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**The Effects of Emotion Regulation Strategies in
Clinical Symptom Presentations**

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Abstract

Preliminary evidence suggests that the effects of regulating emotion may be moderated by both individual differences in trait emotional responding and symptoms of psychopathology. However, more knowledge of how the effects of emotion regulation strategies are moderated by specific symptoms of psychopathology is needed to better diagnose and treat individuals with psychopathology. The purpose of this study was to experimentally test how the effects of emotion suppression (a commonly used emotion regulation strategy) are moderated by individual differences in psychopathology (in this case, depressive symptoms and comorbid clinical worry predictive of generalized anxiety disorder). Results indicated no effect of suppression on self-reported sadness compared to control condition for any participants, and all participants recovered from sadness equally after a delay in time. Suppressing anxiety led to a reduction in self-reported anxiety compared to control condition for nonclinical participants, but this difference was not observed for those showing depressive symptoms, regardless of level of comorbid clinical worry. Results reveal that normative individuals may be able to mitigate state anxiety compared to other emotions like sadness, thus demonstrating the potential of a unique relationship between emotion suppression and anxious responding. However, the lack of ability for those with elevated depression and any level of comorbid worry to mitigate state anxiety indicates the potential for a shared emotion regulatory factor among depressive and anxiety pathology. Implications for theoretical understanding, assessment of psychopathology, and clinical practice are discussed.

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The Effects of Emotion Regulation Strategies in Clinical Symptom Presentations

The human and economic costs of mental illness in the United States are staggering. It has previously been estimated that mental illness affects approximately 26% of Americans (Kessler, Chiu, Demler, & Walters, 2005) and costs up to 60 billion dollars per year (Soni, 2009). Major depression, for example, has been found to be one of the leading causes of disability in the United States and around the world (Murray, 1996). These burdens are likely underestimated given underreporting due to stigma, creating to a barrier to systematic health prevention efforts because those with psychological disorders are at higher risk for developing diseases like cardiovascular disease, respiratory disease, and diabetes (Bloom, Cafiero, Jané-Llopis, Abrahams-Gessel, Bloom et al., 2011). Psychological disorders, like major depression, are shown to negatively impact cardiovascular disease, especially as individuals experience increased stress in response to environmental demands (Joynt, Whellan, & O'Connor, 2003). Both major depression and anxiety disorders have been found to be higher in individuals with diabetes than in normative populations (Li, Barker, Ford, Zhang, Strine et al., 2008), to lead to increased risk of developing Type II diabetes (Golden, Lazo, Carnethon, Bertoni, Schreiner, et al., 2008), and to negatively impact the effects of diabetes treatment for those with comorbid anxiety disorders and diabetes (Li et al., 2008).

Research from a number of psychological perspectives has long suggested that the maladaptive use of strategies used to regulate or cope with negative affect is central to the development and maintenance of psychological disorders. Freud posited that individuals repress memories of events that are too anxiety-provoking to be handled by the ego, causing them to experience emotional discomfort and to impede their ability to deal with

difficult situations (Freud, 1946). Mowrer (1935) found that avoidance of fear responses is negatively reinforced and thus maintains anxious responding to aversive situations. Maintained anxious responding due to negatively reinforced avoidance is strongly hypothesized to contribute to a number of psychosocial difficulties and forms of psychopathology, including posttraumatic stress disorder and depression (Foa, Hembree, & Rothbaum, 2007; Martell, 2013). In examining coping with stress, Lazarus and Folkman (1984) conceptualized an individual's perception of resources for coping with stress as a main determinant of how they might approach attaining resources for coping with that stress. Individuals who perceive fewer resources to deal with stressful situations may avoid utilizing problem-solving techniques and may isolate themselves from potential social support resources that might buffer them against adjustment difficulties and depressed affect (Lazarus & Folkman, 1984). More recently, Hayes, Strosahl, and Wilson (1999) have proposed that individuals' attempts to avoid negative affect and other types of difficult internal experience (e.g., thoughts, memories) creates a barrier to engaging in behaviors that help the individual live in accordance with their personal values. Such a barrier to valued-living can lead to high levels of negative affect and to psychopathology.

A new generation of research on specific strategies individuals use to regulate their emotional responses has arisen in the past twenty years. The most well studied emotion regulation (ER) strategies include changing the way one thinks about an emotion-inducing event to minimize its emotional impact (known as cognitive reappraisal; e.g., Gross, 1998), allowing oneself to experience negative emotions without trying to change their frequency or form (known as emotional acceptance; e.g., Hayes,

Strosahl, & Wilson, 1999), or attempting to inhibit the expression or experience of an emotion to avoid how aversive that emotion may be (known as emotion suppression; e.g., Gross, 1998).

Most research on ER strategies has used experimental paradigms to examine basic outcomes of utilizing such strategies for individuals from nonclinical populations. For instance, suppressing negative emotion has been linked to social adjustment difficulties, depletion of cognitive resources, increased physiological arousal, and delayed recovery from negative affect in studies where subjects are asked to suppress their emotions in response to a range of tasks meant to induce specific negative emotions or distress generally (e.g., Baumeister, Bratslavsky, Muraven, & Tice, 1998; Butler, Lee, & Gross, 2007; Campbell-Sills et al., 2006b; Gross, 1998). In similar experimental paradigms, cognitive reappraisal and emotional acceptance have been linked to positive outcomes. For example, reappraisal has been shown to yield positive social outcomes when compared to suppression, decreased depletion of cognitive resources and no physiological consequence; and both reappraisal and acceptance have shown decreases in negative affect compared to less adaptive strategies or to controls (e.g., Baumeister, et al., 1998; Boland, Shallcross, Papa, & Mauss, in preparation; Butler et al., 2007; Gross, 1998).

Despite the long-held theoretical link between individuals' methods of responding to negative affect and functional problems found in psychopathology, as well as negative psychological effects of emotion suppression demonstrated in experimental studies, only a few empirical studies have sought to test the effects of specific strategies used to regulate emotion for those with psychological disorders compared to those without them.

More empirical research is therefore needed in order to draw conclusions about the nature of ER strategies within clinical populations. A small but growing body of research suggests that these ER strategies may indeed function differently (1) for those with symptoms of psychological disorders compared to those without, and (2) between different symptom presentations (e.g., major depression vs. anxiety disorders). As a number of emotional features (e.g., neuroticism, trait negative affect) are shown to differentiate between disorders (e.g., GAD versus MDD; Mineka, Watson, & Clark, 1998), understanding the psychological effects of ER strategies (e.g., emotion suppression) for individuals with different symptom presentations may help clarify both distinctions and areas of overlap between existing disorders (Mennin, Holoway, Moore, & Heimburg, 2007).

The goal of this study is to examine emotion regulation strategies in the context of specific mental health problems in order to enhance our knowledge of disorder-specific contextual effects of different ER strategies to improve our understanding of online effects of emotion regulation for those with psychopathology. This understanding should improve our ability to accurately diagnose psychological disorders and improve mental health treatments targeting specific regulatory processes that maintain psychopathology. The extant literature in this area has only recently begun to scratch the surface of this problem. Given the range of problems and the range of regulation strategies available, the current study will focus on emotion suppression in the context of depressive symptoms and anxiety disorder symptoms because suppression and other forms of avoidance are hypothesized to be key strategies used by individuals with these psychological disorders (Allen, McHugh, & Barlow, 2008; Hayes, Wilson, & Strosahl,

1999).

This paper first reviews the existing research on the role of emotion suppression in the development and maintenance of psychopathology. This review will highlight the conceptual and methodological limitations that have inhibited definitive conclusions about suppression's contribution to specific types of psychopathological responding. It will also include a discussion of how empirical examination of emotion suppression in multiple symptom presentations is important for understanding disorder-specific emotional responding and thus improving diagnostics and treatment targeting. This discussion will be followed by a description of the methods used and the results of an empirical study conducted on the effects of suppression on multiple emotions among individuals with elevated levels of depression and anxiety disorder symptoms and among normative individuals. Finally, a discussion of the study results will focus on its implications for current emotion research and theory, for clinical diagnostics, and for treatment of individual clients.

Emotion regulation is linked to psychological adjustment in nonclinical populations

Most theoretical efforts in social and clinical psychology have focused attention on the impact of negative emotions on individuals because distressing situations typically require more physical and psychological resources, are more difficult to cope with, lead to greater frequency and intensity of interpersonal difficulties, and provide stronger motivating operations for behavior than positive emotions (Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001). The greater impact of negative emotion on multiple physical and psychological domains may be one reason that maladaptive attempts to avoid negative emotions has been such a strong theoretical focus throughout the study of

psychopathology in particular (see Baumeister et al., 2001 for an extensive review).

Gross' process model of emotion regulation (1998) constitutes an effort to conceptualize individuals' responses to their own negative emotional reactions. The model proposes that emotion suppression is the conscious or subconscious attempt to inhibit ongoing emotional responses to minimize their impact (Gross, 1998), which gives individuals a way to shape their emotional experience in response to an event (e.g., avoiding emotional responses that may be difficult or socially undesirable; Gross & John, 2003). Since this model was introduced, a boom of research on the effects suppression and other ER strategies has found numerous negative effects of suppressing emotion (Gross & Feldman-Barrett, 2011). For instance, a number of these studies have found that suppression is associated with social adjustment difficulties (e.g., Butler, Egloff, et al., 2003; Butler, Lee, & Gross, 2007; Gross & John, 2003). One study found that those who suppressed their emotions during a conversation dyad were reported to be highly unlikable by their dyad partner and that partner reported never wanting to see the individual who suppressed ever again (Butler et al., 2003). Other studies found that suppressing emotions led to greater depletion of cognitive resources, or "ego depletion," compared to reappraisal or controls (Baumeister, Bratslavsky, Muraven, & Tice, 1998; Muraven, Tice, & Baumeister, 1998). Muraven and colleagues (1998) found that those instructed to suppress their emotions in response to a negative emotion induction spent less time on a subsequent cognitively taxing task than those instructed to reappraise the content of the induction. Gross and others have found many correlates of negative health consequences like increased heart rate and increased blood pressure when comparing suppression to more adaptive ER strategies (e.g., Gross, 1998; Gross & Levenson, 1997;

Mauss et al., 2007). For example, when viewing a disgusting film clip, those instructed to suppress showed increased heart rate compared to those instructed to reappraise or to controls (Gross, 1998). The proposed function of suppression to minimize the impact of negative emotion (Gross, 1998) may constitute an attempt to avoid negative emotion, and research has also found that those who typically avoid their internal experiences (e.g., emotion as well as other internal experiences like thoughts and memories) show greater levels of adjustment difficulties and psychological symptoms, especially difficulties with anxiety and stress (Hayes et al., 2004; Roemer et al., 2005).

The studies described above are informative for understanding the unique role of suppression in a number of psychological domains. They suggest that not only can suppression have negative effects on individual functioning, but also that suppression may have an important role in psychopathology. Despite the importance of studying suppression in those experiencing high levels of distress, the studies described above are limited in scope by their predominant reliance on nonclinical populations and their inability to account for external factors that may moderate the hypothesized negative effects of suppressing emotion (Papa, Boland, & Sewell, 2012). Bonanno and colleagues (2004) designed one of the first ER studies to account for such external factors. They used a longitudinal design with a Time 1 experimental manipulation to show that the inability to flexibly use high levels of both suppression and enhancement of emotional facial expressions appropriately in response to contextual demands predicted adjustment difficulties 2 years later. First, this finding supports the link between maladaptive use of ER strategies and psychological difficulties. Second and more importantly, because individuals with a greater ability to suppress their expressions showed better mental

health outcomes, the effects of ER strategies do not appear to stem from the type of strategy used (i.e., suppression on its own is not inherently maladaptive), but from the ways one uses them. Results of the Bonanno et al. (2004) study demonstrated the need for ER research to account for contextual factors (e.g., when an ER strategy is used), boundary conditions (e.g., the effect of time on ER strategies), and individual differences (e.g., in emotional reactivity, in habitual ways one regulates emotion) to more fully understand the effects of ER strategies. More recent research has supported the need to examine the moderating effects of individual differences and other contextual factors when testing the effects of ER (Aldao, 2013; Bonanno & Burton, 2013; Hayes et al., 1996; Papa, Boland, & Sewell, 2012).

Individual differences in trait emotional reactivity and the ability to regulate emotion have more recently been shown to be an important moderator of ER strategies. For instance, a higher tendency to habitually engage in cognitive reappraisal has been shown to predict greater well-being and fewer depressive symptoms after exposure to a stressful life-event (Troy, Wilhelm, Shallcross, & Mauss, 2010). Similar predictions have been found for those who frequently accept their emotions in response to stressful life-events (Shallcross, Troy, Boland, & Mauss, 2010). This same group of researchers (Troy, Shallcross, & Mauss, 2013) later found that individual differences in the tendency to reappraise certain types of situations moderates the effects of reappraisal. They found that the tendency to reappraise uncontrollable stressors was associated with lower depressive symptoms, but tendency to reappraise controllable stressors (i.e., those that could be influenced or modified with overt behavioral problem-solving) was associated with higher levels of depression (Troy, Shallcross, & Mauss, 2013).

In the case of trait emotional responding, research has found differences in the effects of ER strategies for those high in trait negative affect (NA) compared to those low in trait NA, suggesting that suppression is more impactful for individuals with high levels of emotional disruption found in many psychological disorders. In a study on emotional acceptance by Boland and colleagues (in preparation), those high in trait NA instructed to accept their emotions in response to a mixed anxiety/sadness film clip did not differ in anxiety from those low in NA given the same instructions, whereas those high in NA in a control group experienced significantly greater levels of anxiety than those low in NA in the same control group. This result suggests that those high in NA instructed to accept their emotions may have experienced as little anxiety as control participants who were low in NA. Shallcross, Troy, Boland, & Mauss (2010) also found that acceptance predicted lower levels of depressive symptoms three months after a stressful life-event for those people reporting the experience of high, but not low, levels of ongoing life-stress.

Boland & Papa (under review), also found that those high in trait NA instructed to suppress the expression of their emotions experienced less anxiety than control participants immediately following a film clip that induced both sadness and anxiety. However, suppression and control participants low in trait NA experienced no differences immediately after the film clip or after a resting period. This short-term adaptive effect of suppression for those high in trait NA is contrary to outcomes from previous studies showing maladaptive effects of suppression among nonclinical participants. There are a few explanations for this incongruent finding. For example, it is possible that those with high trait NA are well practiced in suppression and thus able to limit their anxiety after

using it. It is also possible that the maladaptive effects of suppression are mediated by other processes that allow for more long-term effects (e.g., suppression leads to lower levels of emotion in the short-term but creates a barrier to using more adaptive or approach behaviors to engage with environmental demands; Hayes et al., 1999; Llewelyn, Dolcos, Iordan, Rudolph, & Dolcos, 2013).

Another explanation for these incongruent findings is methodological. Many previous studies do not include manipulation control groups and/or population control groups (i.e., nonclinical controls). The inclusion of a control manipulation as well as low trait NA participants against which to compare the effects of suppression for those high in NA provided strong comparison groups not seen in a number of previous studies on suppression (e.g., Campbell-Sills et al., 2006b; Liverant et al., 2008). The inclusion of a low trait NA comparison group may contribute to why results in our study counter assumptions that suppression or avoidance of negative emotion has detrimental effects on negative emotion for those with high trait negative affect. Regardless, the inclusion of manipulation control groups and population control groups are crucial for drawing comparisons in any type of experimental research, including suppression research. More work using control groups on both dimensions (i.e., manipulation controls and population controls) is therefore necessary in emotion regulation research to more clearly uncover which conditions suppression is detrimental for and whether suppression leads to immediate or delayed detrimental effects on adjustment for certain individuals.

Suppression is linked to outcome in clinical populations

Research has uncovered differences in emotional responding among clinical populations suggesting that further research is warranted to understand the role of ER

strategies in the context of individual differences in psychopathological responding. For instance, individuals with symptoms of psychopathology have been shown to have higher levels of trait negative emotion in general and in response to stressors (Dennis, 2007; Mennin et al., 2007), more negative appraisals in response to a range of situations (Beck & Emery, 2005), and a greater tendency to avoid negative emotions and stimuli associated with those emotions (Borkovec, Alcaine, & Behar, 2004; Foa, Turk, & Heimberg, 2007; Hayes et al., 2004; Mennin, McGlaughlin, & Flanagan, 2009). However, research examining the interaction of psychopathology and suppression of negative emotions remains preliminary.

The research that is available suggests that suppression and other ER strategies play an important role in the maintenance of anxiety and mood disorders. For instance, a series of studies by Campbell-Sills and colleagues found that suppressing emotional experience is a common strategy used to deal with difficult emotional reactions among individuals with clinical anxiety and depression (2006a). In a follow-up study, the same authors found that clinical participants had difficulty recovering from elevated negative emotion after suppressing compared to after accepting emotion induced by a distressing film clip (2006b). Results from a meta-analysis by Aldao and colleagues (2010) also found that those with major depression and anxiety disorders often used emotion suppression but infrequently used other strategies thought to be adaptive, like cognitive reappraisal.

Effects of ER may differ across symptom presentations

Although suppressing emotion has been correlated with psychopathology, basic research has yet to uncover whether suppression would have the same effect regardless of

an individual's diagnosis or regardless of which specific emotion an individual is suppressing. This lack of understanding highlights a number of important questions. For instance, do individuals with depressive symptoms respond differently to suppressing negative emotion than those with anxiety symptoms? Does suppression increase or maintain anxiety disorder symptoms but not depressive symptoms? Suppression has shown different effects on different emotions initially and over time for those high compared to low in trait NA, but how those effects would be moderated by different types of high trait NA found in different specific disorders is unclear (Boland & Papa, in preparation). Additionally, how do different disorders moderate the effect of suppression on specific emotions (e.g., state sadness versus state anxiety)? A number of these questions are outside the scope of a single research project, and so highlight the need to begin extensive research on the effects of common ER strategies within and between each disorder to gain a greater understanding of disorder-specific effects of suppression.

Clinical research has found that those with different disorders (e.g., major depression vs. anxiety disorders) respond differently to their own emotional reactions, suggesting they may show differences in how they are affected by ER strategies. Preliminary studies that have found disorder-specific patterns of emotional processes *related to* ER strategies support the existence of disorder-specific effects of ER strategies like suppression. Mennin and colleagues (2005) posited that emotion dysregulation is a key factor in the maintenance of generalized anxiety disorder (GAD) after finding that students with GAD reported heightened intensity of emotions and less ability to self-soothe in response to negative emotions than controls. Students with GAD also showed more difficulty managing their emotions in response to music-induced anxiety and

sadness than controls. Roemer and colleagues (2005) further uncovered that those with GAD fear their emotional reactions and make active attempts to avoid the experience of those reactions. Their results led these researchers to develop an acceptance-based behavior therapy to help GAD patients deal with distressing emotions and other internal experiences associated with high anxiety (e.g., catastrophic thoughts). This treatment showed preliminary support for alleviating GAD symptoms (Roemer & Orsillo, 2007; Roemer, Orsillo, & Salter-Pednault, 2008) and a promising role of acceptance for alleviating anxious responding specifically.

Mennin and colleagues (2009) further substantiated findings that emotional intensity and impaired attempts to implement ER strategies are predictive of GAD diagnosis regardless of whether or not participants also had a social anxiety disorder (SAD) diagnosis, but that frequent non-acceptance of emotions specifically predicted having diagnoses of both GAD and SAD at the same time. Other research has substantiated that avoiding the experience of emotions predicted an increase in social anxiety, but emotional avoidance was not associated with depressive symptoms or anger over a three-month period (Kashdan, Breen, Afram, & Tehrar, 2010). These studies suggest that avoidance of emotional responses may have a unique role in maintaining anxiety symptoms but may have different effects on other types of psychopathology.

A study by Aldao and colleagues (2010) also uncovered similar patterns of emotional responding for those with clinical anxiety that did not occur for depressed participants. They found that positive emotion, goal motivation, emotion intensity, and intolerance of uncertainty were higher for those with GAD compared to those with major depression. Differences in emotion intensity and positive affect between disorders would

suggest differences in frequency and nature of attempts to regulate emotions. Indeed, Mennin and colleagues (2007) found that heightened intensity and maladaptive management of emotions were related to GAD, whereas poor understanding and negative reactivity of emotions (beliefs that negative emotions are harmful and will lead to detriment for the individual) were related to major depression. This is in line with Leahy's (2002) finding that depression was associated with expectations of long mood duration whereas anxiety was associated with less acceptance of emotions. These findings suggest that those with higher levels of anxiety may therefore take active, albeit ineffective, steps to avoid their emotions, whereas those with depression may be more passive in not understanding their negative emotions or how to respond them.

Taken together, these results show general differences in the consequences of attempts to control emotions between different emotional disorders. These differences suggest that the differentiation of various strategies to regulate emotions is an important predictor across different clinical symptom presentations. More specifically, the finding that unacceptability of emotions and maladaptive attempts to modulate them are unique to anxiety disorders suggests that suppression may have specific effects on anxious responding characteristic of anxiety disorders on the one hand, but may have a lesser effect on emotional responses related to depression (e.g., sadness or depressed affect) on the other hand.

Liverant and colleagues (2008) found that experimentally instructed suppression actually produced short-term reductions in participant sadness for clinically depressed individuals. However, suppression was no longer effective for those individuals experiencing moderate to severe levels of anxiety about future depressive symptoms.

This result suggests that the effects of suppression on anxiety may be different than on sadness for depressed individuals. The potential for unique differences in the effects of suppressing anxious responding (i.e., anticipation of future outcomes) compared to that of sadness or depressed affect (i.e., reflections of loss or a sense of failure) may also generalize to individuals experiencing low or no depressive symptoms. However, the Liverant et al. study did not include a manipulation control group (e.g., simply paying attention to the emotion induction) or a nonclinical population control group (those low in or without depressive symptomatology). This lack of inclusion impedes our ability to understand how effects of suppression differ from doing nothing at all or how effects on emotion differ for individuals without elevated levels of depression.

A recent study on emotion suppression discussed above (Boland & Papa, in preparation) also found that suppression was uniquely associated with different types of emotional responding. Suppressing facial expressions of one's emotion (e.g., frowns for sadness, "wide eyes" for anxiety) was found to be effective for decreasing experienced sadness for all participants, but is only effective for decreasing experienced anxiety for those high in trait NA. Different effects of ER strategies for different emotions is also supported by a pilot study reviewed above (Boland et al., in preparation) that found that acceptance led to the same decreased level of anxiety as controls for those high in trait NA but not those low in trait NA. Interestingly, acceptance had no effect on sadness in that study even though it did alleviate anxiety for those high in NA. First, this highlights a need to test the effects of any ER strategy on multiple emotions. Second, this further demonstrates the existence of potential differences in disorder-specific effects of any ER strategy. As sadness and anxiety are experienced at variable frequency for those with

major depression compared to those with anxiety disorders (i.e., sadness is more frequent in major depression whereas anxiety is more frequent in anxiety disorders; Sadock & Sadock, 2011), the effect of a given ER strategy likely depends on which disorder an individual experiences. It is therefore possible that individuals with major depression will respond differently to suppression of negative emotion compared to those with symptoms of anxiety disorders.

Comorbidity poses difficulties for examining disorder-specific effects of ER

Comorbidity between major depression and anxiety disorders is estimated to be about 60% in a 12-month period (Kessler et al., 2005). This number does not include subthreshold symptoms, so the presence of comorbid symptoms that still impact functioning may be even higher (Cameron, 2006). The high rate of comorbidity between depression and anxiety poses methodological challenges for comparing the effects of ER strategies between disorders. First, collecting a sample of individuals with one disorder but not the other (e.g., clinical levels of major depression but low levels of anxiety) makes it difficult for a researcher to collect a diverse clinical sample in a feasible period of time (e.g., one year or less). Second, collecting such a diverse sample makes it difficult to recruit a large enough number of participants with each distinct disorder to have adequate statistical power to detect existing effects.

A potential solution to this challenge may be derived from a few previous studies that examined aspects of emotional arousal associated with comorbidity of depression and anxiety symptoms. A recent study by Burkland and colleagues (2014) used fMRI to compare levels of the up- and down-regulation of amygdala activity in response to an affect-labeling task for social phobia participants. After recruiting and testing their social

phobia sample, they then examined how effects differed for those with varying comorbid disorders. Their results revealed a unique up-regulation of amygdala activity for social phobia participants with comorbid depression that was greater than that of social phobia participants with no comorbid diagnosis or with another comorbid anxiety disorder.

Cameron (2006) also examined unique patterns of biological markers of arousal in a sample of depressed, anxious, and comorbid depressed/anxious participants to understand differences in arousal unique to each disorder. He found noradrenergic dysfunction in those with anxiety disorders and comorbid depression but not in those with depression only. On the other hand, he found elevated adrenocorticotrophic hormone and cortisol responses to the Trier Social Stress Task for those with comorbid anxiety and depression, but not for those with only depression or an anxiety disorder. These studies not only show unique patterns of emotional arousal for comorbid individuals versus those with singular disorders, but also demonstrate the ability to examine the unique role of certain disorders on emotional responding by recruiting clinical populations and then comparing levels and types of comorbidity within those populations.

Following these designs, examining a specific clinical population with varying levels of a certain comorbid pathology may be an effective way to get at methodological problems associated with recruiting a necessarily very large diverse clinical sample of major depression and anxiety participants. For instance, recruiting a population of depressed individuals and then examining how those with varying levels of comorbid anxious pathological responding (e.g., pathological worry) would allow examination of the specific role of each pathology in moderating the effects of an ER strategy (like suppression) without having to recruit separate clinical populations and thus fill

exponentially increasing experimental group cells. Varying levels of comorbid anxiety symptoms among a depressed sample could be analyzed statistically as a continuous variable or as a covariate to uncover the degree to which anxiety symptoms moderate the effects of suppression above and beyond that of depression. This approach would allow a much smaller sample size to be recruited than a large-scale between-subjects design, while still maintaining adequate statistical power.

Lastly, examining populations with comorbidity has an important conceptual advantage in ER research. That is, researchers have recently posited that the level of comorbidity between major depression and anxious responding is a sign that common and specific factors of emotional responding exist that contribute to symptom presentations of each disorder (Aldao, 2013; Mennin et al., 2007). For example, nonacceptance of emotions has predicted GAD and SAD comorbidity, whereas emotional intensity has predicted GAD specifically and regardless of comorbidity with SAD (Mennin et al., 2009). Examining those who have depression as well as comorbid anxiety would be an effective way to further understanding specific and common factors of their varied responses to suppressing emotions.

An additional problem with high levels of comorbidity found in previous research may rest in the way each disorder is measured within each study. In terms of self-report measures, a high number of items overlap between common depression and trait anxiety measures. For instance, the Beck Depression Inventory, 2nd Ed. (BDI-II) and the State Trait Anxiety Inventory, Trait Version (STAI-T) are two commonly used measures within studies and have been found to have numerous overlapping items, and to both tap constructs related to depression and general negative affectivity (T.A. Brown, personal

communication, May 27, 2014; Groth-Marnat, 2009; J. Joorman, personal communication, May 9, 2014). Therefore, it will be beneficial to include measures of specific types of pathological responding which are paramount to certain disorders but that are distinct constructs (thus avoiding problems of criterion contamination). For instance, measuring depression and pathological worry, as paramount to GAD, may be a good way to discriminate between types of disordered responding found to be highly comorbid (e.g., major depression and GAD) but that may not overlap heavily as a product of measurement artifact. Fresco and colleagues (2003) found that measuring worry using the common and highly used Penn State Worry Questionnaire (PSWQ) reliably predicted which participants had a full diagnosis of GAD compared to which did not, while initial validation of the PSWQ found that it did not overlap with the construct of major depression (Meyer, Miller, Metzger, & Borkovec, 1990). Taken together, these studies suggest that worry, as reliably predictive of GAD, and major depression, would be valid representations of symptom presentations across which to compare the effects of suppression. Furthermore, comparing worry and depression flows conceptually from multiple studies by Mennin and colleagues (discussed above) that found differences in the existence of ER-related emotional processes between MDD and GAD (e.g., maladaptive management of emotions as related to GAD versus poor understanding of emotions as related to MDD).

The proposed study

Despite suggestions of different effects of ER strategies for different disorders, direct experimental testing of suppression on multiple emotions between depressed and highly anxious individuals is still lacking. However, testing ER strategies in depressed

and anxious individuals is sorely needed to understand just how these effects differ (e.g., does suppression lead to decreased sadness for depressed individuals whereas it leads to increase or maintenance of anxiety for those with high levels of trait anxiety?). Early studies in the attempt to understand the link between emotional responding (e.g., maladaptive management of emotions) and individual disorders (e.g., GAD) are highly informative, but have focused more on ER-related factors (e.g., how intense or how often certain emotions are experienced) as opposed to specific strategies often employed by individuals in response to specific emotions (e.g., suppressing emotions). Furthermore, the few studies that have tested the effects of specific strategies in psychological disorders have done so in the context of a unitary disorder only (e.g., suppression among depressed individuals only in Liverant et al., 2008) or without also including nonclinical participants on whom to draw comparisons (e.g., only clinical participants in Campbell-Sills et al., 2006b or in Liverant et al., 2008).

Suppression itself is thought to be a major form of avoidance that impedes the ability to enact adaptive regulation strategies (Gross, 1998) that are used naturally by individuals or after being taught to individuals engaged in any number of empirically supported treatments. That is, suppression leads to a lack of approach to cognitive content or actual content induced by aversive situations (Llewelyn et al., 2013). Approach to such content is considered necessary for engaging in adaptive responses like cognitive reappraisal, as emphasized in cognitive therapies (Campbell-Sills et al., 2006a). Suppression may also constitute an escape from exposing oneself to aversive stimuli (e.g., expressing naturally felt emotions in social situations) as emphasized in exposure therapies (Allen, McHugh, & Barlow, 2008; Campbell-Sills et al., 2006a). It constitutes

a form of avoidance or lack of acceptance of internal responses (e.g., emotions), and thus creates a barrier to living toward values emphasized in Acceptance and Commitment Therapy (ACT; Hayes et al., 1999). Suppression also leads to a lack of awareness and mindfulness for promoting regulated emotional responses in Dialectal Behavior Therapy for Borderline personality disorder (DBT; Fruzzetti et al., 2005; Linehan, 1993) and other mindfulness-based therapies (e.g., Mindfulness-based Cognitive Therapy for Depression; Segal, Williams, & Teasdale, 2013). Research on the moderating effects of different disorders on suppression is therefore needed to better understand how suppression might be specifically targeted by existing treatments.

The purpose of this study was to test the subjective and physiological effects of suppression after multiple emotion inductions and after respective subsequent resting periods among individuals with no or low depressive symptoms on the one hand, and individuals with mild to severe depressive symptoms who were both high and low in comorbid worry. The effects of suppression immediately after an emotion induction and after a subsequent resting period was examined because previous research has uncovered important effects of how individuals recover from negative emotion over time following attempts to suppress (e.g., delayed recover from negative emotion; Campbell-Sills et al., 2006b; Hofmann, et al., 2009). However, it is still unknown how suppressing emotions will affect individuals with mild to severe depressive symptoms and comorbid anxiety symptoms as compared to normative participants.

Major depression (determined by clinical cutoff on the BDI-II) and degree of comorbid worry (determined by clinical cutoff on the PSWQ) will be the clinical foci in this study because these conditions are highly prevalent in the U.S. (Kessler et al., 2005)

and locally in the State of Nevada (SAHMSA, 2007). Focus on these conditions will also enhance feasibility of conducting this study. First, there is a high prevalence of major depression in the State of Nevada community (ranked 6th highest in the nation for incidence of major depression, SAHMSA, 2007; 18% for clinical levels of depressive symptoms in a study recently conducted in our lab at UNR, Boland & Papa, under review; and estimated to be as high as 37% for depression in many university samples; Miller, Vaillancourt, & Hanna, 2009). Second, the very high rate of comorbidity between major depression and anxiety symptoms ensures that a recruited clinical population (those with major depression) will also exhibit an acceptable range of scores on a measure of worry. Therefore, the proposed study will recruit depressed individuals but will examine how clinical worry comorbidity will moderate the effect of suppression on sadness and anxiety.

Direct experimental testing will allow us to understand how psychological disorders generally and specifically influence the effect of emotion regulation strategies on negative emotion for the purpose of improving existing treatments, developing new ones where necessary, and efficiently tailoring any treatment to the needs of each individual seeking mental health services.

Hypotheses

Given data on the effects of suppression on different emotions (e.g., sadness versus anxiety) for those high versus low in trait NA (Boland & Papa, under review; Boland, Shallcross, Papa, & Mauss, in preparation; Shallcross et al., 2010), and correlational research suggesting that those with anxiety symptoms relate to and understand their emotions differently than those with depressive symptoms (e.g., active

versus passive responses to one's emotions; Aldao et al., 2010; Kashdan et al., 2010; Mennin et al., 2007), it is hypothesized that suppression will 1) reduce the experience of state sadness to the same degree for all participants. 2a) However, suppression will have no effect on state anxiety for normative participants and elevated depression participants with low comorbid worry compared to controls, but 2b) suppression will significantly reduce the level of state anxiety for those with elevated depression and comorbid worry compared to control. A group (suppression versus control) by trait (participants with elevated depressed with high worry versus all other participants) interaction will therefore emerge for state anxiety. Exploratory analyses will be conducted to examine the effect of time on state emotion, and to examine the effects of suppression and symptom level on cardiac responding in order to observe an objective outcome commonly associated with negative affect.

In sum, suppression will prove effective for reducing state sadness for all participants, but will only prove effective for reducing state anxiety for those with both depression and high trait worry compared to other groups. Therefore, a specific role of suppression unique to state anxiety and trait anxiety pathology (in this case, worry) will emerge.

Methodological Approach

Study overview

The purpose of this research is to investigate how emotion suppression might show different outcomes on multiple emotions for those with major depression and comorbid anxiety compared to nonclinical participants. This study used a mixed factorial pretest-posttest design (i.e., measuring the same participants use of both strategies over

multiple times points, and later comparing those with symptoms of major depression with and without comorbid worry to those without depression or worry) to examine the effects of suppression in a university sample of clinically depressed and healthy individuals.

Participants completed validated measures of depression and trait worry traditionally used with both clinical and nonclinical populations (BDI-II and PSWQ) before beginning the experimental manipulation to later determine their level of psychopathology. These measures were chosen because of their widespread use in clinical studies and for their sound psychometrics (Beck & Steer, 1984; Fresco et al, 2003; Meyer et al., 1990; Miller, Vaillancourt, & Hanna, 2009).

Participants were then randomly assigned to one of four orders of experimental presentation. All participants received instructions to suppress and pay attention (control) in response to a sadness induction (a sad film clip) and an anxiety induction (an impromptu speech) that were both followed by resting periods. The order of instructions and the order of inductions were both randomized to control for order effects. All participants viewed a neutral baseline film clip at the beginning of the experimental portion and after the resting period that followed the first induction (but before the second induction took place). Four experimental orders were as follows: pay attention in response to a sad film and later suppress emotions in response to giving an impromptu speech; suppress emotions in response to a sad film and later pay attention in response to an impromptu speech; pay attention in response to an impromptu speech and later suppress emotion in response to a sad film clip; suppress emotion in response to an impromptu speech and later pay attention to a sad film clip (see Figure 1 for a visual depiction of counterbalanced orders of procedures). This study's mixed factorial design

allowed for multiple factors (e.g., 2 sets of instructions, 2 time points) to be tested within-subject on each emotion induction separately, thus maximizing statistical power.

Dependent variables included levels of self-reported emotions (e.g., anxiety, sadness, fatigue, worry, etc; rated on a 1-9 Likert-type scale) taken after each neutral clip, after each emotion induction, and after each resting period. DVs also included interbeat interval recorded throughout the study to observe physiological effects of suppression across major depression and comorbid worry.

Participants

One hundred six participants (72% female) were recruited from the UNR university undergraduate population (via undergraduate psychology courses, flyers placed on campus, and flyers placed at the UNR Student Counseling Center). Participants were required to be fluent in English. They were paid (\$20 each) using research scholarship monies previously awarded to the principal investigator, but they could choose to receive extra credit toward psychology course work instead of cash.

Mean age for this sample was 21.21 years ($SD=4.32$). Participants identified as being European-American (47.2%), Hispanic (15.1%), African-American (6.6%), Asian American (14.2%), Pacific Islander (0.9%, 1 participant), Native American (0.9%, 1 participant), Other (4.7%), and Mixed Ethnicity (10.4%). Participants were randomly assigned to receive some combination of instructions: those instructed to suppress during sadness induction ($N=54$) and pay attention during anxiety induction ($N=54$), and those instructed to pay attention during sadness induction and suppress during anxiety induction ($N=52$). Participants were assigned to three symptom level groups based on clinical cutoffs on a measure of depression and a measure of clinical worry: those who showed a low level

of depressive symptoms or none at all (referred to in this study as “normative” participants; $N=38$), those who showed mild to severe depressive symptoms and were below a clinical cutoff for worry pathology (referred to in this study as “elevated depression-low worry,” $N=31$), and those who showed mild to severe depressive symptoms and were above a clinical cutoff for worry pathology (referred to in this study as “elevated depression-high worry,” $N=36$).

It took approximately two semesters and a summer term to recruit this sample. This was slightly longer than our 1.5 semester estimate, which was based on a rate calculated from a previous experimental study on suppression conducted at UNR (Boland & Papa, under review). In that study, approximately 72 participants per semester were recruited over 2 academic semesters (not including Summer and Winter break), when scheduling 9 experiments per week. The need for participants experiencing symptoms of major depression was advertised in recruitment efforts. Clinical cut-off scores on the BDI-II were used to determine that an adequate range of major depressive symptom severity was observed throughout the sample (also see “Measures” section), so the recruitment took longer for the current study because of the need for a large portion of the sample to score on or above clinical cutoff on the BDI-II.

Procedures

Participants arrived at the laboratory and were provided consent information as approved by the UNR Internal Review Board (IRB). They were informed that the purpose of the study was to understand the effects that television as seen on the Internet (e.g., on YouTube or Hulu) has on viewers, but were informed of the true nature of the study when they were debriefed at the end of the study. Following consent to participate

in the study, electrodes were attached to them to measure heart rate and skin conductance levels. They then completed measures of psychopathology and were randomly assigned to one of the four possible orders of ER conditions.

Participants then viewed a neutral film designed to establish baseline emotional responding. State emotion ratings and emotion process ratings (e.g., “I tried to not think about my emotions as I viewed the clips in the study”) were taken after the neutral film clip. Emotion process ratings assessed the degree to which participants regulated their emotions in a number of ways during the induction, including the degree to which they suppressed their emotions.

Manipulation. After the first neutral film clip, a cover story and a script with instructions appeared on the computer screen for participants to read (up to 30 seconds reading time for each instruction). The cover story described how internet television companies (e.g., YouTube, Hulu) are interested learning more about how viewers respond to content they place on their websites. This cover story has been shown to be successful in previous studies conducted by the author (Boland & Papa, under review; Boland et al., 2012; Mauss, Tamir, Anderson, & Savino, 2011). The instructions described how regulating one’s emotions in a certain way shows certain benefits and then described how participants should regulate their emotions (either pay attention or suppress), in response to the following film clip or task they encounter (either watch sad film clip or give impromptu speech). Giving instructions to participants on how to regulate in response to emotion inductions has been widely successful for previous studies (Boland & Papa, under review; Campbell-Sills et al., 2006b; Gross, 1998). Instructions were relatively similar between groups in length, formatting style, and

information expressed to participants. The following are the instructions were given to each participant depending on the manipulation given:

1. *Suppression*: “Internet television companies like Hulu and YouTube are interested in researching ways that viewers respond to their programming. Many research findings show that individuals who suppress, or try not to outwardly express the emotions they experience, are more able to pay attention to the content of television programs and television clips. Actively hiding feelings from others can also help you to more easily accomplish tasks you perform. Additionally, hiding the feelings you are experiencing will enable you to not have to show any unnecessary emotional discomfort or distress in social situations. Research shows that it is possible for people to hide their emotions from others if they really concentrate on trying not to express them.

Please pay attention to the following film clips. While paying attention to the clips, do everything possible to not express any emotions on your face. Whatever you may feel during the film clips, try to not let those feelings show so that if someone else were watching you, they would not be able to tell that you were feeling anything.”

2. *Control*: “Internet television companies like Hulu and YouTube are interested in researching ways that viewers respond to their programming. Many research findings show that individuals who pay attention to programming are more able to recall content and make accurate judgments about what they see. Therefore, paying attention to film clips may show the benefits of paying

attention to events in real life. For instance, paying attention may help you more easily accomplish tasks you perform. Research shows that it is possible for people to be more accurate about their reactions and their judgments when they pay attention to what is going on in a situation.

With the above in mind, please pay attention to the following film clips as they are shown on the screen.”

Instructions to suppress emotional expression were used in the study as opposed to instructions to suppress emotional experience because a suppression study previously conducted at UNR (Boland & Papa, under review) showed that instructing participants to suppress the experience of emotions mostly had the same effect on emotional experience as simply paying attention (control condition) to a mixed sadness/anxiety film clip. Instructing participants to suppress the expression of emotion actually caused participants to successfully dampen the experience of emotion compared to suppressing experience of emotion and compared to control condition. Therefore, instructing participants to suppress their expression of emotions may actually be a more valid proxy for emotion suppression. Additionally, further exploratory analyses included in that study found that participants instructed to suppress the experience of their emotions actually attempted to feel less negative, attempted to ignore their emotions, attempted to feel more positive, and tried to change their facial expressions, whereas participants instructed to suppress emotional experience only attempted to change their facial expressions. Instructions to suppress emotional expression may therefore be methodologically cleaner in that participants engage in a more unitary regulation process than if they are given instructions to suppress emotional experience. Instructions to simply “pay attention”

were used in this study because they have been shown to be effective as a manipulation control in previous studies (Boland & Papa, under review; Mauss, Troy, Andersen, & Savino, 2011).

Emotionally evocative stimuli. The order of the evocative stimuli described here assumes that participants viewed the sad film clip as first induction and participated in the speech task as second induction. However, in the study they may have taken part in the speech task first (depending on their randomly assigned order of induction stimuli).

The baseline neutral film clip used in this study depicted a nature scene with footage of changing seasons in an arctic forest. Nature clips such as this have been used as a neutral baseline in previous studies on emotion regulation strategies (e.g., Gross, 1998; Rottenberg, Ray, & Gross, 2007). This particular clip has not been used in published research, but was chosen in order to have a modern high-quality digital clip that would support this study's cover story. Guidelines for creating emotion induction film clips provided by Gross and Levenson (1995) were followed to construct this clip. For instance, they recommended using or constructing neutral clips that contain no discrete emotional elements but are somewhat pleasant or relaxing. Participants in a previous suppression study conducted at UNR endorsed low levels of emotional reactivity after the clip followed by significant changes in emotional reactivity after mixed induction (Boland & Papa, under review), thus supporting the effectiveness of this clip as a neutral baseline.

Following post-baseline rationale and first ER instructions, participants viewed a sad film clip shown in previous research to successfully induce sadness. "The Champ" was used, which is approximately a 3-minute film clip that depicts a boy losing his father

after a boxing match. This film has been validated and used successfully as a sadness induction in multiple previous studies and in research specifically examining the effectiveness of film clips to induce sadness (see Rottenberg, Ray, & Gross, 2007). This clip was followed by ratings of state emotion (using the same state emotion ratings and emotion process ratings as after the baseline clip), then a 2-minute resting period to allow participants to return to baseline emotional responding, then more state emotion ratings and emotion process ratings.

Following the first post-clip resting period, participants viewed another neutral film clip to ensure their return to baseline emotional responding. This clip was followed by state emotion ratings. Participants were then instructed that they will give an impromptu speech for “a mock job interview,” and that this will be done in front of a camera and participants will be told their performance will be recorded and later rated by judges. They were given two minutes to prepare for the speech. After this two-minute preparation period, the experimenter entered the room and turned on the camera. Participants were then instructed to turn to the camera and give their speech. After the three minutes were up, a recorded voice on the computer informed them they can stop speaking and to turn back to the computer. This task has been shown to effectively induce anxiety in previous studies (e.g., Boland, Shallcross, Papa, & Mauss, in preparation; Hofmann et al., 2009).

The difference in induction methods between sadness and anxiety did pose a potential limitation for the study. That is, having one task being more passive (viewing a film-clip) and the other being more instrumental (giving a speech) could create differences in participant responding above and beyond that of what suppressing would

purely yield. However, effective anxiety inductions have not been found to exist in the form of film clips and no research could be found that used speaking tasks to induce sadness. Additionally, the proposed methods have been shown to be to be highly effective and powerful inductions for each emotion in numerous studies (discussed above). It was therefore decided to use strong inductions that are also previously validated, even if consistency in induction methods for sadness and anxiety cannot be reached. Furthermore, previous research has found that people are able to effectively follow regulation instructions even while also focusing on either viewing film clips or on completing speeches (e.g., Boland, Shallcross, Papa, & Mauss, Study 2 in preparation; Hofmann et al., 2009), so this difference in induction methods may not be problematic.

Participants were then debriefed and given information about mental health counseling services available at the University of Nevada, Reno after they complete both the film clip and the speech and each subsequent resting period.

Measures

Psychopathology measures. Trait measures of psychopathology included the well validated and widely used Beck Depression Inventory, 2nd ed. (BDI-II; Beck & Steer, 1984) and Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990). The BDI-II is a 21 item measure listing commonly observed symptoms or phenomena associated with major depression that has been found to be sensitive to detecting depressive symptoms in undergraduate samples (some studies have found that up to 37% of their undergraduate sample met clinical levels for depression using the BDI-II; Miller, Vaillancourt, & Hanna, 2009). A standard cutoff of 14 or greater indicates individuals experiencing mild to severe depressive symptoms (Beck & Steer, 1996). A cutoff score

was of 14 was used to indicate depressive symptoms in this study, so that individuals scoring less than 14 were considered “normative” and individuals scoring 14 or greater were considered “depressed.”

The PSWQ is a commonly used measure of worry, with clinical cutoffs to indicate pathological levels of worry indicative of GAD (Fresco et al., 2003). It is a 16-item measure with a 1-5 rating scale to indicate severity of experience of each item (5 items are reverse scored). The PSWQ has demonstrated high internal consistency and good test-retest reliability. It was found to significantly discriminate among those who met GAD on the one hand, and other anxiety disorders (including PTSD) and depression on the other hand (Fresco et al., 2003). This measure’s ability to tap a construct paramount to anxiety disorder symptomatology, but that also does not have measurement overlap with major depression, is a strength of the measure for this study. A number of cutoffs have been used to indicate clinical levels of worry pathology. An established clinical cutoff score of 62 was used for the current study because Behar and colleagues (2003) found that score to be sensitive to detecting clinical worry in an undergraduate population.

The Depression, Anxiety, and Stress Scales (DASS-21) was considered for examining both depression and anxiety symptoms because of its widespread use, relatively short length for the number of conditions it encapsulates, and sound psychometrics. However, similar issues of high comorbidity existing between depression and anxiety disorder symptomatology generally remain with its possible use in this study. Other measures were considered (e.g., semi-structured interviews like the SCID-II or ADIS-IV), but were not chosen because of feasibility considerations and because of the

formulated decision to measure depression and comorbid worry in this study.

State emotion ratings. Participants were asked after each task to rate their current level of 11 different emotions (anxiety, sadness, frustration, relaxation, happiness, worry, contentment, fatigue, nervousness, shame, tension) on a 9-point Likert-style scale from 1 (“low”) to 9 (“high”) as used in similar studies (e.g., Gross, 1998; Mauss et al., 2007). Participant ratings of sadness and anxiety were the primary ratings of interest for this study.

Emotion process rating. Emotion process ratings were used to determine if participants actually followed instructions for regulating emotions as intended and to observe how varying levels of regulating might affect outcomes. Thus, participants were also asked to rate the extent to which they regulated, monitored, or tried to control their emotions in certain ways during the film clips or speech on a 1-9 scale from 1 (“not at all”) to 9 (“extremely”). An example item would read “I tried to alter my emotional reactions to the film clip.” These types of process questions have been used previously in a number of ER studies (e.g., Gross and John, 2003). Emotion process questions can be used as a manipulation check and for potential future exploratory analyses.

Physiological measures. Biopac MP150 (400Hz) was used to measure participant cardiac interbeat interval (IBI). IBI is a well established indicator of the experience and regulation of emotion (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). Measures of cardiac responding have consistently indexed responsiveness across numerous suppression studies in particular (e.g., Campbell-Sills et al, 2006b; Hofmann et al., 2009; Gross & Levenson, 1993), while providing a measure of both sympathetic and parasympathetic activity. IBI was chosen for this study over other measures of cardiac

responding (e.g., heart rate variability) in order to attain single measure averages that closely resemble calculations for average subjective emotion ratings. This between-measure resemblance allowed for greater coherence in analyses and interpretation of study results.

To measure IBI, three sensors were placed on each participant at the beginning of the study and were removed immediately before debriefing. Sensors were placed just below the left and right collar bones and one just below the lowest left rib (ground). Baseline cardiac data were collected during the neutral film clip (for a baseline reading) and was measured continuously throughout the study in order to document changes in physiological activity in response to regulating emotion. Physiological recordings were later partitioned into periods throughout each time point (e.g., baseline, induction, resting period) and mean IBI calculations were made for those time points only (i.e., cardiac responses were not recorded while participants rated their emotional experiences). Average IBI was calculated by averaging intervals (in seconds) between successive R-waves. The R-wave array file was filtered using filter pass methods provided by Acknowledge 3.9 analysis software package and then manually corrected with a mouse by a consulting Physiology Ph.D. candidate and three trained research assistants. Incorrectly specified R-waves (e.g., waves identified as R-waves that were actually artifacts due to movement) were removed and actual R-waves missed by the software package were identified using this method (procedure outlined in Campbell-Sills et al, 2006b).

Data Plan

Power analysis. Power analysis was conducted using the G Power, v 3.1.2

computer program (Faul, Erdfelder, Lang, & Buchner, 2010). The sample size of 76 subjects was estimated for this 3 x 2 x 2 x 2 mixed factorial pretest-posttest design with within-subject randomization (2 emotion inductions, 2 emotion regulation instructions), 2 measurement time points for each induction (post-induction and post-resting period), and a between-subject three group comparison (i.e., normative participants versus elevated depression and low worry participants versus elevated depression and high worry). This analysis was based on an alpha level of 0.05, a high achieved power ($\beta=.95$), and a medium effect size ($f= 0.25$; medium to large effects were found for our results in preliminary research on suppression, Boland & Papa, in preparation). However, additional participants were collected in order to explore the effects of additional moderators outside of the current study.

Data analysis. 3 (normative, elevated depression-low worry, elevated depression-high worry) x 2 (suppress emotion, control) x 2 (self-reported emotion after induction and after resting period) repeated-measures ANOVAs were used to examine differences in outcomes as a result of ER manipulation (suppression versus control) and its interaction with psychopathology (i.e., three different symptom levels) after each induction and subsequent resting period. Effects of suppression were tested separately on each outcome measure (self-reported sadness, self-reported anxiety, cardiac IBI in response to each induction). Data was evaluated for multivariate normality and homogeneity of covariance (sphericity). For each analysis in which the omnibus test is significant, post-hoc analyses evaluated which pair-wise comparisons (e.g., suppression versus control) were significantly different. To test whether individual differences in levels of comorbid trait worry among depressed participants moderated the effects of

instructions on outcome (e.g. state negative emotions), level of trait worry was inserted as a categorical grouping variable (based on an established clinical cutoff) to test if trait worry moderates the effects of suppression on depressed and nonclinical participants. In order to determine magnitude of differences found, effect size analysis was conducted to obtain partial eta squared (partial η^2).

Multivariate outliers were searched for all multivariate analyses and removed based on standard cutoffs for standardized residual, leverage, and Cook's *D* values (see Tabachnik and Fidell, 2012).

Results

Randomization Check

In order to confirm that counterbalanced groups were equivalent in level of self-reported emotion and physiological reactivity at baseline, self-reported sadness and anxiety ratings, and IBI were compared across counterbalanced conditions. Comparing self-reported sadness and anxiety after the baseline film clip across conditions revealed that the four groups did not differ in self-reported sadness, $F(2,104) = .39, p = .80$, self-reported anxiety, $F(2,103) = 1.55, p = .21$, or IBI, $F(3,91) = .08, p = .97$, at baseline. In sum, all outcome measures were equivalent at baseline, allowing for clear comparisons of outcomes as a result of induction.

In order to confirm that symptom level groups were equivalent in level of self-reported emotion and physiological reactivity at baseline, self-reported sadness and anxiety ratings, and IBI were compared across symptom levels. Comparing self-reported sadness and anxiety after the baseline film clip across conditions revealed that the three symptom levels did not differ in self-reported sadness, $F(2,104) = .36, p = .70$, did differ

on self-reported anxiety, $F(2,104) = 3.50, p = .03$, and did not differ on IBI, $F(3,93) = .08, p = .45$, at baseline.

Post-hoc examination of the main effects of symptom level on anxiety at baseline using Least Square Difference revealed that, across both experimental conditions, normative participants reported no difference in anxiety compared to elevated depression-low worry participants, $M_{\text{difference}} = -.42, SE = .53, p = .43$, but less anxiety than elevated depression-high worry participants, $M_{\text{difference}} = -1.33, SE = .51, p = .01$. Elevated depression-low worry participants reported marginally less anxiety than elevated depression-high worry participants, $M_{\text{difference}} = -.91, SE = .54, p = .10$. In sum, elevated depression-high worry showed no difference in anxiety at baseline compared to elevated depression-low worry participants, but greater levels of anxiety than normative participants.

Manipulation Checks

Order Effects. Differences in self-reported sadness and anxiety rated immediately after the film clip and the speech were each compared separately across all four counterbalanced ordered conditions to confirm that there were no effects due to presented order of stimuli or order of instruction. Analyses revealed no differences in self-reported sadness, $F(3,105) = .31, p = .82$, self-reported anxiety, $F(3,104) = .43, p = .73$, or IBI, $F(3,91) = .06, p = .98$, after the film clip across counterbalanced conditions. Analyses also revealed no differences in self-reported sadness, $F(3,104) = 1.50, p = .22$, self-reported anxiety, $F(2,104) = 1.13, p = .34$, or IBI, $F(3,89) = 1.38, p = .25$, after the speech across counterbalanced conditions. Together, this indicates no effect of order of conditions.

In order to account for potential interactions between order of experimental condition, order of inductions, and time, 2 (self-reported emotion or IBI after induction, after resting period) X 2 (sadness clip first and anxiety speech second, anxiety speech first and sadness clip second) X 2 (suppress first and control second, control first and suppress second) two way repeated-measures ANOVAs were also conducted separately on each self-reported emotion and IBI for each induction and subsequent resting period.

For self-reported sadness in response to sadness induction, analysis revealed an effect of time, $F(1,102) = 165.27, p < .001, \eta^2 = .62$, but no time by induction order interaction, $F(1,102) = .71, p = .40, \eta^2 = .007$, no time by condition order interaction, $F(1,102) = 1.58, p = .21, \eta^2 = .02$, and no time by induction order by condition order interaction, $F(1,102) = .59, p = .45, \eta^2 = .006$. There was no main effect of induction order, $F(1,102) = 1.53, p = .22, \eta^2 = .02$, no main effect of condition order, $F(1,102) = .07, p = .80, \eta^2 = .001$, and no induction order by condition order interaction $F(1,102) = .13, p = .72, \eta^2 = .001$. For self-reported anxiety after anxiety induction, four multivariate outliers were removed. Analysis revealed an effect of time, $F(1,98) = 161.49, p < .001, \eta^2 = .62$, but no time by induction order interaction, $F(1,98) = .02, p = .88, \eta^2 < .001$, no time by condition order interaction, $F(1,98) = .78, p = .38, \eta^2 = .008$, and no time by induction order by condition order interaction, $F(1,98) = .89, p = .77, \eta^2 = .001$. There was no main effect of induction order, $F(1,98) = 1.40, p = .24, \eta^2 = .01$, no main effect of condition order, $F(1,98) = .01, p = .91, \eta^2 < .001$, and no induction order by condition order interaction $F(1,98) = .41, p = .52, \eta^2 = .004$.

For IBI in response to sadness induction, two multivariate outliers were removed. Analysis revealed an effect of time, $F(1,82) = 7.29, p = .008, \eta^2 = .08$, but no time by

induction order interaction, $F(1,82) = .01, p = .92, \eta^2 < .001$, no time by condition order interaction, $F(1,82) = .12, p = .73, \eta^2 = .001$, and no time by induction order by condition order interaction, $F(1,82) < .01, p = .97, \eta^2 < .001$. There was no main effect of induction order, $F(1,82) = .10, p = .76, \eta^2 = .001$, no main effect of condition order, $F(1,82) < .01, p = .97, \eta^2 < .001$, and no induction order by condition order interaction $F(1,82) = .05, p = .82, \eta^2 = .001$. For IBI after anxiety induction, four multivariate outliers were removed. Analysis revealed an effect of time, $F(1,80) = 34.04, p < .001, \eta^2 = .30$, but no time by induction order interaction, $F(1,80) = .01, p = .94, \eta^2 < .001$, no time by condition order interaction, $F(1,80) < .01, p = .97, \eta^2 < .001$, and no time by induction order by condition order interaction, $F(1,80) = .59, p = .45, \eta^2 = .007$. There was no main effect of induction order, $F(1,80) = 2.33, p = .13, \eta^2 = .03$, no main effect of condition order, $F(1,80) = 54, p = .47, \eta^2 = .007$, and no induction order by condition order interaction $F(1,80) = .36, p = .55, \eta^2 = .005$. In sum, results revealed no effects of order of induction or experimental condition. Induction order and condition order were therefore not included as factors in primary analyses of self-reported and physiology.

Effectiveness of induction. The effectiveness of each induction to induce sadness and anxiety (respectively) as well as the effectiveness of the resting period to lead to a subsequent reduction in self-reported emotion were then compared. Comparing changes in self-reported sadness ($N=51$) and anxiety ($N=54$) in control condition participants from after neutral film clip to after the emotion induction revealed that self-reported sadness after sadness induction, $M = 5.46, SE = .33$, was significantly higher than at baseline, $M = 2.21, SE = .21, M_{difference} = -3.25, SE = .36, t(51) = -8.98, p < .001$, and that self-reported anxiety after anxiety induction, $M = 7.31, SE = .24$, was significantly higher than at

baseline, $M = 2.35$, $SE = .20$, $M_{\text{difference}} = -4.96$, $SE = .30$, $t(53) = 16.31$, $p < .001$.

Comparisons of self-reported sadness ($N=52$) and anxiety ($N=54$) in the control condition after each induction to levels of the same emotion at the end of each subsequent resting period revealed that each resting period led to a reduction of sadness $M_{\text{difference}} = 2.54$, $SE = .33$, $t(51) = -8.00$, $p < .001$, and anxiety, $M_{\text{difference}} = 2.63$, $SE = .26$, $t(53) = -10.14$, $p < .001$, respectively.

Comparing changes in self-reported anxiety after sadness induction revealed that the sadness induction also induced anxiety, $M = 3.83$, $SE = .30$, compared to anxiety at baseline before sadness induction, $M = 2.69$, $SE = .26$, $M_{\text{difference}} = -1.13$, $SE = .27$, $t(51) = -4.13$, $p < .001$. Therefore, the sadness induction induced anxiety to a much lesser degree than it did sadness. Comparing changes in self-reported sadness after anxiety induction revealed that the anxiety induction also induced sadness, $M = 3.59$, $SE = .33$, compared to sadness at baseline before anxiety induction, $M = 2.02$, $SE = .16$, $M_{\text{difference}} = -1.57$, $SE = .29$, $t(51) = -5.50$, $p < .001$. Therefore, the anxiety induction induced sadness to a much lesser degree than it did anxiety. The fact that each induction induced emotions other than those emotions primarily intended to be induced is consistent with previous research on emotion elicitation. Elicitation research has found that negative emotion inductions typically induce a range of negative affect, but that those emotions that are more strongly induced (e.g., state sadness from sadness induction, state anxiety from anxiety induction) are those on which experimental manipulations (e.g., suppression) will have a primary effect (Rottenberg, Ray, & Gross, 2007).

Differences in the strength of each induction to induce their respective primary emotion were then compared among control participants to explore the variable

effectiveness of each induction. Comparing ratings in self-reported sadness after sadness induction ($N=52$) to self-reported anxiety after anxiety induction ($N=54$) revealed lower levels of sadness after sadness induction, $M = 5.46$, $SE = .33$, compared to anxiety after anxiety induction, $M = 7.31$, $SE = .24$, $M_{\text{difference}} = -1.85$, $SE = .41$, $t(104) = -4.57$, $p < .001$. Participants experienced more anxiety after anxiety induction than they experienced sadness after sadness induction.

Effects of gender on induction. Previous research has found gender differences in emotional reactivity and motivation for regulating emotion (e.g., Timmers, Fischer, & Manstead, 1998). To explore possible gender differences in the effects of suppression in response to each induction and subsequent resting period in this study, 2 (suppress emotion, control) X 2 (female, male) X 2 (self-reported emotion or IBI after induction and after resting period) repeated-measures ANOVAs were conducted separately on each self-reported emotion and IBI for each induction and subsequent resting period.

For self-reported sadness in response to sadness induction, analysis revealed an effect of time, $F(1,100) = 8.42$, $p = .005$, $\eta^2 = .08$, but no time by condition interaction, $F(1,100) = .52$, $p = .47$, $\eta^2 = .005$, no time by gender interaction, $F(2,100) = 1.79$, $p = .17$, $\eta^2 = .04$, and no time by condition by gender interaction, $F(2,100) = .42$, $p = .52$, $\eta^2 = .004$. There was no main effect of experimental condition, $F(1,100) = .15$, $p = .70$, $\eta^2 = .001$, no main effect of gender, $F(1,100) = 1.50$, $p = .23$, $\eta^2 = .03$, and no condition by gender interaction $F(1,100) = 1.48$, $p = .23$, $\eta^2 = .02$. For self-reported anxiety after anxiety induction, five multivariate outliers were removed. Analysis revealed an effect of time, $F(1,96) = 129.05$, $p < .001$, $\eta^2 = .57$, but no time by condition interaction, $F(1,96) = .02$, $p = .88$, $\eta^2 < .001$, no time by gender interaction, $F(2,96) = 1.04$, $p = .31$, $\eta^2 = .01$,

and no time by condition by gender interaction, $F(2,96) = .87, p = .36, \eta^2 = .009$. There was no main effect of experimental condition, $F(1,96) = .35, p = .56, \eta^2 = .004$, no main effect of gender, $F(1,96) = 2.31, p = .13, \eta^2 = .02$, and no condition by gender interaction $F(1,96) = .27, p = .61, \eta^2 = .003$.

For IBI in response to sadness induction, one multivariate outlier was removed. This analysis revealed an effect of time, $F(1,82) = 7.19, p = .009, \eta^2 = .08$, but no time by condition interaction, $F(1,82) = .34, p = .56, \eta^2 = .004$, no time by gender interaction, $F(2,82) = .20, p = .66, \eta^2 = .002$, and no time by condition by gender interaction, $F(2,82) = 1.59, p = .21, \eta^2 = .02$. There was no main effect of experimental condition, $F(1,82) = .05, p = .82, \eta^2 = .001$, no main effect of gender, $F(1,82) = .56, p = .46, \eta^2 = .007$, and no condition by gender interaction $F(1,82) = .23, p = .63, \eta^2 = .003$. For IBI in response to anxiety induction, one multivariate outlier was removed. Analysis revealed an effect of time, $F(1,82) = 33.07, p < .001, \eta^2 = .29$, but no time by condition interaction, $F(1,82) = .58, p = .45, \eta^2 = .007$, no time by gender interaction, $F(2,82) = 1.09, p = .30, \eta^2 = .01$, and no time by condition by gender interaction, $F(2,82) = .01, p = .91, \eta^2 < .001$. There was no main effect of experimental condition, $F(1,82) < .001, p = 1.00, \eta^2 < .001$, no main effect of gender, $F(1,82) = 2.06, p = .16, \eta^2 = .02$, and no condition by gender interaction $F(1,82) = .08, p = .78, \eta^2 = .001$, no main effect of condition, $F(1,104) = .39, p = .53, \eta^2 < .01$, or gender, $F(2,104) = 2.20, p = .12, \eta^2 = .04$, and no condition by gender interaction, $F(1,104) = .37, p = .54, \eta^2 < .01$. For IBI in response to sadness induction, analysis revealed no main effect of condition, $F(1,90) = .02, p = .90, \eta^2 < .001$, or gender, $F(2,90) = .34, p = .71, \eta^2 < .01$, and no condition by gender interaction, $F(1,90) = .004, p = .95, \eta^2 < .001$. For self-reported anxiety in response to anxiety

induction, analysis revealed a marginal main effect of condition, $F(1,104) = 2.95, p = .09, \eta^2 = .03$, no main effect of gender, $F(2,104) = 2.01, p = .14, \eta^2 = .04$, and no condition by gender interaction, $F(1,104) = 1.16, p = .29, \eta^2 = .01$. For IBI in response to anxiety induction, analysis revealed no main effect of condition, $F(1,89) = 1.14, p = .29, \eta^2 = .01$, or gender, $F(2,89) = .12, p = .89, \eta^2 < .01$, and no condition by gender interaction, $F(1,89) = .18, p = .67, \eta^2 < .01$. In sum, there were no effects of gender on self-reported emotion or physiology in this study. Gender was therefore not included as a factor in primary analyses of self-reported and physiology

Level of self-reported suppression. In order to examine whether participants followed instruction to suppress emotion, within-subject ratings of attempted suppression (“I tried to hide my feelings in response to the [film clip or speech].”) were compared between suppression and control participants for each induction. For sadness induction, participants attempting to suppress sadness ($N=54$) endorsed greater attempts to suppress their expression, $M = 8.19, SE = .24$, compared to those instructed to pay attention ($N=51$), $M = 4.42, SE = .36, M_{difference} = 3.76, t(104) = 8.67, p < .001$. For anxiety induction, participants attempting to suppress anxiety ($N=51$) endorsed marginally greater attempts to suppress their expression, $M = 6.00, SE = .32$, compared to those instructed to pay attention ($N=54$), $M = 5.09, SE = .36, M_{difference} = .89, t(104) = 1.83, p = .070$.

Ratings of attempted suppression for those instructed to suppress during sadness induction compared to those instructed to suppress during anxiety induction were then conducted to explore how attempts to suppress emotion differed between inductions. Those instructed to suppress during sadness induction ($N=54$) endorsed greater attempts to suppress their expression, $M = 8.19, SE = .24$, compared to those instructed to suppress

during anxiety induction ($N=51$), $M = 6.00$, $SE = .32$, $M_{difference} = 2.20$, $t(104) = 5.50$, $p < .001$.

In order to check for potential effects of experimental condition order and induction order on participants self-reported attempts to suppress emotion, 2 (sadness clip first and anxiety speech second, anxiety speech first and sadness clip second) X 2 (suppress first and control second, control first and suppress second) two-way univariate ANOVAs were conducted separately on self-reported attempts to suppress during each emotion induction (self-reported attempts to suppress was not measured in response to each subsequent resting period).

For self-reported attempted suppression in response to sadness induction, analysis revealed no main effect of induction order, $F(1,106) = .13$, $p = .72$, $\eta^2 = .001$, no main effect of condition order, $F(1,106) = .65$, $p = .42$, $\eta^2 = .006$, and an induction order by condition order interaction, $F(1,106) = 75.73$, $p < .001$, $\eta^2 = .43$. To analyze the induction order by condition order interaction, independent samples t-tests were used to compare level of self-reported attempted suppression between condition for each induction order. Post-hoc analysis revealed that among participants who experienced sadness induction first, those instructed to suppress their emotions ($N=24$), $M=8.29$, $SE=.32$, endorsed attempting to suppress their emotions to a greater degree than control participants ($N=25$), $M=4.16$, $SE=.44$, $M_{difference} = 4.13$, $SE = .55$, $t(47) = 7.51$, $p < .001$. Among participants who experienced sadness induction second, participants instructed to suppress their emotions ($N=30$), $M=8.10$, $SE=.34$, endorsed attempting to suppress their emotions to a greater degree than control participants ($N=27$), $M=4.67$, $SE=.57$, $M_{difference} = 3.43$, $SE = .65$, $t(55) = 5.26$, $p < .001$. In sum, participants instructed to suppress their

emotion in response to sadness induction endorsed attempting to suppress their emotions to a greater degree than control participants, regardless of the order in which they experienced sadness induction.

For self-reported attempted suppression in response to anxiety induction, five multivariate outliers were removed. Analysis revealed no main effect of induction order, $F(1,101) = .13, p = .72, \eta^2 = .001$, no main effect of condition order, $F(1,101) = .35, p = .56, \eta^2 = .004$, and an induction order by condition order interaction, $F(1,101) = 9.21, p = .003, \eta^2 = .09$. To analyze the induction order by condition order interaction, independent samples t-tests were used to compare level of self-reported attempted suppression between condition for each induction order. Post-hoc analysis revealed that among participants who experienced anxiety induction first, those instructed to suppress their emotions ($N=25$), $M=6.56, SE=.35$, endorsed attempting to suppress their emotions to a greater degree than control participants ($N=30$), $M=4.90, SE=.47$, $M_{\text{difference}} = 1.66, SE = .61, t(53) = 2.74, p = .008$. Among participants who experienced anxiety induction second, participants instructed to suppress their emotions ($N=22$), $M=6.45, SE=.37$, endorsed attempting to suppress their emotions to a marginally greater degree than control participants ($N=24$), $M=5.33, SE=.57, M_{\text{difference}} = 1.12, SE = .69, t(44) = 1.65, p = .10$. In sum, participants instructed to suppress their emotion in response to anxiety induction endorsed attempting to suppress their emotions to a greater degree than control participants when they experienced anxiety induction first, but endorsed attempting to suppress their emotion to a marginally greater degree than control participants when they experienced anxiety induction second. Implications for this result are made in the Limitations section of the Discussion.

Hypothesis Testing

In order to test the first hypothesis (H1) that suppression will reduce the experience of sadness to the same degree across normative, elevated depression-low worry, and elevated depression-high worry participants, and the second hypothesis (H2) that suppression will reduce the experience of anxiety for elevated depression-high worry participants only, 2 (suppress emotion, control) x 3 (normative, elevated depression-low worry, elevated depression-high worry) x 2 (self-reported emotion after induction and after resting period) repeated-measures ANOVAs were conducted separately for self-reported sadness and anxiety. The same analysis was then conducted on IBI after induction and after resting period as the outcome variable in order to explore the effects of instruction and symptom level on cardiac responding.

H1: Effect of instructions, time, and symptom level on self-reported sadness.

Analysis of self-reported sadness ratings across the two time periods after sadness induction revealed three multivariate outliers. However, removal of those outliers did not impact statistical outcomes, so those outliers were retained in the analysis. This analysis revealed a strong effect of time, $F(1,99) = 149.17, p < .001, \eta^2 = .60$, but no time by condition interaction, $F(1,99) = 1.34, p = .25, \eta^2 = .01$, no time by symptom level interaction, $F(2,99) = .93, p = .40, \eta^2 = .02$, and no time by condition by symptom level interaction, $F(2,99) = 1.49, p = .23, \eta^2 = .03$. There was no main effect of experimental condition, $F(1,99) = .01, p = .93, \eta^2 < .001$, no main effect of symptom level, $F(2,99) = 2.00, p = .14, \eta^2 = .04$, and no condition by symptom level interaction $F(2,99) = .87, p = .42, \eta^2 = .02$. H1 was therefore not supported. All participants showed the same recovery from sadness over time, but there was no difference over time between suppression and

control on sadness for different symptom levels (see Figure 2).

H2: Effect of instructions, time, and symptom level on self reported anxiety.

Analysis of self-reported anxiety ratings across the two time periods after anxiety induction revealed four multivariate outliers. Final analysis revealed a strong effect of time, $F(1,95) = 152.99, p < .001, \eta^2 = .62$, but no time by condition interaction, $F(1,95) = .01, p = .93, \eta^2 < .001$, no time by symptom level interaction, $F(2,95) = .66, p = .52, \eta^2 = .01$, and no time by condition by symptom level interaction, $F(2,95) = .66, p = .52, \eta^2 = .01$. There was no main effect of experimental condition, $F(1,95) = .38, p = .54, \eta^2 = .004$, but there was a main effect of symptom level, $F(2,95) = 7.76, p = .001, \eta^2 = .14$, and a condition by symptom level interaction $F(2,95) = 3.55, p = .03, \eta^2 = .07$. H2 was not supported because suppression had an effect on normative participants only (H2a), but no effects on depressed participants with high or low comorbid worry (H2b; see Figure 3).

Post-hoc examination of the main effects of symptom level on anxiety using Least Square Difference revealed that, across both experimental conditions, normative participants ($N=37$) reported less anxiety than elevated depression-low worry participants ($N=29$), $M_{\text{difference}} = -1.14, SE = .45, p = .01$, and elevated depression-high worry participants ($N=35$), $M_{\text{difference}} = -1.73, SE = .43, p < .001$. However, there was no difference in anxiety between elevated depression-low worry and elevated depression-high worry participants, $M_{\text{difference}} = .59, SE = .46, p = .20$. In sum, elevated depression participants with both high and low worry reported greater levels of anxiety across both experimental conditions than normative participants, but equal levels of anxiety to each other.

To analyze the condition by symptom level interaction for Hypothesis 2b, independent samples t-tests were used to compare level of self-reported anxiety between suppression and control conditions within each symptom level. Self-reported anxiety in this analysis was composed of an average of anxiety rated immediately after the speech and after the post-speech resting period. Post-hoc analysis revealed that among normative participants, those suppressing anxiety ($N=21$) experienced less anxiety than controls ($N=17$), $M_{\text{difference}} = -1.66$, $SE = .21$, $t(36) = -2.68$, $p = .01$. There was no difference in self reported anxiety between suppression ($N=9$) or controls ($N=20$) for elevated depression-low worry participants, $M_{\text{difference}} = -.27$, $SE = .78$, $t(27) = .35$, $p = .73$, and no difference in self reported anxiety between suppression ($N=19$) or controls ($N=17$) elevated depression-high worry participants, $M_{\text{difference}} = .33$, $SE = .60$, $t(34) = .55$, $p = .58$, thus not supporting H2b.

Exploratory analysis: Effect of suppression instructions, time, and symptom level on cardiac responding. Analysis of IBI across the sadness induction and subsequent resting period revealed five multivariate outliers. Final analysis revealed an effect of time, $F(1,76) = 10.29$, $p = .002$, $\eta^2 = .12$, but no time by condition interaction, $F(1,76) < .001$, $p = .99$, $\eta^2 < .001$, no time by symptom level interaction, $F(2,76) = .60$, $p = .55$, $\eta^2 = .02$, and a marginal time by condition by symptom level interaction, $F(2,76) = 2.75$, $p = .07$, $\eta^2 = .07$. There was no main effect of experimental condition, $F(1,76) = .01$, $p = .94$, $\eta^2 < .001$, or symptom level, $F(2,76) = 2.20$, $p = .12$, $\eta^2 = .06$, but there was a condition by symptom level interaction $F(2,76) = 5.61$, $p = .005$, $\eta^2 = .13$.

To analyze the condition by symptom level interaction, independent samples t-tests were used to compare length of IBI in response to sadness induction between

suppression and control conditions within each symptom level. Length of IBI in this analysis was composed of an average of IBI during the film clip and during the post-clip resting period. Post-hoc analysis revealed that among normative participants, those suppressing sadness ($N=12$) experienced longer IBIs than controls ($N=17$), $M_{\text{difference}} = .12$, $SE = .04$, $t(27) = 2.87$, $p = .008$. There was no difference in IBIs between suppression ($N=19$) or controls ($N=8$) for elevated depression-low worry participants, $M_{\text{difference}} = -.06$, $SE = .05$, $t(25) = -1.30$, $p = .21$, or between suppression ($N=12$) or controls ($N=14$) for elevated depression-high worry participants, $M_{\text{difference}} = -.05$, $SE = .04$, $t(24) = -1.23$, $p = .23$ (see Figure 4).

Analysis of IBI across the anxiety induction and subsequent resting period revealed five multivariate outliers. Final analysis revealed a strong effect of time, $F(1,76) = 37.89$, $p < .001$, $\eta^2 = .33$, but no time by condition interaction, $F(1,76) = .65$, $p = .42$, $\eta^2 = .008$, no time by symptom level interaction, $F(2,76) = 2.10$, $p = .13$, $\eta^2 = .05$, and no time by condition by symptom level interaction, $F(2,76) = .27$, $p = .76$, $\eta^2 = .007$. There was no main effect of experimental condition, $F(1,76) = .02$, $p = .88$, $\eta^2 < .001$, a marginal main effect of symptom level, $F(2,76) = 2.56$, $p = .08$, $\eta^2 = .06$, and a condition by symptom level interaction, $F(2,76) = 3.14$, $p = .049$, $\eta^2 = .08$.

To analyze the condition by symptom level interaction, independent samples t -tests were used to compare length of IBI in response to anxiety induction between suppression and control conditions within each symptom level. Length of IBI in this analysis was composed of an average of IBI during the speech and during the post-speech resting period. Post-hoc analysis revealed that among normative participants, those suppressing anxiety ($N=17$) experienced shorter IBIs than controls ($N=12$), $M_{\text{difference}} = -$

.07, $SE = .03$, $t(27) = -2.09$, $p < .05$. There was no difference in IBIs between suppression ($N=9$) and control ($N=14$) for elevated depression-low worry participants, $M_{difference} = .03$, $SE = .04$, $t(21) = .76$, $p = .45$, and no difference between suppression ($N=14$) and control ($N=16$) for elevated depression-high worry participants, $M_{difference} = .05$, $SE = .04$, $t(24) = 1.29$, $p = .21$ (see Figure 5). In sum, normative participants experienced longer IBIs (decreased heart rate) compared to controls when suppressing sadness, but shorter IBIs (increased heart rate) compared to controls when suppressing anxiety. There were no differences in IBIs in response to either induction between suppression and control condition for elevated depression-low worry and elevated depression-high worry participants.

In sum, there was only an effect of time on suppressing sadness and no differences in the effects of suppressing sadness between experimental conditions or symptom levels. However, there was an effect of time and a condition by symptom level interaction for suppressing anxiety. Suppressing anxiety led to decreased anxiety compared to control condition for normative individuals but not for elevated depression-low worry or elevated depression-high worry participants. There was also an effect of time and a condition by symptom interaction for the effects of suppression on IBI. Normative participants experienced longer IBIs (decreased heart rate) compared to controls when suppressing sadness, but shorter IBIs (increased heart rate) compared to controls when suppressing anxiety, but there was no effect of suppression on IBI for elevated depression-low worry or elevated depression-high worry participants.

Secondary analyses. To explore the coherence of self-reported emotion with physiology in this study, Pearson r correlations were calculated between self-reported

sadness and IBI after sadness induction, and self-reported anxiety and IBI after anxiety induction. Cohen's (1988) criteria were used to interpret correlations of 0.1-0.3 as small, 0.3-0.5 as moderate, and 0.5 or greater as large effect sizes. Among control participants, self-reported sadness did not correlate with IBI ($r = -.06$), but self-reported anxiety negatively correlated with IBI ($r = -.15$). To understand the coherence of self-reported emotion with physiology when suppressing emotion, the same analyses were conducted among participants instructed to suppress. Among those participants, self-reported sadness did not correlate with IBI ($r = .07$), but self-reported anxiety positively correlated with IBI ($r = .19$).

Exploratory analysis revealed 7 participants fell in the normative range for depressive symptoms but above clinical cutoff for level of worry. In order to determine the effects of those participants on the primary outcomes in this study, primary analyses (i.e., effects on subjective emotion ratings and physiology after induction and resting period) were repeated with those participants removed.

Analysis of self-reported sadness ratings across the two time periods after sadness induction revealed four multivariate outliers. However, removal of those outliers did not impact statistical outcomes for this analysis excluding 7 normative depression-high worry participants, so those outliers were retained in the analysis. This analysis revealed a strong effect of time, $F(1,92) = 135.85, p < .001, \eta^2 = .60$, but no time by condition interaction, $F(1,92) = .95, p = .33, \eta^2 = .01$, no time by symptom level interaction, $F(2,92) = .64, p = .53, \eta^2 = .01$, and no time by condition by symptom level interaction, $F(2,92) = 1.64, p = .20, \eta^2 = .03$. There was no effect of experimental condition, $F(1,92) = .01, p = .91, \eta^2 < .001$, a marginal effect of symptom level, $F(2,92) = 2.54, p = .09, \eta^2 =$

.05, and no condition by symptom level interaction $F(2,92) = .50, p = .61, \eta^2 = .01$.

Similar effects were seen (H1 was still not supported) when removing 7 participants from the normative group who were also above clinical cutoff for worry. All participants showed the same recovery from sadness over time, but there was no difference over time between suppression and control on sadness for different symptom levels.

Analysis of self-reported anxiety ratings across the two time periods after anxiety induction revealed four multivariate outliers. Final analysis revealed a strong effect of time, $F(1,88) = 170.83, p < .001, \eta^2 = .66$, but no time by condition interaction, $F(1,88) < .001, p = .99, \eta^2 < .001$, no time by symptom level interaction, $F(2,88) = 1.61, p = .21, \eta^2 = .04$, and no time by condition by symptom level interaction, $F(2,88) = 1.26, p = .53, \eta^2 = .02$. There was no main effect of experimental condition, $F(1,88) = .004, p = .95, \eta^2 < .001$, but there was a main effect of symptom level, $F(2,88) = 8.77, p < .001, \eta^2 = .17$, and no condition by symptom level interaction $F(2,88) = 1.66, p = .20, \eta^2 = .04$.

Post-hoc examination of the main effects of symptom level on anxiety using Least Square Difference revealed that, across both experimental conditions, normative participants ($N=30$) reported less anxiety than elevated depression-low worry participants ($N=29$), $M_{\text{difference}} = -1.20, SE = .45, p = .009$, and elevated depression-high worry participants ($N=35$), $M_{\text{difference}} = -1.80, SE = .43, p < .001$. However, there was no difference in anxiety between elevated depression-low worry and elevated depression-high worry participants, $M_{\text{difference}} = -.59, SE = .44, p = .18$. In sum, depressed participants with both high and low worry reported greater levels of anxiety than normative participants, but equal levels of anxiety to each other. After 7 participants in the normative range for depressive symptoms but high in worry were removed from the

analysis, suppression had no effect on anxiety for any symptom level. H2 remained unsupported after removing those participants.

Analysis of IBI across the sadness induction and subsequent resting period revealed five multivariate outliers. Final analysis revealed an effect of time, $F(1,72) = 10.69, p = .002, \eta^2 = .13$, but no time by condition interaction, $F(1,72) = .04, p = .84, \eta^2 = .001$, no time by symptom level interaction, $F(2,72) = .78, p = .46, \eta^2 = .02$, and a marginal time by condition by symptom level interaction, $F(2,72) = 2.47, p = .09, \eta^2 = .06$. There was no main effect of experimental condition, $F(1,72) = .02, p = .90, \eta^2 < .001$, or symptom level, $F(2,72) = 1.60, p = .21, \eta^2 = .04$, but there was a condition by symptom level interaction $F(2,72) = 5.43, p = .006, \eta^2 = .13$.

To analyze the condition by symptom level interaction, independent samples t-tests were used to compare length of IBI in response to sadness induction between suppression and control conditions within each symptom level. Length of IBI in this analysis was composed of an average of IBI during the film clip and during the post-clip resting period. Post-hoc analysis revealed that among normative participants, those suppressing sadness ($N=11$) experienced longer IBIs than controls ($N=14$), $M_{\text{difference}} = .12, SE = .04, t(23) = 2.78, p = .01$. There was no difference in IBIs between suppression ($N=19$) or controls ($N=8$) for elevated depression-low worry participants, $M_{\text{difference}} = -.06, SE = .05, t(25) = -1.30, p = .21$, or between suppression ($N=12$) or controls ($N=14$) for elevated depression-high worry participants, $M_{\text{difference}} = -.05, SE = .04, t(24) = -1.23, p = .23$. Results for IBI in response to sadness induction were therefore unchanged after removing 7 participants with normative levels of depression and high worry.

Analysis of IBI across the anxiety induction and subsequent resting period

revealed five multivariate outliers. Final analysis revealed a strong effect of time, $F(1,71) = 41.46, p < .001, \eta^2 = .37$, but no time by condition interaction, $F(1,71) = .01, p = .92, \eta^2 < .001$, no time by symptom level interaction, $F(2,71) = .71, p = .49, \eta^2 = .02$, and no time by condition by symptom level interaction, $F(2,71) = .59, p = .56, \eta^2 = .02$. There was no main effect of experimental condition, $F(1,71) = .22, p = .64, \eta^2 = .003$, a marginal main effect of symptom level, $F(2,76) = 2.33, p = .10, \eta^2 = .06$, and a condition by symptom level interaction, $F(2,71) = 5.04, p = .009, \eta^2 = .12$.

To analyze the condition by symptom level interaction, independent samples *t*-tests were used to compare length of IBI in response to anxiety induction between suppression and control conditions within each symptom level. Length of IBI in this analysis was composed of an average of IBI during the speech and during the post-speech resting period. Post-hoc analysis revealed that among normative participants, those suppressing anxiety ($N=12$) experienced shorter IBIs than controls ($N=11$), $M_{\text{difference}} = -.10, SE = .04, t(21) = -2.62, p = .02$. There was no difference in IBIs between suppression ($N=8$) and control ($N=15$) for elevated depression-low worry participants, $M_{\text{difference}} = .06, SE = .04, t(21) = 1.50, p = .15$, and no difference between suppression ($N=15$) and control ($N=16$) for elevated depression-high worry participants, $M_{\text{difference}} = .07, SE = .04, t(29) = 1.63, p = .11$. Results for IBI in response to anxiety induction were therefore unchanged after removing 7 participants with normative levels of depression and high worry.

In sum, results were mostly unchanged after removing 7 participants with normative levels of depression and high worry, except that suppression did not have an effect on self-reported anxiety compared to controls for any symptom level (i.e., a

condition by symptom level interaction for anxiety induction did not remain when removing those participants).

Discussion

The effects of individuals' attempts to control or cope with difficult negative emotions has been a primary focus of psychology since its infancy (e.g., Freud, 1946; Mowrer, 1935). Avoiding negative emotions has often been theorized to play a key role in the phenomenology of psychological difficulties. Emotion regulation research in the past 20 years has examined emotion suppression as a way individuals attempt to avoid or dampen emotions in response to distressing events. Many suppression studies have tested the effects of suppression in nonclinical populations and have found detrimental effects of suppressing emotion in a number of domains (e.g., impaired social adjustment, cognitive depletion, increased physiological arousal, delayed recovery from negative affect). Apparent detrimental effects of suppression among nonclinical participants have led researchers to hypothesize that suppressing emotion is particularly detrimental for individuals with clinical disorders (e.g., Aldao et al., 2010; Campbell-Sills et al., 2006a). However, only a few studies have directly tested the detrimental effects of suppression among clinical populations, and even fewer studies have done so by including nonclinical participants against whom to compare findings among clinical participants.

The purpose of this study was to test the effects of suppressing emotion on normative and elevated depression individuals with high and low levels of comorbid clinical worry. This study was proposed to further understand the effects of suppressing different emotions for those with symptoms of psychopathology compared to those without symptoms, and between different types of symptoms (e.g., depression versus

clinical worry). To do so, a mixed factorial pretest-posttest experimental design was used to examine the effects of suppression in a university sample of individuals with no depressive symptoms on the one hand, and individuals showing elevated levels of depressive symptoms (on a commonly used measure of depressive symptoms) who were either high and low in comorbid worry (using a common measure of worry) on the other hand.

Based on research on emotion suppression and emotion regulation with clinical and nonclinical samples, and on research on the effects of suppression for those high versus low in trait NA, it was hypothesized that suppression would reduce the experience of state sadness to the same degree for all participants (H1). Additionally, it was hypothesized that suppression would have no effect on state anxiety compared to controls for normative participants and participants with depression and low comorbid worry (H2a), but that suppression will significantly reduce the level of state anxiety compared to control for those with depression and high comorbid worry (H2b). No specific hypotheses were made for the effect of time or for physiological outcomes of suppressing emotion.

H1 was not supported by results. Participants showed large decreases in sadness after a delay in time following sadness induction, but all participants recovered from sadness in the same way regardless of experimental condition. H2 was also not supported. Normative participants instructed to suppress emotion in response to the anxiety induction endorsed less anxiety following induction and subsequent resting period compared to normative participants instructed to pay attention, but there were no differences in anxiety between suppression and paying attention after anxiety induction

for elevated depression-low worry (H2a) and depressed high-worry participants (H2b).

When exploring the effects of suppression and symptom level on physiological responding, normative participants instructed to suppress their emotion showed decreased cardiac responding (larger IBIs) compared to controls in response to sadness induction. However, normative participants instructed to suppress their emotion showed increased cardiac responding (shorter IBIs) compared to controls in response to anxiety induction. There was no effect of suppression on cardiac responding during either induction for elevated depression participants, regardless of level of comorbid worry.

In summary, attempting to suppress emotion does not appear to have any effect on sadness regardless of symptom level, but does appear to lead to less subjective anxiety for normative participants. Additionally, normative participants appear to experience decreased cardiac responding when suppressing sadness but increased cardiac responding when suppressing anxiety compared to controls. However, the mitigating effects of suppression on anxiety and any variable effects of suppression on cardiac responding disappeared for depressed individuals in our sample regardless of level of comorbid worry. Therefore, emotion suppression may not have the same effect across emotions or for individuals with versus without emotional disorder symptoms. The emotion-specific effects of suppression on subjective experience and cardiac responding that are moderated by level of depression in this study have multiple theoretical and clinical implications to consider.

Effects of Suppression on Different Emotions

Theoretically, these results improve our understanding of emotion suppression across sadness and anxiety. The effect of suppression on anxiety, but not sadness,

highlights the potential of a unique relationship between the process of suppressing emotion on subjective anxiety compared to sadness. It is possible that attempting to suppress sadness may not have detrimental effects for any individuals, as all participants in our study recovered from sadness in the same way. On the other hand, only normative participants were able to mitigate the experience of anxiety. This sheds light on a conceptual difficulty that has been prevalent in emotion regulation research. Previous studies have not been consistent on which emotions they have tested suppression (e.g., general “distress” in Campbell-Sills et al., 2006; disgust in Gross, 1998; anxiety in Hofmann et al., 2009; panic in Levitt et al., 2004; also see Aldao, 2013 for a review). Despite finding effects on single emotion inductions, most studies have tested the effects of suppression on a single emotion and then drawn conclusions about what their results imply for the effects of suppression on emotion in general. Findings from this study support the notion that the effects of suppressing emotion may depend on the emotion one tries to suppress, and that anxiety is particularly sensitive to suppression because suppressing anxiety showed effects on multiple modes of measurement.

The ability for nonclinical individuals to actually experience less anxiety when suppressing is consistent with previous research (e.g., Dunn, Billotti, Murphy, & Dalgleish, 2009). This ability raises the potential for suppressing anxiety to be adaptive in the short-term for individuals without depression or pathological worry. If these individuals are able to successfully experience less anxiety during difficult situations, they may be less encumbered by negative emotion and better able to successfully complete emotionally distressing tasks (e.g., public speaking). The ability to be relatively “freed” from anxiety and better able to complete tasks may actually contribute to more

healthy psychological outcomes because being able to address the demands of one's environment despite experiencing anxiety should lead to increased goal completion and increased opportunities for social support (Bonanno et al., 2004; Lazarus & Folkman, 1984). On the other hand, not being able to mitigate anxiety may increase the chances that individuals engage in avoidance and other maladaptive strategies to deal with difficult situations (Allen, McHugh, & Barlow, 2008; Carver & Connor-Smith, 2010; Foa, Hembree, & Rothbaum, 2007). Future research is needed to further elucidate what factors lead some individuals to successfully mitigate their anxiety, while others are unable to do so (e.g., higher levels of arousal when experiencing affect, less skill in successfully mitigating anxiety, less willingness to attempt to voluntarily regulate anxiety, etc.).

The differences in the effects of suppression for sadness and anxiety among normative participants in our study may arise because of specific differences in the function of each emotional response. First, sadness occurs with past-oriented appraisals of loss, whereas anxiety occurs with future-oriented appraisals of threat or catastrophe (Ekman & Friesen, 1972; Levenson, 1999). Appraisals of loss focus on past situations that an individual already has evidence of occurrence (e.g., loss of a family member), so participants at any level of depressive or worry symptoms (including no symptoms) may respond to sadness in the same way regardless of emotion regulation attempts (e.g., suppressing versus paying attention) because every individual has evidence that a loss has already occurred.

On the other hand, future-oriented appraisals of threat are based on anticipated events that have not yet occurred and are therefore speculative (Beck & Emery, 2005).

The perceived need and efforts taken to prepare coping resources for something that has not yet occurred may be greater due to greater uncertainty and anticipation of threat when anxious (Levenson, 1999). Therefore, individuals may be more responsive to suppressed anxiety because greater anticipation of negative consequences of future events (e.g., what others will think about one's public speaking ability) may yield typically greater levels of emotion on which to utilize ER strategies like suppression (e.g., there is "something" to suppress or accept; Shallcross et al., 2010). The unique effects of suppression on anxiety in the current study are consistent with multiple previous study findings suggesting that attempts to regulate anxiety and fear responding have specific effects on state emotional experience and longer-term mood compared to other emotions (Boland & Papa, under review; Dunn et al., 2009; Shallcross, Ford, Floerke, & Mauss, 2013).

Different effects of suppression on sadness versus anxiety in our study could also be due to long-held findings that each of these emotions entail differences in levels of physiological reactivity. Sadness has been found to dampen cardiac responding and other physiological reactions whereas anxious/fearful responses elevate of these reactions (e.g., Gray, 1994; Mauss et al., 2004; Nyklíček et al., 1997; Pignatiello, et al., 1989).

Decreased levels of arousal in the case of sadness may therefore yield decreased experience of affect that one has to suppress compared to other emotions (i.e., there is less emotion to suppress in the first place) and may make recovery from sadness equally possible for all participants. Decreased levels of arousal may have also been why normative participants suppressing during sadness induction experienced even less physiological arousal (i.e., larger IBIs, decreased heart rate) than controls. This conclusion might be viewed as tentative because there was no correlation between

sadness and physiological response during sadness induction in this study. However, previous research has uncovered variable effects between self-reported emotion and physiological arousal in response to emotion induction (Gross, 1998)

On the other hand, normative participants showed a mitigating effect of suppression on the subjective experience of anxiety, but they also showed increased cardiac responding (evidenced by decreased interbeat intervals) in response to anxiety induction. Increased physiological activation when suppressing is consistent with previous studies examining suppression in response to inductions of high-arousal negative affect (e.g., distress in Campbell-Sills, 2006b; disgust in Gross, 1998; anger in Mauss, Evers, Wilhelm, & Gross, 2006). Because anxiety and other high arousal negative emotions have been found to lead to elevations in cardiac responding and other physiological reactions (Mauss et al., 2004; Nyklíček et al., 1997), greater levels of effort may be required to regulate high-arousal negative emotions (Mauss et al., 2006; Mauss & Gross, 2004). The result is the potential for normative participants to suppress the experience of anxiety, but with greater physiological effort than when suppressing the experience of sadness. This possibility is supported by the finding that suppressing emotion during anxiety induction was positively correlated with length of IBI (and thus negatively correlated with cardiac responding). Therefore, differences in physiological activation for each type of emotion may account for the different effects of suppression on self-reported emotion and cardiac responding among normative participants (Gross, 1998).

Suppression's effect on physiological responding is thought to play a role in a number of physical health problems, including hypertension (Roter & Ewart, 1992),

coronary artery disease (CAD; Friedman & Booth-Kewley, 1987), and decreased immune functioning (Kiecolt-Glaser & Glaser, 1991). Gross (1998) proposed that individual instances of suppressing may not take a significant toll on physical health, but that cumulative effects of suppressing over time could account for the relationship between suppression and physical health outcomes. Results of this study suggest that the role of suppression on physical outcomes may depend on the emotion one attempts to suppress. That is, suppressing sadness may not be physically detrimental or contribute to cumulative effects on negative health outcomes, but suppressing anxiety and other high arousal negative emotions may be detrimental for those at risk for hypertension, CAD, and decreased immune functioning.

Effects of Suppression on Clinical Symptoms

Results of this study improve our clinical understanding of suppressing sadness and anxiety for those with different levels of psychopathology. Symptom level did not moderate the effects of suppression on the experience of sadness; all participants recovered from sadness in the same way whether suppressing emotion or simply paying attention (control condition). However, symptom level did moderate the effect of suppression on anxiety. The effect of suppression on anxiety for normative individuals did not hold for depressed individuals regardless of level of comorbid worry. This lack of effect reveals that those with elevated levels of depression may be particularly ineffective at mitigating the experience of state anxiety immediately after it is induced.

It is possible, however, that those with elevated depression-high worry were unable to mitigate anxiety because their baseline anxiety was higher than the normative or elevated depression-low worry symptom groups. However, the inability of elevated

depression-high worry participants to mitigate anxiety is consistent with the lack of effect on anxiety for those with elevated depression-low worry, so the inability to mitigate anxiety using suppression during anxiety induction was likely accounted for by level of depressive symptoms as opposed to level of comorbid worry. Furthermore, lack of effect of suppression for elevated depression-high worry is consistent with the pattern of results seen for IBI in response to anxiety induction. Therefore, the lack of effect of suppression on self-reported anxiety for elevated depression-high worry participants is likely not due to elevations in anxiety for that group at baseline.

It is also possible that differences were not seen between symptom levels because there were low numbers of participants in certain subgroups (e.g., $N=9$ for elevated depression-low worry participants instructed to suppress during anxiety induction). Low numbers of participants in certain subgroups does raise concerns for the ability of statistical analyses to uncover existing effects. However, results of this study showing lack of differences between levels of comorbid worry are consistent with previous research showing that individuals with both depression and anxiety symptoms endorsed frequently suppressing their emotions (Aldao et al., 2010; Campbell-Sills et al., 2006a) and were unable to effectively dampen anxiety in response to experimental manipulation (e.g., Feldner et al., 2006; Liverant et al., 2008). Therefore, lack of differences in self-reported emotion and physiology between levels of comorbid worry in this study is still supported.

Additionally, differences in anxiety may not have existed between comorbid worry groups because those instructed to suppress their emotions in response to anxiety induction as their second induction endorsed suppressing to a marginally greater degree

than control participants. However, those instructed to suppress in response to anxiety induction as their first induction endorsed attempting to suppress to a greater degree than control participants. It is possible this difference in attempted suppression is due to increases in control participants reported attempted suppression at that time point. That is, there may be an increased chance that control participants will call on suppression for a highly distressing anxiety induction because using suppression was suggested during their first induction. Despite the difference in self-reported attempted suppression for order of anxiety induction, effects of suppression on anxiety were still seen for normative participants and were correlated with theoretically consistent IBI results (i.e., seeing increased cardiac responding in response to suppressing high arousal negative emotion; Gross, 1998, Mauss et al., 2006). Additionally, effects of suppression showed a consistent pattern on IBI in response to both emotion inductions for normative, but not elevated depression participants, regardless of order of instruction or induction (i.e., there were no order effects). Therefore, participant attempts to suppress anxiety still appeared to yield effects on subjective anxiety and cardiac responding in response to anxiety induction even when participants were instructed to suppress anxiety as the second induction.

The effects of suppression on anxiety may not have been moderated by level of worry because depressed participants entering the study may have been unable to successfully suppress anxiety simply by virtue of being depressed. That is, depression is known to be associated with many maladaptive affective processes that may interfere with the ability to modulate anxiety in a laboratory setting or in everyday life, including attentional bias toward negative information, increased negative emotional arousal and

decreased positive emotional arousal, rumination and perseveration on negative content, and negative reactivity to emotions, among others (Aldao et al., 2010; Barlow, 2008; Mennin et al., 2007). It is possible that engaging in any number of these processes simply due to experiencing depressive symptoms inhibits individuals' ability to effectively suppress anxiety. This is difficult to say definitively from the current study, because it did not include a comparison group of individuals high in worry but low in depressive symptoms.

Results of the current study do provide some understanding of the correlation between suppressing emotion and depression and anxiety pathology found in previous studies (e.g., Aldao et al., 2010; Campbell-Sills et al., 2006a). The fact that suppressing led depressed participants with any level of comorbid worry to not be able to mitigate induced negative emotion suggests that suppression's relationship to those disorders may be accounted for by an immediate and causal relationship. That is, failed attempts to suppress anxiety by depressed participants in this study suggest that ineffective attempts to suppress negative emotion may leave depressed individuals with increased levels of negative emotion they must cope with. That increased negative emotion could contribute to a cascade of difficulties that in-turn exacerbate depressive symptoms (e.g., increased difficulty regulating negative affect, decreased experience of positive affect, interpersonal difficulties). More research is needed to fully understand the nature and direction of that causal relationship and the reasons that suppression may be ineffective for those who experience depression and anxiety symptoms compared to those who do not.

Results of this study also contribute to our diagnostic understanding of suppression between emotions for those with symptoms of clinical disorders. Ineffective

attempts to dampen anxiety in general or at highly distressing times may define a specific syndrome (e.g., “anxiety dysregulation”) that might indicate depression and/or clinical worry and GAD for clinicians assessing clients for possible psychopathology. The lack of disorder-specific effects found for either emotion in the current study supports some previous calls to combine major depression and generalized anxiety disorder into a single class of “emotional disorders” (Watson, 2005). However, additional research is needed to improve our knowledge of disorder specific effects of emotion regulation that may further aid in defining pathological syndromes and diagnosing classes of psychopathology (e.g., emotional disorders like depression and GAD versus psychotic disorders like schizophrenia and delusional disorder).

Previous research suggests there are shared factors between depression and clinical worry that may also explain the lack of differences in the effects of suppression between those sets of symptoms. First, both conditions share high levels of negative emotional arousal and study samples composed of both depression and anxiety pathology in previous studies have endorsed using suppression often as a “go-to” strategy for regulating their high arousal and negative thought patterns. Furthermore, those studies did not mention differences between depression and anxiety pathology (Aldao et al., 2010; Campbell-Sills et al., 2006a). In fact, evidence for multiple overlapping components of affective responding between GAD (as typified by excessive worry; Fresco et al., 2003; Mennin et al., 2007) and major depression outlined from the Mennin et al. (2009, 2007) and Aldao et al. (2010) studies, may explain the lack of difference in the effects of suppression in this study. That is, the effects of attempts to suppress emotion may be related to an emotional response factor(s) that overlaps mood and

anxiety pathology as opposed to a factor that would indicate unique differences between each type of pathology. Again, this conclusion should be viewed as highly tentative because the current study did not examine the unique moderating effects of high worry pathology or GAD without comorbid depressive symptoms. Future studies could investigate this possibility further if they could attain subsamples with high levels of depressive symptoms with no anxiety symptoms and high levels of anxiety symptoms with no depressive symptoms in the same study.

The high comorbidity between depression and anxiety pathology may also underlie a separate shared variable we have yet to uncover that may explain similar effects of suppression attempts between disorders. Shared effects of suppression may be related to one of any number of important factors that contributes to the high comorbidity observed between mood and anxiety disorders. This is plausible because both types of psychopathology share similar appraisals related to negative affect (Beck & Emery, 2005), attempts to avoid fearful affect (Allen, McHugh, & Barlow, 2008; Campbell-Sills et al, 2006a), and anxiety about their negative experiences in general and specific symptoms they experience (Liverant et al., 2008). Shared factors like these may lead to shared regulatory effects of suppression on anxiety across multiple symptom presentations.

Given these results, it is possible that different effects might be seen across symptom presentations for other ER strategies (e.g., reappraisal, acceptance) not tested in this study. Other ER strategies target different psychological processes than suppressing emotional expression (e.g., reappraisal involves manipulating internal thoughts, while acceptance involves relating to emotional states) and have been found to yield different

effects on measures of effort and psychophysiology (Mauss et al., 2006). Different effects on experience and physiology may therefore be seen across depression and comorbid worry for other ER strategies as well. Different symptom presentations also entail different behavioral, emotional, and cognitive correlates, so different effects of suppression and other emotion regulation strategies may be observed between other sets of symptoms not tested in this study (e.g., depressive versus panic symptoms; depressive versus traumatic stress symptoms).

Another interesting consideration is the effect of suppression over time. This study found strong effects of time for sadness, anxiety, and cardiac responding, but no interaction of time with condition and/or symptom level for any analysis. Since suppression has been correlated with multiple negative outcomes and psychopathology, suppression may result in different changes in outcomes over periods of time longer than was possible to observe after the 2-minute resting period in this study. It may be possible to observe decreases in anxiety compared to control condition or even rebound effects of increased anxiety after longer periods of minutes to hours, for example. Additionally, other psychological processes may occur that account for the positive correlation between depression and anxiety on the one hand, and suppression or avoidance of negative affect on the other (Campbell-Sills et al., 2006a; Hayes et al., 2004; Roemer et al., 2005). For instance, depressed and anxious individuals may have a difficult time enlisting suppression for situations in which effective suppression is actually contextually appropriate (Bonanno et al., 2004; Hayes et al., 1999). They may therefore miss opportunities to respond to situations in ways that lead to adaptive outcomes (e.g., not directing high levels of anger to a stranger in public) or may even create maladaptive

outcomes for themselves (e.g., directing high levels of anger at a new potential friend and then enlisting negative social feedback). In other words, if those individuals have difficulty suppressing effectively, they might not be able to flexibly call on suppression for those situations in which it may actually be adaptive (Bonanno & Burton, 2013).

Suppression may also contribute to psychological problems in a more long-term and indirect sense than may be observed in its immediate effects on negative emotion and physiology. Suppression may allow individuals to avoid social and emotional situations that first cued their anxiety (e.g., being interviewed) and thus lose opportunities to resolve those situations (Llewellyn et al., 2013). That is, suppression leads to a lack of approach to private cognitive content or overt content induced by aversive situations. Approach to such content is considered necessary for engaging in adaptive responses discussed in a number of psychotherapies, such as cognitive reappraisal emphasized in cognitive therapies, exposing oneself to aversive stimuli in exposure therapies (Allen et al., 2008; Foa et al., 2007), accepting internal responses (e.g., emotions) to allow living toward personal values as emphasized in ACT (Hayes et al., 1999), and awareness and mindfulness for promoting regulated emotional responses in DBT (Fruzzetti et al., 2005) and other mindfulness-based therapies (e.g., Mindfulness-based Cognitive Therapy for Depression; Segal, Williams, & Teasdale, 2013).

In a practical sense, results of this study also may be informative for mental health clinicians' understanding of suppressing emotions across negative emotions to inform diagnostics and which interventions may be most effective to maximize mental and physical health for each unique client (in line with Gordon Paul's "clinical question," 1954). The difficulty modulating anxiety may help clinicians recognize the potential that

a given individual is experiencing depression and/or clinical worry and GAD (e.g., “During those times you try to suppress your anxiety, is it effective at not feeling anxious, or does the anxiety remain?”). Clinicians using biofeedback may even be able to directly observe the effects of suppression on heart rate to observe how suppressing anxiety might affect their clients.

Clinicians might also use knowledge of suppression across emotions and symptom levels to inform which skills they might practice with clients. Different emotions are cued by different contexts (Levenson, 1999), so the context will correspond with the emotion one attempts to suppress (e.g., sadness occurs in contexts of loss; anxiety occurs in contexts of anticipated threat or catastrophe) and will inform which skills might be helpful for depressed clients under certain situations. In regards to suppressing sadness, clinicians may not need to instruct clients on any particular action in those contexts because the effects of suppression on sadness appear to be no different for those with psychopathology than for nonclinical participants. Clinicians may simply instruct clients 1) to be “easy” on themselves and to regulate sadness the way they might naturally do so in the moment in order to minimize psychological effort and self-critical meta-cognitive statements often characteristic of depression (e.g., “I couldn’t even control my emotions right.”), 2) while attempting to engage in valued or reinforcing activities that may give the client a sense of purpose and enhance self-concept (Hayes, Strohsahl, & Wilson, 1999; Papa, Sewell, Garrison-Diehn, & Rummell, 2013).

The mitigating effects of suppression on anxiety that appears to be negated close to the moment an emotion is induced for those with high levels of depression and worry symptoms may therefore warrant a different approach by clinicians when discussing how

clients attempt to mitigate their anxiety in particular. Clinicians may benefit clients by helping them learn new strategies that can act as alternatives to suppressing anxiety. For example, clients may benefit from many techniques for relating to thoughts and regulating anxious affect that have been found to be effective in cognitive behavioral and third wave therapies (e.g., cognitive reappraisal, acceptance, mindfulness, problem-solving). These therapeutic techniques are not new, but a focus on when to use them (e.g., in the case of anxiety but not sadness for depressed individuals) could improve therapeutic interventions.

A focus on what contexts suppression may be adaptive versus maladaptive for is supported by recent research demonstrating the variable impact of context on the effects of emotion regulation strategies, and how using suppression inflexibly or inappropriately in the “wrong” contexts has been shown to predict increased levels of psychopathology (see Bonanno & Burton, 2013 for a full review). Clinicians may therefore promote more flexibility regarding the use of suppression across contexts to improve client functioning. For example, they might instruct clients to relate to post-rejection sadness how they normally would while engaging in self-enhancing and valued activities, but to reappraise the potential for catastrophe or accept their anxiety in order to promote approach behaviors and active problem-solving when situations are within their control. These suggestions should currently be viewed as tentative, but continued research in this area should allow increasing basic knowledge of suppression and other ER strategies that can inform diagnostics and treatment targeting.

Strengths, Limitations, and Future Directions

This study had a number of limitations. First, the proposed method of using

comorbidity (e.g., clinical versus nonclinical levels of worry among depressed participants) contributed to not being able to observe effects of suppression unique to high worry or anxiety pathology. Recruiting participants with symptoms of depression or clinical worry only would provide a sample that might allow for “cleaner” comparisons between types of symptoms. However, feasibility issues (e.g., the high rate of comorbidity of depression and anxiety pathology) make such direct between-group comparisons highly difficult.

A related difficulty was attaining a sample that was moderate to severe in depressive symptoms (those who scored 20 or above on the BDI-II) who were also low in comorbid worry. Results were initially analyzed by excluding those demonstrating mild depressive symptoms in order to get further discrimination between normative and more highly depressed individuals (that analysis was completed separately and is not reported here). That analysis revealed distinct differences in suppressing anxiety between the elevated depression-low worry and elevated depression-high worry participants that more closely supported H2, but the low sample size of the elevated depression-low worry subgroup was too unstable to allow for any adequate conclusions about the effects of suppression for that subgroup (N=5 for those suppressing anxiety, N=6 for those paying attention). Low N values for the elevated depression-low worry subgroup in that separate analysis forced the inclusion of mildly depressed participants (those who scored from 14-20 on the BDI-II) into the main analysis (reported here) in order to maximize statistical power. However, inclusion of participants scoring in the mildly depressed range potentially washed out observable effects of suppression manipulation on more strongly depressed individuals (i.e., those moderate to severe in depressive symptoms). The

number of participants scoring in the moderate to severely depressed range with levels of worry below clinical cutoff ($N = 11/105$) in this study sample illustrates the difficulty in attaining a more clinically diverse sample.

Additionally, it was not possible to examine the unique effects of high worry symptoms independent of depressive symptoms. The study sample was only composed of 7 participants who scored below clinical cutoff for depressive symptoms but above clinical cutoff for worry. Seven participants made it impossible to include a separate normative-high worry cell in any analyses. However, as discussed, recruiting a normative-high worry cell was not intended in the study design because recruiting a sample of enough participants with distinctly depressive symptoms *or* anxiety disorder symptoms may take multiple years and hundreds of participants. This feasibility issue in studying distinct factors between emotional disorders inhibited the ability to draw conclusions about the unique moderating effects of high worry symptoms. The difficulty obtaining a subsample that is high in anxiety pathology but low in depressive symptoms or high in depressive symptoms but low in anxiety pathology may be why there are not more studies directly comparing the effects of any psychological processes (including emotion regulation strategies) between mood and anxiety disorders. This raises an interesting but unfortunate problem for research attempting to examine shared and unique factors of emotional disorders in any domain of psychological functioning, and may be a key reason why the high comorbidity rate between depression and anxiety pathology remains relatively unexplained.

In order to observe the effects that normative-high worry participants had on the results of this study, primary analyses were run again with those participants removed

from the normative symptom level group. That analysis mostly revealed similar results as when those participants are included, but with normative participants no longer experiencing less anxiety when instructed to suppress compared to control condition. This change may be due to a true effect those individuals high in worry had on the normative group. However, it may also be due to decreases in statistical power that comes with removing multiple participants ($N=38$ for the normative group in the initial analysis versus $N=31$ after removing 7 participants). Despite changes in results when removing those 7 participants, the initial analysis that included them is still seen as valid for multiple reasons. First, the normative group with those 7 participants included showed lower levels of anxiety at baseline than the elevated depressed-high worry group and no differences in anxiety at baseline compared to the elevated depression-low worry group. Showing lower anxiety than the study's high anxiety pathology group but the same level of anxiety as another low anxiety pathology group demonstrates that the normative symptom group is a valid representation of normative individuals even when those 7 participants are included. Second, any elevated scores that may significantly impact group scores on state emotional responding would have been found and removed when removing multivariate outliers from the analysis. Individual participant scores that might heavily skew results were already removed when the initial analysis was completed.

This study's short-term experimental design also poses a potential limitation. Although a short-term design allows for immediate observation of effects of suppression on multiple emotions, it does not allow observation of long term effects of suppression. This makes it difficult to explore potential mediating variables (e.g., the effects of time

and potential longer-term rebound in levels of negative emotion occurring as a result of suppressing) that may contribute to a set of factors that defines relationships between suppression and psychopathology found in previous correlational studies. Future experimental studies might examine the effects of suppression for depressed participants by measuring self-reported emotion and physiology after a longer period of time (e.g., 30 minutes after induction) or by adding a longitudinal component to see how other factors (e.g., trait variables and response to suppressing emotion) might predict the differential development of mood versus anxiety pathology (cf. Bonanno et al., 2004; Westphal et al., 2010).

The nature of emotion inductions used in the current study raise other potential limitations. First, sadness induction induced lower levels of sadness than the anxiety induction did for anxiety. Relatively lower levels of sadness induced raises the possibility that effects of suppression were not seen for sadness because participants may not have experienced as much sadness on which to bring suppression to bear. Despite this possibility, the unique effects of suppressing anxiety is consistent with previous research showing that suppression of negative emotion is associated with more anxious responding than other emotions (Boland & Papa, under review; Dunn et al., 2009; Levitt et al., 2004). Future research will need to further explore this possibility.

Alternatively, it is possible that the difference in presentation of each induction accounts for different effects seen for suppression. Film clips typically used in emotion regulation research may allow for more passive participation from participants and therefore provide the opportunity to engage in suppression while viewing them, whereas giving a speech may require more active participation and thus dampen participant

attempts to suppress due to multitasking demands. This study induced two emotions using two different types of inductions (i.e., a film clip versus a speech task). Different inductions in this study were purposely chosen to maximize their strength in order to increase external validity and capture more true effects of suppressing emotions induced by actual real-life events in similar contexts (e.g., times of interpersonal loss, anticipating judgments from others). Additionally, previous research has reliably induced sadness with film clips and anxiety with speeches, but not vice versa. However, the difference in presentation of induction could have resulted in reported decreased attempts to suppress for those instructed to suppress anxiety compared those instructed to suppress sadness. It may have been more difficult to suppress emotion while actively giving a speech than while watching a film clip. Although this and previous ER studies have found that impromptu speeches provide a very strong anxiety induction on which to instruct participants to suppress (among other emotion regulation strategies; Hofmann et al., 2008; Mauss et al., 2011), impromptu speeches may create an additional demand characteristic that makes it difficult for participants to fully engage in suppression compared to more passive tasks like viewing film clips. If methods for strongly inducing multiple emotions in the same way can be discovered and validated (e.g., film clips that also strongly induce anxiety), future studies may find the ability to address this issue by instructing participants to suppress their emotions in response to similar types of inductions with similar degrees of strength.

This study had a number of methodological strengths. First, study procedures and suppression manipulation were consistent with previous basic and clinical emotion suppression research. Suppression instructions were chosen based on previous

suppression research finding that suppressing expression of emotion was more effective for dampening emotion than suppressing the internal experience of emotion (Boland & Papa, under review). Additionally, suppressing expression was found to be methodologically “cleaner” in that study compared to suppressing emotional experience, so suppressing expression was used for this study in order to have a strong and methodologically sound method for instructing suppression.

Suppression instructions were also purposely worded so that there were no particular demand characteristics for each induced emotion. That is, no specific emotion was suggested in suppression or control instructions. Participants were instructed to suppress the expression or experience of “any emotions” that may arise, as opposed to any specific emotion being suggested. Additionally, the sadness and anxiety inductions were the same length, ensuring that differences in effects of induction and participants attempts to suppress were not due to the amount of time each emotion was induced or suppressed.

This study also examined multiple factors associated with suppressing emotions (e.g., the effects of suppression on multiple emotions, the moderating effects of symptom level, the effect of time on each type of suppression). The inclusion of multiple emotions, physiology, and symptom level in particular allowed this study to uncover emotion- and symptom- specific effects (or lack thereof) of suppression that have only been minimally examined in emotion regulation research to date. Although the presence of multiple factors or “moving pieces” may make results seem complex, this actually fits well with newly proposed efforts in emotion regulation research to specify multiple factors associated with each regulation strategy (e.g., suppression, cognitive reappraisal,

emotional acceptance, rumination, etc.) to provide more valid and comprehensive analogs of how the effects of these strategies are influenced by context (Aldao, 2013; Bonanno & Burton, 2013).

Lastly, this study's many components are informative in that they comprise an effort to understand exactly what suppression "is." Suppression has long been theorized to be an important process in the development and maintenance of psychopathology. Although there have been many informative studies in suppression research, there has been a dearth of research to understand the differential effects of suppression for individuals with clinical symptoms versus those without. This study's examination of multiple effects of suppressing emotions scratches the surface of understanding the role of suppression as an emotion regulatory process in psychopathology. Future studies should continue to investigate the role of emotion regulation strategies across emotions and psychopathology in order to attain further conceptual clarity and enhance discriminant validity for researchers and clinicians.

Implications

Suppression of negative emotion has been seen as a process contributing to the development and maintenance of psychopathology (Aldao et al., 2010; Bonanno et al., 2004; Campbell-Sills et al., 2006a). First, this study contributes to conceptual clarity concerning suppression as an emotion regulatory process. Many studies have assumed that suppression generally has the same effects across emotions, but this study found that suppression had different effects on anxiety compared to sadness. These effects may indicate that suppression does not have universal subjective and physiological effects on emotion, and that effects of suppression may depend on the emotion suppressed.

Results of this study are supported by previous research showing similar unique effects of suppression on anxiety compared to sadness (Boland and Papa, under review). The observed variable effects of suppression across emotions may indicate a partial solution to recent calls for greater efforts to account for context in research on emotion regulation strategies (Aldao, 2013, Bonanno et al., 2004). Emotions have long been hypothesized to be contextually dependent responses to environment contexts (e.g., sadness is a response to loss, anxiety is a response to perceived threat; Ekman & Friesen, 1972; Levenson, 1999), so testing the effects of ER strategies on different emotions in the same study may provide a simple yet highly ecologically valid proxy for understanding the effects of regulating emotions across different contexts. Continuing to test multiple emotions in the same study could help us understand how context may shape the effects of ER strategies.

Second, when examining contributions of emotion regulation in psychopathology, this study contributes to efforts to understand diagnostic overlap in emotional processes between depressive and anxious responding (Burklund et al., 2014) and how hypothesized unique and shared factors related to emotion (e.g., effects of regulating emotion, typical levels of trait negative affect; e.g., Aldao et al., 2010; Mennin et al., 2007) contribute to that relationship. This study's findings suggest that the effects of suppressing anxiety are similar for depressed individuals regardless of levels of comorbid clinical worry. Therefore, how individuals respond to suppression may not depend on specific forms of psychopathology (in this case, depression and comorbid worry), but simply on experiencing high levels of psychopathology at all.

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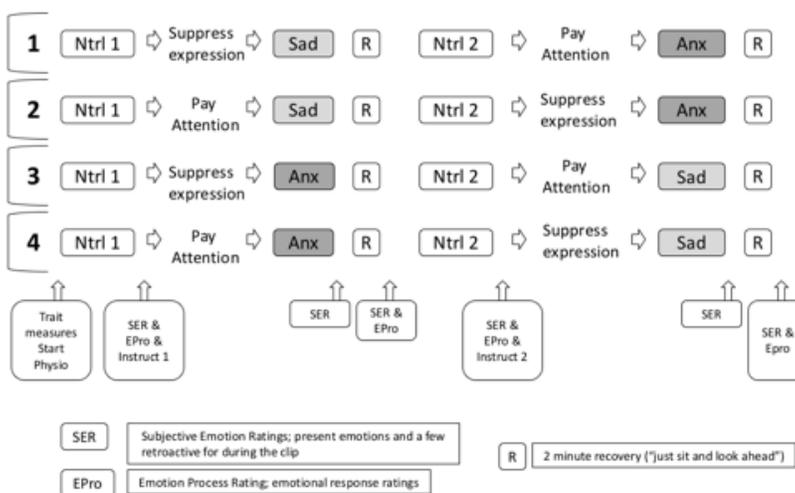


Figure 1. Visual depiction of study procedures, with four counterbalanced conditions.

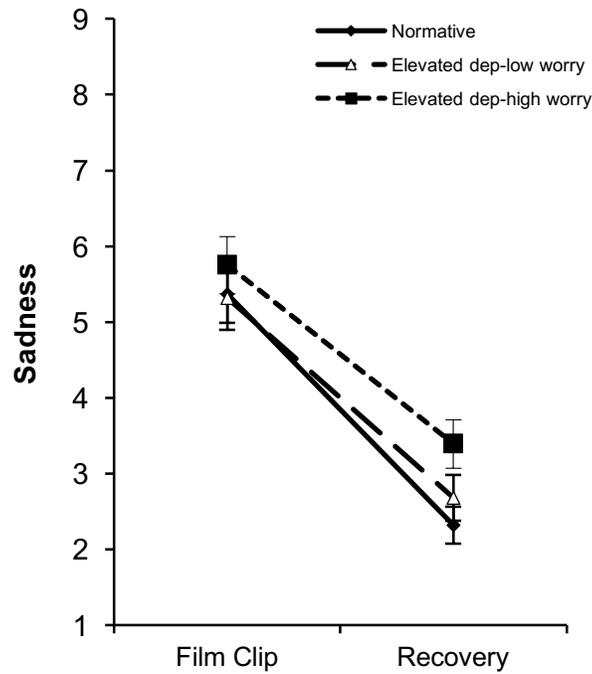


Figure 2. Sadness intensity, presented in mean of self-reported sadness in response to the film clip and in response to the post-clip recovery period for each symptom level (normative, elevated depression-low worry, elevated depression-high worry). Error bars represent standard error of means.

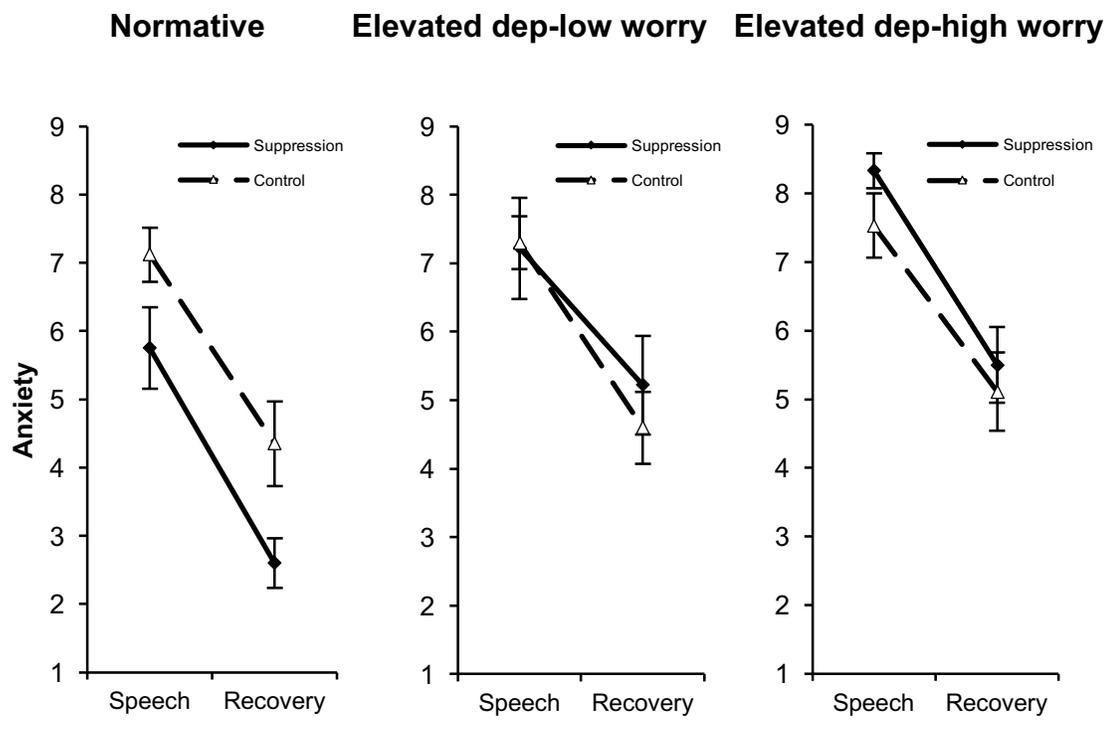


Figure 3. Anxiety experienced in response to the impromptu speech task and in response to the post-speech recovery period for each (suppression versus control) and symptom level (normative, elevated depression-low worry, elevated depression-high worry). Error bars represent standard error of means.

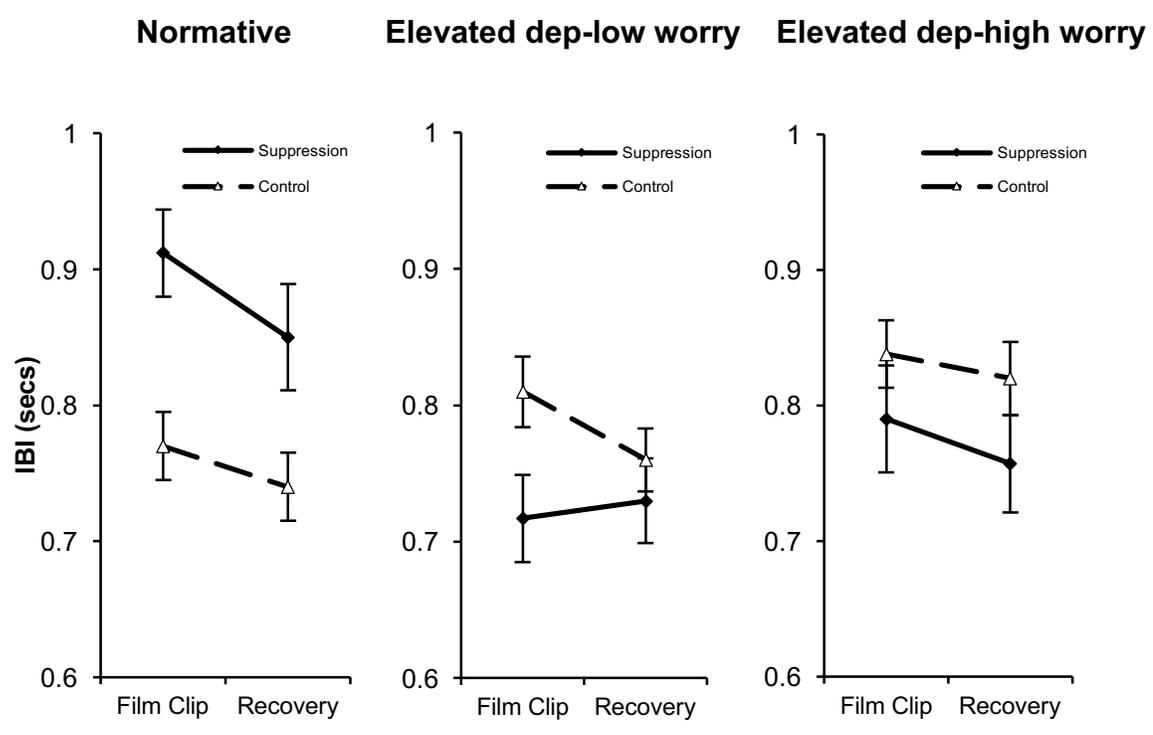


Figure 4. Cardiac interbeat intervals (IBI), presented in mean IBI during the film clip and during the post-clip recovery period for each experimental group (suppression versus control) and symptom level (normative, elevated depression-low worry, elevated depression-high worry). Error bars represent standard error of means.

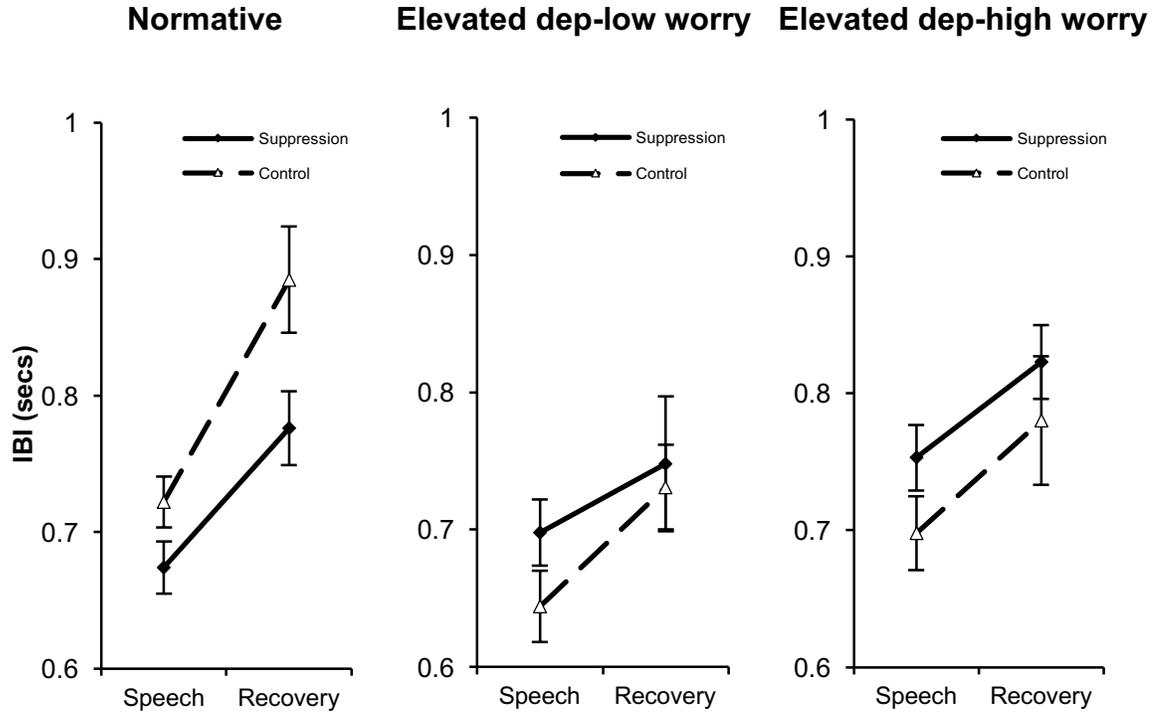


Figure 5. Cardiac interbeat intervals (IBI), presented in mean IBI during the impromptu speech task and during the post-speech recovery period for each experimental group (suppression versus control) and symptom level (normative, elevated depression-low worry, elevated depression-high worry). Error bars represent standard error of means.