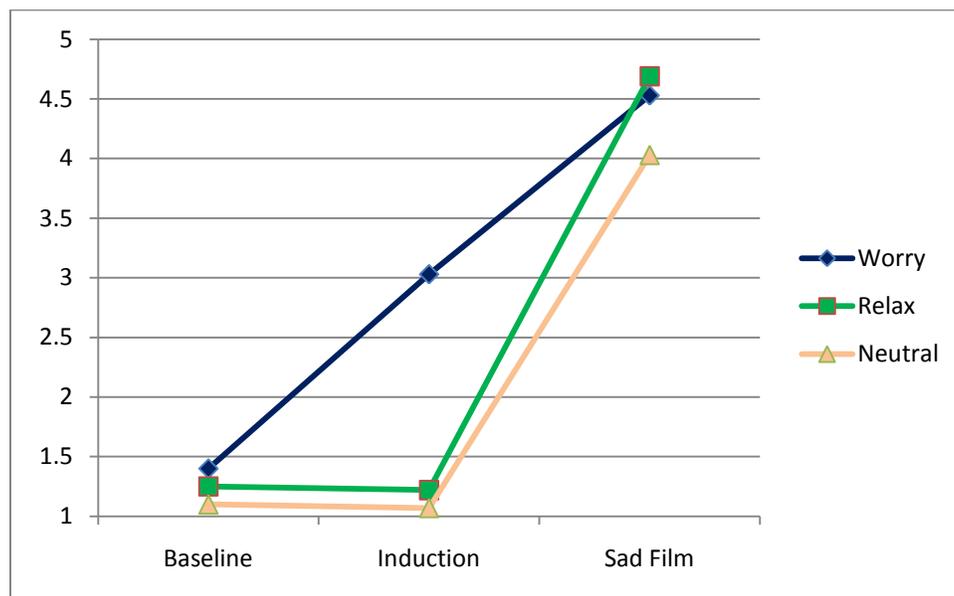
*Figure 5.* Change in negative affect (NA) from the baseline to induction to fearful exposure for both groups.



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*Note.* Scores are reported as change in log-transformed microSiemens. (When #8, a GAD participant in the worry condition using *Celexa*, was removed from analysis.)

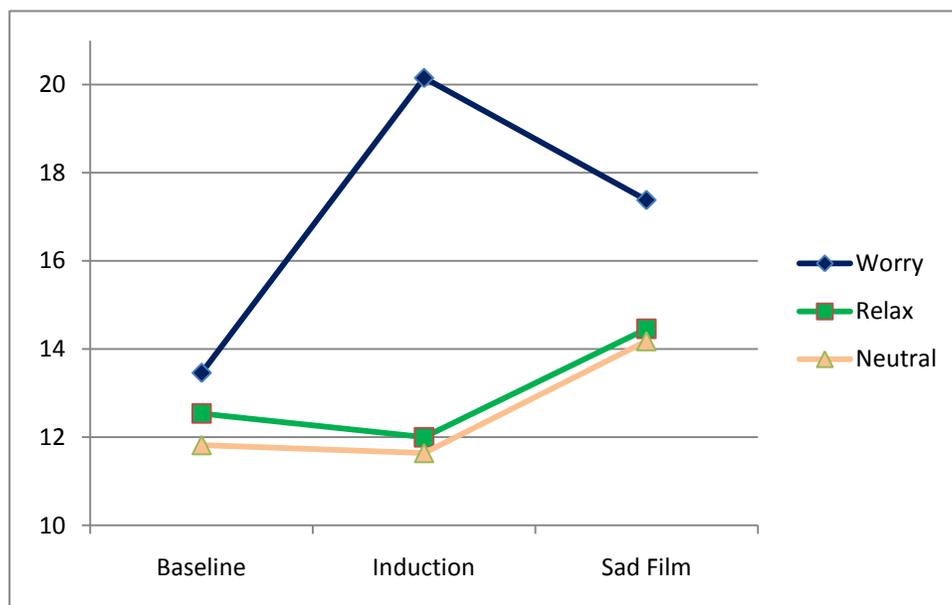
*Figure 6.* Change in amplitude average scores from the induction period to the fearful exposure.



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*Note.* Scores are reported as change in non-transformed values.

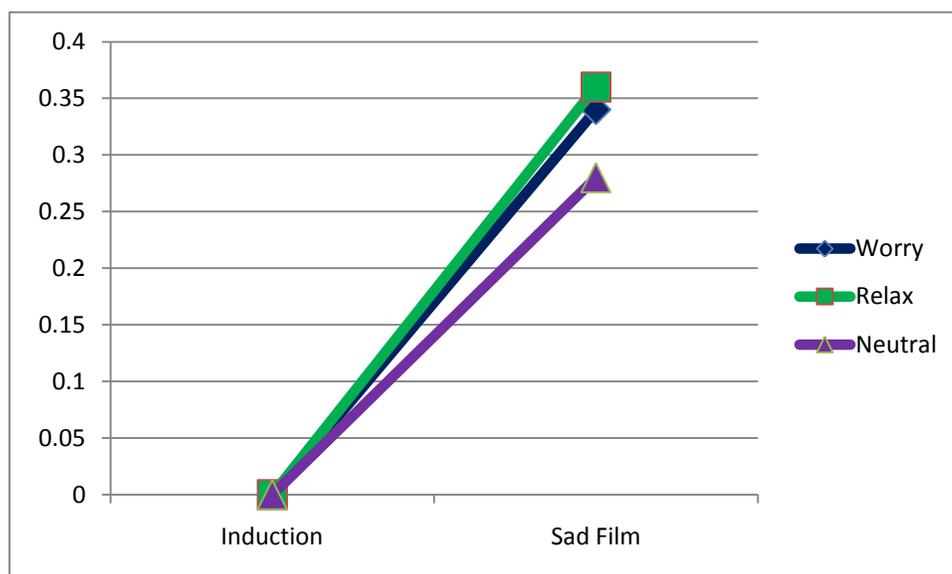
*Figure 7.* Change in sadness from the baseline to induction to sad exposure for both groups.



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*Note.* Scores are reported as change in non-transformed values.

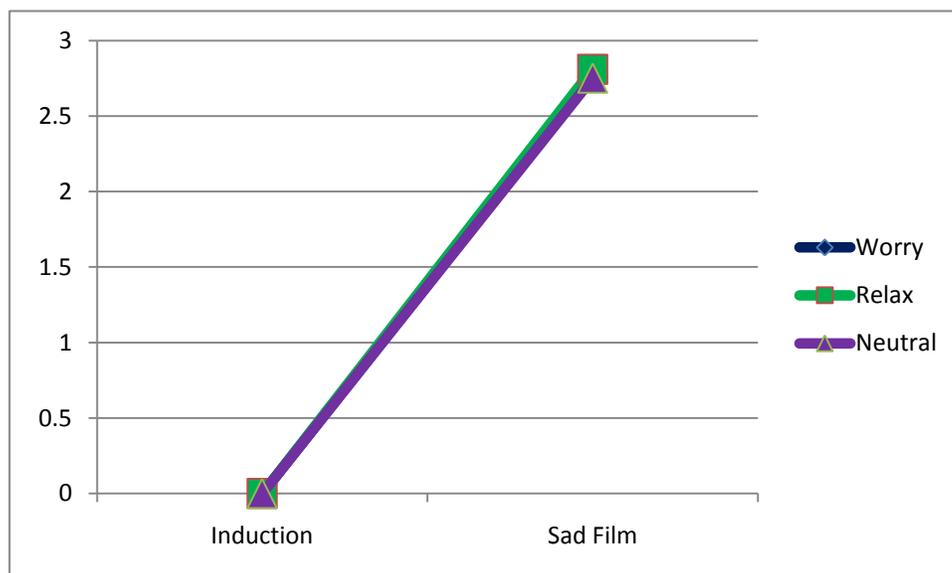
*Figure 8.* Change in negative affect (NA) from the baseline to induction to sad exposure for both groups.



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*Note.* Scores are reported as change in log-transformed microSiemens.

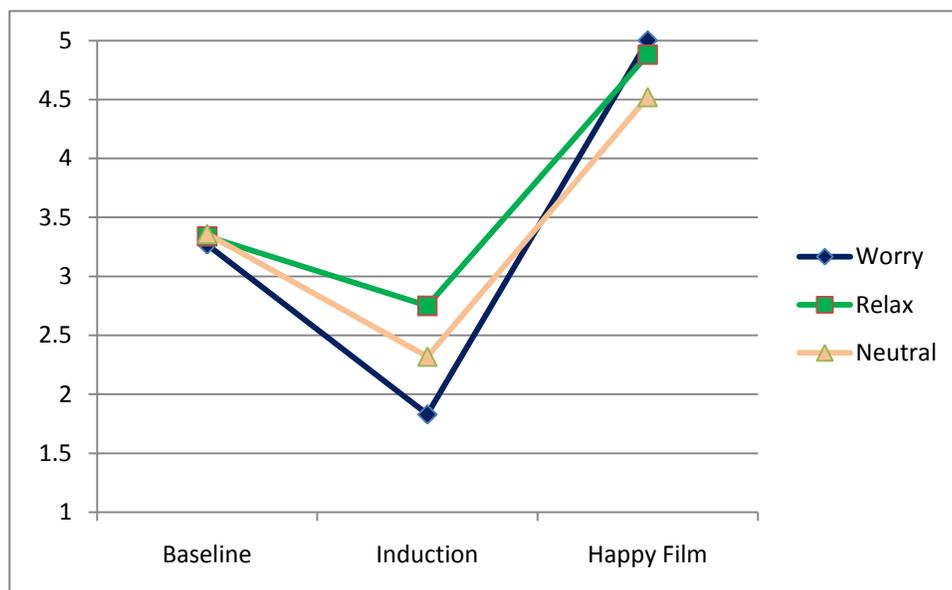
*Figure 9.* Change in amplitude average scores from the induction period to the sad exposure.



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*Note.* Scores are reported as change in log-transformed  $\text{ms}^2$ .

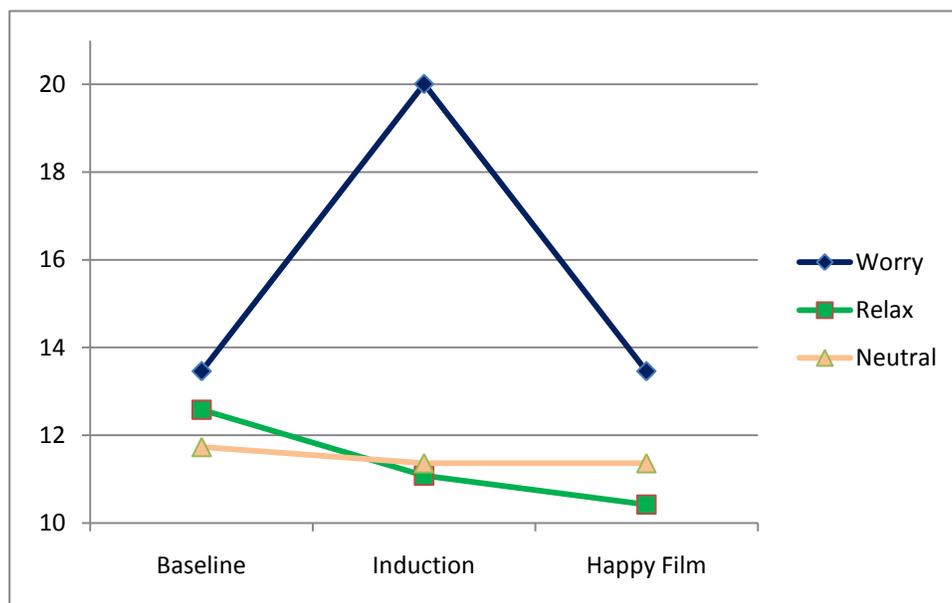
*Figure 10.* Change in RSA from the induction period to the sad exposure.



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*Note.* Scores are reported as change in non-transformed values.

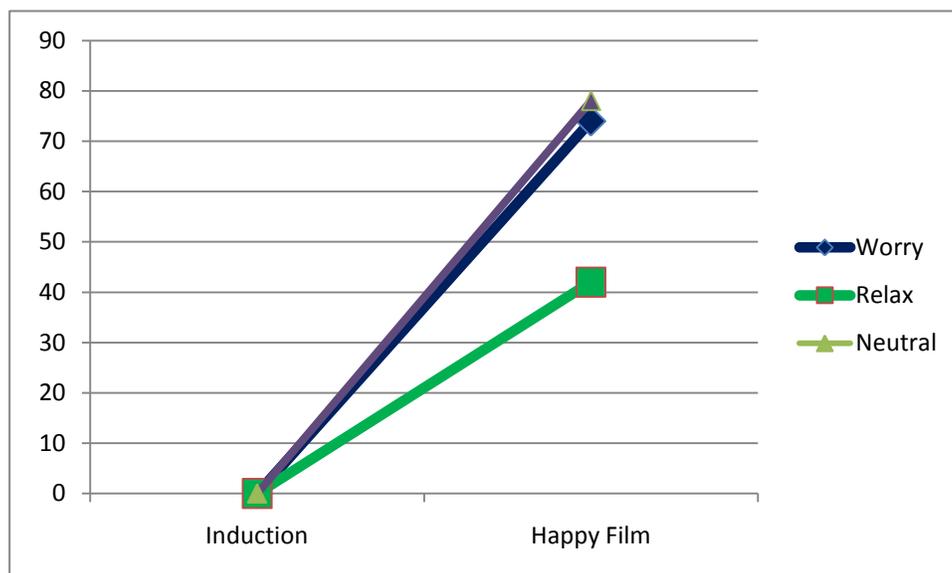
*Figure 11.* Change in happiness from the baseline to induction to happy exposure for both groups.



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*Note.* Scores are reported as change in non-transformed values.

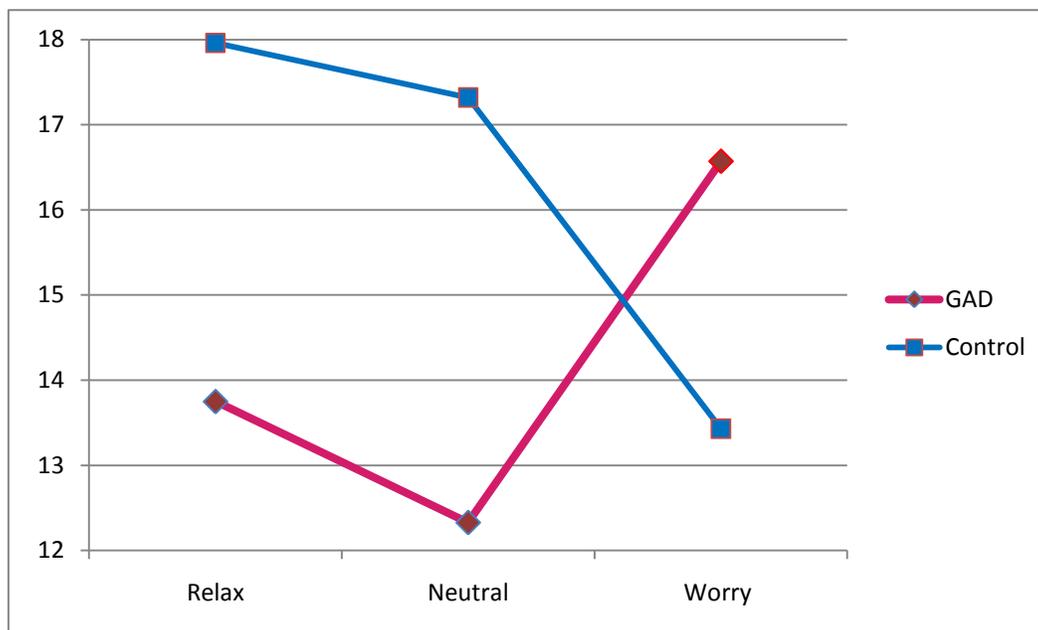
*Figure 12.* Change in negative affect (NA) from the baseline to induction to happy exposure for both groups.



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*Note.* Scores are reported as change in log-transformed  $ms^2$ .

*Figure 13.* Change in RSA from the induction period to the happy exposure.



*Figure 14. Emotion coping scores by group and induction type.*

## VITA

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#### EDUCATION

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#### TEACHING EXPERIENCE

- 2009 Course Instructor, Introductory Psychology, PSU
- 2007, 2009-10 Graduate Lecturer/Lab Instructor, Research Methods, PSU

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- 2009-2010 Brittany Pasay. *The Effects of Worry on Discrepancy Between Reported Interpersonal Problems in Friendship Dyads*. Undergraduate Honors Thesis completed at PSU.

#### PEER-REVIWED PUBLICATIONS

- Newman, M. G. & **Llera, S. J.** (2011). A new theory of experiential avoidance in generalized anxiety disorder: A review and synthesis of research supporting an avoidance of a negative emotional contrast. *Clinical Psychology Review*, *31*, 371-382. doi: 10.1016/j.cpr.2011.01.008.
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