Bates College

SCARAB

Honors Theses

Capstone Projects

5-2017

Subclinical Depression, Anxiety, and Alexithymia: Implications for Implicit and Explicit Emotion Regulation

William N. Koller Bates College, wkoller@bates.edu

Follow this and additional works at: https://scarab.bates.edu/honorstheses

Recommended Citation

Koller, William N., "Subclinical Depression, Anxiety, and Alexithymia: Implications for Implicit and Explicit Emotion Regulation" (2017). *Honors Theses*. 199. https://scarab.bates.edu/honorstheses/199

This Open Access is brought to you for free and open access by the Capstone Projects at SCARAB. It has been accepted for inclusion in Honors Theses by an authorized administrator of SCARAB. For more information, please contact batesscarab@bates.edu.

Subclinical Depression, Anxiety, and Alexithymia: Implications for Implicit and Explicit Emotion Regulation

An Empirical Research Honors Thesis

Presented to The Faculty of the Department of Psychology Bates College

In partial fulfillment of the requirement for the Degree of Bachelor of Arts

By

William N. Koller

Lewiston, Maine March 24, 2017

Acknowledgments

First and foremost, I would like to thank my advisor, Dr. Sara Masland, for her support in this endeavor. I would also like to thank Dr. Kate White for her willingness to take me under her wing and answer endless questions about heart rate variability, as well as Brian Pfohl for his bottomless knowledge of statistics and his mastery of Excel. Finally, a special thank you to Azure Reid-Russell for her undying support, both emotional and statistical, and to my parents, Sharon and Steve Koller for feeding me, housing me, and sending me an edible arrangement in my darkest hour.

Table of Contents

Abstract	5
General Introduction	6
Alexithymia Alexithymia and Psychopathology Measurement of Alexithymia Present Studies	7 8 12 14
Study 1 Introduction	16
Implicit Emotion Regulation Hypotheses	16 19
Study 1 Method	20
Participants Materials Procedure	20 20 23
Study 1 Results	24
Screening Variables of Interest ECT Depression, Anxiety, and Alexithymia	24 24 25 27
Study 1 Discussion	29
Behavioral Data Implicit Emotion Regulation Strengths and Limitations Conclusion and Future Directions	29 33 35 35
Study 2 Introduction	38
Explicit Emotion Regulation Reappraisal Physiological Profiles of Alexithymia Heart Rate Variability Hypotheses	38 39 42 45 47
Study 2 Method	49
Participants Materials Procedure	49 49 51

Study 2 Results	52
Screening Variables of Interest Manipulation Checks Depression, Anxiety, and Alexithymia	52 53 53 55
Study 2 Discussion	65
Reappraisal Subjectively Reported Affect Decoupling of Physiological and Subjective Emotional Experience Strengths and Limitations Conclusion and Future Directions	66 72 75 77 78
General Discussion	81
Strengths and Limitations Conclusion and Future Directions	81 82
References	84
Footnotes	100
List of Tables	101
Tables	101
Table 1: Correlation Matrix – Study 1 Table 2: Correlation Matrix – Study 2 Table 3: Repeated Measures – HRV Table 4: Repeated Measures – Subjectively Reported Negative Affect Table 5: Repeated Measures – Subjectively Reported Positive Affect	102 103 104 105 106
Figures	107
Figure 1: Mediation models Figure 2: Changes to the congruency effect on trial n as a product of trial n-1 Figure 3: Procedural diagram of the Emotion Regulation Task Figure 4: Mean HRV over time Figure 5: Mean subjectively reported affect over time Figure 6: Subjectively reported positive affect – CESD x Time Figure 7: Subjectively reported negative affect – GAD7 x Time Figure 8: A process model of emotion regulation	107 108 109 110 111 112 113 114

Abstract

Alexithymia is defined as an inability to identify or experience emotions, and has been connected to both major depressive disorder (MDD) and generalized anxiety disorder (GAD). Both MDD and GAD are associated with a tendency to select maladaptive emotion regulation (ER) techniques. The present studies explored the relationship between alexithymia and subclinical depression/anxiety by proposing that alexithymia is a mediator of the relationship between depressive/anxious symptoms and ER difficulty. Study 1 examined this relationship in the context of implicit, or automatic, ER, as assessed by an emotional conflict task. While the task failed to index implicit ER ability, this study fostered an understanding of the role of response windows in implicit ER paradigms. Study 2 explored this relationship in the context of explicit, or effortful, ER, as assessed by heart rate variability (HRV) and subjectively reported affect associated with an anxiety-inducing task prior to which participants were instructed to implement reappraisal. This study did not find alexithymia to be a mediator of the relationship between symptoms of depression/anxiety and ER, suggesting that alexithymia does not negatively impact individuals' ability to engage in instructed reappraisal. However, based on the general failure of reappraisal on a physiological level, this study also suggested that the relative success of reappraisal is contextually determined. Finally, this study found depression and anxiety to be associated with heightened subjective distress, and pointed to a decoupling between physiological and subjective emotional experience in subclinical depression and anxiety.

Subclinical Depression, Anxiety and Alexithymia: Implications for Implicit and Explicit Emotion Regulation

Emotion regulation (ER), or the conscious or non-conscious modulation of one's emotion states to meet the contextual demands of daily life (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Cole, Michel, & Teti, 1994), is a process with widespread implications for quality of life and functioning for both general and clinical populations. In the general population, ER predicts adaptive interpersonal sensitivity and prosocial tendencies (Lopes et al., 2005), and among individuals who report high levels of stress, those who are able to regulate their emotions tend to be happier (Extremera & Rey, 2015). Furthermore, deficits in overall emotional intelligence, a construct that assumes the ability to recognize emotions in the self and others as well as to regulate emotions to achieve goals (Colman, 2015), are associated with undesirable outcomes that include substance use, physical fights, and poor interpersonal relations (Brackett, Mayer, & Warner, 2004). ER is also implicitly tied to psychopathology. For example, the inability to successfully regulate emotions increases the likelihood that subclinical symptoms of disorders such as depression evolve into full-blown psychopathology (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Aldao et al., 2010), and can act as a maintaining factor for multiple disorders (Berking & Wupperman, 2012).

With both the benefits of successful ER and the consequences of poor ER in mind, it is clear that an understanding of the psychological constructs implicated in ER difficulty could be instrumental in improving the quality of life of clinical and subclinical populations alike. Whether this knowledge is applied through targeted intervention aimed at training specific populations to use adaptive ER techniques or as a general educational tool, the benefits of a more complete understanding of what causes ER to fail are undeniable. Accordingly, the following studies sought to investigate one construct that may play a role in ER difficulty in the context of depressive and anxious symptoms: *alexithymia*.

Alexithymia

In 1973, psychotherapist Peter Sifneos first used the term alexithymia, literally meaning "no words for mood", to describe observed deficits in the emotional functioning of psychosomatic patients. Since then, alexithymia has come to be known as a multi-dimensional construct that refers to an inability to identify and describe emotions in the self (Sifneos, 1973). More specifically, alexithymia has been conceptualized with a two-factor model that includes a cognitive dimension, in which individuals struggle to identify and verbalize their emotions, and an affective dimension, in which individuals simply have difficulties experiencing these emotions (Bermond et al., 2007). Core areas of deficit associated with alexithymia include dysfunction in emotional awareness of the self and social attachment, where alexithymia is characterized by cold and socially avoidant behavior (Spitzer et al., 2005). In keeping with these broader areas of dysfunction, alexithymia also impairs empathy for and emotional understanding of others, as evidenced by a reduced tendency towards prosocial behavior in individuals with alexithymia (Feldmanhall, Dalgleish, & Mobbs, 2013). Due in part to the tendency for the personal emotional difficulties of alexithymia to manifest themselves as larger social deficits, it has been speculated that individuals with alexithymia may exhibit a reduced capacity to use social interaction as a means of emotion regulation (Spitzer et al., 2005). As such, individuals with alexithymia may rely disproportionately on internalized ER techniques, making the elucidation of the nature of alexithymia's relationship with ER all the more important.

While it was first defined in the context of psychosomatic disorders (Sifneos, 1973), alexithymia has been associated with other disorders, including mood and anxiety disorders

(Onur et al., 2013), and is also highly prevalent in the general population. When using cutoff scores specified by the 20-item Toronto Alexithymia Scale (TAS-20; Bagby, Parker, & Taylor, 1994a) – one of two scales that have largely driven research on alexithymia – the prevalence of alexithymia among the general population is as high as 13% (Salminen, 1999). Despite this cross-population prevalence, past studies have largely failed to explore the interplay between depressive/anxious symptoms and alexithymia on a subclinical level. The present studies sought to investigate this relationship in the context of ER by asking the following questions: could alexithymia engender depressive and anxious symptoms that interfere with ER ability? Or, by contrast, could alexithymia be a product of depressive and anxious symptoms that acts as a barrier to adaptive ER? To conceptualize these research questions, two mediation models were proposed. The first model (Model A; see Figure 1A) proposed that alexithymia would predict depressive and anxious symptoms, which, in turn, would predict difficulties with ER. The second model (Model B; see Figure 1B), on the other hand, proposed that depressive/anxious symptoms would predict alexithymia, which would subsequently act as a psychological mechanism through which such individuals experience difficulty regulating emotion. In order to further explore these models, however, alexithymia's unique role in psychopathology must first be understood.

Alexithymia and Psychopathology

Although the present studies examined depressive and anxious symptoms on a subclinical level, recognizing the nature of alexithymia's relationship with full-blown psychopathology is crucial for understanding its role in the context of depressive and anxious symptoms in the general public. While alexithymia is by no means a purely clinical construct and can be seen in varying populations, converging evidence has suggested that the presentation of alexithymia may be influenced by psychopathology. For example, while alexithymia has been found by meta-

review to be a stable, personality-like trait in nonclinical populations, it is more variable in clinical samples (de Haan et al., 2012; Karukivi & Saarijärvi, 2014). In other words, while healthy controls tend to display constant levels of alexithymia across time, individuals with various psychopathologies may have more fluid, state-dependent levels of alexithymia. These variations are linked to the quality and severity of an individual's symptoms. It is important to note, however, that distinctions between alexithymia in clinical populations and nonclinical populations may be present on more than just a state versus trait level. For example, Diborde et al. (2004) provide evidence for a distinction between the symptom presentation of alexithymia in clinical and nonclinical populations in the context of eating disorders. In their study, individuals categorized as alexithymic in a control group exhibited selective deficits in the emotional cognition dimension of alexithymia while maintaining functioning in the affective dimension. However, individuals with alexithymia in an eating disorder group were characterized by deficits in both the affective dimension and the cognitive dimension, suggesting that alexithymia affected the quality of the emotional experience only in clinical populations.

A similar distinction between the presentation of alexithymia in clinical versus nonclinical populations can be seen on a neurobiological level. In individuals with major depressive disorder (MDD), for example, Ho et al. (2016) found alexithymia to be correlated with decreased functional connectivity in the right prefrontal gyrus and right-lateralized regions of the brain associated with cognitive ER in the default mode network (DMN) – a network that has been associated with emotional self-reflection and understanding emotion in others. However, in controls, alexithymia was associated with increased functional connectivity between the right inferior frontal gyrus triangularis and the right superior occipital lobe, a region associated with emotional response to exterior stimuli. Ho et al. (2016) suggest that this indicates an association between alexithymia and reduced automatic ER and introspective thinking in participants with MDD, but not in healthy controls. By contrast, they posited the increased connectivity seen in healthy controls to be associated with difficulty evaluating emotional salience of external stimuli.

While the studies above describe alexithymia on a broad clinical-versus-nonclinical level, the specific role alexithymia plays in psychopathology can be explored through its appearance as a common substrate of highly comorbid "distress disorders" MDD and generalized anxiety disorder (GAD; Mennin et al., 2007; 2005). Although alexithymia is not a diagnostic construct per se, it has been consistently linked with both MDD (with 32.1% co-occurrence; Honkalampi et al., 2000; Onur et al., 2013) and GAD (Onur et al., 2013). In MDD, alexithymia is associated with symptom severity (Honkalampi et al., 1999). This linkage between alexithymia and distress disorders such as MDD and GAD certainly makes conceptual sense – as outlined in Model A, an inability to label or experience emotions in the self could conceivably play an intimate role in processes related to depression and anxiety, including the formation or preservation of ineffectual strategies of emotion management or maladaptive schemas about the self or the world at large (a feature especially relevant to MDD; Renner et al., 2012). Conversely, as seen in Model B, alexithymia could be a product of depression and anxiety, where chronic feelings of lowered mood or anxious thought lead to difficulty accurately conceptualizing one's emotions. Indeed, Leahy (2002) found that the belief that one's emotions are incomprehensible, uncontrollable, or somehow different than others' was common in both depression and anxiety a view that fits well with the concept of alexithymia.

The prevalence of this construct in two frequently co-occurring disorders suggests that alexithymia may be related to areas of deficits shared by these disorders. Emotion dysregulation

- an integral component of both MDD and GAD (Aldao et al., 2010; Campbell-Sills & Barlow, 2007) – may be one such area of common deficit in which alexithymia plays a critical role. As alexithymia involves deficits in emotional awareness and emotion labeling, it is conceivable that the ER issues that are common to MDD and GAD may be facilitated or exacerbated by the presence of this construct. For example, alexithymia may interfere with one's ability to identify appropriate times to engage in regulation or limit the depth of emotional understanding necessary to properly implement adaptive ER techniques.¹ In support of this notion, ER literature has suggested that alexithymia may be associated with maladaptive ER strategies. Chen et al. (2011), for example, found different manifestations of alexithymia to be associated with unique ER profiles. Of particular note, the general-high alexithymia subtype (GHA; corresponding to high scores on all three components of the TAS-20) predicted suppressive ER techniques. Furthermore, conscious emotional awareness – an area of deficit in alexithymia – appears to be necessary for the proper implementation of reappraisal (Subic-Wrana et al., 2014). Together, these findings suggest that the nature of one's repertoire of ER techniques may be adversely influenced by the presence of alexithymia, offering support for the conceptualization of alexithymia as a mediator of the relationship between depressive/anxious symptoms and emotion dysregulation (Model B).

In further support of Model B's proposed detrimental effect of alexithymia on ER, Mennin et al.'s (2007) model of emotion dysregulation implicated in anxiety and mood symptoms considers *poor emotional understanding*, or an inability to properly evaluate one's emotional state (a key feature of alexithymia), to be an important factor in the failure to engage in ER. What's more, this feature was found to be strongly associated with MDD independently, as well as comorbid GAD. Furthermore, although Mennin et al. (2007) initially predicted *heightened intensity of emotions* to be implicated in emotion dysregulation, it was not found to be in any way predictive of their indices of maladaptive emotion management in MDD. This suggests that emotional intensity may simply be involved in emotion generation rather than emotion regulation, and that emotion dysregulation in individuals with MDD may stem from some other mechanism, such as alexithymia. The role of alexithymia's deleterious role in MDD has further neurobiological support from Ho et al. (2016) who, as previously mentioned, found not only a differentiation between the nature of alexithymia in clinical versus nonclinical contexts, but also functional evidence for an association between alexithymia and reduced automatic ER in MDD.

With the differentiation in alexithymia's presentation in clinical versus nonclinical populations as well as its associations with maladaptive ER in mind, it can be said that the studies at hand were informed by the assumptions that A) alexithymia can be associated with unique deficits in populations with psychopathology, and that B) these deficits can include difficulty regulating emotion.

Measurement of Alexithymia

As previously mentioned, two scales in particular have largely driven research on alexithymia: the Bermond-Vorst Alexithymia Questionnaire (BVAQ; Vorst, 2001) and the 20item Toronto Alexithymia Scale (TAS-20: Bagby et al., 1994a). The BVAQ identifies five traits typical of alexithymia: 1) difficulty *differentiating* between emotions; 2) reduced ability to *fantasize*; 3) difficulty *verbalizing* emotions; 4) difficulty *emotionalizing*, referring to one's degree of emotional arousal in response to emotion-eliciting stimuli; and 5) reduced tendency or ability to *analyze* or otherwise reflect upon one's feelings (Bermond et al., 2007). The TAS-20, on the other hand, splits alexithymia into three distinct sub-dimensions: Difficulty Describing Feelings (DDF), Difficulty Identifying Feelings (DIF), and Externally-Oriented Thinking (EOT; Bagby et al., 1994a). The BVAQ and the TAS-20 have both been psychometrically validated in clinical and subclinical populations alike, and tend to correlate well with each other (Vorst, 2001; Parker et al., 1993; Bagby et al., 1994a; 1994b). However, despite their apparent similarities, the BVAQ was developed to overcome several shortcomings of the TAS-20. For example, the TAS-20 contains a non-consistent number of items in each sub-dimension, which result in some factors contributing more to the total score than others (Vorst, 2001). What's more, the TAS-20 has no explicit measure for "reduced fantasizing" and "reduced experiencing of emotional feelings" – both of which have been identified as key characteristics of alexithymia (Mann, Wise, & Shay, 1992).

Sub-dimensions aside, it has also been speculated that the TAS-20 fails to measure a larger grouping principle of alexithymia. As previously indicated, alexithymia includes both a cognitive dimension and an affective dimension (Bermond et al., 2007). A study performed by Vorst (2001) suggests that the TAS-20 fails to measure this latter component of alexithymia. According to this study, the sum score of the cognitive subscales of the BVAQ (analyzing, identifying, and verbalizing of emotions) was found to be nearly statistically identical to the *total* score of the TAS-20, while the affective subscales of the BVAQ (emotionalizing and fantasizing) had little correlation with the TAS-20 sub-dimensions. Together, this suggests that the BVAQ may provide a more complete picture of the affective dimension of alexithymia than its counterpart. It is unclear, however, if the addition of this dimension produces significant changes in the predictive validity of alexithymia. The TAS-20's EOT sub-dimension has been found to be predictive of more severe symptomatology after inpatient treatment for major depression (Günther et al., 2016), suggesting that certain aspects of alexithymia may attenuate the success of

treatment intervention. Furthermore, Karukivi et al. (2014) found the DIF sub-dimension to be predictive of state and trait anxiety in late adolescence. Together, these findings suggest that the sub-dimensions proposed by the TAS-20 have some level of predictive value in the context of depressive and anxious symptoms, regardless of whether or not they successfully measure the affective side of alexithymia.

Present Studies

While past studies have identified a link between alexithymia and psychopathology, both as a risk factor and as a predictor of symptoms, its precise role in ER difficulty for individuals with subclinical depression and anxiety remains unclear. Accordingly, the present studies sought to explore the specific roles that subclinical symptoms of depression, anxiety, and alexithymia play in ER difficulties that are typical of MDD and GAD on a clinical level. As depression and anxiety disorders are often associated with emotion dysregulation, and alexithymia involves a shortcoming in one's understanding or experience of emotion that may interfere with ER processes, these studies posited that A) subclinical symptoms of MDD and GAD would be negatively associated with ER ability, that B) symptoms of MDD and GAD would predict alexithymia, and that C) alexithymia would act as a mechanism through which individuals with symptoms of MDD and GAD fail to engage in ER. More specifically, the present studies predicted that alexithymia would mediate a negative relationship between depressive/anxious symptoms and ER ability, whereby the proposed relationship between symptom severity and ER ability would be attenuated when alexithymia was included as a predictor of ER ability. This hypothesis is in line with Model B (Figure 1B), and is informed by Chen et al.'s (2011) findings of distinct alexithymic profiles associated with different ER techniques, as well as Subic-Wrana et al.'s (2014) findings that suggest a role of emotional awareness in the selection of ER

strategies. Although the studies at hand predicted a specific outcome, the results were analyzed to examine Model A – which suggested alexithymia to be a predisposing factor for maladaptive beliefs about one's self or one's emotions that develop into depression or anxiety and subsequent ER difficulties – as well as Model B.

While the relationship between alexithymia, depressive/anxious symptoms, and ER can be conceptualized using multiple models, it is equally important to note that ER can exist on more than one level. Namely, ER can be assessed through the examination of implicit (i.e., automatic) or explicit (i.e., effortful) regulation. While much of the research outlined above has examined alexithymia's relationship with ER in general, this relationship has rarely been deconstructed to explore implicit versus explicit ER. To that end, Study 1 examined the proposed relationship between symptoms of depression/anxiety, alexithymia, and ER by focusing on implicit ER, while Study 2 explored this relationship in the realm of explicit ER.

Study 1 Introduction

The question posed by Study 1 concerned implicit, or automatic, ER and its relationship with alexithymia as a potential mediator of the proposed association between symptoms of depression/anxiety and ER difficulty: is a lack of conscious awareness of emotions a barrier to successful subconscious ER, or are automatic tendencies unaffected by higher-level deficits? In order to explore this question, implicit ER must first be understood.

Implicit Emotion Regulation

ER occurs both implicitly, or automatically, and explicitly, or through effortful engagement. While these two processes occur on different levels of awareness, both implicit and explicit ER are necessary for wellbeing. Implicit ER, for its part, is automatically evoked by a stimulus, occurs subconsciously, and is important for healthy functioning in day-to-day life as it can effectively meet the high demand for moment-to-moment regulation without requiring the same level of conscious awareness as explicit ER (Gyurak, Gross, & Etkin, 2011). To illustrate this point, imagine a scenario where an individual is running late for a meeting at work. Without the capacity to implicitly regulate emotion, the individual would have to devote mental resources to engage in some form of explicit ER, such as reappraisal (e.g., "Being late for this meeting helped me miss the morning traffic and improved my drive to work."). This may cause him to be distracted or unengaged during the meeting. If this process were able to occur automatically, however, the individual would be able to focus on the meeting while simultaneously processing the negative emotions generated by his late arrival.

Although implicit ER occurs outside of one's awareness, it can be just as useful as more effortful strategies at modulating emotion. In response to an anxiety-producing task, for example, unconscious regulation (elicited by subtly exposing participants to reappraisal-related words prior to the task) was equally effective at diminishing physiological response when compared to explicit implementation of reappraisal (Williams et al., 2009). What's more, (Yang et al., 2015) found that implicit ER reduced subjective emotional experience to both gains and losses in a gambling task without requiring cognitive resources, effectively insulating participants against the emotional effects of successes and failures in a decision-making context. Overall, these findings suggest that while implicit ER may not have historically received as much research as explicit ER, it is an important aspect of successful regulation in the course of daily functioning and bears significance to one's experience of the world at large.

Implicit ER involves a number of different processes, each of which corresponds to a unique area of the brain. As a whole, the implicit ER system likely includes the amygdala, hippocampus, and areas of the medial prefrontal cortex, including the subgenual anterior cingulate cortex (sgACC), the ventromedial prefrontal cortex (vmPFC), the rostral/pregenual anterior cingulate cortex(r/pgACC), the dorsal anterior cingulate cortex (dACC), and the dorsomedial prefrontal cortex (dmPFC; Phillips et al., 2015). Growing support suggests that this system – particularly the amygdala and medial prefrontal cortical regions – is modulated by serotonin (Phillips et al., 2015). The different regions comprising this system are related to the unique aspects of implicit ER, with the sgACC and vmPFC being implicated in automatic behavioral control, the r/pgACC in automatic attentional control, and the dACC, dmPFC, the hippocampus, and the vmPFC being important for automatic cognitive change necessary for subconscious error monitoring and rule-learning. Each of these components plays a critical role in one's ability to subconsciously and automatically amplify, attenuate, or maintain a given emotion.

In the context of this study, implicit ER was operationalized using an adaptation of the classic Stroop task (Stroop, 1935) designed to create emotional conflict. This task - called the Emotional Conflict Task (ECT) – is composed of images of emotional faces with superimposed emotional labels (i.e., "fear", "happy") that either match the face's affect or are incongruent. For incongruent trials, participants must suppress the emotional information presented by the word in order to correctly identify the affect of the face (Etkin et al., 2006). This process can be analyzed through behavioral data collected during this task, and acts as an index of implicit ER as it occurs without instruction and without participant knowledge (Etkin et al., 2010). The ECT has also been supported by neuroimaging studies. In the original study, Etkin et al. (2006) used the ECT in conjunction with fMRI to associate the phenomenon of emotional conflict resolution with the rostral anterior cingulate cortex (rACC) – a brain region that has been shown to be implicated in implicit ER processes and, more specifically, in automatic attentional control (Phillips et al., 2015). Past research with the emotional conflict task has revealed slowed performance in clinical samples, including among samples with GAD as well as comorbid MDD and GAD (Etkin & Schatzberg, 2011 and Etkin et al., 2010, as seen in Robinson et al., 2015). Slower performance on the ECT is thought to indicate reduced ability to engage in implicit ER as it suggests that participants are taking longer to successfully resolve emotional conflict. Although faster performance may also reflect abnormal ER (e.g., hypersensitivity to emotional stimuli), this phenomenon has not been associated with a particular disorder other than binge-eating disorder (Robinson et al., 2015). Based on these findings, participants with more depressive and anxious symptoms were expected to exhibit slower ECT performance in the context of this study.

Hypotheses

Past research on alexithymia and ER has left the relationship between alexithymia and implicit ER largely unexplored. However, some inferences may be made based on the relationship between implicit and explicit ER. While implicit and explicit ER may be dissociable processes, they do have some level of interconnectivity. Rather than being confined to one level of awareness, ER processes can fluctuate between conscious and subconscious domains. Of particular note, the frequent use of an explicit ER strategy can render it implicit (Gyurak et al., 2011). This is an important concept to consider as it implies that habitual utilization of any technique – adaptive or maladaptive – may have long lasting effects on one's automatic regulation tendencies. As individuals with alexithymia are more likely to explicitly implement maladaptive ER strategies (Chen et al., 2011), this assumption suggests that alexithymia may be associated with a tendency to automatically implement maladaptive ER strategies, and therefore may act as a barrier to successful implicit ER. This notion is further supported by Ho et al.'s (2016) fMRI study that suggests that alexithymia may be associated with difficulty automatically regulating emotions on a neurobiological level. Together, these findings informed my hypothesis: alexithymia was predicted to mediate a negative relationship between depressive/anxious symptoms and implicit ER ability, whereby the proposed relationship between symptom severity and implicit ER ability would be attenuated when alexithymia was included as a predictor of implicit ER ability. This hypothesis was consistent with Model B (Figure 1B).

Study 1 Method

Participants

Participants were 65 Bates College students (16 men, 49 women) recruited via an email announce system, Facebook events, or a Bates introductory psychology course. The study was approved by the Bates College IRB and standardized consent forms were made available before the study. Participants received either course credit or \$5 as compensation. All participants were at least 18 years of age (with the exception of one 17-year-old who continued with the study despite being informed of the study requirements) and were comfortable reading directions and questions in English. The sample was 12.3% Black, 72.3% White, and 12.3% Asian. 10.8% of the sample identified as Hispanic or Latina/Latino.

Materials

The Toronto Alexithymia Scale (TAS-20; Bagby et al., 1994a). As previously mentioned, the TAS-20 proposes three distinct sub-dimensions of alexithymia: Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF), and Externally-Oriented Thinking (EOT; Bagby et al., 1994a). While it has been psychometrically validated in clinical and general populations (Parker et al., 1993; Bagby et al., 1994a; 1994b), it has been suggested that the TAS-20 fails to measure the affective component of alexithymia (Vorst, 2001). Alexithymia is a dimensional variable, and the score on the TAS-20 is therefore best analyzed as a continuous variable (as it was in this experiment). However, the authors of the TAS-20 also established categorical cut-off points, with scores equal to or less than 51 corresponding to nonalexithymia, scores equal to or greater than 61 corresponding to alexithymia, and scores of 52 to 60 corresponding to possible alexithymia (Bagby et al., 1994a). The TAS-20 judges alexithymia using a 5-point Likert scale (1 = strongly disagree and 5 = strongly agree) and includes 20 items, such as "I am often confused about what emotion I am feeling" and "When I am upset, I don't know if I am sad, frightened, or angry". The TAS-20 has been characterized by a Cronbach's alpha coefficient of .81 and has shown good test-retest reliability (.77; Bagby et al., 1994a), as well as strong convergent and moderate discriminant validity (Bagby et al., 1994b).

The Center for Epidemiologic Studies Depression scale (CES-D; Radloff, 1977). The CES-D is a scale used to assess depressive symptoms in the general population. However, the CES-D has also been shown to operate comparably to the Beck Depression Index as a screening tool for depression in clinical samples (Zich, Attkisson &, Greenfield, 1990). The CES-D asks participants to indicate how often they have felt a certain way during the past week, and uses a scale with 4 response categories (*Rarely or none of the time* [less than 1 day], *Some or a little of the time* [1-2 days], *Occasionally or a moderate amount of time* [3-4 days], and *All of the time* [5-7 days]). There are 20 items on the CES-D in the form of statements such as "I was bothered by things that usually don't bother me", and higher scores indicate higher levels of depression. The CES-D has been characterized by Cronbach's alpha coefficients of .81-.84 in community samples and .90 in clinical samples, and has shown strong convergent and discriminant validity (Radloff, 1977).

The 7-item Generalized Anxiety Disorder scale (GAD-7; Spitzer et al., 2006). The GAD-7 is a brief clinical measure used for assessing GAD. The GAD-7 has been psychometrically validated and exhibits good agreement between administration via interview and self-report (Spitzer et al., 2006). While it was designed as a diagnostic instrument, the GAD-7 has also been validated as a dimensional measure of GAD severity (Rutter & Brown, 2016) – the form in which GAD was primarily analyzed in the present study. The GAD-7 asks participants how often they have been bothered by a list of problems within the last 2 weeks, and

uses a scale with 4 response categories (0 = Not at all sure, 1 = Several days, 2 = Over half the days, 3 = Nearly every day). The 7 items that comprise this measure include problems such as "Feeling nervous, anxious, or on edge", and are followed by a question regarding functional impairments. Higher scores indicate higher levels of anxiety. The GAD-7 has been characterized by a Cronbach's alpha coefficient of .92 in community samples and .90 in clinical samples, and has shown good test-retest reliability (.83) as well as strong convergent (Spitzer et al., 2006) and moderate discriminant validity (Beard & Björgvinsson, 2014).

Emotional Conflict Task (ECT; Etkin et al., 2006). The ECT is a reinterpretation of the classic color/word Stroop task: a paradigm which involves a color word printed in incongruently colored ink (e.g., the word "red" printed in blue ink), and asks participants to identify the ink color. The ECT follows a similar design, but uses emotion words and emotional faces in the place of color words and colored ink. Namely, happy or fearful facial expressions are presented with the words "happy" or "fear" superimposed on them, and participants are asked to identify the facial expression while ignoring the emotion words. Stimuli are either congruent (e.g., happy expression/"fear" word).

148 stimuli were presented in ECT. Unlike the original task, which presented each stimulus for 1000 ms, the current version of the ECT was modified to have a response-dependent window of presentation with each stimulus remaining on the screen until the participant responded. This change was made in an attempt to emphasize differences in response times while reducing the number of inaccurate trials that had to be discarded. Stimuli were followed by a varying interstimulus interval of 3000-5000 ms (mean interval = 4000 ms) during which a central fixation cross appeared on the screen. Stimuli appeared in a pseudorandom order, with equal numbers of congruent-congruent, congruent-incongruent, incongruent-congruent, and incongruent-incongruent pairings. The gender, affect, and identity of the faces were randomized throughout the trials. Finally, in order to avoid repetition priming caused by repeated images, there were neither direct repetitions of images of the same face in combination with different words nor images of the same face/word combination. Participants were instructed to identify the affect of the faces as quickly and as accurately as possible by pushing response buttons that corresponded to "fearful" ("1" key) and "happy" ("2" key).

Like classic Stroop tasks, this task is associated with slower response times for incongruent stimuli. This effect is known as the congruency effect. Implicit ER is operationalized in this paradigm through changes in the congruency effect. Specifically, when the previous trial (trial n -1) is incongruent, emotional control is triggered. This reduces susceptibility to response conflict on trial n, leading to faster response times on trial n if presented with another incongruent stimulus.² Implicit ER can therefore be quantified in this paradigm by contrasting response times on incongruent trials preceded by a congruent trial (cI; expected to be slower) with incongruent trials preceded by another incongruent trial (iI; expected to be faster). In this case, better implicit ER corresponds to a larger difference between iI and cI. This process has been suggested to occur outside of conscious awareness (Gyurak et al., 2011).

Procedure

After reviewing and signing the consent form with the researcher, participants responded to a number of questionnaires via the online survey program Qualtrics (Qualtrics, 2015), including CES-D, the GAD-7, the TAS-20, and a set of demographic questions. Following the initial questionnaires, participants completed the ECT, which was integrated into the computer program e-Prime. Finally, after completing the ECT, the participants received a debriefing script

and either course credit or \$5. *All* participants, regardless of symptom endorsement, were provided with a list of mental health resource.

Study 1 Results

Screening

Participants with total accuracy of 85% or less were excluded from further analysis (*n* = 4) in an attempt to screen the data for individuals who did not successfully engage with the emotional conflict task (ECT). One exception was made for a participant who responded to one of the four blocks of the ECT with reversed key binding for fearful and happy faces (i.e., pressed the "2" key when presented with a fearful face and the "1" key when presented with a happy face instead of the opposite). Once the scores for this block were adjusted accordingly, the participant was included in further analysis as their corrected accuracy was above 85%. For the remaining 61 participants, only accurate trials were considered.

In order to screen the data for within-participant outliers, mean response times (RT) and standard deviations were computed for each participant. Using these values, z-scores were calculated for each trial. Trials with RTs that deviated from an individual's mean RT by more than two z-scores in either direction were excluded from further analysis.

Variables of Interest

In order to assess the conflict resolution effect seen in Etkin et al. (2006), a variable was created by contrasting response times on incongruent trials preceded by another incongruent trial (iI) with response times on incongruent trials preceded by a congruent trial (cI; $RT_{cI} - RT_{iI}$). In this case, better implicit ER corresponded to a larger difference between iI and cI, or a larger $RT_{cI} - RT_{iI}$ value.

Although RT_{cI} - RT_{iI} was the main variable of interest, implicit ER was also assessed through two other variables. One of these variables was simply mean RT for incongruent trials (RT_{I}), which represented how long on average it took participants to respond to incongruent trials. Here, smaller RT_{I} values corresponded to better implicit ER as it represented a faster response to trials in which the resolution of emotional control was required. In order to correct for within-participant variation of response time, RT_{I} was also incorporated into a variable that contrasted RT of congruent trials with RT of incongruent trials (RT_{I} - RT_{C}). In this case, smaller values of RT_{I} - RT_{C} corresponded to better implicit ER.

ECT

In order to assess the general effectiveness of the ECT, the mean RT for trials that required emotional control (incongruent trials; I) was contrasted with the mean RT for trials that required no emotional control (congruent trials; C). As expected, the mean RT of congruent trials (M = 891.47 ms, SD = 243.02 ms) was significantly smaller than the mean RT of incongruent trials (M = 989.24 ms, SD = 279.74 ms), t(60) = 10.00, p < .001 according to a paired samples t-test. Consistent with the goal of the paradigm, this analysis demonstrated that, on average, participants responded more quickly to trials in which there was no emotional conflict (C) compared to trials in which there was emotional conflict (I). At a basic level, this suggests that the suppression of an emotional response to an incongruent emotion word increased the amount of time it took to accurately identify the affect of facial images.

To explore the main implicit ER variable of interest ($RT_{iI} - RT_{cI}$), the effect of conflict resolution priming in incongruent trials was assessed by contrasting the mean RT of incongruent trials preceded by congruent trials (cI) with the mean RT of incongruent trials preceded by incongruent trials (iI). Unexpectedly, no conflict resolution effect was seen in incongruent trials. While the mean RT of iI trials (M = 985.11 ms, SD = 272.39 ms) was smaller than the mean RT of cI trials (M = 989.52 ms, SD = 290.27 ms), this difference was not found to be significant by a paired samples t-test, t(60) = -0.43, p = .671. A similar analysis run in the context of congruent trials ($RT_{cC} - RT_{iC}$), however, revealed a significant difference where the mean RT of cC trials (M = 871.83 ms, SD = 231.35 ms) was significantly smaller than that of iC trials (M = 901.48 ms, SD = 251.27 ms) according to a paired samples t-test, t(60) = -3.38, p = .001 (see Figure 2). These findings stand in direct opposition to Etkin et al.'s (2006) original results, which showed a conflict resolution effect in the case of incongruent trials (where the mean RT of iI trials was significantly smaller than the mean RT of cI trials), but found no evidence of such an effect in the case of congruent trials as there was no emotional conflict present and therefore no priming of conflict resolution. As emotional conflict resolution could not have been influencing the RTs of trials in which there was no emotional conflict (i.e., congruent trials), further analyses were conducted to determine what was driving these effects.

Due to the unexpected results outlined above, analyses were run to determine whether the observed effects were related to conflict resolution, as posited by Etkin et al. (2006), or whether they were driven by simple repetition priming. While there were no direct repetitions of images in the ECT, there were instances of *category repetition* (e.g., fearful face with happy word followed by another fearful face with happy word). To explore the possibility that category repetition affected participants' responses to experimental stimuli, a paired samples t-test was conducted to compare the RTs of high conflict resolution trials (i.e., iI trials) in which category repetition occurred to trials in which repetition did not occur. If repetition priming were driving the observed effects, the mean RT of category repetition trials would be expected to be *lower* due to priming of a specific word category/image category combination. Interestingly, while the

mean RTs of the two conditions were significantly different, this difference was not in the expected direction. Instead, the mean RT of category repetition trials (M = 1016.41 ms, SD = 282.17 ms) was significantly *higher* than the mean RT of non-category repetition trials (M = 935.66 ms, SD = 341.21 ms), t(60) = 2.62, p = .011. A similar analysis was run in the context of congruent trials to compare RTs of cC trials in which category repetition occurred to cC trials in which repetition did not occur. In this case, the observed effects were consistent with what would be expected from repetition priming, with the mean RT of category repetition trials (M = 816.15 ms, SD = 301.55 ms) being significantly lower than the mean RT of non-category repetition trials (M = 816.15 ms, SD = 301.55 ms) being significantly lower than the mean RT of non-category repetition trials (M = 816.15 ms, SD = 301.55 ms) being significantly lower than the mean RT of non-category repetition trials (M = 908.93 ms, SD = 259.76 ms), t(60) = -3.16, p = .002. Together, these findings suggest that the ECT may not have acted as a valid index of implicit ER ability in the context of this study, and that subsequent behavioral results should be interpreted with caution.

Depression, Anxiety, and Alexithymia

All scales demonstrated acceptable levels of internal consistency. Cronbach's alphas for the CES-D and the GAD-7 were .89 and .85, respectively. Cronbach's alpha for the TAS-20 as a whole was .85. The DIF, DDF, and EOT subscales contained 7, 5, and 8 items and had Cronbach's alphas of .87, .67, and .69, respectively.

Due to the subclinical nature of the sample, depression, anxiety, and alexithymia were regarded as dimensional constructs. However, in order to get a sense of the overall levels of pathology within this sample, the CES-D, the GAD-7, and the TAS-20 were briefly examined on a categorical basis. Of the 65 total participants, 28 were above the cutoff point for depression, 18 for anxiety, and 14 for both depression and anxiety using cutoff scores of 16 and 10, respectively (Kroenke et al., 2007; Radloff, 1977). Ten participants were above the cutoff point (61; Bagby et al., 1994a) for high alexithymia.

Correlations. In order to assess the relationships between the independent variables (IVs; total score on the CES-D, the GAD-7, and the TAS-20) and several dependent variables (DVs) of interest, a number of bivariate correlation analyses were performed. The full results of these correlation analyses can be found in Table 1.

To assess the relationships between participants' scores on depression (M = 16.58, SD = 8.89), anxiety (M = 6.82, SD = 4.27), and alexithymia (M = 46.94, SD = 11.46) scales – the CES-D, GAD-7, and TAS-20, respectively – bivariate correlation analyses were run between these variables. As expected, these variables were highly positively correlated. Total score on the CES-D was significantly correlated with total score on the GAD-7, r(62) = .71, p < .001, as well as total score on the TAS-20, r(62) = .59, p < .001. GAD-7 and TAS-20 scores were also significantly correlated with one another, r(62) = .49, p < .001.

Contrary to predictions, depression, anxiety, and alexithymia were not significantly correlated with variables indexing implicit ER ability. Bivariate correlation analyses were run between total CES-D, GAD-7, and TAS-20 scores and multiple implicit ER variables including incongruent adaptation (RT_{cI} - RT_{iI}), the difference between emotional and non-emotional control trials (RT_{I} - RT_{C}), and mean RT of incongruent trials alone (RT_{I}). These analyses yielded no significant correlations.

Regression analyses. In order to explore the mediation models depicted in Figure 1 in the context of implicit ER, a number of simple linear regressions were conducted. The difference between the mean RT for incongruent trials and the mean RT for congruent $[RT_I - RT_C]$ was used as a DV in these analyses as it was the only index of implicit ER that accounted for withinsubjects variability in RT via subtraction while also demonstrating robust significant effects that were in line with what would be expected from emotional conflict resolution (i.e., the mean of incongruent trials was significantly larger than the mean of congruent trials). For this variable, smaller values corresponded to faster responses to incongruent trials relative to congruent trials, which in turn indicated better implicit ER.

First, to explore Model A (see Figure 1A), a simple linear regression was calculated to predict RT_1 - RT_C based on total score on the TAS-20, yielding a nonsignificant regression equation, $R^2 = .01$, F(1, 59) = 1.79, p = .186. As this relationship was not found to be significant, no further mediation analyses were conducted.

In the exploration of mediation Model B (see Figure 1B), a simple linear regression found RT_I - RT_C to be predicted by neither total score on the CES-D, $R^2 = -.01$, F(1, 59) = 0.18, p = .676, nor total score on the GAD-7, $R^2 = .00$, F(1, 59) = 0.73, p = .396. As these relationships were not found to be significant, no further mediation analyses were conducted.

Study 1 Discussion

The purpose of this study was to examine the nature of the relationship between depressive/anxious symptoms, alexithymia, and implicit ER ability. Two mediation models were evaluated. While total scores on the CES-D, GAD-7, and TAS-20 were highly significantly correlated, demonstrating the expected association between these three constructs, none of these scores were significantly associated with variables corresponding to implicit ER success. However, unexpected dissociation of behavioral data suggested that this finding might have been influenced by a failure of the experimental protocol rather than a lack of impact of depressive/anxious symptoms and/or alexithymia on implicit ER ability.

Behavioral Data

Etkin et al. (2006) – the original author of the emotional conflict task (ECT) – found that emotional conflict on trial *n* -1 led to a decreased RT in the face of emotional conflict on trial *n*.

Through further exploration of behavioral data, Etkin et al. (2006) attributed this effect to an anticipatory mechanism whereby participants' ability to engage in emotional control was bolstered after being exposed to incongruent emotional stimuli on the previous trial. Etkin et al. (2006) dubbed this phenomenon *emotional conflict resolution*, and localized it to the rostral anterior cingulate cortex (rACC). In the context of this study, the emotional conflict paradigm was used as an index of implicit ER as it implicates subconscious modulation of emotion participants unknowingly regulate their emotional response to the emotion word in order to accurately identify the affect of the face. However, while the behavioral data from Etkin et al.'s (2006) original study was consistent with what would be expected from emotional conflict resolution – with iI trials being significantly faster than cI trials, and cC and iC trials being statistically identical (as there is no emotional conflict in congruent trials and therefore no effect of emotional conflict resolution) – these behavioral effects were not seen in the present study. In fact, the behavioral data collected in the present study yielded an almost-opposite pattern of results: while there was *no* significant difference between mean RTs of il trials and iC trials, cC trials were, on average, significantly faster than iC trials.

In order to explore what was driving this effect, the behavioral data were analyzed to explore forms of priming outside of the realm of emotional conflict. While there were no direct repetitions of facial images in the ECT, instances of category repetition (e.g., fearful face with happy word followed by another fearful face with happy word) were unavoidable. This being the case, it is possible that the RTs of participants performing the ECT may have been more heavily influenced by the simple repetition of category pairs than by the effect of emotion conflict resolution. Failure to account for the influence of repetition priming has been a major criticism of the ECT's non-emotional counterparts. For example, Mayr, Awh, and Laurey (2003) suggest that

certain cognitive control tasks – paradigms that, similar to the ECT, rely on the assumption that conflict monitoring processes trigger top-down control on trial n when presented with a (nonemotional) incongruent stimuli on trial n-1 – may be failing to recognize the influence of bottomup, or stimulus-driven, effects such as repetition priming. Indeed, Mavr et al. (2003) demonstrate that repetition priming accounts for a larger proportion of the behavioral effects typically conflated with conflict monitoring than was reflected in past research with cognitive control paradigms. Fortunately, Etkin et al. (2006) had the foresight to check for this effect. In their study, mean RTs of category repetition trials would be expected to be significantly faster than mean RTs of non-category repetition trials if repetition priming were driving the observed effects - a comparison that was performed in Etkin et al. (2006) and yielded no significant difference between the category repetition and non-category repetition groups. In the context of the present study, however, this analysis revealed category repetition trials to be significantly faster than non-category repetition trials within the context of low conflict resolution trials (i.e., cC trials). This suggests that the unexpected significant difference observed when comparing cC and iC trials may have been driven by repetition priming, whereby the responses of participants susceptible to the quickening effects of category repetition – a phenomenon that, by definition, can occur only in cC trials and not in iC trials – drove the average RT of cC trials down compared to iC trials that involved no instances of category repetition.

In the case of incongruent trials, however, a different pattern emerged. Rather than being faster than their non-category repetition counterpart (as would have been expected from repetition priming), the mean RT of iI trials in which there was category repetition was, somewhat paradoxically, significantly *slower* than the mean RT of iI trials in which there was no

category repetition. This suggests that something other than repetition priming was driving the unexpected lack of emotional conflict resolution seen within the context of incongruent trials.

At this point, is important to remember that the version of the ECT used in this study differed from the original task on the basis of response window. While Etkin et al. (2006) used both response time and accuracy as metrics of validation for the task, the present study sought to operationalize one's ability to engage in implicit emotion regulation using only RT. As such, the RT data acquired from this paradigm was more highly valued compared to accuracy data. In pursuit of more clearly distilled RT data, the relationship between speed and accuracy in the experimental paradigm was manipulated: while Etkin et al. (2006) imposed a 1000 ms response window on each stimulus, with the stimuli disappearing and being marked as inaccurate if no response was given within the set time constraints, the present study removed this response window, meaning the stimuli progressed only when a response was given. Participants were, however, still instructed to respond both as quickly and as accurately as possible, and no participant took longer than the allotted 35 minutes to complete the protocol as a whole. The goal of this alteration was to afford participants adequate time to respond to more time-consuming stimuli such that any difficulties in resolving emotional conflict would be fully reflected in the behavioral data. In other words, if a participant with deficits in the realm of implicit emotion regulation had such difficulty with emotional conflict resolution that the incongruent trials took, on average, over 1000 ms to resolve, an unlimited response window would allow this effect to be registered in terms of RT instead of being marked as an incorrect response.

While the intent of this change was specific and goal-oriented, it is possible that it may have given rise to unintended downstream effects that fundamentally altered the manner in which participants interacted with the ECT. For example, as the removal of the 1000 ms response window afforded participants more time with which to familiarize themselves with each set of stimuli, this change could have rendered participants more susceptible to repetition priming – a phenomenon that was seen in the context of congruent trials and may have been responsible for driving the unexpected behavioral dissociation therein. Why this change would cause an opposite phenomenon to manifest itself within incongruent trials, however, proves to be a more difficult question. It is possible that increased opportunity for familiarization with the stimuli produced opposite effects within incongruent trials due to a sense of distrust associated with the presence of incongruent stimuli. The difficulty of responding to incongruent trials (reflected in the results by significantly higher mean RTs of incongruent trials compared to congruent trials) could have caused participants to second-guess category repeats (e.g., fearful face with happy word followed by another fearful face with happy word), opting to spend more *time* to double check their answer on category repetition trials to make sure they weren't falling into an incorrect pattern of response. This suspicion of category repetition could be equated to a "multiple-choice test effect", where students begin to get nervous after they fill in too many of any one letter choice on a multiple-choice exam.

Implicit Emotion Regulation

More detrimental in terms of the specific goals of this study, the removal of the response window present in the original ECT may have inadvertently destroyed the implicit component of this task. Although outliers in the behavioral data were removed through the process described in the Results section (meaning any trials to which participants took a disproportionately long time responding were not considered for further analysis), the potential downstream effects of such an alteration are undeniable. Indeed, the behavioral data revealed average RTs that were higher than Etkin et al.'s (2006) 1000 ms response window (the mean RT of incongruent category repetition

trials was, for example, 1016.41 ms), showing concrete and widespread effects of the altered response window on participant behavior. With these effects in mind, it seems likely that this paradigm failed in its goal to index implicit ER. This notion is supported on the level of the behavioral dissociation within the data, which shows little to no evidence of emotional conflict resolution, as well as on a conceptual level – as mentioned above, the absence of a response window may have allowed participants to familiarize themselves with the stimuli rather than forcing them to respond before more conscious processes take hold. In other words, rather than exhibiting participants' ability to engage in top-down emotional control, the odd patterns of dissociation within the behavioral data beg a more stimulus-driven explanation of the task's effects.

Clearly, the proposed mediation models and predicted associations were not supported. However, due to the reasons enumerated above, it is difficult to draw concrete conclusions about the relationship between alexithymia, depressive/anxious symptoms, and implicit ER using this data. The one index of implicit ER that may have retained some of its validity (as explained in the Results section) is the contrast between incongruent and congruent trials ($RT_1 - RT_C$). Based on the expected significant difference between these groups, it could be said that this measure maintained its intended role as an indicator of automatic emotional control. However, while regression analyses using $RT_1 - RT_C$ as a DV did not find significant relationships between this variable and total scores on the CES-D, GAD-7, or TAS-20, these results should also be tempered by the fact that the alteration to the ECT changed the way participants interacted with the task as a whole.

Strengths and Limitations

Besides the obvious limitation associated with the ECT's failure to index implicit ER ability, it is also important to note that the sample, being made up of majority White college students between the ages of 17 and 22, was fairly homogeneous. While this homogeneity may contribute to the internal consistency of the data and allow stronger conclusions to be drawn about populations congruent with this sample, it certainly curtails the degree of generalizability of the findings. What's more, this study was conducted using a non-clinical sample. This was consistent with the study's goal of exploring the effects of alexithymia and depressive/anxious symptoms on implicit ER on a subclinical level – a series of relationships that have been left largely uninvestigated by past research. However, due to the subclinical nature of the sample, it is possible that the present study lacked the sensitivity necessary to registers effects that may have been more apparent in a more clinical sample.

Furthermore, in addition to historical criticisms of the TAS-20 outlined in the general introduction, Cronbach's alpha scores of less than .70 on both the DDF and EOT subscales of the TAS-20 call into question the internal reliability of the scale. This suggests that these particular subscales of the TAS-20 may not be measuring a unitary construct. It is important to note, however, that the Cronbach's alpha for the scale as a whole was well over .70, indicating a more robust overall internal consistency.

Conclusion and Future Directions

Overall, these speculations on the interaction between emotional conflict resolution and response windows act as both an exploratory examination of the effects of a response window on conflict monitoring tasks and as a cautionary tale. Despite undermining the original intent of the task, the removal of the response window provided compelling findings on the nature of the ECT and other conflict-monitoring tasks. Interestingly, past research using non-emotional conflict monitoring tasks has not necessarily imposed a response window as part of the experimental design. These experiments, however, report only on the basic congruency effect, not the congruency effect as a function of the identity of the previous trial (e.g., Cohen & Shoup, 1997). Experiments that *do* break down their analysis to the level of cI, cC, iC, and iI (and witness appropriate dissociation of behavioral data as a result) tend to implement a response window that ranges from 30 ms in the context of simple stimuli to 1000 ms for more complex stimuli (e.g., Ullsperger, Bylsma, & Botvinick, 2005; Botvinick et al., 1999; Etkin et al., 2006). This suggests that while the basic congruency effect may be a durable effect, the congruency effect as a function of the identity of the previous trial may be more fragile. This conclusion is supported by my data, in which the simple removal of the response window seemed to have significantly interfered with the more nuanced, previous-trial-dependent congruency effect while maintaining the more basic incongruent-versus-congruent congruency effect.

While the present study failed to link the ECT to implicit ER, future studies that rely on this task to study implicit ER in alexithymia should be aware of several potentially confounding effects that alexithymia may have on this task. First, past research has found that individuals with alexithymia experience difficulties identifying a range of facial expressions, including fear and enjoyment (Parker et al., 1993, as seen in Gilbert, 2009). This could conceivably affect one's ability to engage with the emotional faces used in the ECT, resulting in poor performance that is unrelated to actual implicit ER ability. Additionally, individuals high in alexithymia have demonstrated difficulty matching verbal descriptions to emotional images (Lane et al. 1996). In the context of the ECT, this may cause individuals higher in alexithymia to experience less conflict between the emotional word and the emotional face during incongruent trials, which would decrease their need to engage in emotional control in the first place.

In general, future research should continue exploring the interplay between depressive/anxious symptoms and alexithymia in the context of implicit ER while considering the limitations of the ECT mentioned above. A replication of this study with an intact response window may provide more informative results about the nature of this relationship.

Study 2 Introduction

In Study 2, the relationship between ER and alexithymia in depression and anxiety was examined with regard to explicit, or effortful, ER. More specifically, Study 2 sought to determine whether or not conscious awareness of emotional processes was necessary to reap the benefits of an adaptive ER technique in which participants were explicitly instructed to engage: cognitive reappraisal. While Chen et al. (2011) found alexithymia to be associated with a reduced *tendency* to select adaptive ER techniques such as reappraisal, this study explored whether alexithymia affects one's *ability* to put said techniques into practice when prompted. To explore this question further, explicit ER must first be understood.

Explicit Emotion Regulation

As opposed to implicit ER – a process that occurs subconsciously – explicit ER involves the conscious and effortful modulation of one's emotional state and, on a theoretical level, requires some level of awareness and insight to properly implement (Gyurak et al., 2011). However, whether this insight involves an understanding of the specific emotions at hand or simply an understanding that engagement in ER is necessary in a given moment remains to be seen.

While the ability to modulate one's emotions in an effortful manner is, like other forms of ER, an important day-to-day skill, explicit ER is especially important in learning-based treatment settings. In techniques such as Cognitive Behavioral Therapy (CBT), where therapy facilitates the learning of adaptive cognitive strategies and schemas, a patient's ability to consciously engage in ER techniques is crucial for learning and overall therapeutic success. One form of CBT-inspired therapy in particular – Emotion Regulation Therapy, or ERT – focuses on instructing individuals with distress disorders such as GAD and MDD in the use of adaptive ER

techniques (Mennin, 2006), and is especially dependent on an individual's ability to explicitly regulate their emotions. Similarly, Dialectical Behavior Therapy (DBT; Linehan, 1993) includes skills that directly target distress tolerance and ER. What's more, as mentioned in Study 1, the boundary between the realms of implicit and explicit ER is porous: habitual use of an explicit ER strategy can render it implicit (Gyurak et al., 2011). This dynamic highlights the importance of explicit ER, as it suggests that one's ability to engage in explicit techniques may affect the quality of one's ultimate repertoire of automatic responses to emotion.

Reappraisal

There is a wide range of strategies that can be implemented, implicitly or explicitly, in the pursuit of regulating emotion. Research in ER has identified many such techniques, including cognitive reappraisal, distraction, labeling, suppression, and rumination. However, while their common goal may be to modulate emotion in some way, not all ER strategies lead to the same outcome. While certain strategies, such as suppression and rumination, have been found to be largely maladaptive and are implicated in various forms of psychopathology including depression and anxiety (Aldao et al., 2010), others have been associated with more successful reduction of negative affect. One ER technique that has been consistently identified as an adaptive method of regulation is cognitive reappraisal (Wolgast, Lundh, & Viborg, 2011). *Cognitive reappraisal* (reappraisal for short) refers to the generation of benign or positive interpretations of otherwise stressful situations (Gross, 1998b), and has been shown to reduce physiological signs of stress (Kim & Hamann, 2012; Yuan et al., 2015). Habitual use of reappraisal is related to greater positive affect, interpersonal functioning, and general wellbeing when compared to suppression (Gross & John, 2003).

It is important to note, however, that like most ER strategies, there is a contextual component to the success of reappraisal. For example, while reappraisal seems to be an adaptive ER strategy when dealing with uncontrollable stress (i.e., stress that stems from events outside of the individual's control), cognitive reappraisal ability was found by Troy et al. (2013) to be paradoxically associated with greater levels of depression in individuals with controllable stress. This suggests that, at least in the context of controllable stressors, taking direct action to change the stressor may be more effective than using a less action-based cognitive technique to simply rethink the stressor. What's more, its association with depression suggests that the use of reappraisal in this context may be a mechanism through which a sense of self-efficacy – a protective factor against depression (Maciejewski, Prigerson, & Mazure, 2000) – is undermined. In other words, if individuals with depression consistently use reappraisal as an alternative to more action-based problem solving techniques, it may contribute to a perceived lack of agency that may in turn maintain depressive symptoms. Overall, this study helps to qualify reappraisal's status as an infallible ER technique – rather than being a universally adaptive regulatory strategy, reappraisal may be most useful in the face of situational stressors that are outside of one's control.

In addition to being moderated by situational factors, the success of reappraisal can depend on the population in question. For example, in a task where participants monitored by electroencephalogram (EEG) were instructed to engage in reappraisal prior to viewing a number of negative and neutral words and images, Han et al. (2014) found reappraisal instantaneously helped to reduce attentional bias towards negative words in healthy controls. However, this effect was not immediately seen in participants with GAD. Instead, the negative bias persisted in the early stages of perception before the eventual initiation of successful down-regulation. In addition to the delayed onset of down-regulation, the effect of reappraisal differed between these populations by way of lateralization – down-regulation of negative emotion was found to be a left-lateralized process only in the case of individuals with GAD, where heightened left dorsolateral prefrontal cortex activity was observed during ER. These findings suggest that the act of reappraising may manifest itself differently in individuals with GAD than in healthy controls, with healthy controls being more able to reap the immediate benefits of reappraisal. For nonclinical symptoms of anxiety (i.e., state anxiety), however, reappraisal seems to be an effective technique. When asked to perform an impromptu speech, the implementation of reappraisal significantly reduced pre-speech anxiety when compared to other techniques such as suppression (Hofmann et al., 2009).

Reappraisal also plays an important role in depression. According to Troy et al. (2010), at high levels of stress, successful engagement in cognitive reappraisal (indexed by changes in skin conductance and self-reported sadness in response to sad film clips) was associated with fewer depressive symptoms. This suggests that the ability to successfully implement cognitive reappraisal may be an important protective factor and mediator in the relationship between stress and depression. Despite these benefits, past research has consistently shown that reappraisal is more frequently used by healthy controls (Garnefski & Kraaij, 2007), while individuals with depression are more likely to implement less adaptive ER techniques such as rumination and expressive suppression (D'Avanzato et al., 2013). While this dissociation manifests itself on a behavioral level, it may be rooted in neurobiology. According to Johnstone et al. (2007), who explicitly instructed individuals to engage in reappraisal during various tasks, healthy controls exhibited a negative, ventromedial prefrontal cortex (vmPFC)-mediated relationship between the left ventrolateral prefrontal cortex (vIPFC) and the amygdala during regulation. However, an

entirely different phenomenon was seen in depressed individuals, where a *positive* linkage between vmPFC and amygdala activity was observed. Additionally, by overlaying pupil dilation and functional MRI data, Johnstone et al. (2007) found that individuals with depression who expended more effort during reappraisal exhibited heightened amygdala, insula, and thalamus activation. The opposite was true of healthy controls. These findings are evidence of underlying pathophysiology that interferes with the ability of individuals with depression to engage in cognitive reappraisal – a deficit the authors suggest stems from maladaptive engagement of the lateral PFC-VMPFC-amygdala inhibitory circuit during regulation.

Physiological Profiles of Alexithymia

It is important to note that the precise nature of the physiological profile of alexithymia remains a point of contention in the literature. Certain bodies of research on the physiology of alexithymia suggest that individuals with alexithymia tend to have *decreased* responsiveness to emotional stimuli. Wehmer et al. (1995), for example, found that higher alexithymia corresponded to a reduced increase in heart rate and lower electrodermal response to emotion-provoking images, while Constantinou, Panayiotou, and Theodorou (2014) observed decreased autonomic reactivity in individuals with alexithymia as indexed by skin conductance and startle responses to emotional stimuli. Based on these studies, it is conceivable that a potential association with hyposensitivity may represent a physiological mechanism through which individuals with alexithymia exhibit decreased recognition for situations that require ER and subsequently fail to successfully regulate affect. If this were the case, and alexithymia was associated with a failure in ER *recognition* only, explicit instruction in ER may be particularly effective in the facilitation of successful regulation in alexithymia.

Despite the evidence for a pattern of decreased reactivity in alexithymia, a *hyper*-arousal model of alexithymia has also emerged, with multiple studies citing increased arousal states in individuals high in alexithymia (e.g., Eastabrook, Lanteigne, & Hollenstein, 2013; Friedlander et al., 1997; Luminet et al., 2004). An important clarification to be made in Friedlander et al. (1997), however, is that while higher *baseline* physiological arousal was found to be associated with alexithymia, individuals with alexithymia exhibited reduced changes in heart rate in response to disgusting scenes, suggesting lower *reactivity* to emotion-eliciting stimuli. One possible explanation for this phenomenon could be an interaction between baseline arousal and reactivity – in other words, it could be that higher baseline arousal may increase the threshold at which stimuli elicit a response, thereby decreasing reactivity. However, while Stone and Nielson (2001) also found an increased baseline level of arousal in individuals with high alexithymia in response to emotion-eliciting videos, *all* participants, regardless of alexithymia levels, showed an increase in physiological arousal in response to negative stimuli. Interestingly, although their physiological reactivity was comparable to their less-alexithymic counterparts, individuals high in alexithymia failed to report any change in subjective emotional intensity. This suggests that alexithymia may be associated with some level of decoupling between physiological arousal and subjective experience of emotion - a phenomenon that the authors posit may put individuals with alexithymia at risk for stress disorders. The presence of a decoupling between subjective emotional experience and physiological response to emotion in alexithymia has been more recently validated (Peasley-Miklus, Panayiotou, & Vrana, 2016), and serves as further evidence for alexithymia's proposed detrimental effect on ER: individuals who fail to recognize physiological changes in response to emotion may miss crucial bodily cues to engage in ER in a given moment. Keeping the bidimensional aspect of alexithymia in mind, the presence of this

decoupling may be reflective of individuals affected solely by the cognitive deficits of alexithymia, whose physiology reflects the presence of an emotional response but who have difficulty identifying or describing it on a subjective level.

Overall, these physiological findings suggest that alexithymia may be best characterized by some combination of the hypo- and hyper-arousal theories. Namely, alexithymia may be associated with higher baseline arousal, but less reactivity (or less awareness/identification of emotions, depending on the type of alexithymia in question) in the face of emotion-inducing stimuli. Bermond et al. (2010) help to clarify this point of contention by arguing that past studies fail to break apart alexithymia into its cognitive and affective components. Their findings suggest that the two dimensions of alexithymia affect different aspects of physiological response, with the affective component influencing galvanic skin response (GSR) peak amplitude while the cognitive component influences baseline GSR values. Further research suggests that these dimensions and their effects on physiology may be split along clinical/nonclinical lines. For example, Herbert, Herbert, and Pollatos (2011) found that within nonclinical samples, individuals with alexithymia demonstrate reduced interoceptive awareness and physiological reactivity that may reflect attenuated emotional experience. In MDD, however, alexithymia has been associated with heightened physiological arousal that may correspond to displacement behavior, or a compensatory reaction to difficulties in the cognitive regulation of emotions (Troisi et al., 2000). In this circumstance, it could be that nonclinical samples exhibit cognitive and affective deficits associated with alexithymia and therefore have diminished physiological reactivity and a subsequent reduced need for ER. By contrast, individuals with MDD may exhibit high capacities for experiencing affect but reduced cognitive functioning as a result of alexithymia, resulting in a positive feedback loop of intensified and unregulated negative

44

emotions (van der Velde et al., 2013).³ This explanation helps to reconcile the contrasting physiological evidence regarding alexithymia while also providing support for a potential alexithymia-mediated model of emotion dysregulation in psychopathology consistent with mediation model B (see Figure 1B).

Heart Rate Variability

Heart rate variability (HRV) refers to beat-to-beat variation in heart rate, and was first implemented in a clinical setting by Hon and Lee (1963), who noticed that distress in fetuses was preceded by alterations in the interval between heartbeats. Since then, HRV has come to be known as a non-invasive measure of autonomic nervous system activation invaluable to clinical research (Malik et al., 1996).

The heart is innervated by multiple sources. The parasympathetic nervous system (PNS), or the "rest and digest" system, affects heart rate via the vagus nerve, which releases acetylcholine that acts on the sino-atrial and atrio-ventricular nodes, resulting in decreased heart rate (Karim, Ali, & Hasan, 2011; Malik et al., 1996). The sympathetic nervous system (SNS), or the "fight or flight" system, influences heart rate through the release of epinephrine and norepinephrine, acting on the same nodes as the PNS and causing increased heart rate (Karim et al., 2011). HRV has been speculated to measure the ratio of the sympathetic versus the parasympathetic influence on heart rate, and is an indicator of the body's ability to regulate and respond adaptively to stress. In the context of this study, however, HRV data were analyzed with regards to parasympathetic influence only. This was done through the use of the high frequency component (HF): a frequency band of oscillations that corresponds, in normal subjects and under controlled conditions, to respiratory arrhythmia (Lombardi, 2002).

Although somewhat counterintuitive at first glance, *higher* HRV is more adaptive as it is an indicator of heightened flexibility in the face of environmental stressors (Dishman et al., 2000). For example, if an individual with well-developed regulation abilities becomes stressed, their heart rate will undergo an initial increase followed by a rapid return to baseline as they engage in regulatory processes. This process would be reflected in high HRV in this individual. In keeping with this notion, research has shown HRV to have an inverse relationship with perceived stress, such that individuals who perceive more stress have lower HRV (Dishman et al., 2000). Furthermore, the HF component of HRV has been shown to decrease under conditions of emotional strain and time pressure (Nickel & Nachreiner, 2003) as well as in anxiety-inducing situations (Jönsson, 2007) within the general population.

In terms of its relationship with psychopathology, low HRV is common in anxiety disorders (including GAD, panic disorder, and social phobia; Licht et al., 2009) and unmedicated depression with and without comorbid anxiety (Kemp et al., 2012). It is important to note, however, that the observed relationship between HRV and anxiety disorders may be mediated by medication – when controlling for the use of tricyclics, selective serotonin reuptake inhibitors (SSRIs), or other antidepressants, the relationship between HRV and anxiety disorders was reduced to the point of non-significance in one study (Licht et al., 2009). This finding calls into question whether HRV is related to the features of anxiety disorders themselves or simply the treatment associated with such disorders. In spite of this, Kemp et al. (2012) found depression with comorbid anxiety to have a stronger inverse relationship with HRV than depression alone, even in un-medicated populations. This finding suggests that comorbid anxiety may have a different physiological profile than anxiety alone. In the context of this study, HRV acted as a measure of one's ability to engage in explicitly instructed reappraisal by indexing the changes in PNS activation over the course of the experimental protocol via the HF component. Past research on ER and HRV has shown that individuals with greater regulatory abilities have higher baseline HRV (Appelhans & Luecken, 2006). What's more, according to Butler, Wilhelm, and Gross (2006) successful engagement in ER appears to increase HRV. In conjunction with the aforementioned negative relationship between HF and emotional strain (Nickel & Nachreiner, 2003), these findings informed the use of the HF component of HRV to operationalize participants' ER ability, with higher HF acting as an index of more successful ER and lower HF indicating less successful ER.

Hypotheses

While Johnstone et al. (2007) demonstrate the causes of reappraisal's failure on a neurobiological level, it remains unclear what psychological factors may influence an individual's ability to engage in reappraisal. Furthermore, past studies have associated the ability to engage in reappraisal with fewer depressive symptoms (Troy et al., 2010) and found reappraisal use to be more prevalent in healthy controls than individuals with depression or anxiety (Garnefski & Kraaij, 2007). Although these studies point to the difficulty of successful cognitive reappraisal in depression and anxiety, none broach the question of *why* these differences are seen.

As previously mentioned, Subic-Wrana et al. (2014) assessed participants' levels of emotional awareness and ER preferences and found emotional awareness to be positively correlated with reappraisal use. As the scale assessing emotional awareness (adapted from the Levels of Emotional Awareness Scale, or LEAS; Lane et al., 1990) ranged from implicit, or subconscious, to explicit, or conscious, emotional awareness, this means that participants with explicit emotional awareness were more likely to implement reappraisal. As such, Subic-Wrana et al. (2014) provide preliminary evidence for a model of emotion dysregulation that implicates a lack of explicit emotional awareness as a barrier to successful engagement in reappraisal. Indeed, it is conceivable that reappraisal requires some level of understanding of the emotion being experienced in order to fully conceptualize A) in what way the situation at hand is producing negative affect and/or B) how to reframe the situation in a way that will change or improve one's mood state. In this way, alexithymia could be said to mediate the relationship between depressive/anxious symptoms and ER ability consistent with Model B (Figure 1B). This is further supported by Chen et al. (2011), who established that the presence of alexithymia decreases one's likelihood to implement reappraisal. Based on these findings and in accordance with Model B, I predicted that alexithymia would mediate a negative relationship between depressive/anxious symptoms and explicit ER ability, indexed here by changes in HRV following stress induction and subsequent explicitly instructed cognitive reappraisal. In other words, I predicted that the proposed relationship between symptom severity and explicit ER ability would be attenuated when alexithymia was included as a predictor of explicit ER ability.

As it was performed in the context of explicit ER – in this case via instructions to engage in reappraisal – this study also sought to elucidate whether the association between alexithymia and ER failure that has been seen in past studies⁴ was due to individuals with alexithymia *missing the cue* to initiate regulation or *lacking the ability* to successfully regulate even when prompted to do so. In this case, the predicted mediating role of alexithymia in a negative relationship with ER corresponds to the latter, while a lack of association between alexithymia and emotion dysregulation may be indicative of the former.

Study 2 Method

Participants

Participants were 62 Bates College students (28 men, 33 women, 1 agender) recruited via an email announce system, Facebook events, or a Bates introductory psychology course, 12 of whom also participated in Study 1. Standardized consent forms were made available before the study. Participants received either course credit or \$5 as compensation. Additionally, all participants were entered in a raffle for one of three \$10 gift cards to an on-campus restaurant. All participants were at least 18 years of age (with the exception of one 17-year-old who continued with the study despite being informed of the study requirements) and were comfortable reading directions and questions in English. The sample was 17.7% Black, 69.4% White, and 19.4% Asian. 9.7% of the sample identified as Hispanic or Latina/Latino.

Materials

As in Study 1, the Toronto Alexithymia Scale (TAS-20; Bagby et al., 1994a), the Center for Epidemiologic Studies Depression scale (CES-D; Radloff, 1977), and the 7-item Generalized Anxiety Disorder scale (GAD-7; Spitzer et al., 2006) were used. For complete descriptions of these scales, see the Materials section of Study 1.

The Positive and Negative Affect Schedule (PANAS; Crawford & Henry, 2004). The PANAS is a self-report questionnaire that consists of two 10-item subscales – one for the measurement of positive affect, and one for the measurement of negative affect. For both subscales, higher scores correspond to higher levels of corresponding affect. The PANAS lists a series of emotion words, such as "Interested" and "Determined" in the case of positive affect, and "Upset" and "Scared" in the case of negative affect. While reading the emotion words, participants use a scale with 5 response categories (1 = *very slightly or not at all* and 5 = *extremely*) to indicate the extent to which they feel that way in the present moment.

Emotion Regulation Task (ERT; adapted from White & Soto, 2017). In this study, participants performed a modified emotion regulation task (ERT) adapted from White and Soto (2017). In the adapted ERT, a subjective and physiological baseline⁵ (B1) was established in which participants responded to the PANAS before sitting still for 2 minutes while their heart rate was recorded using an electrocardiogram (EKG). The EKG continued to record participants' heart rates for the remainder of the protocol. Next, participants performed an emotionally neutral writing task in which they were instructed to describe a nearby glass bowl by typing about it for three minutes (CW).

The investigator then informed participants that they would soon be performing a spurof-the-moment speech that would last for 2 minutes. Participants were told that this speech would be filmed and that the investigator would be evaluating their performance based on persuasiveness. A second subjective and physiological baseline was established after the presentation of this information (B2). Next, participants were presented with a prompt that instructed them to engage in a reappraisal writing task (R; "Write about all of the positive aspects of giving this speech, including what you may learn and how you may grow through the experience"; White and Soto, 2017). After writing for 3 minutes, the participants provided a third subjective and physiological baseline (B3).

Finally, participants received the topic of the speech ("Should the death penalty be a legal form of punishment for severe crimes?"; White & Soto, 2017) and performed a speech for 2 minutes in front of the investigator and a device that was set up to appear to be filming (S). The

participants then provided a final subjective and physiological baseline (B4). Figure 3 includes a diagram of the overall procedure.

PowerLab 4/25T Electrocardiogram and LabChart 8© (**ADInstruments, 2016**). The electrocardiogram used in this study was a PowerLab 4/25T device used to collect physiological data by means of an eight-channel polygraph and a microcomputer. The data extracted from this device were analyzed and cleaned using LabChart 8© software (ADInstruments, 2016). Data were analyzed to examine HRV, which acted as a measure of parasympathetic nervous system (PNS) arousal (Katona & Jih, 1975) with high HRV indexing high levels of PNS arousal. This was done through the use of the high frequency component, or HF – a frequency band of oscillations that corresponds, in normal subjects and under controlled conditions, to respiratory arrhythmia (Lombardi, 2002). In the context of this study, lower HF represented less successful ER, while higher HF represented more successful ER.

Procedure

After reviewing and signing an informed consent form with the researcher, participants responded to a number of questionnaires, including the CES-D, the GAD-7, and the TAS-20. All self-report measures and subsequent experimental stimuli were integrated into an online survey created using Qualtrics survey software (Qualtrics, 2015).

After responding to the initial questionnaires, the participants were acquainted with the EKG as a noninvasive device that causes no significant discomfort and is used to measure heart rate variability (HRV). They were then instructed to remove any jewelry. Next, the participants were asked if they are comfortable having electrodes placed on their lower ribcage and clavicle by the investigator. If so, the investigator adhered three pre-gelled electrodes to the right clavicle at the midclavicular line, on the last bone of the ribcage at the left midaxillary line and on the last

bone of the ribcage at the right midaxillary line, swabbing each area with alcohol beforehand. If the participant was uncomfortable with this procedure, the investigator clearly instructed the participant on how to adhere the electrodes, and, if participant comfort allowed, checked to make sure they were properly positioned. Once the electrodes were attached and the EKG was recording, participants performed the ERT (see Figure 3).

Finally, before receiving a debriefing form and reimbursement, participants responded to a demographics questionnaire. Like in Study 1, *all* participants, regardless of symptom endorsement, were provided with a list of mental health resources.

Study 2 Results

It is important to note that the term "HRV" in the following sections is synonymous with high frequency component, or HF, measured in units of microseconds squared (μ s²). In the context of this study, lower HF values represented less successful ER, while higher HF values represented more successful ER.

Screening

The SPSS outlier analysis function was used to identify and remove outliers from the raw HRV data. Only outliers classified as "extreme values" were removed, with the program using a formula of 1.5x interquartile range to identify these problematic data points.

In order to assess the level of engagement with the reappraisal task, all responses to the reappraisal writing prompt were carefully examined to determine whether or not the participant followed the instructions of the task (i.e., "Write about all of the positive aspects of giving this speech, including what you may learn and how you may grow through the experience"). Appropriate responses showed that the participant had read the prompt and followed instructions, and were classified as responses that listed at least one positive aspect associated with giving the

speech. Examples of appropriate responses seen in the data include "[The speech] will allow me to learn how to think on my feet" and "I can learn how to improvise more effectively and efficiently". An example of an inappropriate response was "I don't think I will grow much because of this speech". Responses that did not list any positive aspects associated with giving the speech were noted (n = 1). However, due to the low number of inappropriate responses, and the fact that an inability to follow reappraisal instructions could be considered an index of ER difficulty, the participant associated with this response was *not* removed from further analysis. This exercise showed, however, that all participants at least read the prompt and that all but one participant successfully followed the prompt's instructions.

Variables of Interest

To assess the success of the reappraisal exercise, the change in HRV between the postspeech-instruction baseline (B2) and the post-regulation baseline (B3) was examined (B3 - B2). The change in HRV between B2 and the final baseline of the protocol (B4) was also examined as an index of the longer-lasting effects of reappraisal (B4 - B2). For both change scores (B3 - B2 and B4 - B2), larger positive values corresponded to a decreased stress response and more successful engagement in reappraisal.

Manipulation Checks

In order to characterize the overall changes in HRV and subjectively reported affect over the course of the four baselines, three sets of paired samples t-tests were conducted to compare both mean HRV and mean subjectively reported affect at these time points. A Bonferroni correction was applied to account for Type I error associated with multiple comparisons, resulting in an alpha-level of .017 for the following paired samples t-tests. Consistent with the goal of the stress induction protocol, participants displayed significantly lower mean HRV (as indexed by a lower HF value) at B2 (post stress induction; $M = 1330.81 \mu s^2$, $SD = 1256.85 \mu s^2$) than at B1 (pre stress induction; $M = 1984.60 \mu s^2$, $SD = 2161.23 \mu s^2$) as revealed by a paired samples t-test contrasting the mean HF value of B1 with the mean HF value of B2, t(59) = 3.32, p = .002. This suggests that participants became significantly more stressed after learning that they would soon be performing a speech. This finding was supported by paired samples t-test that contrasted subjectively reported affect at B1 with subjectively reported affect at B2, where negative affect was significantly higher at B2 (M = 16.66, SD = 5.56) than at B1 (M = 13.39, SD = 3.77), t(60) = -5.97, p < .001, and positive affect was marginally significantly lower at B2 (M = 27.03, SD = 8.25) than at B1 (M = 28.80, SD = 7.82), t(60) = 2.38, p = .020.

Surprisingly, mean HRV at B3 (post reappraisal; $M = 1091.32 \ \mu s^2$, $SD = 900.40 \ \mu s^2$) was significantly *lower* than mean HRV at B2 (post stress induction; $M = 1330.81 \ \mu s^2$, $SD = 1256.85 \ \mu s^2$) as shown by a paired samples t-test, t(58) = 2.53, p = .014. This suggests that rather than reducing stress, the reappraisal task was associated with a persisting stress response. This finding, however, was not supported by subjectively reported affect, as shown by paired samples t-tests that revealed a significant difference in neither negative affect at B2 (M = 16.73, SD = 5.57) versus B3 (M = 16.25, SD = 6.24), t(59) = 1.26, p = .214, nor positive affect at B2 (M = 27.03, SD = 8.25) versus B3 (M = 26.33, SD = 9.00, t(60) = 1.62, p = .111.

Finally, a paired samples t-test comparing mean HRV at B3 (post reappraisal; $M = 1091.32 \ \mu s^2$, $SD = 900.40 \ \mu s^2$) with mean HRV at B4 (post speech; $M = 1082.82 \ \mu s^2$, $SD = 893.77 \ \mu s^2$) revealed no significant difference between these two points, suggesting that participants were equally stressed immediately following reappraisal as they were after

completing the speech. This finding was congruent with subjective data, as shown by paired samples t-tests that revealed a significant difference in neither negative affect at B3 (M = 16.21, SD = 6.19) versus B4 (M = 16.11, SD = 6.39), t(60) = 0.17, p = .870, nor positive affect at B3 (M = 26.29, SD = 9.00) versus B4 (M = 25.25, SD = 9.06, t(60) = 1.84, p = .071.

These analyses provided an understanding of a typical response to the experimental protocol, demonstrating how HRV and subjectively reported affect fluctuated and interacted over the course of the experiment. For a graphical representation of overall changes in HRV and in subjectively reported affect over time, see Figures 4 and 5, respectively.

Depression, Anxiety, and Alexithymia

All scales demonstrated acceptable levels of internal consistency. Cronbach's alphas for the CES-D and the GAD-7 were .80 and .85, respectively. Cronbach's alpha for the TAS-20 as a whole was .82. The DIF, DDF, and EOT subscales contained 7, 5, and 8 items and had Cronbach's alphas of .83, .76, and .65, respectively. In the present study, the PANAS was used at four time points. At the first time point, Cronbach's alphas for positive and negative affect were .87 and .78, respectively. At the second time point, Cronbach's alphas for positive and negative affect were .91 and .86, respectively. At the third time point, Cronbach's alphas for positive and negative affect were .92 and .88, respectively. Finally, at the fourth time point, Cronbach's alphas for positive and negative affect were .92 and .87, respectively.

Correlations. In the pursuit of a preliminary understanding of the relationships between the IVs (total score on the CES-D, the GAD-7, and the TAS-20) and the DVs of interest, a number of bivariate correlation analyses were performed. These and all following analyses use an alpha-level of .05 unless otherwise specified. The full results of these correlation analyses can be found in Table 2. To assess the relationships between participants' scores on depression (M = 13.84, SD = 6.20), anxiety (M = 5.36, SD = 3.97), and alexithymia (M = 42.29, SD = 9.86) scales – the CES-D, GAD-7, and TAS-20, respectively – bivariate correlation analyses were run between these variables. As in Study 1, these variables were highly positively correlated. Total score on the CES-D was significantly correlated with total score on the GAD-7, r(60) = .63, p < .001, as well as total score on the TAS-20, r(60) = .61, p < .001. GAD-7 and TAS-20 scores were also significantly correlated with one another, r(60) = .41, p = .001.

Bivariate correlation analyses were also performed to explore the relationships between depression, anxiety, alexithymia and subjective/physiological distress during the task. For clarity's sake, variables with no significant relationship to any of the IVs were excluded from Table 2. Of particular note, total score on the CES-D was significantly correlated with HRV during the speech (S), r(55) = -.30, p = .024, as well as during the control writing task (CW), r(56) = -.30, p = .022. Similarly, total score on the GAD-7 was significantly correlated with HRV during CW, r(56) = -.27, p = .042. In all cases, increased depression/anxiety corresponded to decreased HRV at these time points.

In the context of subjectively reported affect, total score on the CES-D was significantly correlated with subjectively reported negative affect at the first baseline (B1) and final baseline (B4), where higher depression corresponded with higher levels of negative affect at both B1, r(60) = .31, p = .016, and B4, r(60) = .33, p = .008. Total score on the GAD-7 was also significantly correlated with subjectively reported negative affect at these time points, where higher anxiety corresponded with higher levels of negative affect at B1, r(60) = .41, p = .001 and B4, r(60) = .44, p < .001. No such phenomenon was seen in the context of the TAS-20.

Contrary to predictions, bivariate correlation analyses did not find total scores on the CES-D, GAD-7, and TAS-20 to be significantly correlated with HRV change scores indexing relative success of reappraisal (B3 - B2, B4 - B2). However, when these change scores were calculated using subjectively reported negative affect instead of HRV, total score on the GAD-7 was found to be significantly correlated with B4 - B2, r(59) = .39, p = .002, suggesting some level of decoupling between perceived stress and physiological arousal in the context of anxiety.

Regression analyses.

Mediation analyses. In order to explore the proposed mediation models (see Figure 1) in the context of explicit ER, a number of simple linear regressions were conducted using both HRV and subjectively reported affect.

HRV. In the following analyses, the HRV change score B3 - B2 was used as the DV to index the immediate physiological effects of reappraisal, with larger values of this variable corresponding to a decreased stress response and more successful regulation.

First, to explore Model A (see Figure 1A), a simple linear regression was calculated to predict B3 - B2 based on total score on the TAS-20, yielding a marginally significant regression equation, $R^2 = .03$, F(1, 57) = 2.95, p = .091, where, surprisingly, higher total score on the TAS-20 predicted a *larger* B3 - B2 change score, $\beta = 15.83$, t(56) = 1.72, p = .091. In other words, alexithymia predicted a better reappraisal outcome. In order to determine whether this trend, though not very robust, would be reflected elsewhere in this model, the mediation analysis continued with further simple linear regressions that were calculated to predict total scores on the CES-D and the GAD-7 based on total score on the TAS-20. Both analyses yielded significant regression equations, $R^2 = .36$, F(1, 60) = 35.47, p < .001 with total score of the CES-D as the DV; $R^2 = .41$, F(1, 60) = 12.10, p = .001 with total score of the GAD-7 as the DV. Higher total

score on the TAS-20 predicted higher total scores on both the CES-D, $\beta = 0.38$, t(59) = 5.96, p < .001, and the GAD-7, $\beta = .17$, t(59) = 3.48, p = .001. Finally, simple linear regressions were calculated to predict B3 - B2 based on total scores on the CES-D and the GAD-7. A significant regression equation was yielded in neither the context of the CES-D, $R^2 = -.01$, F(1, 57) = 0.28, p = .602, nor the context of the GAD-7, $R^2 = .00$, F(1, 57) = 1.00, p = .320, and no further meditation analyses were performed.

These latter two simple linear regressions also failed to support mediation model B (see Figure 1B), as they established no significant relationship between total scores on the CES-D/GAD-7 and B3 - B2 change values. As such, no further meditation analyses were performed.

Subjectively reported affect. In the following analyses, the subjective change score PANAS3 – PANAS2 was used as the DV to index the immediate subjective effects of reappraisal, with larger values of this variable corresponding to a decreased stress response and more successful regulation in the case of positive affect and an *increased* stress response and *less* successful regulation in the case of negative affect.

First, to explore Model A (see Figure 1A) in the context of subjectively reported affect, two simple linear regressions were calculated to predict positive and negative affect, respectively, based on total score on the TAS-20. A significant regression equation was yielded in neither the context of positive affect, $R^2 = .03$, F(1, 59) = 2.04, p = .158, nor the context of negative affect, $R^2 = .00$, F(1, 58) = 2.07, p = .651, and no further mediation analyses were performed in this context.

To explore Model B (see Figure 1B), two simple linear regressions were calculated to predict positive and negative affect, respectively, based on total score on the GAD-7. A significant regression equation was yielded in neither the context of positive affect, $R^2 = .00$, F(1, R)

59) = 0.20, p = .659, nor the context of negative affect, $R^2 = .01$, F(1, 58) = 0.54, p = .466, and no further mediation analyses were performed in this context. Finally, two simple linear regressions were calculated to predict positive and negative affect, respectively, based on total score on the CES-D. A significant regression equation was yielded in neither the context of positive affect, $R^2 = .04$, F(1, 59) = 2.39, p = .127, nor the context of negative affect, $R^2 =$.02, F(1, 58) = 0.95, p = .335, and no further mediation analyses were performed in this context.

Task-specific analyses. Finally, simple linear regressions were calculated in an attempt to better characterize certain relationships that were established by correlation analysis (namely correlations between HRV [S] and CES-D scores, as well as HRV [CW] and both CES-D and GAD-7 scores; see Table 2).

First, a simple linear regression was calculated to predict HRV during the speech based on total score on the CES-D. In line with the correlational relationship that inspired it, this analysis established total score on the CES-D to be a predictor of HRV during the speech, yielding a significant regression equation, $R^2 = .07$, F(1, 56) = 5.37, p = .024, where higher score on the CES-D predicted lower HRV during the speech, $\beta = -24.71$, t(54) = -2.32, p = .024. Simple linear regressions were also calculated to predict HRV during the control writing task based on total score on both the CES-D, $R^2 = .07$, F(1, 56) = 5.54, p = .022, and the GAD-7, $R^2 =$.06, F(1, 57) = 4.33, p = .042. In both cases, higher total scores significantly predicted lower HRV during this task, $\beta = -34.00$, t(54) = -2.35, p = .022 in the case of the CES-D; $\beta = -51.14$, t(55) = -2.08, p = .042 in the case of GAD-7

Categorical Analyses. In order to investigate the effects of depressive/anxious symptoms and alexithymia on subjective and physiological emotional response over time, these constructs were examined on a categorical basis, with depression and anxiety being categorized based on

their recommended diagnostic cut-off points of 16 and 10, respectively (Radloff, 1977; Kroenke et al., 2007). Due to the fact that only 2 participants were above the TAS-20's recommended cutoff point for high alexithymia (61; Bagby et al., 1994a), alexithymia was categorized by dividing the sample into groups representative of "low" and "high" alexithymia after using median split techniques to establish a cut-off point of 42. Based on these methods of categorization, 20 of the 62 total participants were above the cutoff point for depression, 10 for anxiety, and 7 for both depression and anxiety. The groups representing low (corresponding to TAS-20 scores between 0 and 41) and high (corresponding to TAS-20 scores of 42 and above) alexithymia both contained 31 participants. With the largely subclinical nature of the sample in mind, it is important to temper the results outlined below with the knowledge that the power of these analyses was limited due to the small number of people above diagnostic cutoff points, especially in the context of the GAD-7.

Heart rate variability over time. Three one-way repeated measures ANOVAs were conducted to compare the effects of categorical depression, anxiety, and alexithymia, respectively, on HRV across all time points of the experimental protocol. In all cases, HRV was examined across seven time points – B1, CW, B2, R, B3, S, and B4 – in order to examine physiological distress associated with the experimental protocol.

CES-D. First, a one-way repeated measures ANOVA was run with CES-D group (high depression [n = 18] vs. low depression [n = 33]) as the between-subjects variable (IV) and HRV as the DV. Time (i.e., all seven time points) was included as the repeated-measures within-subject variable. The results of this analysis and subsequent analyses using HRV as the DV are presented in Table 3. As Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(20) = 202.05$, p < .001), degrees of freedom were corrected using Greenhouse-

Geisser estimates of sphericity ($\epsilon = 0.38$). While this analysis revealed a significant withinsubjects effect of time, F(2.25, 110.44) = 14.10, p < .001, neither a between-subjects effect of CES-D group membership on HRV nor a CES-D x Time interaction were found.

GAD-7. A second one-way repeated measures ANOVA was run with GAD-7 group (high anxiety [n = 8] vs. low anxiety [n = 43]) as the between-subjects IV and HRV as the DV. As before, time was included as the repeated-measures within-subject variable. As Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(20) = 200.55$, p < .001), degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = 0.38$). Similar to the previous analysis, while this analysis revealed a significant within-subjects effect of time, F(2.44, 111.26) = 5.72, p = .003, neither a between-subjects effect of GAD-7 group membership on HRV nor a GAD-7 x Time interaction were found.

TAS-20. Finally, a third one-way repeated measures ANOVA was run with TAS-20 group (high alexithymia [n = 25] vs. low alexithymia [n = 26]) as the between-subjects IV and HRV as the DV. Time was included as the repeated-measures within-subject variable. As Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(20) = 205.73$, p < .001), degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\epsilon = 0.37$). Similar to the previous two analyses, while this analysis revealed a significant within-subjects effect of time, F(2.23, 109.41) = 14.91, p < .001, neither a between-subjects effect of TAS-20 group membership on HRV nor a TAS-20 x Time interaction were found.

Subjectively reported affect over time. Three sets of one-way repeated measures ANOVAs were conducted to compare the effects of categorical depression, anxiety, and alexithymia, respectively, on subjectively reported affect across all baseline time points. In all

cases, subjectively reported affect was examined across four baseline time points – B1, B2, B3, and B4 – in order to examine subjective distress associated with the experimental protocol.

CES-D. First, a one-way repeated measures ANOVA was run with CES-D group (high depression [n = 20] vs. low depression [n = 40]) as the IV and subjective negative affect as the DV. Time (i.e., all four baseline time points) was included as the repeated-measures withinsubject variable. The results of this analysis and subsequent analyses using subjective negative affect as the DV are presented in Table 4. As Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(5) = 19.68, p = .001$), degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = 0.85$). This analysis revealed a significant within-subjects effect of time, F(2.55, 147.80) = 14.04, p < .001. A marginally significant main effect of CES-D group membership on negative affect was also found, F(2.55, 147.80) = 3.44, p = .069, indicating that members of the high depression group tended to report higher levels of negative affect (M = 17.28, SE = 0.76) than did members of the low depression group (M = 14.84, SE = 1.08) across time. No CES-D x Time interaction was found.

A one-way repeated measures ANOVA was also run with CES-D group (high depression [n = 18] vs. low depression [n = 42]) as the IV and subjective positive affect as the DV. As before, time was included as the repeated-measures within-subject variable. The results of this analysis and subsequent analyses using subjective positive affect as the DV are presented in Table 5. As Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(5) = 61.51, p < .001$), degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = 0.63$). This analysis revealed a significant within-subjects effect of time, F(1.90, 110.22) = 3.60, p = .033. Although analysis revealed no between-subjects effect of CES-D group membership on positive affect, a significant CES-D x Time interaction was found, F(1.90,

110.22) = 3.69, p = .030. To further characterize this interaction effect, simple effects were examined using a number of independent samples t-tests that compared positive affect of members of the high depression group to positive affect of members of the low depression group at B1, B2, B3, and B4. These analyses revealed a marginally significant simple effect of depression group membership on subjectively reported positive affect at B1 only. Namely, positive affect at B1 was reported to be marginally significantly lower by members of the high depression group (M = 26.10, SD = 7.55) than by members of the low depression group (M =30.19, SD = 7.62), t(60) = -1.98, p = .052. This effect was not seen at any other time point. For a visual representation of this effect, see Figure 6.

GAD-7. Next, a one-way repeated measures ANOVA was run with GAD-7 group (high anxiety [n = 10] vs. low anxiety [n = 50]) as the IV and subjective negative affect as the DV. Time was included as the repeated-measures within-subject variable. As Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(5) = 19.96$, p = .001), degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = 0.85$). As seen in Table 4, this analysis revealed a significant within-subjects effect of time, F(2.27, 111.26) = 5.72, p = .003, as well as a significant between-subjects effect of GAD-7 group membership on negative affect, F(1, 58) = 8.56, p = .004, indicating that members of the high anxiety group tended to report higher levels of negative affect (M = 19.63 SE = 1.46) than did members of the low anxiety group (M = 14.86, SE = 0.65) across time. A significant GAD-7 x Time interaction was also found F(2.54, 147.57) = 3.02, p = .040. To further characterize this interaction effect, simple effects were computed using a number of independent samples t-tests that compared negative affect of members of the high anxiety group to negative affect of members of the low anxiety group at B1, B2, B3, and B4. These analyses revealed a significant simple effect of anxiety

group membership on subjectively reported negative affect at B1 and B4 only. At B1, negative affect was reported to be significantly higher by members of the high anxiety group (M = 17.10, SD = 5.95) than by members of the low anxiety group (M = 12.69, SD = 2.70), t(60) = -2.30, p = .045. A similar phenomenon was seen at B4, where negative affect was reported to be significantly higher by the high anxiety group (M = 22.30, SD = 8.55) than by the low anxiety group (M = 14.83, SD = 5.15), t(60) = -2.67, p = .023. This effect was not seen at any other time point. For a visual representation of these effects, see Figure 7.

A one-way repeated measures ANOVA was also run with GAD-7 group (high anxiety [n = 9] vs. low anxiety [n = 51]) as the IV and subjective positive affect as the DV. As before, time was included as the repeated-measures within-subject variable. As Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(5) = 65.95$, p < .001), degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = 0.62$). As seen in Table 5, this analysis revealed no significant effects other than a marginally significant within-subjects effect of time, F(1.84, 106.95) = 2.88, p = .065.

TAS-20. Finally, a one-way repeated measures ANOVA was run with TAS-20 group (high alexithymia [n = 31] vs. low alexithymia [n = 29]) as the IV and subjective negative affect as the DV. Time was included as the repeated-measures within-subject variable. As Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(5) = 19.62, p = .001$), degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon = 0.85$). As seen in Table 4, this analysis revealed no significant effects other than a significant within-subjects effect of time, F(2.56, 148.54) = 14.02, p < .001.

A one-way repeated measures ANOVA was also run with TAS-20 group (high alexithymia [n = 31] vs. low alexithymia [n = 29]) as the IV and subjective positive affect as the

DV. As before, time was included as the repeated-measures within-subject variable. As Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(5) = 65.74$, p >.001), degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity ($\varepsilon =$ 0.62). As seen in Table 5, this analysis revealed no significant effects other than a significant within-subjects effect of time, F(1.85, 107.12) = 7.54, p = .001.

Study 2 Discussion

The purpose of this study was to examine the nature of the relationship between depressive/anxious symptoms, alexithymia, and explicit ER ability through two mediation models. More specifically, this study aimed to build a more complete understanding of how depressive/anxious symptoms and alexithymia affected one's ability to benefit from explicit instructions to engage in reappraisal.

While total scores on the CES-D, GAD-7, and TAS-20 were highly correlated with and predictive of one another, demonstrating the expected association between these three constructs, these relationships were consistent with neither proposed model (A or B). This was largely due to a lack of association between these variables and physiological change scores B3 - B2 and B4 - B2. The meaning of these change scores, however, were difficult to interpret due to an unexpected persisting stress response following reappraisal. It is important to note that alexithymia was, surprisingly, marginally associated with a *more positive* (or at least less negative) reappraisal outcome.

In order to explore the relationships between variables of interest outside of the context of change scores, categorical analyses were also conducted. While these analyses revealed no significant effects of group membership on HRV over time, several main and interaction effects of group membership on subjective affect were seen. These effects included a tendency for

individuals high in depression to report higher negative affect across time and lower positive affect particularly at B1, as well as a tendency for individuals high in anxiety to report higher negative affect across time, particularly at B1 and B4. These findings were supported by positive correlations between scores on the CES-D and GAD-7 and subjectively reported negative effect at B1 and B4. No such finding was seen in the context of alexithymia.

Reappraisal

The marked failure of reappraisal was perhaps the most striking finding in the context of the greater experimental design of this study – not only did explicitly-instructed reappraisal fail to increase HRV when comparing B3 (post-reappraisal) to B2 (post-stress induction), it paradoxically corresponded to a further drop in HRV. This failure was widespread, manifesting itself on some level across all participants, regardless of their relative endorsement of symptoms of alexithymia, depression, or anxiety. This pronounced post-reappraisal stress response indicated that, contrary to the goal of this manipulation, even participants on the healthier side of the spectrum failed to receive any benefits from reappraisal on a physiological level, instead becoming increasingly distressed. Interestingly, although average subjectively reported affect showed no subjective benefits to be associated with reappraisal, it did not reflect the same pattern of increased distress following reappraisal as the physiological data. However, this dissociation may be best explained by examining this phenomenon on a group-by-group basis, and will be discussed presently.

This unexpected pattern of physiological distress following reappraisal invites the question: what went wrong? Why did performing an ER technique that is widely held to be adaptive correspond with increased distress? To answer this question, an understanding of the goals and stipulations of reappraisal must be understood. As previously mentioned, reappraisal is

a technique in which individuals attempt to generate benign or positive interpretations of otherwise stressful situations (Gross, 1998b), and has been shown in the past to reduce physiological signs of stress (Kim & Hamann, 2012; Yuan et al., 2015) as well as increase HRV (Denson, Grisham, & Moulds, 2011). However, while reappraisal can be an effective ER strategy, its relative success may depend on when it is implemented. A process model of ER put forward by Gross (1998a) suggests that emotional cues elicit behavioral, experimental, or physiological emotional response tendencies that aim to facilitate adaptive responses to perceived stressors, eventually informing the emotional response itself. Within this model, it is speculated that regulation can occur at more than one stage. Namely, Gross (1998a) identified two major classes of ER – antecedent-focused and response-focused (see Figure 8). As seen in Figure 8, antecedent-focused regulation occurs before response tendencies are fully formed, while response-focused regulation attempts to manipulate response tendencies *after* their formation. For example, a student who was concerned about an upcoming paper could anticipate experiencing writing-induced stress and engage in an antecedent-focused technique to reframe the task as a fun, creative writing exercise (in this case, a form of reappraisal) before engaging with the paper. Alternatively, this student could use a response-focused technique to try to limit outward signs of anxiety (e.g., fidgeting; a form of suppression) while writing.

As indicated by the above example, reappraisal has been historically identified as an antecedent-focused ER strategy (Gross, 1998a), and past research has largely examined it as such (e.g., Denson et al., 2011; Kim & Hamann, 2012; Yuan et al., 2015). The present study, however, was based on past work that used reappraisal as a more response-focused ER strategy. For example, both Hofmann et al. (2009) and White and Soto (2017) asked participants to engage in reappraisal *after* stress induction and both associated reappraisal exercises with a

physiological change corresponding to reduced pre-speech distress. Similarly, in the context of this study, reappraisal was implemented after stress induction as a form of response-focused regulation. As such, reappraisal may have occurred after the formation of initial response tendencies, to use the language of Gross (1998a). Indeed, by examining the significant decrease in HRV from B1 to B2, along with corresponding changes in subjectively reported affect indicating increasing distress, it is clear that stress-related response tendencies were formed on both physiological and subjective levels prior to the reappraisal writing task taking place. With a stress response already formulating, it may have been too late to reframe the stressor in a positive light, rendering reappraisal ineffectual. In other words, based on Gross' (1998a) model of ER, a stressor that has already elicited a "locked in" response may be more resistant to antecedentfocused reframing – once the damage caused by the stressor is already done, decreasing distress may become more dependent on changing the response itself rather than reframing the cause. As to why reappraisal *increased* physiological indicators of stress, it stands to reason that an antecedent-focused ER strategy implemented too late in the emotional process may not only fail to alter the course of the emotional response, but may act as a reminder of the imminent stressful event. As the nature of reappraisal dictates, the prompt used in this experiment ("Write about all of the positive aspects of giving this speech, including what you may learn and how you may grow through the experience") required participants to actively reengage with the looming stressor of the speech. As this prompt A) failed to reduce negative affect and B) forced participants to think about a stressor, it is conceivable that, rather than helping participants to feel better about the situation, this task acted as a form of imposed rumination, focusing attention on the causes and consequences of distress rather than it's solutions (Nolan, Roberts, & Gotlib, 1998).

Overall, the findings of Study 2 point to the failure of reappraisal, at least in a responsefocused context,⁶ and serve to challenge its stereotypical role as an infallible ER technique. These unexpected results have several important implications regarding the adaptive use of reappraisal. First, although reappraisal is often categorized as an adaptive ER strategy, its relative success appears to be largely situational – individuals must understand not only how to implement reappraisal, but when. These findings are in line with a growing body of ER literature that suggests that no form of ER is inherently "good" or inherently "bad." Rather, its value is defined by the situation in which it is used. For example, Wolgast and Lundh (2017) found that distraction (an ER technique that has long been ambiguously placed on the spectrum from "maladaptive" to "adaptive") was adaptive (i.e., associated with well-being) when combined with active acceptance, or behavioral flexibility in the face of negative emotions, but maladaptive when combined with more avoidant strategies. On the other hand, while examining daily experience of positive and negative emotion using a daily diary technique, Brockman et al. (2017) found that strategies often presumed to be adaptive (such as reappraisal) conferred little to no benefit in the regulation of negative affect for close to half their sample. Troy et al. (2013) lend support to the presence of a contextual component of reappraisal by suggesting that the type of stressor involved in ER has an effect on its outcome, and that reappraisal used in the wrong situations can be associated with higher levels of depression. Similarly, the present study highlights the importance of choosing the right technique at the right time – not only can inappropriate engagement in reappraisal fail to decrease negative affect, it can actually *increase* distress on a physiological level. Together, these findings suggest that emotion dysregulation can occur not only on the level of strategy selection (e.g., choosing to use reappraisal over suppression), but also on the level of contextual strategy implementation (e.g., choosing when to

use reappraisal). This more nuanced model of emotion dysregulation conveys the import of ER education involved in therapies and other interventions – the individual must know not only the cognitive steps necessary to perform a certain ER strategy, but also at what point in the emotional process it should be implemented and under what circumstances it will be most effective. According to this model, reappraisal should be one of a larger repertoire of ER technique, each valuable in certain contexts. For example, this study found that reappraisal may fail in a context where the stressor cannot be anticipated or where Gross's (1998a) emotional response tendencies are already formulated, thereby highlighting the need for contextually adaptive response-based ER techniques.

Regardless of how it was conceptualized – antecedent-focused or otherwise – HRV data clearly indicated that nobody benefitted from reappraisal in this study, regardless of symptom severity or level of alexithymia. This phenomenon was reflected by the repeated measures ANOVAs performed with HRV as the DV that revealed no significant between subjects differences at any time point. The investigation of the proposed mediation models did, however, reveal a surprising trend towards significance: higher levels of alexithymia predicted a larger change from B2 to B3 at a marginally significant level. This suggests that individuals with alexithymia may have had more positive reappraisal outcomes. It is important to note, however, that due to the stress-inducing qualities of reappraisal in this study, this simply means that alexithymia was associated with a *less negative* reaction to reappraisal. As to why this effect was seen, individuals with alexithymia may have been spared from the generally detrimental effects of reappraisal by, ironically, the very feature that was expected to hinder their ER ability: a lack of emotional insight. These individuals, having presumably limited understanding of their emotions, may have had attenuated associations between the onset of negative affect and the

upcoming speech task. In other words, if reappraisal acted as a reminder of an imminent stressful event, it may not have caused as strong a resurgence of negative emotions in individuals with alexithymia as it did in the sample at large.

In any case, the fact that individuals high in alexithymia experienced the same effects of reappraisal, if not a marginally less-negative outcome, as their less-alexithymic counterparts answers a major research question of this study. Namely, this study asked if previously observed associations between alexithymia and a reduced tendency to utilize reappraisal were due to individuals with alexithymia missing the cue to initiate regulation or lacking the ability to perform the technique itself. The present results demonstrate that alexithymia has little effect on explicit ER *ability*, at least in the case of response-based reappraisal, and suggest that past studies (e.g., Chen et al., 2011; Subic-Wrana et al., 2014) may have been identifying an association between alexithymia/reduced emotional awareness and limited recognition of ER situations. This finding has important implications for the treatment of alexithymia, as it points to explicit ER strategies as a potentially effective method of overriding the ER deficits associated with alexithymia. For example, particular emphasis could be put on coaching individuals with alexithymia in the recognition of situations where engagement in ER is necessary, perhaps by relying on more concrete, non-emotional cues (e.g., teaching an individual to ask themselves "Was this an outcome I wanted or did not want?" to decide whether to engage in ER, rather than "Do I feel sad/disappointed/angry/etc.?"). It is important to note that this finding should be tempered by the fact that reappraisal generally manifested itself as a maladaptive ER strategy in this study. This means that a potential relationship between ER ability and alexithymia could have gone undetected due to the fact that *nobody* benefitted from reappraisal. Furthermore, this

implication should not be generalized to the context of antecedent-focused reappraisal until more research has been conducted.

Subjectively Reported Affect

Due to the failure of reappraisal on a general level, the stress induction at B2 marked the beginning of a downward spiral of distress (as shown by physiological data and largely agreeing subjective negative reports) that leveled off only after the speech was complete (see Figures 4 and 5). However, while patterns of physiological distress were consistent between depression, anxiety, and alexithymia groups, a different pattern emerged in subjectively reported affect.

Depression. For example, correlations between depression and subjectively reported negative affect suggested an association between depression and distress at B1 and B4, and a marginally significant between-subjects effect of depression on negative affect suggested an overall trend of increased subjective distress for individuals high in depression. Together, these findings point to an overall tendency for individuals high in depression to report more distress at multiple points during the task – a phenomenon that fits well with our understanding of depression as being characterized by persistent low mood (American Psychiatric Association, 2013). However, in addition to being closely associated with depressed mood, depression is also characterized by anhedonia, or a diminished ability to experience pleasure (Treadway, & Zald, 2011). This particular feature may be best indexed by the positive component of the PANAS, which, in addition to measuring the absence of distress, is indicative of an individual's level of engagement and excitement (using emotion words such as "interested, "enthusiastic, "inspired"; Crawford & Henry, 2004). In the context of subjectively reported positive affect, a significant interaction between depression and time was seen. This interaction was characterized by a tendency for individuals in the high depression group to report lower positive affect at the

beginning of the experiment (B1; see Figure 6). Based on these reports, it can be said that depression corresponded to a diminished sense of excitement about and interest in the upcoming study – a dissociation that may be indicative of some level of anhedonia among individuals high in depression. This finding is congruent with a growing body of literature that associates not only increased negative affect, but also dampened positive affect as a feature of depression, identifying both blunted momentary enjoyment of positive stimuli (Barch et al., 2016) and reduced ability to sustain subjective and neural responses to positive stimuli (McMakin, Santiago, & Shirk, 2009; Heller et al., 2009) among depressed individuals. Together, these phenomena involving both positive and negative affect serve to highlight the subjectively reported presence of classic MDD symptoms in a subclinical sample, shedding light on the difficulties associated with depression even on a nonclinical level.

Finally, the significant interaction effect exhibited in the context of subjectively reported positive affect may serve as evidence for decreased subjective reactivity among individuals high in depression. While participants in the low depression group began with relatively high reports of positive affect, the stress induction task brought them to levels of positive affect as low as those of the high depression group. However, no parallel change was seen among individuals high in depression. This suggests that, while they may report lower positive affect to begin with, individuals high in depression are less susceptible to further decreases in positive affect caused by changes in the environment. This phenomenon lends support to a theory of reactivity called emotion context insensitivity (ECI; Rottenberg, 2007). ECI suggests that depressed individuals exhibit reduced reactivity to emotional stimuli, both positive and negative – an effect that is exemplified in this study by the failure of a negative stimulus (stress induction via speech

instruction) to elicit a change in positive affect comparable to the change seen in the low depression group.

Anxiety. When comparing individuals high in anxiety to their healthier counterparts, similar dissociations in subjectively reported affect were seen. However, while members of the high anxiety group tended to report significantly higher levels of negative affect in general, no such effect was seen in terms of positive affect. Combined with the findings outlined above, this is supportive of past research that found elevated negative affect to be associated with both anxiety and depression, but reduced levels of positive affect to be associated with depression only (i.e., facilitated by depression-specific symptoms such as anhedonia; Watson, Clark, & Carey, 1988).

While positive affect was largely unaffected by anxiety group membership, a significant interaction effect between anxiety and time revealed individuals in the high anxiety group to report increased negative affect at both B1 and B4 (see Figure 7). In other words, unlike the low anxiety group, whose stress spiked following stress induction before leveling off after the speech was completed, members of the high anxiety group both started off on a more distressed level (at B1) and continued to increase in stress through the end of the experiment (B4). While the reports of increased negative affect at B1 could be explained by the heightened anticipatory anxiety characteristic of anxiety disorders such as GAD⁷ (Paulus & Stein, 2006), this effect did not hold true at B2, where both the high *and* low anxiety group reported comparable levels of distress in anticipation of the upcoming speech. A more nuanced explanation of this effect, therefore, is informed by past research that has associated GAD with *hypersensitivity to uncertain threat* (Gorka et al., 2017). In this scenario, this could mean that while the nature of the experiment was still uncertain (at B1), members of the high anxiety group were disproportionately distressed.

However, when the speech was announced and uncertainty was attenuated, this distress, while still high, was proportionate to that of the low anxiety group. As for the persisting subjective experience of distress at B4, this study suggests that anxiety may be associated with difficulty in returning to baseline after a stressful event. This conclusion is supported by Paulesu et al. (2010), who found that activation in brain regions corresponding to worry in individuals with GAD (medial prefrontal and anterior cingulate regions) persisted during resting states following exposure to worry-inducing sentences. Overall, these findings are indicative of the adverse effects that even subclinical anxiety can have on simple tasks performed in day-to-day life (e.g., participation in a psychology experiment leading to persisting subjective distress even after the experiment is over).

Decoupling of Physiological and Subjective Emotional Experience

While the findings outlined above point to a differentiation of subjective affective experience as a function of symptom severity, it is important to remember that no such dissociation was seen on a physiological level. This suggests that subclinical depression and anxiety may be associated with a decoupling of physiological and subjective emotional experience. Interestingly, while a similar phenomenon has been identified in alexithymia (Stone & Nielson, 2001; Peasley-Miklus et al., 2016), the decoupling effects seen in this study appeared to be independent of alexithymia group membership, between which subjective and physiological data largely agreed. What's more, this effect existed in the opposite direction of what would have been expected in the context of alexithymia – rather than failing to report a change in subjective affect, individuals high in depression and anxiety tended to over-report, expressing higher negative affect and lower positive affect despite displaying HRV that was statistically identical to healthier individuals. In the case of depression, this decoupling phenomenon may be indicative of a negatively biased self-focus, where individuals with symptoms of depression overestimate signs of their own distress. The presence of heightened self-focus has been linked to MDD in the past, and has often been paired with a negative attentional bias (Lemogne et al., 2011). For example, fMRI studies have found depression to be associated with increased resting state functional connectivity between the default mode network (DMN) – a brain region associated with self-referential thought – and brain regions such as the thalamus and other areas responsible for cognitive control (Sheline et al., 2010; Singh & Gotlib, 2014). This increased connectivity reflects a preoccupation with internally generated thought in depression – which may be negatively biased and ruminative – and represents a potential mechanism through which individuals with symptoms of depression may report negative affect that is disproportionate to their actual physiological arousal.

While it is certainly conceivable that a similar internally oriented negative bias is present in individuals high in anxiety, this phenomenon is less well documented in the context of GAD. A similar physiological/subjective decoupling effect could, however, be caused by an entirely different mechanism in anxiety. For example, one model of panic disorder (PD) that has received equivocal empirical support suggests that panic attacks stem from a "catastrophic misinterpretation" of autonomic arousal stimuli. This initial misinterpretation sets off a positive feedback loop of physiological distress and further misinterpretations, which eventually result in a panic attack (Austin & Richards, 2006). While PD and subclinical GAD are by no means identical (and the results of this study reflect no positive feedback loop), this model suggests that individuals with anxiety may exhibit heightened sensitivity to physiological arousal. In other words, rather than the cognitive bias towards negative and ruminative thought that is associated with depression, individuals high in anxiety may be more likely to read into their more physical bodily cues. In support of this notion, Hoehn-Saric et al., 2004 used ambulatory monitors to track physiological data over the course of 4 days, and found individuals with both PD and GAD to be more sensitive to bodily changes than non-anxious individuals. Together, these studies are indicative of an attentional bias to physiological arousal among individuals high in anxiety that may result in the decoupling of physiological and subjective emotional experience.

Overall, the comparison of subjective and physiological data indicates a common decoupling phenomenon within subclinical depression and anxiety. While the mechanisms behind this phenomenon may be unique to the separate constructs, an understanding of both may prove beneficial in therapeutic settings, where individuals with depressive and anxious symptoms could be instructed in techniques that combat tendencies to engage in negative, selffocused thinking and read into physiological cues, respectively.

Strengths and Limitations

First, it is important to note that certain potential influencers of participants' HRV were not controlled for in this study. Namely, the participants' body mass index (BMI) and psychiatric medication history was unknown. However, while studies such as Licht et al. (2009) found an observed relationship between HRV and anxiety disorders to be mediated by medication, the lack of significant relationship between symptoms of depression, anxiety, alexithymia and HRV suggests that this may be a nonissue. In other words, as HRV data were already considered as statistically identical across groups, further attenuation of group differences caused by controlling for medication would have no confounding effect on this study or its implications.

Furthermore, it would be negligent not to mention that several of the effects outlined above were of only marginal significance (with some teetering on the brink of significance, e.g., p = .054). While these findings and their implications were discussed within this study, they should be interpreted with caution. However, the interpretation of these marginally significant findings is still valuable, especially through a Fisherian lens, and may be indicative of trends that would be more pronounced in a more clinical sample. Categorical analyses should also be regarded critically due to A) low sample size in certain groups (e.g., high anxiety, n = 10) and correspondingly low power,⁸ and B) the use of categorical analysis to examine a largely dimensional variable (alexithymia). More specifically, alexithymia was categorized using median split techniques, which are inherently prone to power reduction (McClelland et al., 2015).

Finally, while this study examines both symptoms of depression and anxiety, analyses using these variables occurred independently. In other words, no analyses were conducted to compare comorbid anxiety and depression symptoms with the symptoms of either disorder as an orthogonal construct. The presence of symptom comorbidity should be considered when interpreting the results corresponding to one specific disorder or the other.

Conclusion and Future Directions

Overall, this study identified several interesting trends, each with an important implication. First, this study demonstrated the failure of reappraisal, across groups, as a response-focused ER strategy. This finding lends support to a contextual model of ER that, rather than suggesting that certain techniques are universally adaptive while others are universally maladaptive, determines the value of ER strategies on a situational basis. This model of ER holds important implications for the future treatment of emotion dysregulation as it suggests that "adaptive" ER techniques implemented at the wrong time may have unintended negative effects, and points to the need for a flexible and diverse repertoire of ER strategies. Second, this study *did not* find alexithymia to be associated with greater explicit ER difficulty. Although this finding should be interpreted with the knowledge of reappraisal's failure in mind, it could point to instructed reappraisal as a viable ER technique for individuals with alexithymia, especially if paired with a non-emotional strategy aimed at identifying when to use it in the first place. Third, this study found subjective trends corresponding to deficits of clinical depression and anxiety on a subclinical level. These included subjectively reported depressed mood and anhedonia in the case of subclinical depression, and a heightened subjective sensitivity to uncertain threat as well as difficulty returning to baseline following a stressful event in the case of subclinical anxiety. This is indicative of an intensified subjective emotional experience for individuals suffering from symptoms of depression and anxiety, and points to a need for symptom alleviation and intervention even on a subclinical level. Finally, this study demonstrated a decoupling of physiological and subjective emotional experience in the context of subclinical depression and anxiety, but not alexithymia. This decoupling phenomenon may represent increased negative self-referential thought (in the case of high depression) and a bias to over-interpret or read into physiological arousal (in the case of high anxiety). These potential mechanisms could be addressed by interventions designed to limit focus on and reduce negative biases regarding internal states and physiological cues.

Future research should continue to focus on ER from a contextual standpoint, identifying precisely when certain ER techniques are adaptive, and when they may backfire. Furthermore, a similar study conducted with reappraisal as an antecedent-focused strategy rather than a response-focused strategy may yield more telling results about the role of alexithymia in ER. Namely, such a study could foster an understanding of the effect of alexithymia on explicitly instructed reappraisal within a presumably more adaptive context. Finally, future research should

examine in more detail the potential decoupling between physiological and subjective emotional experience in depression and anxiety in the pursuit of intervention techniques that target the misor over-interpretation of physiological distress in these disorders.

General Discussion

The goal of the present studies was to explore the nature of the relationship between depressive/anxious symptoms and alexithymia in the context of both implicit and explicit ER. These studies yielded results that bear significant implications regarding the measurement of implicit ER in the case of Study 1, and the nature of reappraisal and its intersection with alexithymia, as well as the specific influences of depression and anxiety on emotional experience in the case of Study 2. While both studies yielded interesting findings independently, the failure of the implicit ER paradigm in Study 1 makes it difficult to draw broader comparisons between the effects of depression, anxiety, and alexithymia on implicit versus explicit ER. However, the findings of Study 2 do answer this research question in part, suggesting that alexithymia may not be a negative influence on explicit ER ability, at least in the case of response-based reappraisal. Furthermore, the highly significant relationships between scores on depression, anxiety, and alexithymia scales in both Study 1 and Study 2 are consistent with past research (Honkalampi et al., 2000; Onur et al., 2013) and are indicative of a strong association between these constructs even within subclinical populations.

Strengths and Limitations

As mentioned in Study 1, the use of subclinical samples in these studies was both a strength and a limitation. While it allowed for the validation of clinically supported deficits on a subclinical level, the nature of these samples may have attenuated certain effects that would have been more robust within a clinical sample. Especially in terms of alexithymia, the manifestation of which varies according to the nature of the population in which it is observed (de Haan et al., 2012; Karukivi & Saarijärvi, 2014), the findings of these studies cannot necessarily be generalized to clinical populations, where alexithymia may produce drastically different effects

on ER ability. Similarly, the relative homogeneity of the samples (72.3% White in Study 1, 69.4% White in Study 2), while beneficial in terms of internal validity, limits the generalizability of these studies' results.

Concerning the measurement of alexithymia, both studies exhibited limited internal consistency associated with the Externally Oriented Thinking (EOT) subscale of the TAS-20. This suggests that revision to the items making up this subscale may be beneficial to the measurement of this particular aspect of alexithymia. Finally, with the historical limitations of the TAS-20 in mind, neither study was able to deconstruct alexithymia into its cognitive and affective components, which limited the level of specificity that could be achieved in analysis. As a whole, however, both Study 1 and Study 2 found the overall scale to be internally consistent, suggesting that the TAS-20 succeeded at measuring a unitary construct whether or not it comprised both the cognitive and affective dimension of alexithymia.

Conclusion and Future Directions

Overall, these studies provide important insight into the relationships between depression, anxiety, alexithymia, and ER in a college environment – a setting where awareness of mental health issues has been rapidly on the rise. More specifically, Study 1 offers implications regarding the ECT and the measurement of implicit ER, while Study 2 helps to qualify reappraisal as a contextually adaptive ER technique while demonstrating unique effects of depression and anxiety on the emotional experience of a subclinical sample. While questions regarding the effects of depression, anxiety, and alexithymia on ER remain, especially in the context of implicit ER, these studies offer an initial glimpse into the intersections between these constructs and establishes a number of findings that can inform both future research as well as the treatment of subclinical psychopathology and emotion dysregulation.

Future research should continue to explore the relationships outlined in these studies, focusing on implicit ER and other forms of explicit ER, including antecedent-focused reappraisal. Furthermore, these experiments should be replicated within clinical populations to determine whether the effects seen in these studies are amplified or altered by the level of psychopathology within a given sample. Finally, the findings of the present studies (Study 2 in particular), should be further investigated in the pursuit of developing therapies and interventions that take into account the effects of alexithymia on ER as well as the unique challenges presented by depression and anxiety on a subclinical level.

References

ADInstruments. (2016). LabChart 8 [Computer software].

- Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review*, *30*(2), 217-237. doi:10.1016/j.cpr.2009.11.004
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. Washington, D.C: American Psychiatric Association.
- Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology*, *10*(3), 229-240. doi:10.1037/1089-2680.10.3.229
- Austin, D. W., & Richards, J. C. (2006). A Test of Core Assumptions of the Catastrophic
 Misinterpretation Model of Panic Disorder. *Cognitive Therapy and Research*, *30*(1), 53–68. https://doi.org/10.1007/s10608-006-9010-4
- Bagby, R. M., Parker, J. A., & Taylor, G. J. (1994). The twenty-item Toronto Alexithymia Scale:
 I. Item selection and cross-validation of the factor structure. *Journal of Psychosomatic Research*, *38*(1), 23-32. doi:10.1016/0022-3999(94)90005-1
- Bagby, R. M., Taylor, G. J., & Parker, J. A. (1994). The twenty-item Toronto Alexithymia Scale:
 II. Convergent, discriminant, and concurrent validity. *Journal of Psychosomatic Research*, 38(1), 33-40. doi:10.1016/0022-3999(94)90006-X
- Barch, D. M., Pagliaccio, D., & Luking, K. (2016). Mechanisms Underlying Motivational
 Deficits in Psychopathology: Similarities and Differences in Depression and
 Schizophrenia. In E. H. Simpson & P. D. Balsam (Eds.), *Behavioral Neuroscience of*

Motivation (pp. 411–449). Cham: Springer International Publishing. Retrieved from http://dx.doi.org/10.1007/7854_2015_376

- Beard, C., & Björgvinsson, T. (2014). Beyond generalized anxiety disorder: Psychometric properties of the GAD-7 in a heterogeneous psychiatric sample. *Journal of Anxiety Disorders*, 28(6), 547-552. doi:10.1016/j.janxdis.2014.06.002
- Berking, M., & Wupperman, P. (2012). Emotion regulation and mental health: Recent finding, current challenges, and future directions. *Current Opinion in Psychiatry*, 25(2), 128-134. doi:10.1097/YCO.0b013e3283503669
- Bermond, B., Bierman, D. J., Cladder, M. A., Moormann, P. P., & Vorst, H. M. (2010). The cognitive and affective alexithymia dimensions in the regulation of sympathetic responses. *International Journal of Psychophysiology*, 75(3), 227-233. doi:10.1016/j.ijpsycho.2009.11.004
- Bermond, B., Clayton, K., Liberova, A., Luminet, O., Maruszewski, T., Bitti, P. R., & ...
 Wicherts, J. (2007). A cognitive and an affective dimension of alexithymia in six
 languages and seven populations. *Cognition and Emotion*, 21(5), 1125-1136.
 doi:10.1080/02699930601056989
- Botvinick, M., Nystrom, L. E., Fissell, K., Carter, C. S., & Cohen, J. D. (1999). Conflict monitoring versus selection-for-action in anterior cingulate cortex. *Nature*, 402(6758), 179-181. doi:10.1038/46035
- Brackett, M. A., Mayer, J. D., & Warner, R. M. (2004). Emotional intelligence and its relation to everyday behaviour. *Personality and Individual Differences*, *36*(6), 1387-1402. doi:10.1016/S0191-8869(03)00236-8

- Brockman, R., Ciarrochi, J., Parker, P., & Kashdan, T. (2017). Emotion regulation strategies in daily life: Mindfulness, cognitive reappraisal and emotion suppression. *Cognitive Behaviour Therapy*, 46(2), 91-113. doi:10.1080/16506073.2016.1218926
- Butler, E. A., Wilhelm, F. H., & Gross, J. J. (2006). Respiratory sinus arrhythmia, emotion, and emotion regulation during social interaction. *Psychophysiology*, *43*(6), 612-622. doi:10.1111/j.1469-8986.2006.00467.x
- Campbell-Sills, L., & Barlow, D. H. (2007). Incorporating Emotion Regulation into
 Conceptualizations and Treatments of Anxiety and Mood Disorders. In J. J. Gross, J. J.
 Gross (Eds.), *Handbook of emotion regulation* (pp. 542-559). New York, NY, US:
 Guilford Press.
- Chen, J., Xu, T., Jing, J., & Chan, R. K. (2011). Alexithymia and emotional regulation: A cluster analytical approach. *BMC Psychiatry*, *11*, doi:10.1186/1471-244X-11-33
- Cole, P. M., Michel, M. K., & Teti, L. O. (1994). The development of emotion regulation and dysregulation: A clinical perspective. *Monographs of the Society for Research In Child Development*, 59(2-3), 73-100. doi:10.2307/1166139
- Colman, A. M. (2015). A dictionary of psychology (4th ed.). Oxford: Oxford University Press.
- Constantinou, E., Panayiotou, G., & Theodorou, M. (2014). Emotion processing deficits in alexithymia and response to a depth of processing intervention. *Biological Psychology*, *103*, 212-222. doi:10.1016/j.biopsycho.2014.09.011
- Cohen, A., & Shoup, R. (1997). Perceptual dimensional constraints in response selection processes. *Cognitive Psychology*, *32*(2), 128-181. doi:10.1006/cogp.1997.0648
- Crawford, J. R., & Henry, J. D. (2004). The Positive and Negative Affect Schedule (PANAS): Construct validity, measurement properties and normative data in a large non-clinical

sample. *British Journal of Clinical Psychology*, *43*(3), 245-265. doi:10.1348/0144665031752934

- D'Avanzato, C., Joormann, J., Siemer, M., & Gotlib, I. H. (2013). Emotion regulation in depression and anxiety: Examining diagnostic specificity and stability of strategy use. *Cognitive Therapy and Research*, 37(5), 968-980. doi:10.1007/s10608-013-9537-0
- de Haan, H., Joosten, E., Wijdeveld, T., Boswinkel, P., van der Palen, J., & De Jong, C. (2012).
 Alexithymia is not a stable personality trait in patients with substance use
 disorders. *Psychiatry Research*, *198*(1), 123-129. doi:10.1016/j.psychres.2011.09.027
- Denson, T. F., Grisham, J. R., & Moulds, M. L. (2011). Cognitive reappraisal increases heart rate variability in response to an anger provocation. *Motivation and Emotion*, 35(1), 14-22. doi:10.1007/s11031-011-9201-5
- Diborde, A., Berthoz, S., Perdereau, F., Godart, N., Corcos, M., & Jeammet, P. (2004). Validité du questionnaire d'alexithymie de Bermond et Vorst : Étude chez des sujets présentant des troubles du comportement alimentaire et chez des témoins. = Validity of the BVAQ:
 A study in eating disorder patients and controls. *L'encéphale: Revue de Psychiatrie Clinique Biologique et Thérapeutique*, *30*(5), 464-473. doi:10.1016/S0013-7006(04)95461-9
- Dishman, R. K., Nakamura, Y., Garcia, M. E., Thompson, R. W., Dunn, A. L., & Blair, S. N.
 (2000). Heart rate variability, trait anxiety, and perceived stress among physically fit men and women. *International Journal of Psychophysiology*, *37*(2), 121-133.
 doi:10.1016/S0167-8760(00)00085-4
- Eastabrook, J. M., Lanteigne, D. M., & Hollenstein, T. (2013). Decoupling between physiological, self-reported, and expressed emotional responses in

alexithymia. *Personality and Individual Differences*, *55*(8), 978-982. doi:10.1016/j.paid.2013.08.001

- Extremera, N., & Rey, L. (2015). The moderator role of emotion regulation ability in the link between stress and well-being. *Frontiers in Psychology*, 6
- Feldmanhall, O., Dalgleish, T., & Mobbs, D. (2013). Alexithymia decreases altruism in real social decisions. *Cortex, 49*(3), 899-904. doi:10.1016/j.cortex.2012.10.015
- Friedlander, L., Lumley, M. A., Farchione, T., & Doyal, G. (1997). Testing the alexithymia hypothesis: Physiological and subjective responses during relaxation and stress. *Journal of Nervous and Mental Disease*, *185*(4), 233-239. doi:10.1097/00005053-199704000-00003
- Garnefski, N., & Kraaij, V. (2007). The Cognitive Emotion Regulation Questionnaire:
 Psychometric features and prospective relationships with depression and anxiety in adults. *European Journal of Psychological Assessment, 23*, 141–149.
- Gilbert, A. M. (2009). The physiological response to implicit and explicit fear faces in alexithymia. *Dissertation Abstracts International*, 69, 7139.
- Gorka, S. M., Lieberman, L., Shankman, S. A., & Phan, K. L. (2017). Startle potentiation to uncertain threat as a psychophysiological indicator of fear-based psychopathology: An examination across multiple internalizing disorders. *Journal of Abnormal Psychology*, *126*(1), 8-18. doi:10.1037/abn0000233
- Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology*, 74(1), 224-237. doi:10.1037/0022-3514.74.1.224

- Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology*, 2(3), 271-299. doi:10.1037/1089-2680.2.3.271
- Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes:
 Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology*, 85(2), 348-362. doi:10.1037/0022-3514.85.2.348
- Günther, V., Rufer, M., Kersting, A., & Suslow, T. (2016). Predicting symptoms in major depression after inpatient treatment: The role of alexithymia. *Nordic Journal of Psychiatry*, 70(5), 392-398. doi:10.3109/08039488.2016.1146796
- Gyurak, A., Gross, J. J., & Etkin, A. (2011). Explicit and Implicit Emotion Regulation: A Dual-Process Framework. *Cognition & Emotion*, 25(3), 400–412. http://doi.org/10.1080/02699931.2010.544160
- Han, H. Y., Gan, T., Li, P., Li, Z. J., Guo, M., & Yao, S. M. (2014). Attentional bias modulation by reappraisal in patients with generalized anxiety disorder: an event-related potential study. *Brazilian Journal of Medical and Biological Research*, 47(7), 576–583. http://doi.org/10.1590/1414-431X20143622
- Heller, A. S., Johnstone, T., Shackman, A. J., Light, S. N., Peterson, M. J., Kolden, G. G., & ...
 Davidson, R. J. (2009). Reduced capacity to sustain positive emotion in major depression reflects diminished maintenance of fronto-striatal brain activation. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, 106(52), 22445-22450. doi:10.1073/pnas.0910651106
- Herbert, B. M., Herbert, C., & Pollatos, O. (2011). On the relationship between interoceptive awareness and alexithymia: Is interoceptive awareness related to emotional

awareness?. *Journal of Personality*, 79(5), 1149-1175. doi:10.1111/j.1467-6494.2011.00717.x

- Hofmann, S. G., Heering, S., Sawyer, A. T., & Asnaani, A. (2009). How to Handle Anxiety: The Effects of Reappraisal, Acceptance, and Suppression Strategies on Anxious Arousal. *Behaviour Research and Therapy*, *47*(5), 389–394.
 http://doi.org/10.1016/j.brat.2009.02.010
- Hoehn-Saric, R., McLeod, D. R., Funderburk, F., & Kowalski, P. (2004). Somatic symptoms and physiologic responses in generalized anxiety disorder and panic disorder: An ambulatory monitor study. *Archives of General Psychiatry*, *61*(9), 913-921.
 doi:10.1001/archpsyc.61.9.913
- Hon, E. H., & Lee, S. T. (1963). Electronic evaluation of the fetal heart rate. VIII. Patterns preceding fetal death, further observations. *Am J Obstet Gynecol*, 15(87), 814-826.
- Honkalampi, K., Hintikka, J., Tanskanen, A., Lehtonen, J., & Viinamäki, H. (2000). Depression is strongly associated with alexithymia in the general population. *Journal of Psychosomatic Research*, 48(1), 99-104. doi:10.1016/S0022-3999(99)00083-5
- Honkalampi, K., Saarinen, P., Hintikka, J., Virtanen, V., & Viinamäki, H. (1999). Factors associated with alexithymia in patients suffering from depression. *Psychotherapy and Psychosomatics*, 68(5), 270-275. doi:10.1159/000012343
- Ho, N. P., Wong, M. C., & Lee, T. C. (2016). Neural connectivity of alexithymia: Specific association with major depressive disorder. *Journal of Affective Disorders*, 193362-372. doi:10.1016/j.jad.2015.12.057
- Johnstone, T., van Reekum, C. M., Urry, H. L., Kalin, N. H., & Davidson, R. J. (2007). Failure to regulate: Counterproductive recruitment of top-down prefrontal-subcortical circuitry in

major depression. *The Journal of Neuroscience*, *27*(33), 8877-8884. doi:10.1523/JNEUROSCI.2063-07.2007

- Jönsson, P. (2007). Respiratory sinus arrhythmia as a function of state anxiety in healthy individuals. *International Journal of Psychophysiology*, *63*(1), 48-54. doi:10.1016/j.ijpsycho.2006.08.002
- Karim, N., Ali, S., & Hasan, J. A. (2011). Heart rate variability A review. Australian Journal of Basic and Applied Sciences, 7(1), 71-77.
- Karukivi, M., & Saarijärvi, S. (2014). Development of alexithymic personality features. *World Journal of Psychiatry*, 4(4), 91–102. http://doi.org/10.5498/wjp.v4.i4.91
- Karukivi, M., Vahlberg, T., Pölönen, T., Filppu, T., & Saarijärvi, S. (2014). Does alexithymia expose to mental disorder symptoms in late adolescence? A 4-year follow-up study. *General Hospital Psychiatry*, *36*(6), 748-752.
 doi:10.1016/j.genhosppsych.2014.09.012
- Katona, P. G., & Jih, F. (1975). Respiratory sinus arrhythmia: noninvasive measure of parasympathetic cardiac control. *Journal of Applied Physiology*, 39(5).
- Kemp, A. H., Quintana, D. S., Felmingham, K. L., Matthews, S., & Jelinek, H. F. (2012).
 Depression, comorbid anxiety disorders, and heart rate variability in physically healthy, unmedicated patients: Implications for cardiovascular risk. *Plos ONE*, 7(2), doi:10.1371/journal.pone.0030777
- Kim, S. H., & Hamann, S. (2012). The effect of cognitive reappraisal on physiological reactivity and emotional memory. *International Journal of Psychophysiology*, 83(3), 348-356. doi:10.1016/j.ijpsycho.2011.12.001

- Kroenke, K., Spitzer, R. L., Williams, J. B., Monahan, P. O., & Löwe, B. (2007). Anxiety disorders in primary care: prevalence, impairment, comorbidity, and detection. *Annals of Internal Medicine*, *146*(5), 317. doi:10.7326/0003-4819-146-5-200703060-00004
- Lane, R. D., Quinlan, D. M., Schwartz, G. E., Walker, P. A., & Zeitlin, S. (1990). The Levels of Emotional Awareness Scale: A cognitive-developmental measure of emotion. *Journal of Personality Assessment*, 55(1-2), 124-134. doi:10.1207/s15327752jpa5501&2_12
- Leahy, R. L. (2002). A model of emotional schemas. *Cognitive and Behavioral Practice*, *9*(3), 177-190. doi:10.1016/S1077-7229(02)80048-7
- Lemogne, C., Gorwood, P., Bergouignan, L., Pélissolo, A., Lehéricy, S., & Fossati, P. (2011). Negative affectivity, self-referential processing and the cortical midline structures. *Social Cognitive and Affective Neuroscience*, 6(4), 426-433. doi:10.1093/scan/nsq049
- Licht, C. M., de Geus, E. C., van Dyck, R., & Penninx, B. H. (2009). Association between anxiety disorders and heart rate variability in the Netherlands Study of Depression and Anxiety (NESDA). *Psychosomatic Medicine*, *71*(5), 508-518.
 doi:10.1097/PSY.0b013e3181a292a6
- Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York, NY, US: Guilford Press.

Lombardi, F. (2002). Clinical Implications of Present Physiological Understanding of HRV Components. *Cardiac Electrophysiology Review*, 6(3), 245–249. https://doi.org/10.1023/A:1016329008921

Lopes, P. N., Salovey, P., Côté, S., Beers, M., & Petty, R. E. (2005). Emotion regulation abilities and the quality of social interaction. *Emotion*, *5*(1), 113-118. doi:10.1037/1528-3542.5.1.113

- Luminet, O., Rimé, B., Bagby, R. M., & Taylor, G. J. (2004). A multimodal investigation of emotional responding in alexithymia. *Cognition and Emotion*,18(6), 741-766. doi:10.1080/02699930341000275
- Maciejewski, P. K., Prigerson, H. G., & Mazure, C. M. (2000). Self-efficacy as a mediator between stressful life events and depressive symptoms: Differences based on history of prior depression. *The British Journal of Psychiatry*, *176*373-378. doi:10.1192/bjp.176.4.373
- Malik, M., Bigger, J. T., Camm, A. J., Kleiger, R. E., Malliani, A., Moss, A. J., & Schwartz, P. J. (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *European Heart Journal*, *17*(3), 354-381.
 doi:10.1093/oxfordjournals.eurheartj.a014868
- Mann, L. S., Wise, T. N., & Shay, L. (1992). Factor analysis of the Toronto Alexithymia Scale:
 Elucidation of a polythetic construct. *Psychotherapy and Psychosomatics*, 58(1), 40-45.
 doi:10.1159/000288608
- Mayr, U., Awh, E., & Laurey, P. (2003). Conflict adaptation effects in the absence of executive control. *Nature Neuroscience*, *6*(5), 450-452.
- McClelland, G. H., Lynch, J. J., Irwin, J. R., Spiller, S. A., & Fitzsimons, G. J. (2015). Median splits, Type II errors, and false–positive consumer psychology: Don't fight the power. *Journal of Consumer Psychology*, 25(4), 679-689. doi:10.1016/j.jcps.2015.05.006
- McMakin, D. L., Santiago, C. D., & Shirk, S. R. (2009). The time course of positive and negative emotion in dysphoria. *The Journal of Positive Psychology*, 4(2), 182-192. doi:10.1080/17439760802650600

- Mennin, D. S. (2006). Emotion Regulation Therapy: An Integrative Approach to Treatment-Resistant Anxiety Disorders. *Journal of Contemporary Psychotherapy*, 36(2), 95-105. doi:10.1007/s10879-006-9012-2
- Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2002). Applying an emotion regulation framework to integrative approaches to generalized anxiety disorder. *Clinical Psychology: Science and Practice*, 9(1), 85-90. doi:10.1093/clipsy/9.1.8
- Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2005). Preliminary evidence for an emotion dysregulation model of generalized anxiety disorder. *Behaviour Research and Therapy*, 43(10), 1281-1310. doi:10.1016/j.brat.2004.08.00
- Mennin, D. S., Holaway, R. M., Fresco, D. M., Moore, M. T., & Heimberg, R. G. (2007).
 Delineating components of emotion and its dysregulation in anxiety and mood
 psychopathology. *Behavior Therapy*, 38(3), 284-302. doi:10.1016/j.beth.2006.09.001
- Mennin, D. S., McLaughlin, K. A., & Flanagan, T. J. (2009). Emotion regulation deficits in generalized anxiety disorder, social anxiety disorder, and their co-occurrence. *Journal of Anxiety Disorders*, 23(7), 866–871. http://doi.org/10.1016/j.janxdis.2009.04.006
- Nickel, P., & Nachreiner, F. (2003). Sensitivity and diagnosticity of the 0.1-Hz component of heart rate variability as an indicator of mental workload. *Human Factors*, *45*(57), 5–590.
- Nolan, S. A., Roberts, J. E., & Gotlib, I. H. (1998). Neuroticism and ruminative response style as predictors of change in depressive symptomatology. *Cognitive Therapy and Research*, 22(5), 445-455. doi:10.1023/A:1018769531641
- Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science*, 3(5), 400-424. doi:10.1111/j.1745-6924.2008.00088.x

- Onur, E., Alkın, T., Sheridan, M. J., & Wise, T. N. (2013). Alexithymia and emotional intelligence in patients with panic disorder, generalized anxiety disorder and major depressive disorder. *Psychiatric Quarterly*, 84(3), 303-311. doi:10.1007/s11126-012-9246-y
- Parker, J. A., Bagby, R. M., Taylor, G. J., Endler, N. S., & Schmitz, P. (1993). Factorial validity of the 20-item Toronto Alexithymia Scale. *European Journal of Personality*, 7(4), 221-232. doi:10.1002/per.2410070403
- Paulesu, E., Sambugaro, E., Torti, T., Danelli, L., Ferri, F., Scialfa, G., & ... Sassaroli, S. (2010).
 Neural correlates of worry in generalized anxiety disorder and in normal controls: A functional MRI study. *Psychological Medicine*, 40(1), 117-124.
 doi:10.1017/S0033291709005649
- Paulus, M. P., & Stein, M. B. (2006). An Insular View of Anxiety. *Biological Psychiatry*, 60(4), 383-387. doi:10.1016/j.biopsych.2006.03.042
- Peasley-Miklus, C. E., Panayiotou, G., & Vrana, S. R. (2016). Alexithymia predicts arousalbased processing deficits and discordance between emotion response systems during emotional imagery. *Emotion*, 16(2), 164-174. doi:10.1037/emo0000086
- Qualtrics. (2015). Qualtrics survey software [Computer software]. Provo, Utah: Retrieved from http://www.qualtrics.com.
- Radloff, L. S. (1977). The CES-D scale: A self report depression scale for research in the general population. Applied Psychological Measurement 1: 385-401.
- Renner, F., Lobbestael, J., Peeters, F., Arntz, A., & Huibers, M. (2012). Early maladaptive schemas in depressed patients: Stability and relation with depressive symptoms over the

course of treatment. Journal of Affective Disorders, 136(3), 581-590.

doi:10.1016/j.jad.2011.10.027

- Robinson, A., Safer, D. L., Austin, J. L., & Etkin, A. (2015). Does implicit emotion regulation in binge eating disorder matter?. *Eating Behaviors*, 18186-191.
 doi:10.1016/j.eatbeh.2015.05.011
- Rottenberg, J. (2007). Major Depressive Disorder: Emerging Evidence for Emotion Context Insensitivity. In J. Rottenberg, S. L. Johnson, J. Rottenberg, S. L. Johnson (Eds.), *Emotion and psychopathology: Bridging affective and clinical science* (pp. 151-165). Washington, DC, US: American Psychological Association. doi:10.1037/11562-007
- Salminen, J. K., Saarijärvi, S., Äärelä, E., Toikka, T., & Kauhanen, J. (1999). Prevalence of alexithymia and its association with sociodemographic variables in the general population of Finland. *Journal of Psychosomatic Research*, 46(1), 75-82. doi:10.1016/S0022-3999(98)00053-1
- Sheline, Y. I., Price, J. L., Yan, Z., & Mintun, M. A. (2010). Resting-state functional MRI in depression unmasks increased connectivity between networks via the dorsal nexus. *Proceedings of the National Academy of Sciences of the United States of America*, 107(24), 11020–11025. http://doi.org/10.1073/pnas.1000446107
- Sifneos, P. E. (1973). The prevalence of 'alexithymic' characteristics in psychosomatic patients. *Psychotherapy and Psychosomatics*, *22*(2-6), 255-262. doi:10.1159/000286529
- Singh, M. K., & Gotlib, I. H. (2014). The neuroscience of depression: Implications for assessment and intervention. *Behaviour Research and Therapy*, 6260-73. doi:10.1016/j.brat.2014.08.008

- Spitzer, C., Siebel-Jürges, U., Barnow, S., Grabe, H. J., & Freyberger, H. J. (2005). Alexithymia and Interpersonal Problems. *Psychotherapy and Psychosomatics*, 74(4), 240-246. doi:10.1159/000085148
- Spitzer, R. L., Kroenke, K., Williams, J. B., & Löwe, B. (2006). A Brief Measure for Assessing Generalized Anxiety Disorder. *Arch Intern Med*, 166(10), 1092. doi:10.1001/archinte.166.10.1092
- Stone, L. A., & Nielson, K. A. (2001). Intact physiological responses to arousal with impaired emotional recognition in alexithymia. *Psychotherapy and Psychosomatics*, 70(2), 92-102. doi:10.1159/000056232
- Subic-Wrana, C., Beutel, M. E., Brähler, E., Stöbel-Richter, Y., Knebel, A., Lane, R. D., &
 Wiltink, J. (2014). How is emotional awareness related to emotion regulation strategies and self-reported negative affect in the general population?. *Plos ONE*, *9*(3), doi:10.1371/journal.pone.0091846
- Treadway, M. T., & Zald, D. H. (2011). Reconsidering anhedonia in depression: Lessons from translational neuroscience. *Neuroscience and Biobehavioral Reviews*, 35(3), 537-555. doi:10.1016/j.neubiorev.2010.06.006
- Troisi, A., Belsanti, S., Bucci, A. R., Mosco, C., Sinti, F., & Verucci, M. (2000). Affect regulation in alexithymia: An ethological study of displacement behavior during psychiatric interviews. *Journal of Nervous and Mental Disease*, *188*(1), 13-18. doi:10.1097/00005053-200001000-00003
- Troy, A. S., Shallcross, A. J., & Mauss, I. B. (2013). A person-by-situation approach to emotion regulation: Cognitive reappraisal can either help or hurt, depending on the context. *Psychological Science*, 24(12), 2505-2514. doi:10.1177/0956797613496434

- Troy, A. S., Wilhelm, F. H., Shallcross, A. J., & Mauss, I. B. (2010). Seeing the Silver Lining: Cognitive Reappraisal Ability Moderates the Relationship Between Stress and Depressive Symptoms. *Emotion (Washington, D.C.)*, *10*(6), 783–795. http://doi.org/10.1037/a0020262
- Ullsperger, M., Bylsma, L. M., & Botvinick, M. M. (2005). The conflict adaptation effect: It's not just priming. *Cognitive, Affective & Behavioral Neuroscience*, 5(4), 467-472. doi:10.3758/CABN.5.4.467
- van der Velde, J., Servaas, M. N., Goerlich, K. S., Bruggeman, R., Horton, P., Costafreda, S. G.,
 & Aleman, A. (2013). Neural correlates of alexithymia: A meta-analysis of emotion processing studies. *Neuroscience and Biobehavioral Reviews*, *37*(8), 1774-1785. doi:10.1016/j.neubiorev.2013.07.008
- Vorst, H. M. (2001). Validity and reliability of the Bermond—Vorst Alexithymia Questionnaire. *Personality and Individual Differences*, 30(3), 413-434. doi:10.1016/S0191-8869(00)00033-7
- Watson, D., Clark, L. A., & Carey, G. (1988). Positive and negative affectivity and their relation to anxiety and depressive disorders. *Journal of Abnormal Psychology*, 97(3), 346-353. doi:10.1037/0021-843X.97.3.346
- Wehmer, F., Brejnak, C., Lumley, M., & Stettner, L. (1995). Alexithymia and physiological reactivity to emotion-provoking visual scenes. *Journal of Nervous and Mental Disease*, 183(6), 351-357. doi:10.1097/00005053-199506000-00001
- White, K. H., & Soto, J. A. (in preparation). The Role of Trait Anxiety in the Selection of Emotion Regulation Strategies in Response to Anticipatory Anxiety.

- Williams, L. E., Bargh, J. A., Nocera, C. C., & Gray, J. R. (2009). The unconscious regulation of emotion: Nonconscious reappraisal goals modulate emotional reactivity. *Emotion*, 9(6), 847-854. doi:10.1037/a0017745
- Wolgast, M., & Lundh, L. (2017). Is distraction an adaptive or maladaptive strategy for emotion regulation? A person-oriented approach. *Journal of Psychopathology and Behavioral Assessment*, 39(1), 117-127. doi:10.1007/s10862-016-9570-x
- Wolgast, M., Lundh, L., & Viborg, G. (2011). Cognitive reappraisal and acceptance: An experimental comparison of two emotion regulation strategies. *Behaviour Research and Therapy*, 49(12), 858-866. doi:10.1016/j.brat.2011.09.011
- Yang, Q., Tang, P., Gu, R., Luo, W., & Luo, Y. (2015). Implicit emotion regulation affects outcome evaluation. *Social Cognitive and Affective Neuroscience*, 10(6), 824-831.
- Yuan, J., Ding, N., Liu, Y., & Yang, J. (2015). Unconscious emotion regulation: Nonconscious reappraisal decreases emotion-related physiological reactivity during frustration. *Cognition and Emotion*, 29(6), 1042-1053. doi:10.1080/02699931.2014.965663
- Zich, J. M., Attkisson, C. C., & Greenfield, T. K. (1990). Screening for depression in primary care clinics: The CES-D and the BDI. *International Journal of Psychiatry in Medicine*, 20(3), 259-277. doi:10.2190/LYKR-7VHP-YJEM-MKM2

Footnotes

¹The strengths and weakness of "adaptive" and "maladaptive" ER techniques are discussed in more detail in the context of Study 2.

² In this study, this effect is referred to as both "emotional conflict priming" and "emotional conflict resolution".

³ This theory is at odds with Diborde et al.'s (2004) aforementioned finding that nonclinical samples with alexithymia exhibited deficits corresponding to the cognitive dimension of alexithymia *only* while both the cognitive *and* affective dimensions of alexithymia were implicated within a clinical population. This particular finding, however, was studied in the context of eating disorders and therefore cannot necessarily be generalized to distress disorders. ⁴ Past studies have largely examined a general association between alexithymia and ER techniques (e.g., Chen et al., 2011; Subic-Wrana et al., 2014) without explicitly instructing individuals to actually engage in ER.

⁵ In this study, the term "baseline" refers to inter-task periods in which participants completed the PANAS before being instructed to sit still for 2 minutes as to establish an unadulterated HRV data point. This term does not necessarily denote the absence or presence of an experimental manipulation.

⁶ It is possible that reappraisal succeeded as an antecedent-focused ER strategy by reducing negative affect during the speech itself, which took place *after* the reappraisal exercise. However, because all participants engaged in reappraisal, there was no control group and thus no way to determine the relative effectiveness of reappraisal at this time point.

⁷ Though more well-validated in social anxiety disorder.

⁸ Although power was optimized through the use of repeated-measures ANOVAs.

List of Tables

Table 1: Correlation Matrix – Study 1	102
Table 2: Correlation Matrix – Study 2	103
Table 3: Repeated Measures – HRV	104
Table 4: Repeated Measures – Subjectively Reported Negative Affect	105
Table 5: Repeated Measures – Subjectively Reported Positive Affect	106

2	3
5	۵
5	3
C	D
H	_

Sun
nmary
l fo
Interc
orrel
lations,
, Means
s, and
t Sta
, and Standard
Devi
iation.
sfor
Inde
penden
int Va
aria
bles c
ınd D
0
dent V
ari
ables
of Int
erest
- Sti
udy 1

Measure	I	2	3	4	S	6
1. CES-D Score						
2. GAD-7 Score	.71**					
3. TAS-20 Score	.59**	.49**	1			
4. RT _{el} - RT _{il}	09	02	.23	1		
5. RT _I - RT _C	06	.11	17	01	-	
6. RT ₁	.12	.22	04	22	.59**	l
M	16.58	6.82	46.94	4.41	97.76	989.24
SD	8.89	4.27	11.46	80.68	76.36	279.74

Tables

*. Indicates *p* < .05 **. Indicates *p* < .01

Н
50
9
5
\sim

Sun	
nmary	
of In	
of Intercorrel	
nrela	
elations, M	
5	
, and	
ans, and Standard Deviations for Indep	
rd D	
eviati	
ons f	
ts for In	
depei	
ıdent	
lent Variables and Dep	
ibles	
and D	
) eper	
ıdent	
Varid	
ibles	
of In	
ariables of Interest	
- St	
udy 2	

.63**	I									
.61**	.41**	I								
30*	27*	07	-							
30*	03	20	.36**	I						
.31*	.41**	21	20	Ĥ	I					
.33**	.44**	.19	21	18	**69.	I				
.07	.13	.22	34*	03	.15	.34**	I			
.01	.07	.09	24	13	.05	.13	.64**	I		
.13	.10	.06	12	.03	.11	.16	05	.09		
.21	.39**	.16	.01	07	.20	.52**	.13	.10	.26*	I
13.84	5.36	42.29	698.07	592.03	13.40		-233.33	-147.30		-0.54
6.20	3.97	9.86	690.65	507.53	3.74	6.37	708.91	810.80		4.44
Note: all HRV variables are expressed in units of µs ²	lls ²									
	.63** .61** 30* .31* .31* .01 .13 .13 .21 13.84 13.84	$.63^{**}$ $.61^{**} .41^{**}$ $30^{*}27^{*}$ $.30^{*}03$ $.31^{*} .41^{**}$ $.33^{**} .44^{**}$ $.07 .13$ $.01 .07$ $.13 .10$ $.21 .39^{**}$ $13.84 5.36$ $6.20 3.97$		** ** .41** *27*07 * .41**21 * .41** .19 .13 .22 .07 .09 .10 .06 .39** .16 5.36 42.29 698 3.97 9.86 690	*** .41** ** .41** ** .27* .07 ** .41** .20 .36** . ** .41** .21 .20 .36** . ** .41** .19 21 .20 . ** .44** .19 21 .20 . .13 .22 34* . .07 .09 24 .10 .06 12 .01 .01 .01 .01 .01 .5.36 42.29 698.07 59 50 50 3.97 9.86 690.65 50 50 50 50	*** .41** ** .41** ** .27* .07 ** .03 .20 .36** ** .41** .21 .20 .11 ** .41** .19 21 .10 .13 .22	***1**.41** $27*$ 07*0307 <t< td=""><td>*** *** .41** ** .41** * ** ** ** ** .41** ** .41** ** .41** </td><td>******$.41^{**}$**$.41^{**}$07$7^{*}$$.07$*$.03$$.20$$.36^{**}$$3$$.20$$.36^{**}$$.13$$.21$$20$$.11^{*}$$.19$$12$$.13$$.22$$34^{*}$$.07$$.09$$12$$.10$$.06$$12$$.39^{**}$$.16$$.39^{**}$$.16$$.39^{**}$$.16$$.39^{**}$$.16$$.39^{*}$$.16$$.397$$9.86$$690.65$$507.53$$3.74$$6.37$$708.91$$810.80$</td><td>*** *** .41*** *** .41*** * ** ** ** .41** <th< td=""></th<></td></t<>	*** *** .41** ** .41** * ** ** ** ** .41** ** .41** ** .41**	****** $.41^{**}$ ** $.41^{**}$ 07 7^{*} $.07$ * $.03$ $.20$ $.36^{**}$ 3 $.20$ $.36^{**}$ $.13$ $.21$ 20 $.11^{*}$ $.19$ 12 $.13$ $.22$ 34^{*} $.07$ $.09$ 12 $.10$ $.06$ 12 $.39^{**}$ $.16$ $.39^{**}$ $.16$ $.39^{**}$ $.16$ $.39^{**}$ $.16$ $.39^{*}$ $.16$ $.397$ 9.86 690.65 507.53 3.74 6.37 708.91 810.80	*** *** .41*** *** .41*** * ** ** ** .41** <th< td=""></th<>

**. Indicates p < .01

\$	t)	Ι.
¢		ľ
5	-	
¢	D	
¢		۵

Repea	Table
ited Measur	ω
ures for Heart Rate	
Heart Ra	
⁷ ariability Across All Time Point	
ross All	
Time Po	
vints	

		CES-D (depression vs. no depression)	s. no depression)	
Effect	Mean Square	df	F	q
Time	14276904.55	2.25	14.10	< .001
CES-D	4647013.04	1	1.78	.189
Time × CES-D	114455.47	2.25	0.11	.913
Error	1012433.30	110.44		
		GAD-7 (anxiety vs. no anxiety)	s. no anxiety)	
Effect	Mean Square	df	F	q
Time	5700377.81	2.44	5.72	.003
GAD-7	3913298.01	1	1.49	.228
Time × GAD-7	500541.98	2.27	0.50	.630
Error	997017.00	111.26		
		TAS-20 (high alexithymia vs. low alexithymia)	ı vs. low alexithymia)	
Effect	Mean Square	df	F	q
Time	5624820.89	2.23	14.91	< .001
TAS-20	15406.86	1	0.01	.940
Time × TAS-20	509296.20	2.23	0.50	.627
Error	1013864.24	109.41		

		CES-D (depression vs. no depression)	. no depression)	
Effect	Mean Square	df	F	q
Time	157.14	2.55	14.04	< .001
CES-D	320.13	1	3.44	.069
Time × CES-D	15.77	2.55	1.41	.246
Error	11.20	147.80		
		GAD-7 (anxiety vs. no anxiety)	no anxiety)	
Effect	Mean Square	df	F	р
Time	103.33	2.54	9.47	<.001
GAD-7	756.84	1	8.86	.004
Time × GAD-7	32.97	2.54	3.02	.040
Error	10.92	147.57		
		TAS-20 (high alexithymia vs. low alexithymia)	vs. low alexithymia)	
Effect	Mean Square	df	F	q
Time	158.65	2.56	14.02	<.001
TAS-20	72.42	1	0.75	.392
Time × TAS-20	5.50	2.56	0.49	.663
Error	11.32	148.54		

Repeated Measures for Subjectively Reported Negative Affect from B1 to B4

Table 4

		CES-D (depression vs	s. no depression)	
Effect	Mean Square	df F	F	q
Time	89.12	1.90	3.60	.033
CES-D	205.22	1	0.83	.366
Time × CES-D	91.38	1.90	3.69	.030
Error	24.78	110.22		
		GAD-7 (anxiety vs. no anxiety)	s. no anxiety)	
Effect	Mean Square	df	F	q
Time	77.74	1.84	2.88	.065
GAD-7	1.91	1	0.01	.931
Time × GAD-7	11.87	1.84	0.44	.117
Error	26.96	106.95		
		TAS-20 (high alexithymia vs. low alexithymia)	vs. low alexithymia)	
Effect	Mean Square	df	F	q
Time	203.05	1.85	7.54	.001
TAS-20	266.37	1	1.08	.302
Time × TAS-20	10.27	1.85	0.38	.108
Error	26.95	107.12		

Repeated Measures for Subjectively Reported Positive Affect from B1 to B4

Table 5



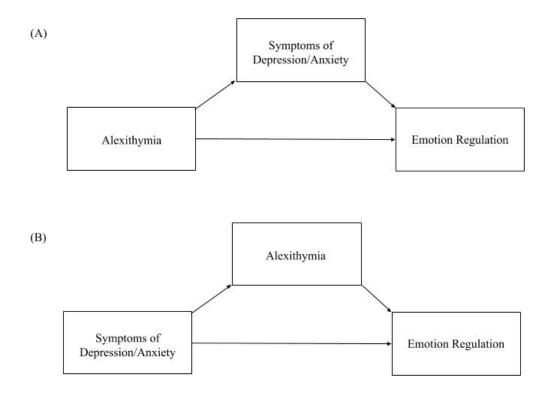


Figure 1. Two proposed mediation models for the relationship between alexithymia, symptoms of depression/anxiety, and ER.

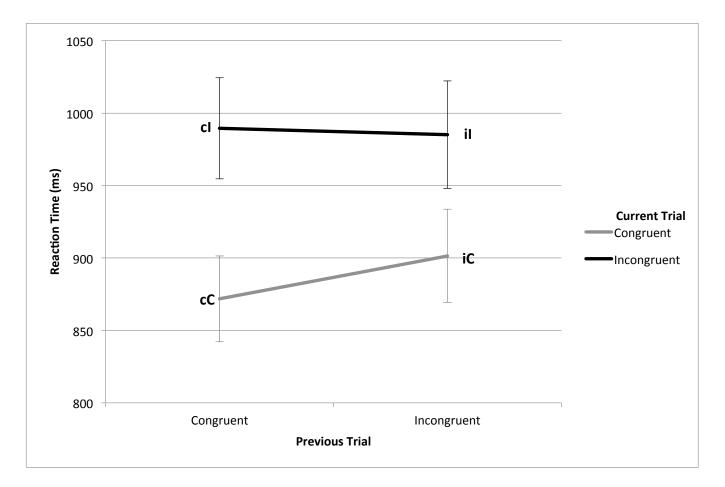
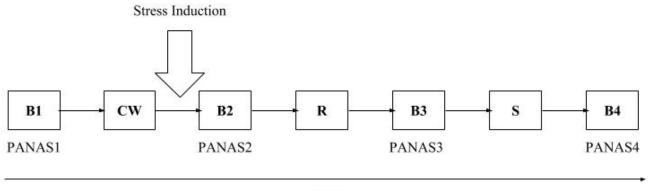
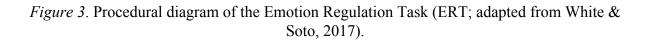


Figure 2. Changes to the congruency effect on trial *n* as a product of trial *n*-1.







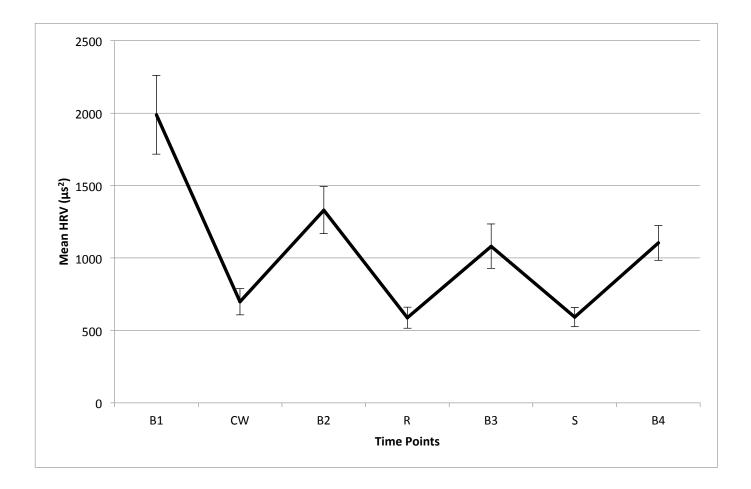


Figure 4. Mean heart rate variability (HRV) over time, across all participants.

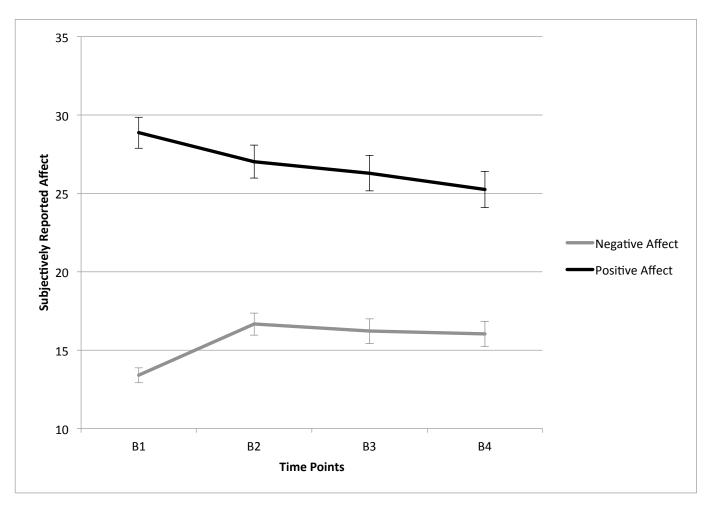


Figure 5. Mean subjectively reported affect over time, across all participants.

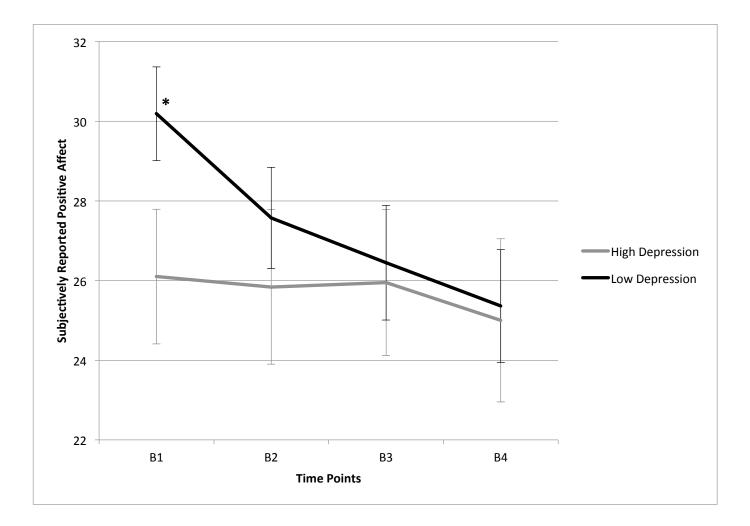


Figure 6. Subjectively reported positive affect as a factor of Group (high depression vs. low depression) x Time. Note: points of marginal significant difference (p = .054) are marked with an *.

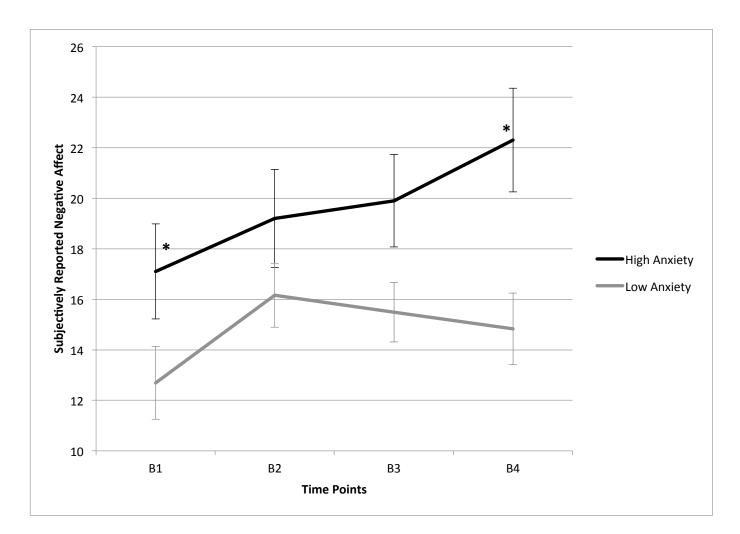


Figure 7. Subjectively reported negative affect as a factor of Group (high anxiety vs. low anxiety) x Time. Note: points of significant difference (p < .05) are marked with an *.

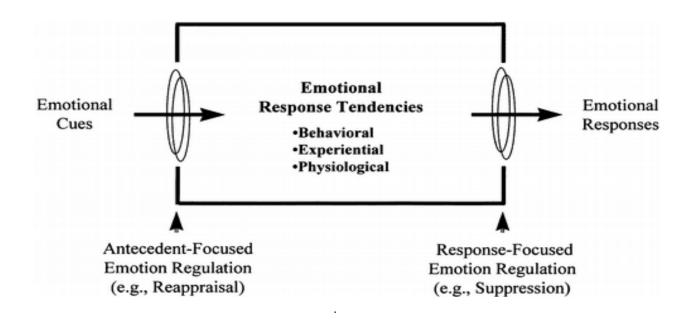


Figure 8. A process model of emotion regulation (adapted from Gross, 1998a).