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Cognitive Reappraisal Ability as a Protective Factor: Resilience to Stress Across Time and Context

Abstract

Emotion regulation is crucially involved in individuals' psychological health. For example, the frequent use of cognitive reappraisal, or changing the way one thinks about an emotional event, is positively associated with psychological health. Recent cross-sectional findings have shown that the ability to use cognitive reappraisal (cognitive reappraisal ability; CRA) is associated with lower depression in the context of high stress. However, two important questions about CRA remain unexamined: 1) Does CRA predict long-term adjustment to stress? 2) Do the protective effects of CRA depend upon the type of stress encountered? To examine these questions, a community sample of men and women (n=181) who had recently experienced a stressful life event was recruited and a prospective longitudinal design was employed. Life stress severity, stressor controllability, and depressive symptoms were measured at Time 1 and at Time 2 (6 months later). CRA was measured using a multi-method laboratory challenge at Time 1. The results of Question 1 revealed a significant prospective relationship in which CRA interacted with changes in life stress to predict changes in depression between Time 1 and Time 2. Specifically, among individuals with greater increases in stress, those with high CRA reported significantly smaller increases in depressive symptoms relative to those with low CRA. For Question 2, results indicated that the protective effects of CRA depend upon the stressors' controllability. Specifically, in highly stressful contexts that were uncontrollable, the protective effects of CRA remained, such that high CRA was associated with lower levels of depressive symptoms. In highly stressful contexts that were controllable, the protective effects of CRA were reversed, such that high CRA was associated with higher levels of depressive symptoms. Thus, CRA appears to be adaptive in uncontrollable but maladaptive in controllable stressful contexts. Overall, these results suggest that, for highly stressed individuals, CRA is an important protective factor against long-term increases in depression. Importantly, however, these protective effects depend upon the type of stressful context encountered. These results have important implications for understanding how emotion regulation ability contributes to risk and resilience in the face of stress, for clinical interventions and prevention programs, and for understanding what constitutes adaptive emotion regulation across contexts.

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COGNITIVE REAPPRAISAL ABILITY AS A PROTECTIVE FACTOR: RESILIENCE
TO STRESS ACROSS TIME AND CONTEXT

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Presented to
the Faculty of Social Sciences
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In Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

by

Allison S. Troy

June, 2012

Advisor: Iris B. Mauss

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Abstract

Emotion regulation is crucially involved in individuals' psychological health. For example, the frequent use of cognitive reappraisal, or changing the way one thinks about an emotional event, is positively associated with psychological health. Recent cross-sectional findings have shown that the *ability* to use cognitive reappraisal (cognitive reappraisal ability; CRA) is associated with lower depression in the context of high stress. However, two important questions about CRA remain unexamined: 1) Does CRA predict long-term adjustment to stress? 2) Do the protective effects of CRA depend upon the type of stress encountered? To examine these questions, a community sample of men and women ($n=181$) who had recently experienced a stressful life event was recruited and a prospective longitudinal design was employed. Life stress severity, stressor controllability, and depressive symptoms were measured at Time 1 and at Time 2 (6 months later). CRA was measured using a multi-method laboratory challenge at Time 1. The results of Question 1 revealed a significant prospective relationship in which CRA interacted with changes in life stress to predict changes in depression between Time 1 and Time 2. Specifically, among individuals with greater increases in stress, those with high CRA reported significantly smaller increases in depressive symptoms relative to those with low CRA. For Question 2, results indicated that the protective effects of CRA

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Table of Contents

Chapter One: Introduction1
 Reappraisal is Effective3
 Reappraisal is Adaptive7
 Reappraisal and psychological health.....7
 Reappraisal in the context of stress.....8
 The Importance of Cognitive Reappraisal *Ability*12
 Question One: Does Cognitive Reappraisal Ability Have *Long-Term Effects* on
 Psychological Health?.....17
 Question Two: Is Cognitive Reappraisal Ability Maladaptive in Specific Types of
 Stressful Contexts?.....21

Chapter Two: Method29
 Participants.....29
 Procedure30
 Measures32
 Life stress32
 Stressor controllability33
 Depression symptoms35
 Cognitive reappraisal ability36

Chapter Three: Results.....39
 Outlier Analysis39
 Data Analysis Strategy.....39
 Manipulation Check: Sadness Induction39
 Manipulation Check: Cognitive Reappraisal Instruction.....40
 Discriminant Validity of Cognitive Reappraisal Ability41
 Discriminant Validity of Stressor Controllability.....42
 Question One: Does Cognitive Reappraisal Ability Have Long-Term Effects on
 Psychological Health?43
 Cross-sectional effects of CRA.....43
 Secondary analyses of cross-sectional effects44
 Prospective effects of CRA.....46
 Secondary analyses of prospective effects.....47
 Question Two: Do the Protective Effects of Cognitive Reappraisal Ability Differ
 Depending on How Controllable the Context is?48
 Cross-sectional analysis using CRA-SAD.....48
 Cross-sectional analysis using CRA-SCL50
 Secondary analyses of cross-sectional effects51
 Longitudinal prospective effects of CRA across contexts.....52

Chapter Four: Discussion.....54
 The Prospective Effects of CRA.....55

Theoretical implications.....	56
Clinical implications	58
Discussion of null effects.....	59
The Protective Effects of CRA in Controllable Versus Uncontrollable Contexts.	61
Theoretical implications.....	63
Clinical implications	67
Discussion of null effects.....	67
Limitations and Future Directions	68
Concluding Comment	72
References.....	74
Appendices.....	85
Appendix A: Tables	85
Appendix B: Figures	98

Chapter One: Introduction

Humans are emotional beings. In many situations, emotions -- even intense ones -- can be adaptive: they allow us to avoid or escape threats in the environment, to take advantage of resources that may aid our survival, and to function socially (Ellsworth & Smith, 1988; Izard, 1977; Keltner & Kring, 1998). In some situations, however, emotions may be maladaptive. For instance, feelings of intense anxiety may prevent someone from delivering an effective presentation, or feelings of extreme anger may negatively affect performance in the workplace. In such cases, it is often more adaptive to *regulate* our emotions. Emotion regulation involves the use of behavioral and cognitive strategies to change the duration and/or intensity of an emotion (Gross & Thompson, 2007). Although human beings have likely engaged in emotion regulation since antiquity, this topic was not heavily studied using empirical methods until the twentieth century. The 1990's in particular saw a tremendous increase in research on emotion regulation, and the field has continued to grow. According to the online database PsycInfo, over 1400 articles listing "emotion regulation" as keywords have been published since 1999 (search conducted April 1, 2012).

Scientific interest in this topic may be due to the fact that emotion regulation is a common phenomenon in people's lives -- in modern society, humans encounter a wide range of situations that require some form of emotion regulation. Failure to effectively regulate emotions could have devastating consequences for psychological health. In

addition, individuals have a large number of different emotion regulation strategies to choose from (for reviews see Gross & Thompson, 2007; Koole, 2009) and each unique strategy appears to have a different profile of consequences in terms of benefits (e.g., decreases in the experience of unwanted emotions) and costs (e.g., maladaptive physiological responding or impaired social functioning). This repertoire of possible emotion regulation strategies includes strategies focused on changing behavior (e.g., suppression), attention (e.g., attention control, distraction), cognitions (e.g., cognitive reappraisal, acceptance), and physiological reactions (e.g., deep breathing techniques).

With such a wide variety of emotion regulation strategies at one's disposal, it is important to engage in strategies that are both effective and adaptive when used.

Throughout this paper, I define "effective" as an emotion regulation strategy that allows individuals to change their subjective experience of emotions in the desired direction without experiencing short-term costs. That is, effective emotion regulation strategies allow individuals to change their subjective experience of emotion without any detrimental physiological, social, or cognitive side effects. In contrast, I define "adaptive" emotion regulation strategies as strategies that contribute to longer-term beneficial psychological health outcomes. Based on both theoretical and empirical considerations, I will focus on one specific emotion regulation strategy that appears to be both effective in terms of allowing people to manage their emotions, and adaptive in terms of leading to positive psychological outcomes: cognitive reappraisal. This strategy has been defined as reframing the way one thinks about a stimulus in order to change its emotional impact (Gross & Thompson, 2007).

Reappraisal is Effective

The question of what constitutes effective emotion regulation may be best approached from the perspective of appraisal theory. The importance of appraisals in the generation and regulation of emotion has been written about and discussed for thousands of years, starting with philosophers such as Plato and Aristotle (cf. Schorr, 2001). Specifically, the Platonic view holds that emotions are weaknesses that occur outside of people's control and are antithetical to logic and reason. In other words, emotions simply happen without any regard to specific appraisals of the world around us (Lazarus, 2001). Aristotle argued, in contrast, that emotions are simply reflections of judgments and beliefs about the world – in other words, emotions are rooted in reason. Because people can control their judgments and beliefs, he argued, they can also control their emotions (Aristotle, 1941). This emphasis on evaluations as elicitors of emotion is in contrast to theories of emotion that have hypothesized that events themselves automatically elicit emotion (Watson, 1919), perhaps through automatically generated patterns of physiological arousal (James, 1890; Lindsley, 1951).

Inspired by the Aristotelian view of emotions, modern-day appraisal theorists have broadly defined appraisals as the meaning and significance a person assigns to an event or stimulus in the environment and are influenced by a person's current goals or needs (Roseman & Smith, 2001). Broadly, appraisal theorists believe that “the way we evaluate an event determines how we react emotionally” (Lazarus, 1999, p. 87). Thus, a person's appraisal of an event, *not the event itself*, causes an emotional reaction (Lazarus, 1991; Lazarus & Folkman, 1984; Scherer, Schorr, & Johnstone, 2001). In this way,

appraisal theory can explain the observation that the same event can lead to divergent emotional responses for different individuals.

Further, specific appraisals of an event should lead to the experience of specific emotions. For example, Lazarus theorized that events appraised as a loss are associated with the experience of sadness, while events appraised as a threat are associated with the experience of anger or fear (Lazarus, 1991). Research on appraisals supports Lazarus' argument -- people exposed to comparable events, either in the laboratory or in a naturalistic setting, display a wide variety of emotional reactions depending on their specific appraisals of the event (Folkman & Lazarus, 1985; Scherer & Ceschi, 1997; Siemer, Mauss, & Gross, 2007; Smith & Ellsworth, 1987). Conversely, individuals faced with very different events may experience the same emotional reactions if they engage in the same pattern of appraisals. For example, completely different events such as a breakup and a job loss could both lead to feelings of sadness if they are both appraised as losses, or could lead to feelings of relief and happiness if they are appraised as beneficial (Roseman & Smith, 2001).

Within this theoretical framework, then, appraisals are of critical importance in the emotion generation process. Thus, emotion regulation strategies that target appraisals should be particularly effective because these strategies would change the underlying cause of the emotion itself. Without a change to the underlying appraisal, however, it should be much more difficult to change the experience of an emotion because the existing cause of the emotion will persist. With this theoretical framework in mind,

cognitive reappraisal should be a particularly effective strategy because, by definition, it directly targets appraisals.

Cross-sectional laboratory studies have supported this hypothesis. Specifically, it has been shown that the use of cognitive reappraisal is effective in two important ways. First, cognitive reappraisal is associated with the successful down-regulation of negative emotions and second, the use of reappraisal is not associated with negative “side effects.” For example, Gross (1998) showed disgusting film clips to undergraduates and asked them to either reappraise their emotions, suppress their emotions, or simply watch the film. Results indicated that participants who suppressed their reactions to the film were able to decrease their outward expressions of disgust, but still experienced the same amount of disgust as those who simply watched the film. Those who suppressed their emotional responses also experienced more peripheral physiological responding than the reappraisal or control groups. In contrast, the group that reappraised had no significant increase in physiological responding and reported experiencing less disgust. Several other laboratory studies that have induced other negative emotions have found the same pattern of results: the use of reappraisal is associated with decreases in the experience of negative emotion without any increases in maladaptive physiological responding (Dandoy & Goldstein, 1990; Egloff, Schmukle, Burns, & Schwerdtfeger, 2006; Jackson, Malmstadt, Larson, & Davidson, 2000; Lazarus, Opton, Nomikos, & Rankin, 1965). Subsequent studies have found that cognitive reappraisal has no negative effects on memory for emotional events (Egloff et al., 2006; Richards & Gross, 2000) or social functioning (Butler, Egloff, Wilhelm, Smith, Erickson, & Gross, 2003; Lopes, Salovey, Cote, &

Beers, 2005), while other types of emotion regulation do. Thus, reappraisal seems to be a particularly effective emotion regulation strategy – it leads to decreases in the experience of negative emotion, but those who use it do not pay any physiological, cognitive, or social price.

Subsequent studies have illustrated that, in addition to down-regulating negative emotions, reappraisal can also be used to increase the experience of positive emotions. For example, Mauss and colleagues (Mauss, Cook, Cheng, & Gross, 2007) found that individuals who reported frequently using reappraisal as an emotion regulation strategy experienced less anger and less maladaptive physiological responding in a laboratory anger induction than those who did not report using reappraisal frequently. In addition, the frequent reappraisers reported experiencing more positive emotions during the anger task. Similarly, Haga and colleagues (Haga, Kraft, & Corby, 2009) found that the self-reported use of reappraisal was associated with both lower levels of negative affect as well as higher levels of positive affect. This lends support to the idea that reappraisal can be used effectively to decrease unwanted negative emotions, as well as to increase the experience of positive emotions.

Overall then, cognitive reappraisal appears to provide a very effective way to regulate emotions. People can use it to either down-regulate unwanted negative emotions, or to up-regulate positive emotions. In addition, those who engage in cognitive reappraisal do not appear to experience unwanted physiological, behavioral, or social costs.

Reappraisal is Adaptive

As illustrated above, cognitive reappraisal appears to be very effective for regulating emotions. In addition, cognitive reappraisal also appears to be adaptive – its use is associated with beneficial long-term psychological health outcomes. Broadly, I use the term “psychological health” to refer to greater positive (e.g., well-being, satisfaction with life) as well as lower negative (e.g., depression, anxiety) psychological health outcomes. Below, I review empirical evidence that supports the hypothesis that the use of reappraisal is associated with both of these facets of psychological health.

Reappraisal and psychological health. Few studies have examined the role of reappraisal and psychological health outcomes in healthy populations. The small number of studies that have been conducted, however, seem to converge on a common pattern. For example, Gross and John (2003; John & Gross, 2004) have examined the relationship between cognitive reappraisal use and positive outcomes over time. They found that the use of cognitive reappraisal, as measured with a self-report trait measure, was associated with greater overall levels of well-being, including measures of self-esteem, life satisfaction, and optimism. Haga et al (2009) similarly found that the use of CR was associated with higher levels of satisfaction with life in an international sample of undergraduates. These studies suggest that there are individual differences in daily reappraisal use, and that those who report using cognitive reappraisal are more likely to experience increased psychological health.

A large body of research has also examined the role of reappraisal use and decreases in negative psychological health outcomes. In several studies, Garnefski and

colleagues have found a robust negative relationship between self-reported use of reappraisal and depression (Garnefski, Baan, & Kraaij, 2005; Garnefski, Boon, & Kraaij, 2003; Garnefski, Kraaij, & Spinhoven, 2001; Garnefski & Kraaij, 2006). In a longitudinal study using a sample of older adults, the negative relationship between cognitive reappraisal and depression was replicated at a two and a half year follow-up session, which suggests that this relationship persists over time (Kraaij, Pruymboom, & Garnefski, 2002). The self-reported use of cognitive reappraisal is also associated with less trait anxiety (Egloff et al., 2006). Indeed, a recent meta-analysis found that self-reported reappraisal use (in both clinical and non-clinical samples) is associated with fewer symptoms of depression and anxiety, which suggests that the link between reappraisal and these two outcomes is robust (Aldao, Nolen-Hoeksema, & Schweizer, 2010). In addition, the use of reappraisal has been associated with fewer symptoms of acute stress disorder and post-traumatic stress disorder (Bryant, Moulds, & Guthrie, 2001; Fairbank, Hansen, & Fitterling, 1991). Taken together, these studies suggest that there is a robust association between self-reported reappraisal use and decreased negative psychological health outcomes such as depression. Specifically, those who report frequently engaging in reappraisal are less likely to experience a range of psychological health problems.

Reappraisal in the context of stress. The studies reviewed so far have examined the main effect between reappraisal use and psychological health. However, the use of reappraisal should be particularly important in one specific context – that of high life stress. That is, reappraisal may act as an important protective factor against negative

outcomes by supplying an effective way to manage the negative emotions that accompany stress.

Exposure to stressful life events is associated with increased risk for a host of negative outcomes, including negative physical health outcomes (Feldman, Bensing, & deRuijter, 2007; Koh, Choe, Song, & Lee, 2006; Miller & Blackwell, 2006) and a host of negative psychological health outcomes including depression (Caspi, Sugden, Moffitt, Taylor, Craig, Harrington, et al., 2003; Hawley, Ho, Zuroff, & Blatt, 2007; Monroe, Slavich, Torres, & Gotlib, 2007; Tennant, 2002), anxiety (Tsoory, Cohen, & Richter-Levin, 2007), and post-traumatic stress disorder (Brewin & Holmes, 2003). Importantly, however, this is only true *on average*. Indeed, most people who are exposed to stressful life events do not exhibit any of these unwanted consequences. In a recent study, for example, 60% of people who had recently experienced traumatic injury displayed low levels of depressive symptomatology over a six month period (deRoon-Cassini, Mancini, Rusch, & Bonanno, 2010). These results are in line with existing evidence that has shown that the majority of people exposed to very serious stressful events such as the death of a loved one can be characterized as resilient – that is, even after exposure to severe stress or trauma, most people maintain normal levels of psychological functioning (Bonanno, 2004; 2005; Ong, Bergeman, Bisconti, & Wallace, 2006). Impressively, many people even experience positive outcomes or personal growth after stress exposure (Park, Mills-Baxter, & Fenster, 2005; Woodward & Joseph, 2003).

These findings raise an important question: Could the use of cognitive reappraisal be a particularly important contributor to psychological health in the context of high

stress? Recently, there has been increasing interest in emotion regulation as a critical contributor to resilience in the context of stress (for a review see Troy & Mauss, 2011). This increased interest may be due to the finding that the experience of stress is inherently emotional – exposure to stress tends to lead to increases in negative emotions (Feldman, Cohen, Lepore, Matthews, Kamarck, & Marsland, 1999). From this perspective then, unregulated negative emotions may serve as an important mediator of the relationship between stress and psychological health problems. Within this framework then, those individuals who can effectively regulate their emotions using a strategy like cognitive reappraisal may be protected against psychological health problems in the context of stress by directly targeting and regulating this increase in negative emotions (Troy & Mauss, 2011). Specifically, the use of positive reappraisal, which consists of reframing emotional events in a more positive light, may be a particularly adaptive strategy in the context of high stress (Troy et al., 2010).

Several studies on positive reappraisal in highly stressed populations provide initial support for this hypothesis. For example, in a sample of highly stressed people caring for patients with Multiple Sclerosis, Pakenham (2005) found that the self-reported use of reappraisal was associated with a lower incidence of negative psychological health outcomes. Similar results have been found in patient populations with chronic medical problems (van der Veek, Kraaij, Van Koppen, Garnefski, & Joeke, 2007; Young & McNicoll, 1998) and individuals exposed to trauma (Moore, Zoellner, & Mollenholt, 2008). Additionally, Folkman & Moskowitz (2000a) noted that among caregivers for

AIDS patients, those who frequently engaged in cognitive reappraisal experienced more positive emotions both during care giving and after the death of the patient.

Extending these findings by using an experimental intervention in a sample of HIV positive men, Carrico and colleagues examined the moderating effects of a cognitive-behavioral stress management (CBSM) intervention on the relationship between stress and depression (Carrico, Antoni, Weaver, Lechner, & Schneiderman, 2005). Over the ten-week treatment period, the men who received the CBSM intervention showed significant decreases in depressive symptomatology, and this decrease was mediated by self-reported increases in the use of reappraisal, which suggests that the use of reappraisal may have been the mechanism of change in these patients. A subsequent study administered an internet based intervention for individuals experiencing complicated grief (Wagner, Knaevelsrud, & Maercker, 2007). Importantly, one of the three modules was dedicated solely to cognitive reappraisal exercises. At the end of the 5-week treatment period, those in the intervention group reported lower levels of depression and anxiety symptoms, and increases in post-traumatic growth relative to the wait-list control group (Wagner et al., 2007).

All of these studies converge on the conclusion that the use of cognitive reappraisal in highly stressful circumstances is associated with better psychological health outcomes. Thus, these studies support the hypothesis that the use of cognitive reappraisal could serve as a critical protective factor against psychological health problems, and that this protective function may be particularly important in highly stressful, relative to less stressful, contexts.

The Importance of Cognitive Reappraisal *Ability*

While the literature reviewed above is critical in providing insight into the effectiveness and adaptiveness of cognitive reappraisal, many important open questions remain. One particularly important area of inquiry is related to the fact that nearly all of the research on this topic to date has relied on self-report trait measures of cognitive reappraisal *use* in daily life. The most commonly used self-report measure is the Emotion Regulation Questionnaire (ERQ), a 10-item questionnaire in which individuals are asked to report how frequently they use cognitive reappraisal and suppression to regulate their emotions (John & Gross, 2003). The ERQ has shown high reliability (John & Gross, 2003), and, as shown above, is associated with increased psychological health. There are, however, several possible limitations that could be associated with self-reports of reappraisal use.

First, previous research has shown that retrospective self-report measures are subject to social desirability and self-presentational biases (Schwarz, 1999). For this reason, measures like the ERQ may over-estimate how frequently reappraisal is used. Additionally, more recent research has found that retrospective self-reports of emotion regulation are prone to biases caused by one's current emotional state (Wilhelm & Grossman, 2010), or by personality factors such as self-esteem (Robinson & Feldman Barrett, 2010). Overall then, retrospective self-reports of emotion regulation use may not be entirely accurate due to reporting biases.

In addition to these reporting biases, it may also be that many individuals cannot accurately introspect upon their use of emotion regulation strategies (cf. Todd, Tennen,

Carney, Armeli, & Affleck, 2004). For example, there is a growing body of evidence that many aspects of emotion regulation operate on an implicit level, below the level of conscious awareness (Hopp, Troy, & Mauss, 2011; Mauss, Evers, Wilhelm, & Gross, 2006). Additionally, van Reekum and colleagues (van Reekum, Johnstone, Urry, Thurow, Schaefer, Alexander, et al., 2007) found that the effects of reappraisal instructions on neural activation were significantly reduced when they accounted for eye gaze behavior. Thus, although participants were instructed to use reappraisal, many individuals appeared to instead be using attentional deployment strategies to change their emotions by, for example, averting their eyes from negative images. These results suggest that some individuals may simply not understand the definition of reappraisal, or may be unable to use reappraisal effectively. If this is the case, it would be unreasonable to ask people to report on the use of this strategy.

In addition to the limitations associated with self-report measures described above, it remains unclear what construct(s) the ERQ and similar measures are truly measuring. For instance, the face validity of the items on the ERQ suggests that it measures frequency of reappraisal use in daily life (e.g., “When I want to feel less negative emotion, I change the way I’m thinking about the situation.”). It is possible, however, that individuals’ answers on the ERQ capture other facets of reappraisal such as motivation and desire to use reappraisal, or the ability to use reappraisal. In this way, the ERQ may be collapsing several different facets of reappraisal into one measure.

If the ERQ is indeed collapsing across several different facets of cognitive reappraisal, existing research using this measure does not allow conclusions to be drawn

about the nature of these different facets or how they may relate to psychological health. It may be, for instance, that the frequency of reappraisal use predicts psychological health outcomes differently than the motivation or the ability to use reappraisal. Although many potential facets of reappraisal could be studied, one facet stands out as being theoretically important in terms of long-term resilience: *ability*. That is, individuals' actual ability to successfully change their experience of unwanted emotions may contribute to positive psychological health outcomes. Further, the ability to use reappraisal may be a particularly important predictor of psychological health in contexts that are highly emotionally charged, such as high stress contexts. In this way, individuals high in reappraisal ability could effectively down-regulate their negative emotions by changing the appraisals that cause them.

Indeed, Kashdan and Rottenberg (2010) have suggested that a person's ability to flexibly use effective regulation strategies as required by situational demands is a particularly important contributor to psychological health. In support of this hypothesis, Bonanno (2004) has found that the ability to flexibly express or suppress emotional expressions is a long-term predictor of resilience in the face of severe stress. Although this work focused only on the regulation of behavior and not of subjective emotional experience, it is important in suggesting that emotion regulation ability may be an important predictor of psychological health. Thus, reappraisal ability may uniquely predict mental health outcomes over and above other facets of reappraisal in the context of high stress.

To begin testing the hypothesis that *ability* may be a particularly important facet of cognitive reappraisal, recent work has focused specifically on measuring cognitive reappraisal ability (CRA). That is, instead of using self-reported use, this research has used laboratory measures of emotional responding to gauge how successful individuals are when they attempt to use reappraisal to change their subjective experience of emotion. For example, Wager and colleagues were among the first researchers to develop a measure of CRA (Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008). They showed participants in an fMRI study negative pictures and instructed participants to just look at half of the pictures and to use reappraisal while viewing the other half of the pictures. CRA was operationalized as the decrease in self-reported negative emotion from the just watch condition to the reappraisal condition. Using a similar paradigm, McRae and colleagues (McRae, Jacobs, Ray, John, & Gross, 2012) found that performance on a laboratory challenge measure of CRA was empirically distinct from self-reported reappraisal use, supporting the hypothesis that the *frequency* of using reappraisal and the *ability* to use reappraisal are separate constructs.

Similarly, Shiota and Levenson (2009) used disgusting and sad film clips to induce emotions in the laboratory. They experimentally manipulated the use of reappraisal by instructing participants to use positive reappraisal during some of the film clips. CRA was indexed by changes in self-reported subjective emotional response, as well as physiological and behavioral response. Shiota and Levenson also measured self-reported reappraisal ability. Results indicated that self-reported ability was not strongly correlated with actual ability as measured by the laboratory paradigm. There was also a

high degree of variability in reappraisal ability across individuals. This study provides support for the idea that people are not able to accurately report on their *ability* to use reappraisal and highlights the importance of using laboratory paradigms for providing accurate measures of reappraisal ability.

Building upon Shiota and Levenson's methods, Troy and colleagues (Troy, Wilhelm, Shallcross, & Mauss, 2010) examined how individual differences in CRA relate to psychological health. Similar to the paradigm used by Shiota and Levenson (2009), Troy and colleagues used film clips to induce sadness in the laboratory. During one of the film clips, participants received instructions to use positive reappraisal. CRA was indexed by changes in self-reported sadness and skin conductance level from the uninstructed sad clip to the instructed sad clip. Results indicated that CRA interacted with life stress to predict depressive symptoms. Specifically, individuals high in CRA exhibited less depressive symptoms under high stress circumstances as compared to individuals low in CRA. Reappraisal ability was unrelated to depression in the context of low life stress. These results held when controlling for self-reported trait reappraisal use, which suggests that reappraisal ability predicts depressive symptoms above and beyond self-reported use of reappraisal.

This research is an important first step in investigating how people's *ability* to change their emotional states using reappraisal relates to psychological health outcomes. Its results suggest that CRA may be an important protective factor against negative outcomes such as depression, particularly among highly stressed populations. It may be that CRA is particularly important in high stress contexts because it provides people with

an effective way to manage the negative emotions that accompany highly stressful circumstances. These findings also suggest that the ability to use positive reappraisal (reframing events more positively) may be a particularly adaptive process in the context of high stress. For this reason, the present study specifically examines the ability to use positive reappraisal. For the remainder of the paper, the term “cognitive reappraisal ability” refers specifically to the ability to use positive reappraisal, rather than other subtypes of reappraisal.

Taken together, the literature reviewed so far highlights four important insights about cognitive reappraisal: 1) Cognitive reappraisal is effective – it provides a way to successfully regulate the subjective experience of emotions without any accompanying physiological, cognitive, or social costs, 2) The dispositional use of cognitive reappraisal is adaptive – it is associated with better psychological health both cross-sectionally and longitudinally, and it appears to contribute to resilience in the context of high stress, 3) The ability to use reappraisal may be a critical contributor to psychological health over and above the contributions of dispositional cognitive reappraisal use, 4) Cognitive reappraisal ability may be particularly important for psychological health in highly stressful, relative to less stressful, contexts.

However, several questions pertaining to cognitive reappraisal remain unanswered. The current study seeks to provide answers to two particularly important questions related to CRA, which will allow for a better understanding of how this construct relates to psychological health over time and across contexts.

Question One: Does Cognitive Reappraisal Ability Have *Long-Term Effects* On Psychological Health?

As noted above, the construct of cognitive reappraisal ability (CRA) is fairly new and has not been widely studied. The small amount of research that has been conducted, however, suggests that in the immediate aftermath of a stressor, CRA is associated with better psychological health outcomes (Troy et al., 2010). Unfortunately, however, all of the research on CRA to date has been cross-sectional (Shiota & Levenson, 2009; Troy et al., 2010; Wager et al., 2008). For this reason, it remains unclear whether the beneficial cross-sectional effects of CRA on psychological health would translate into longer-term psychological health outcomes.

Additionally, cross-sectional designs do not allow for strong causal claims. Based on the existing findings, it is not clear that CRA causally contributes to psychological health outcomes and not the other way around. For example, low CRA may simply be a side-effect of elevated depression in the context of high stress. Indeed, previous research on individuals with anxiety and mood disorders has found that these disordered individuals are more likely to rely on maladaptive coping strategies such as suppression or avoidance, rather than adaptive strategies like reappraisal (Betts, Gullone, & Allen, 2009; Campbell-Sills & Barlow, 2007). Although this line of research did not examine CRA directly, these findings could be explained by the hypothesis that disordered individuals are lower in CRA and are thus forced to rely on less effective regulation strategies.

Research that has directly measured CRA and psychological health outcomes provides some argument against the hypothesis that low CRA is simply a side effect or a consequence of low psychological health. First, in the study of CRA conducted by Troy and colleagues (2010), there was no direct relationship between CRA and depression – only the interaction between CRA and life stress was significant. Similarly, a study conducted by Ehring and colleagues (Ehring, Fischer, Schnülle, Bösterling, & Tuschen-Caffier, 2008) found that recovered-depressed individuals reported high levels of maladaptive emotion regulation relative to never-depressed individuals, which suggests that low CRA may not simply be a side effect of one's current depressive state. These findings add support to the hypothesis that deficits in emotion regulation ability act as risk factors for the development of psychopathology (Gross & Muñoz, 1995; Kring & Werner, 2004).

While the existing cross-sectional evidence provides some support for the notion that CRA causally contributes to improved psychological health, the current lack of longitudinal studies does not allow for strong causal conclusions. Thus, prospective longitudinal designs are needed in order to examine CRA as a predictor of changes in psychological health over time. Specifically, if CRA interacts with changes in stress over time to predict changes in depressive symptoms over time, this would support the hypothesis that increased CRA causes decreases in depression over time, particularly for those with larger increases in stress. That is, if individual differences in CRA precede changes in both stress and depression, we can be more confident that CRA plays a causal role in psychological health. In addition, longitudinal designs would provide evidence

about the time course of the relationship between CRA and psychological health in the context of stress.

To address these open questions, the current study used a prospective longitudinal design in a community sample of adults who had recently been exposed to a stressful life event. During Time 1, participants completed the laboratory measure of CRA, in addition to self-report measures of life stress severity and depressive symptoms. Time 2 took place six months later, and participants completed self-report measures of current depressive symptoms and current life stress severity.

This study design includes several key elements that extend existing research. First, the use of a large (N=181) community sample (both men and women aged 21-60) yields highly generalizable results. Second, the sample consists of people who have been exposed to a recent stressful life event, which will allow CRA to be measured in a context that appears to be critically important: high stress. Third, life stress and depressive symptoms were measured at two time points, allowing for the assessment of change over time. Fourth, this study used the previously validated (see Troy et al., 2010) laboratory measure of CRA rather than self-reports.

The current study tests the hypothesis that high CRA, in interaction with change in life stress over time, is associated with greater decreases in depressive symptoms over a six-month period. This hypothesis is examined both cross-sectionally (to replicate past results) and longitudinally (to extend past results). The hypothesis will be supported if individuals with higher levels of CRA and higher levels of life stress at Time 2 (controlling for life stress at Time 1) report larger decreases in depressive symptoms over

time compared to those individuals who are high in life stress at Time 2 but low in CRA. No differences in depressive symptoms as a function of CRA are predicted for individuals low in life stress at Time Two.

Question Two: Is Cognitive Reappraisal Ability Maladaptive In Specific Types of Stressful Contexts?

As discussed above, existing evidence suggests that CRA is most protective in highly stressful contexts relative to less stressful contexts (Troy et al., 2010). Importantly, these results were significant in a sample that included a very diverse range of stressful life events, including sudden job loss, divorce, and experiencing the death of a close family member. Thus, CRA may be an adaptive process across a wide range of stressful contexts. The relative usefulness of CRA in high stress contexts may be due to the fact that stress exposure leads to increases in negative emotion (Feldman, Cohen, Lepore, Matthews, Kamarck, & Marsland, 1999). From this perspective, one's ability to regulate negative emotions is important in all highly stressful contexts because there will likely be more negative emotions that need to be regulated effectively.

Although the protective effects of CRA appear to hold across a wide range of stressful circumstances, there may be important exceptions to this observation. Indeed, theorists across the social sciences have argued that no psychological process is inherently adaptive in all contexts (Darley, 1992; Folkman & Moskowitz, 2000b; Mischel, 1968; O'Mara, McNulty, & Karney, 2011). In addition, many personality psychologists have emphasized the importance of person by situation interactions in explaining human behavior (Blass, 1991; Ender & Magnusson, 1976; Murray &

Kluckhohn, 1953). This raises the question of whether there are contexts in which CRA may be relatively less adaptive, or may even be maladaptive (i.e., associated with negative psychological health outcomes). What contextual factors might moderate the adaptiveness of reappraisal? From a theoretical perspective, CRA would appear to be more useful in stressful contexts in which it is important to change one's emotional reactions to a stressor. With this in mind, are there any stressful circumstances in which it might not be critical -- or maybe even counterproductive -- to regulate one's emotions?

Research on self-control may provide an answer to this question. Many self-control researchers have made the distinction between primary and secondary control (Heckhausen & Schulz, 1993; 1995; Rothbaum, Weisz, & Snyder, 1982; Schulz & Heckhausen, 1996). Heckhausen and Schulz (1995) defined primary control as "bringing the environment into line with one's wishes" (p. 285). Based on this definition, examples of primary control include active problem solving such as putting out a fire in one's home with a fire extinguisher. Secondary control, on the other hand, involves "bringing oneself in line with the environment" (Heckhausen & Schulz, 1995, p. 285). That is, secondary control involves making changes within oneself, often by changing one's goals, expectations, or cognitions, in order to cope with stress. Based on this definition, examples of secondary control often involve attempts at emotion regulation, such as downplaying the importance or impact of a negative event. Within this definition, then, the use of cognitive reappraisal would be considered a secondary control strategy.

In the coping literature, the distinction between primary and secondary control is paralleled by the distinction between problem-focused and emotion-focused coping,

respectively (see Folkman, 1984 and Folkman & Lazarus, 1980 for reviews). For example, Folkman (1984) defined problem-focused coping as attempting to directly manage the problem that is causing distress through strategies such as active problem solving, decision making, and seeking instrumental support from others. On the other hand, Folkman defined emotion-focused coping as attempting to regulate the distress that has been caused by using strategies like cognitive reappraisal. Indeed, Folkman (1984) explicitly states that she considers secondary control a form of emotion-focused coping because it involves “efforts to accommodate oneself to uncontrollable events” (Folkman, 1984, p. 844). Based on these definitions then, it appears that the use of CRA could be considered an example of emotion-focused coping.

Importantly, Folkman (1984) notes the context in which she believes emotion-focused coping is particularly well suited: uncontrollable stress. That is, in circumstances where an individual has no control over the stressor that is occurring, problem-focused coping may be ineffective or even counterproductive. By engaging in emotion-focused coping, however, an individual may be able to decrease their levels of distress and increase “perceptions of control in ostensibly uncontrollable circumstances” (Folkman, 1984, p. 844)¹.

¹ Although the theoretical distinction between primary and secondary control maps on to the distinction between problem-focused and emotion-focused coping, only the coping literature has made specific predictions about controllable and uncontrollable contexts. For this reason, I will use the terms problem-focused and emotion-focused coping for the remainder of the paper, rather than primary and secondary control.

For example, consider someone who is about to lose her job because of poor performance at work. This kind of stressor could be considered at least somewhat controllable because this individual could attempt to improve her performance. To cope with this stressor then, the most adaptive course of action would be to engage in problem-focused coping by, for instance, putting in longer hours at work and meeting with her bosses to see how she can improve her performance. Engaging in emotion-focused coping, on the other hand, would probably not be adaptive. In fact, engaging in an emotion-focused strategy like cognitive reappraisal may even hinder her ability to engage in problem-focused coping because this may use precious time and cognitive resources that could otherwise be used for more active coping. In addition, if this individual is able to successfully down-regulate her negative emotions using cognitive reappraisal, she may no longer be motivated to engage in direct action or other forms of active coping. That is, the presence of negative emotions in the context of controllable stressors may be adaptive, in that they may provide the motivation to actively cope with the stressor at hand.

Now consider a more uncontrollable circumstance – someone who is about to lose her job due to layoffs during a difficult economic period. This context is considerably less controllable than the first – there may be nothing this individual can do in order to prevent the job loss. In this context then, it may be more adaptive to engage in emotion-focused coping by, for instance, attempting to reframe the situation more positively in order to regulate her emotions and come to terms with the reality of the situation. Engaging in problem-focused coping, however, would seem maladaptive because direct

attempts to change the situation would most likely fail. Within this theoretical framework, problem-focused coping would be particularly adaptive in controllable stressful contexts and emotion-focused coping would be particularly adaptive in uncontrollable stressful contexts.

Research using self-report checklist measures of stress and coping provides some initial support for the idea that emotion-focused coping is more adaptive in uncontrollable stressful contexts. In stressful circumstances perceived as uncontrollable, people are more likely to report engaging in emotion-focused coping strategies like reappraisal, relative to situations that are perceived as controllable (Carver, Scheier, & Weintraub, 1989; Folkman & Lazarus, 1980). In addition, Collins, Baum, & Singer (1983) studied the residents of Three Mile Island (survivors of an uncontrollable nuclear accident) and found that those who reported using more emotion-focused coping in response to the event reported lower levels of psychological distress.

In stressful situations that are uncontrollable, however, cognitive reappraisal may be associated with worse psychological health. For example, in a four-year longitudinal study, participants with the tendency to form positive appraisals of stressful events were significantly more depressed when they experienced severe, controllable stressors, relative to those who experienced less severe, controllable stressors (O'Mara et al., 2011). No studies to date, however, have examined cognitive reappraisal ability simultaneously in both controllable and uncontrollable stressful contexts.

Based on the theoretical arguments and the literature reviewed above, CRA appears to provide a particularly effective way to regulate emotions in the context of

stress. Further, based on the theoretical framework described in this section, because CRA is an emotion-focused strategy, it should be particularly protective in stressful situations that are uncontrollable, relative to stressors that are controllable. More specifically, CRA may be particularly well-suited to allow people to manage their negative emotions in uncontrollable circumstances because more active forms of coping are not possible. In the context of controllable stress, however, CRA should be less adaptive or even maladaptive, because this context is better suited for the use of active coping strategies, rather than emotion-focused ones.

In order to address this important question of whether the protective effects of CRA differ depending upon the controllability of stressors that have been experienced, the current study uses the same community sample that was described in the **Question One** section above. Importantly, a measure of stressor controllability (for stressors encountered by the participants in the community sample) was provided by a matched sample of independent coders, which yields a relatively objective measure of stressor controllability.

A large body of research has examined stressor controllability, which has been defined as the degree to which “potential outcomes ...can be influenced by human actions” (Heth & Somer, 2002, p. 885). However, much of this research has used non-human subjects (Brown, Hurley, Repucci, & Drugan, 2001; Wellman, Cullen, & Pellemounter, 1998), or has experimentally manipulated stressor controllability in the lab, rather than studying naturally occurring stressful life events (Friedland, Keinan, & Regev, 1992; Peters, Godaert, Ballieux, van Vliet, Willemsen, Sweep, & Heijnen, 1998;

Watanabe, Iwanaga, & Ozeki, 2002). Additionally, the small body of research that has studied naturally occurring life events has relied on subjective self-report measures of perceived controllability (Folkman, 1984; Puente-Diaz & Anshel, 2005; Terry, 1991). Previous research has shown that an individual's subjective perception of stressor controllability can be confounded with several personality factors, including locus of control, pessimism, and self-efficacy (Heth & Somer, 2002). Additionally, one's perception of controllability may be confounded with one's ability to respond to and actively cope with stress (Hiroto, 1974; Rosenbaum & Ben-Ari, 1985). For these reasons, subjective ratings of controllability may yield biased or inaccurate estimates that may be confounded with other study variables such as depression.

For this reason, I developed a relatively objective measure of stressor controllability for the present study (see **Measures** section below). This measure of controllability was collected in addition to the laboratory measure of CRA and the self-report measures of cumulative life stress and depressive symptoms. Each of these self-report measures was collected during the laboratory session at Time 1 and at Time 2, which will allow for the assessment of changes in cumulative stress, stressor controllability, and depressive symptoms over a six month period. The addition of a measure of stressor controllability allowed me to test the hypothesis that CRA interacts with both stress level and controllability of stress to predict depressive symptoms. Specifically, I hypothesized that CRA will be most protective against negative outcomes under high stress conditions that are relatively uncontrollable. In the context of relatively controllable stress, on the other hand, I expect the protective effect of CRA to be

diminished or even reversed, because this is a context in which CRA should be less protective against negative outcomes. Because I expect these relationships to predict changes in psychological health over time, I predict that this three-way interaction will be present cross-sectionally (at Time 1), as well as prospectively (when examining changes in cumulative stress, stressor controllability, and depressive symptoms) at Time 2.

Chapter Two: Method

Participants

A community sample of 181 men and women between the ages of 21 and 60 participated in this study. To qualify, all participants were required to be native English speakers and to have experienced a stressful life event (SLE) within the past eight weeks. For the purposes of this study, an SLE was defined as an event with a clearly defined starting point within the past eight weeks (i.e., an acute instead of a chronic stressor) that has had a significant, negative impact on participants' lives. Examples of SLEs included in the sample are: death of a spouse or close family member, injury of self or close family member, divorce, sudden unemployment, and exposure to crime. The inclusion of many different types of stressors allows us to study the role of cognitive reappraisal ability as a general mechanism across a wide range of stressors and stress levels. Participants were recruited through postings online such as Craigslist, and by posting flyers in public areas such as laundromats, libraries, and local hospitals. The sample mirrors the racial and ethnic makeup of the Denver Metro area. In terms of race, the sample was 85% European American, 6% African American, 1% Asian American, and about 8% reported other or multiple races. In terms of ethnicity, the sample was 7% Hispanic/Latino. Participants were compensated \$55 for the completion of Time 1 and \$15 for the completion of Time 2.

Procedure

The study consisted of two time points over the course of six months. Time 1 took place within eight weeks of each participant's SLE. Time 2 occurred six months after Time 1. At both time points, participants completed questionnaires measuring cumulative life stress and depressive symptoms. At the end of Time 1, the laboratory reappraisal ability task was administered (see Appendix B, Figure 1). Participants were seated alone in front of a computer monitor in a quiet lab room. The experimenter connected sensors to each participant's fingers and stomach in order to collect physiological measurements during the task. Each participant was presented with a short (three minutes) emotionally neutral video clip depicting scenes from a nature film. After the film clip, participants rated the amount of 13 different emotions, including sadness, they felt during the film clip on nine-point Likert scales in order to establish baseline levels of emotion. Next, participants were presented with three film clips pretested to induce moderate amounts of sadness. These clips came from the following films: *Fatal Attraction*, *I Am Sam*, and *Kramer vs. Kramer*. Film clips have been widely used in previous research to induce sadness (Fredrickson & Levenson, 1998; Rottenberg, Ray, & Gross, 2007) and are considered more ecologically valid and more intense than still pictures or words (Rottenberg, Ray, & Gross, 2007). Each of these film clips is approximately two minutes long, depicts a personal interaction (each of these films show two people discussing an emotional event) and have received similar normative ratings of moderate sadness in previous research (Troy et al., 2010).

During two of the sad film clips, participants were simply asked to “please watch the following film clip carefully.” During one of the sad film clips, participants were asked to reappraise the situation they are watching in order to decrease its emotional impact. The instructions for this condition are presented as follows:

Please watch the following film clip carefully. This time, as you watch, try to think about the situation you see in a more positive light. You can achieve this in several different ways. For example, try to imagine advice that you could give to the characters in the film clip to make them feel better. This could be advice that would help them think about the positive bearing this event could have on their lives. Or, think about the good things they might learn from this experience. Keep in mind that even though a situation may be painful in the moment, in the long run, it could make one’s life better, or have unexpected good outcomes. In other words, try to think about the situation in as positive terms as you possibly can. This can be difficult at times, so it is very important that you try your best. Please ask the research assistant if you have any questions about this task. It is very important that you carefully watch the film clip, but think about it from a positive perspective.

These instructions are based on writing techniques used in clinical research to encourage patients to reframe a stressful event in a more positive way. (Lange, van de Ven, & Schrieken, 2003; Lange et al., 2003; Pennebaker & Chung, 2007). Although other techniques have been used previously for reappraisal instructions, such as self-distancing (Ayduk, Mendoza-Denton, Mischel, Downey, Peake, & Rodriguez, 2000), or denying the reality of the event (Gross & Levenson, 1993) the reframing technique we use seems to provide a high degree of ecological validity, and closely mirrors the definition of cognitive reappraisal as laid out by Gross (Gross & Thompson, 2007). These instructions also avoid explicitly telling the participant to feel less sad while reappraising, thus avoiding potential demand characteristics in the self-report data. In addition, these

instructions give specific examples of how one could use reappraisal while watching the film clip. Thus, it should be more likely that each person will be able to successfully use reappraisal when instructed to.

After the presentation of each film clip, participants were asked to report the greatest amount of 13 different emotions, including sadness, that they experienced during the clip. In order to account for possible habituation to the sad film clips, participants were randomly assigned to two groups. The order of the film clips was the same for both groups, but the order of the emotion regulation instructions differed (see Appendix B, Figure 1). Group 1 was given instructions to reappraise on the second sad film, whereas Group 2 was given instructions to reappraise on the third sad film. The remaining sad films for each group served as controls. By changing the film in which participants reappraised, I am able to account for possible habituation (decrease in sadness over each subsequent film clip) by comparing sadness ratings on the films between groups. This design also allows for the examination of changes within individuals. Because neither group is reappraising during the first sad film clip, sadness ratings during this film are used as a sadness baseline.

Measures

Life stress. The cumulative negative impact of stressful life events was measured with the Life Experiences Survey at Time 1 and Time 2 (LES; Sarason, Johnson, & Siegel, 1978). This measure consists of 46 items about a wide range of different stressors, both positive (e.g. marriage) and negative (e.g. death of a spouse). Each item asks if one has experienced a particular stressor within the past 18 months and the degree to which

the stressor has affected you (in both positive and negative ways). The LES has been widely used as a measure of stress in previous literature (Roth, Goode, Williams, & Faught, 1994; Harrington, Matheny, Curlette, McCarthy, & Penick, 2005, Schmidt, Demulder, & Denham, 2002). This measure supplies both the number of events that have occurred in past months, as well as the sum of the perceived impact of negative events. For the purposes of this study, we use the perceived negative impact of events occurring in the past 18 months as our measure of cumulative life stress at Time One and Time Two. The LES produces test-retest reliability coefficients ranging between .56 and .88 for the negative impact scale over a five to six week time period. Responses to the LES also appear to be relatively free of response bias (Sarason et al., 1978). The present sample included individuals who had experienced a wide range of cumulative life stress at Time One ($M = 15.87$, $SD = 10.50$, Range: 1 - 46) and Time Two ($M = 9.82$, $SD = 9.33$, Range: 0 - 40). For illustrative purposes, an individual who experienced 3 stressful events that were perceived as “extremely negative” in the past 18 months would have a score of 9 on the LES.

Stressor controllability. Previous researchers have defined stressor controllability in a wide variety of ways (Fleming, Baum, & Singer, 1984; Folkman, 1984; Heth & Somer, 2002; Roth & Cohen, 1986). For the purposes of the present study, I use Heth & Somer’s (2002) definition of stressor controllability, which defines this construct as the degree to which “potential outcomes ...can be influenced by human actions” (Heth & Somer, 2002, p. 885). Using this definition, the more an individual can

exert influence over the occurrence and outcome of a stressful event, the more control this individual has.

As mentioned in the **Question 2** section above, self report measures of stressor controllability may be subject to confounds that may result in biased estimates of personal control. Indeed, previous research has shown that many individuals provide inaccurate estimates of their personal control across a wide range of contexts (Langer, 1975; Langer & Roth, 1975). Thus, it was important to develop a more objective measure of stressor controllability that yields a relatively unbiased estimate of the controllability of participants' real life stressors.

To create such an objective measure, a sample of independent coders from the community ($N=22$) provided ratings of the controllability of the original participants' stressors. As shown in Appendix A Table 1, this new sample of coders was matched to the original sample on age, sex, race, years of education, and family income. In addition, this sample was significantly less depressed than the original sample, suggesting that the coders' controllability ratings were less likely to be driven by high levels of depression. Each rater was given the definition of controllability described above, and all coders provided ratings for all 46 possible stressors on the LES (the same measure that was used to collect the measure of cumulative life stress, described above). Ratings were assigned on a 1 to 4 scale, with 1 indicating a very uncontrollable event and 4 indicating a very controllable event. Across all 22 coders, the single measure intra-class correlation coefficient was 0.54, $F(34, 680) = 28.19, p < .001$, indicating a moderate degree of reliability across coders.

The controllability ratings from all coders were averaged to create an overall controllability score for each item on the LES. To calculate a total stressor controllability score for each participant in the original sample, the controllability scores for each stressor on the LES that the participant had experienced in the past 18 months and perceived as having a *negative* impact were averaged. Thus, this composite indicates the average amount of control each participant had over the stressors they experienced in the past 18 months. The mean controllability score for Time 1 was 2.78 (SD=.36, Range: 1.23 – 3.33) and for Time 2 it was 2.97 (SD=.39, Range: 1.27 – 3.70).

Depression symptoms. Depression symptoms were measured at Time One and Time Two using the Beck Depression Inventory (BDI; Beck & Steer, 1984). This allows for the measurement of changes in depressive symptoms over time. The BDI is a self-report measure consisting of 21 items. Each question consists of four grouped statements (for example, “I do not feel sad,” “I feel sad,” “I am sad all of the time and I can’t snap out of it,” and “I am so sad or unhappy that I can’t stand it”). Participants are instructed to select the item that best describes how they have felt in the past week. One question, which pertains to suicidal thoughts, was not included in the current study, leaving a total of 20 questions. Each item is scored on a zero to three scale and the total BDI score is calculated by summing the scores across all items. The BDI has been shown to have adequate internal consistency (Beck & Steer, 1984) and has been very widely used in research to measure current depressive symptoms (Brands et al., 2007; Thombs et al., 2007; Pearlstein, Zlotnick, Battle, Stuart, O’Hara, & Price, 2006; O’Donnell, Wardle, Dantzer, & Steptoe, 2006). Because the current sample was, on average, highly stressed,

average BDI scores were elevated at Time One ($M = 11.85$, $SD = 9.83$) and Time Two ($M = 11.64$, $SD = 10.30$).

Cognitive reappraisal ability. Cognitive reappraisal ability, or the amount that individuals are able to decrease the amount of sadness they feel when instructed to reappraise, was measured in a laboratory paradigm at Time One. Two separate indices of CRA were collected: changes in self-reported sadness and changes in skin conductance level.

Self-reported sadness was measured immediately after each film clip. Participants rated, on a nine-point Likert scale, the highest amount of 13 different emotions that were experienced during the film that was just watched. Change scores in self-reported sadness were calculated by subtracting sadness ratings made after the reappraised sad film from sadness ratings made after the baseline sad film. Because the reappraisal film is not the same for everyone, raw film ratings were converted to z-scores before the change score for each individual was calculated. This variable is referred to as CRA-SAD. Mean scores on CRA-SAD were 0.31 ($SD = 1.02$).

As in previous research (Troy et al., 2010), I decided a priori that individuals who responded to the baseline sad film clip with no sadness (an answer of 1 on the 9 point scale) would be excluded from all analyses. Because the baseline sadness induction failed for these participants, their CRA scores would be difficult to interpret. 17 participants were excluded for this reason, leaving 164 participants for analysis.

During the experimental session, physiological channels were sampled continuously at 1000 Hz using laboratory software. Later, customized analysis software

(Wilhelm, Grossman & Roth, 1999) was applied to physiological data reduction, artifact control, and computation of average physiological scores for each participant for each film clip. Although multiple physiological measures were obtained, the primary physiological measure of interest is skin conductance level (SCL) because it has been used previously as an indicator of CRA (Troy et al., 2010).

SCL is a measure of electrodermal activation. SCL was derived from a signal using a constant-voltage device to pass 0.5 V between Beckman electrodes (using an electrolyte of sodium chloride in Unibase) attached to the palmar surface of the middle phalanges of the first and second fingers of the non-dominant hand. Skin conductance level was indexed by the mean level after movement and electrode contact artifacts had been edited out using a customized detection procedure (Wilhelm, Grossman & Roth, 1999). Previous research has found that increases in sadness are associated with decreases in SCL (Kreibig, Wilhelm, Roth & Gross, 2007; Kunzmann & Gruhn, 2005; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). Based on these findings, we believe that greater reappraisal ability will be associated with increases in SCL, while a lack of reappraisal ability will be characterized by decreases in SCL. This index of CRA is referred to as CRA-SCL. Mean scores on CRA-SCL were -0.01 ($SD = 0.55$). Due to technical problems, SCL data was not available for 20 participants. This left 134 participants available for analyses with CRA-SCL.

Possible group effects were examined to make sure that there were no significant differences between experimental groups on either measure of CRA. T-tests revealed that

CRA did not differ significantly between experimental groups using either index of CRA,
CRA-SAD: $t(162) = 1.15, p = 0.25$, or CRA-SCL: $t(125) = -0.43, p = .67$.

Chapter Three: Results

Outlier Analysis

Each variable that is used in the analyses discussed below was examined for outliers, which are defined as values greater than three times the inter-quartile range above or below the mean (Tukey, 1977). Three variables contained outliers: Time 1 stressor controllability ($n=3$), Time 2 stressor controllability ($n=4$), and CRA-SCL ($n=7$). To ensure that our results were not driven by these extreme values, these outliers were removed for all analyses. This left a total of 161 values for analyses with Time 1 stressor controllability, 121 values for analyses with Time 2 stressor controllability, and 127 values for analyses with CRA-SCL.

Data Analysis Strategy

For all regression analyses described below, all continuous predictors were mean centered before calculating interaction terms and before entering effects into the models.

Manipulation Check: Sadness Induction

To confirm that the three sad film clips induced moderate amounts of sadness, I examined mean sadness ratings for each film clip for unmanipulated (uninstructed) film viewings (the whole sample for the first sad film, Group 2 for the second sad film, Group 1 for the third sad film). The mean (SD) sadness ratings were 6.40 (2.21; Film 1), 6.46 (2.24; Film 2), and 6.26 (2.36; Film 3). The results of paired-samples *t*-tests indicated that all three sad film clips induced significantly greater reports of sadness than the neutral

film clip ($M = 1.27$, $SD = 1.13$, all $ps < .01$). In addition, each of the three film clips induced significantly greater amounts of sadness than anger, fear, or happiness (all $ps < .01$). Lastly, unmanipulated sadness ratings for Film 1 were not significantly different from sadness ratings for Film 2, $t(82) = -.10$, $p = .92$, or Film 3, $t(81) = .47$, $p = .64$, and there were no significant differences between Film 2 and Film 3, $t(136) = .31$, $p = .75$.

Manipulation Check: Cognitive Reappraisal Instruction

To test whether the reappraisal instructions affected sadness reports consistent with instructions (i.e., led to lower levels of sadness on average), we conducted a repeated measures ANOVA with film (Sad Film 2 vs. 3) as a within-individual factor and experimental group (reappraisal instruction vs. no instruction) as a between-subjects factor. To take into account each individual's sadness baseline, we entered change scores from the sad baseline to Sad Films 2 and 3, respectively. These scores are in z-units, and a negative score indicates that the individual reported less sadness on the film in question than on the baseline sad film. As illustrated in Appendix B Figure 2, the interaction between film clip and experimental group was significant, $F(1, 162) = 16.85$, $p < .01$. The differences observed between groups were in the expected directions (see Appendix B, Figure 1): during the second sad film, when Group 1 was asked to use cognitive reappraisal and Group 2 was just watching, Group 1 reported significantly lower levels of sadness relative to Group 2, $t(162) = -3.82$, $p < .01$. During the third sad film clip, when Group 2 was asked to use cognitive reappraisal and Group 1 was just watching, Group 2's mean sadness ratings were lower than Group 1's. This group difference, however, was not statistically significant, $t(162) = 0.64$, $p = .52$.

Although not all of the predicted group differences were significant, the direction of changes in means on this task suggest that participants were attempting to use cognitive reappraisal and at least some were succeeding in their attempts. Additionally, it may not be surprising that the group difference on the third sad film was not significant, given that Group 1 had been asked to reappraise on an earlier film. This group might have persevered using cognitive reappraisal during the last film clip, resulting in lower sadness ratings than would have otherwise been observed. Indeed, 45% of participants in Group 1 reported that they used reappraisal during the third film clip, even though they had not been instructed to do so.

Discriminant Validity of Cognitive Reappraisal Ability

The two indices of CRA, CRA-SAD and CRA-SCL, were not significantly correlated with one another ($r = -.10, p = .22$). Therefore, I conducted separate analyses for these two indices of CRA.

Correlations between CRA-SAD, CRA-SCL, and measures of cumulative stress, depressive symptoms, stressor controllability, demographic variables, emotion regulation and personality variables, emotional reactivity, and verbal intelligence are shown in Appendix A, Table 2. As indicated in Table 2, CRA-SAD was negatively correlated with Time 2 cumulative stress and Time 2 depressive symptoms². CRA-SAD was also

² Given this pattern of correlations, I also examined whether Time 2 cumulative stress mediates the relationship between CRA-SAD and Time 2 depressive symptoms (controlling for Time 1 cumulative stress and Time 1 depressive symptoms). The partial correlation between CRA-SAD and Time 2 depressive symptoms, controlling for Time 2 stress, was not significant ($r = -.12, p = .17$), which suggests that Time 2 stress is a significant mediator. However, the Sobel Test was not significant ($p > .05$).

positively correlated with family income, self-reported cognitive reappraisal ability, social desirability, and sadness reactivity. CRA-SAD was also marginally negatively correlated with Time 1 Depression ($p = .07$), and marginally positively correlated with extraversion ($p = .08$). CRA-SCL showed slightly different patterns of association with the other variables: there was a significant positive correlation with SCL reactivity, and a marginally significant positive correlation with Time 2 depressive symptoms ($p = .06$). CRA-SCL was not significantly associated with any of the other measured variables. Independent samples t-tests confirmed that there were no significant gender differences on either measure of CRA (both $ps >.26$). A series of one-way ANOVAs also showed no significant relationship between either measure of CRA and race or ethnicity, respectively (all $ps >.32$)

Discriminant Validity of Stressor Controllability

Because this is the first study to use this measure of stressor controllability, it was important to examine how this new measure relates to other constructs. The correlation between Time 1 and Time 2 stressor controllability was significant ($r=.31, p<.01$). Correlations between controllability at both time points and measures of depressive symptoms, life stress, CRA, demographic variables, emotion regulation and personality factors, emotional reactivity, and verbal intelligence are shown in Appendix A, Table 3.

As shown in Table 3, Time 1 controllability was positively related to Time 1 life stress. In addition, there was a marginally significant correlation between Time 1 controllability and Time 2 life stress ($p=.10$). Time 1 controllability was not significantly correlated with any of the other measured variables. Time 2 controllability was positively

related to extraversion, but was not significantly correlated with any of the other measured variables.

A t-test was conducted to examine whether there were significant gender differences in controllability scores at both time points. There were no significant gender differences in stressor controllability at Time 1, $t(159)=-1.36, p=.17$. At Time 2, there was a significant gender difference, $t(115)=-2.28, p=.03$, with women exhibiting higher levels of stressor controllability ($M=3.07, SD=.21$) compared to men ($M=2.96, SD=.32$). A series of one-way ANOVAs revealed that stressor controllability was not significantly associated with race or ethnicity at either time point (all $ps>.20$). At Time 1, participants also provided self-report ratings of how much control they thought they had over the most stressful event they had experienced in the past three months on a 1-5 scale. These self-report ratings at Time 1 were not significantly correlated with Time 1 stressor controllability, $r=.12, p=.12$, although it is important to note that the measure of stressor controllability assessed *all* negative events that had occurred in the past 18 months, rather than just the most stressful event.

Question One: Does Cognitive Reappraisal Ability Have Long-Term Effects on Psychological Health?

Cross-sectional effects of CRA. First, I examined the cross-sectional effects of CRA on depressive symptoms. Depressive symptoms at Time 1 were entered as the dependent variable, and mean centered values for CRA, Time 1 cumulative life stress, and the interaction between the two were entered simultaneously into the regression model. CRA-SAD and CRA-SCL were examined in two separate regression models. The

results of these two regressions are shown in Appendix A, Table 4 and Table 5. For CRA-SAD, there was a significant main effect of cumulative stress, but no significant main effect of CRA-SAD, and no significant interaction between cumulative stress and CRA-SAD. The same pattern was observed for CRA-SCL: there was a significant main effect of cumulative stress, but no significant main effect of CRA-SCL, and no significant interaction. Thus, the cross-sectional interaction between CRA and cumulative stress that has been previously reported (Troy et al., 2010) was not replicated.

Secondary analyses of cross-sectional effects. A series of secondary analyses were performed to investigate the role of trait reappraisal, age, sex, and reactivity in the cross-sectional relationship between cumulative stress, CRA, and depressive symptoms. For each of the secondary variables that was examined, the mean centered variable was added as a main effect to the original regression model described above, along with all two-way interactions, and the three-way interaction between cumulative stress, CRA, and each control variable, respectively.

For models using CRA-SAD, when each of these models were examined, the two way interaction between CRA-SAD and cumulative stress remained non-significant (all $ps > .10$). There were, however, significant three-way interactions between CRA-SAD, cumulative stress, and trait reappraisal, $\beta = .13$, $t(156) = 1.98$, $p = .05$, CRA-SAD, cumulative stress, and sex, $\beta = .49$, $t(156) = 2.15$, $p = .03$, and CRA-SAD, cumulative stress, and sadness reactivity, $\beta = -.22$, $t(156) = -2.87$, $p = .005$.

When plotting these three-way interactions, the pattern of the relationship between CRA-SAD and cumulative stress in predicting depression was similar (i.e., the

relationship was not flipped) for those who were high versus low in trait reappraisal, as well as for both men and women. In both cases, the three-way interaction appears to be significant because of differences in the strength of the interaction between cumulative stress and CRA. For instance, the two-way interaction between cumulative stress and CRA-SAD was stronger for those who were high in trait reappraisal, relative to those who were low in trait reappraisal. Similarly, the two-way interaction between cumulative stress and CRA was marginally significant for women ($p=.08$), but not significant for men ($p=.18$). Because the pattern of results was not dramatically different depending upon trait reappraisal use or sex, I did not examine these 3-way interactions further.

An examination of the three-way interaction with sadness reactivity revealed that CRA-SAD was associated with lower levels of depression at high levels of stress for individuals who were high in reactivity, but this protective effect was not present for those who were low in reactivity. Although this interaction may indicate an interesting phenomenon, it does not appear to be a general pattern across measures – as discussed below, the same pattern is not present using CRA-SCL as the indicator of CRA, or when examining Time 2 depression. For this reason, the three-way interaction with reactivity was not examined further. There was not a significant three-way interaction between cumulative stress, CRA, and age ($p=.41$).

For models using CRA-SCL, when each of the control variables except for SCL-reactivity were added to the regression model, the two-way interaction between CRA-SCL and cumulative stress remained non-significant (all $ps>.32$). When SCL-reactivity was added to the model, the two-way interaction between CRA-SCL and cumulative

stress was significant ($p=.03$). This two-way interaction was qualified by a significant three-way interaction between cumulative stress, CRA-SCL, and SCL-reactivity, $\beta=.30$, $t(125)=3.44$, $p=.001$. Post-hoc examination of this interaction revealed that CRA-SCL was not associated with lower levels of depression for highly stressed individuals at both high and low levels of SCL-reactivity. Given the significant three way interaction with CRA-SAD, cumulative stress, and reactivity mentioned above, this counterintuitive finding might point to a very interesting phenomenon, the reliability of which should be examined in future research. There were no significant three-way interactions between CRA-SCL, cumulative stress, and age, sex, or trait reappraisal, respectively (all $ps > .36$).

Prospective effects of CRA. To investigate the prospective effects of CRA, another set of multiple regressions was performed. Depressive symptoms at Time 2 were entered as the dependent variable, and depressive symptoms at Time 1 were entered as a main effect to the regression model. Additionally, cumulative stress at Time 1, cumulative stress at Time 2, and CRA (both indices examined in separate regression models) were all entered as main effects. Finally, the interaction between CRA and cumulative stress at Time 2 was entered into the model. The results of these regressions are shown in Appendix A, Table 6 and Table 7. For the model with CRA-SAD, there was a significant main effect of Time 1 depressive symptoms, a significant main effect of Time 2 cumulative stress, and a significant interaction between Time 2 cumulative stress and CRA-SAD. To examine the interaction, the relationship was plotted using values ± 1 standard deviation on CRA-SAD and Time 2 cumulative stress, following the procedures outlined by Aiken and West (1991). This interaction is shown in Appendix B, Figure 3.

Simple slopes analyses revealed that the effect of Time 2 cumulative stress at low levels of CRA-SAD was significantly greater than zero, $\beta=.58$, $t(136)=7.86$, $p<.001$. Likewise, the effect of Time 2 cumulative stress at high levels of CRA-SAD was also significantly greater than zero, $\beta=.37$, $t(136)=4.23$, $p<.001$. Importantly, however, the effect of CRA-SAD at low levels of Time 2 cumulative stress was not significantly different from zero, $\beta=.04$, $t(136)=.58$, $p=.57$, while the effect of CRA-SAD at high levels of Time 2 cumulative stress was significantly less than zero, $\beta=-.17$, $t(136)=-2.33$, $p=.02$. Thus, at high levels of Time 2 stress, participants with higher levels of CRA-SAD had significantly smaller increases in depressive symptoms from Time 1 to Time 2, relative to those who were low in CRA-SAD.

For the model with CRA-SCL (shown in Appendix A, Table 7), there were significant main effects of Time 1 depressive symptoms and Time 2 cumulative stress. The main effect of Time 1 cumulative stress was marginally significant ($p=.06$). Neither the main effect of CRA-SCL nor the interaction between CRA-SCL and Time 2 cumulative stress were significant.

Secondary analyses of prospective effects. To ensure that the significant prospective interaction between Time 2 cumulative stress and CRA-SAD was not driven by potential confounds, the model for CRA-SAD described above was re-run, this time with sadness reactivity, trait reappraisal, age, and sex, added to the model, respectively.

When sadness reactivity, trait reappraisal, and age were each added to the regression model, the prospective two-way interaction between Time 2 cumulative stress and CRA-SAD remained significant (all $ps < .05$). When sex was added to the model, the

prospective interaction was marginally significant, $\beta=-.09$, $t(129)=-1.71$, $p=.09$. There was no significant main effect of sex on Time 2 depressive symptoms, $\beta=-.06$, $t(129)=-1.17$, $p=.24$, and no significant three-way interaction between Time 2 cumulative stress, CRA-SAD, and sex, $\beta=.15$, $t(129)=0.94$, $p=.35$. Therefore, the effect of sex on the prospective findings was not examined further.

Question Two: Do the Protective Effects of Cognitive Reappraisal Ability Differ Depending on How Controllable the Context is?

To answer Question Two, a series of multiple regressions was conducted to examine the cross-sectional effects of cumulative stress, CRA, and stressor controllability on depression. Depressive symptoms at Time 1 were entered as the dependent variable. CRA, Time 1 cumulative stress, and Time 1 stressor controllability were each entered as main effects, along with all possible two-way and three-way interactions. Two models were run to examine the effects of CRA-SAD and CRA-SCL separately. The results of these regressions are shown in Appendix A, Table 8 and Table 9.

Cross-sectional analysis using CRA-SAD. For the model with CRA-SAD (Appendix A, Table 8), there was a significant main effect of cumulative stress, a significant main effect of CRA-SAD, and a significant two-way interaction between CRA-SAD and stressor controllability. There was also a significant three-way interaction between cumulative stress, CRA-SAD, and stressor controllability. To examine the three-way interaction, the relationship was plotted using values ± 1 standard deviation on each of the independent variables, following the procedures outlined by Aiken and West (1991). This interaction is shown in Appendix B, Figure 4.

In the context of more *uncontrollable stress* (Figure 4, Panel A), simple slopes analyses revealed that the effect of cumulative stress at low levels of CRA-SAD was significantly greater than zero, $\beta=1.14$, $t(153)=5.66$, $p<.001$. Likewise, the effect of cumulative stress at high levels of CRA-SAD was significantly greater than zero, $\beta=.38$, $t(153)=2.62$, $p=.01$. Importantly, however, the effect of CRA-SAD at low levels of cumulative stress was not significantly different from zero, $\beta=-.04$, $t(153)=-.32$, $p=.75$, while the effect of CRA-SAD at high levels of cumulative stress was significantly less than zero, $\beta=-.80$, $t(153)=-3.63$, $p<.001$. Thus, in the context of relatively *uncontrollable stress*, the pattern that has been reported in previous research (Troy et al., 2010) was replicated; those who were highly stressed and high in CRA-SAD were significantly less depressed than those who were highly stressed and low in CRA-SAD.

In the context of more *controllable stress*, however, a different pattern was observed (Appendix B, Figure 4, Panel B). The effect of cumulative stress at low levels of CRA-SAD was significantly greater than zero, $\beta=.39$, $t(153)=3.35$, $p=.001$. Likewise, the effect of cumulative stress at high levels of CRA-SAD was significantly greater than zero, $\beta=.83$, $t(153)=6.01$, $p<.001$. Importantly, however, the effect of CRA-SAD at low levels of cumulative stress was not significantly different from zero, $\beta=-.10$, $t(153)=-.93$, $p=.35$, while the effect of CRA-SAD at high levels of cumulative stress was significantly greater than zero, $\beta=.33$, $t(153)=2.61$, $p=.01$. Thus, compared to uncontrollable contexts, the relationship between CRA-SAD and depressive symptoms at high levels of stress is *reversed* in the context of *controllable stress*. Specifically, at high levels of stress, those

who are high in CRA-SAD are significantly more depressed than those who are highly stressed and low in CRA-SAD.

Cross-sectional analysis using CRA-SCL. For the model with CRA-SCL (Appendix A, Table 9), there was a significant main effect of cumulative stress. In addition, there was a significant three-way interaction between cumulative stress, CRA-SCL, and stressor controllability. This interaction is depicted in Appendix B, Figure 5 using values ± 1 SD from the mean on each of the independent variables.

In the context of more *uncontrollable stress* (Figure 5, Panel A), simple slopes analyses revealed that the effect of cumulative stress at low levels of CRA-SCL was significantly greater than zero, $\beta=.99$, $t(131)=5.70$, $p<.001$. Likewise, the effect of cumulative stress at high levels of CRA-SCL was also significantly greater than zero, $\beta=.62$, $t(131)=3.97$, $p<.001$. The effect of CRA-SCL at low levels of cumulative stress was not significantly different from zero, $\beta=-.08$, $t(131)=.70$, $p=.49$. At high levels of cumulative stress, the effect of CRA-SCL was negative, indicating that those who were higher in CRA-SCL had lower levels of depression compared to those with lower CRA-SCL, however, this difference was not statistically significant, $\beta=-.30$, $t(131)=-1.45$, $p=.15$. Thus, in the context of uncontrollable stress, the pattern observed using CRA-SCL is similar to the pattern observed using CRA-SAD. Specifically, when moving from low to high stress, the increase in depressive symptoms is smaller for those who are higher in CRA-SCL, relative to those who are lower in CRA-SCL.

Compared to uncontrollable stress, in the context of more *controllable stress*, a different relationship was observed (Appendix B, Figure 5, Panel B). The effect of

cumulative stress at low levels of CRA-SCL was not significantly greater than zero, $\beta=.20$, $t(131)=1.07$, $p=.29$. However, the effect of cumulative stress at high levels of CRA-SCL was significantly greater than zero, $\beta=.80$, $t(131)=5.09$, $p<.001$. In addition, the effect of CRA-SCL at low levels of cumulative stress was not significantly different from zero, $\beta=-.15$, $t(131)=-.88$, $p=.38$, while the effect of CRA-SCL at high levels of cumulative stress was significantly greater than zero, $\beta=.46$, $t(131)=2.61$, $p=.01$. Thus, in uncontrollable contexts, the relationship between CRA-SCL, cumulative stress, and depressive symptoms is very similar to the relationship observed using CRA-SAD. Specifically, when moving from low to high stress, individuals who are low in CRA-SCL did not exhibit significant increases in depressive symptoms. Additionally, at high levels of stress, those who are low in CRA-SCL were significantly less depressed than those who were high in CRA-SCL.

Secondary analyses of cross-sectional effects. To ensure that the significant three way interactions described above were not driven by potential confounds, a new series of regression models was examined, which control for the effects of reactivity, trait reappraisal use, sex, and age. For models using CRA-SAD, when each of these control variables was entered into the regression model, the three-way interaction between CRA-SAD, cumulative stress, and stressor controllability remained significant (all $ps < .01$). In addition, there were no significant 4-way interactions between CRA-SAD, cumulative stress, stressor controllability, and each of the control variables, respectively (all $ps > .24$, except for the 4-way interaction with sex, $p=.09$).

For regression models using CRA-SCL, when each of the control variables were added to the model, the three way interaction between CRA-SCL, cumulative stress, and stressor controllability remained significant (all $ps < .03$). There were no significant 4-way interactions between CRA-SCL, cumulative stress, stressor controllability, and any of the control variables (all $ps > .38$, except for the 4-way interaction with age, $p = .06$).

Longitudinal prospective effects of CRA across contexts. To investigate whether the relationship between CRA, cumulative stress, and stressor controllability (described above) prospectively predicts changes in depression, another set of multiple regressions were performed. This time, Time 2 depressive symptoms were entered as the dependent variable. Time 1 depressive symptoms, Time 1 cumulative stress, and Time 1 stressor controllability were each entered as independent variables. In addition Time 2 cumulative stress, Time 2 stressor controllability, and CRA were entered as independent variables. This allowed me to assess change in depressive symptoms, change in cumulative stress, and change in stressor controllability over time. I additionally added all possible two-way interactions between Time 2 cumulative stress, Time 2 stressor controllability, and CRA to the model, in addition to the three-way interaction between these variables. As above, separate regressions were conducted for CRA-SAD and CRA-SCL, respectively. The results of these regressions are shown in Appendix A, Table 10 and Table 11.

For the model with CRA-SAD (Table 10), there were significant main effects of Time 1 depressive symptoms, Time 1 stressor controllability, and Time 2 cumulative stress. None of the other main effects or interactions in this model were significant.

For the model with CRA-SCL (Table 11), there were significant main effects of Time 1 depressive symptoms and Time 2 cumulative stress. None of the other main effects or interactions in the model were significant.

Chapter Four: Discussion

Previous research has consistently shown that the trait use of cognitive reappraisal allows people to effectively regulate their emotions, and is associated with positive psychological health outcomes (Garnefski et al., 2003; Gross & John, 2003). More recent research has focused on cognitive reappraisal *ability*, or the degree to which individuals can change their emotions when using reappraisal (McRae et al., 2012b, Shiota & Levenson, 2009; Troy et al., 2010). CRA has been shown to be empirically distinct from trait reappraisal use (McRae et al., 2012b), and to predict psychological health over and above the effects of trait reappraisal use (Troy et al., 2010). Specifically, CRA appears to be an important protective factor against depression in the immediate aftermath of stress (Troy et al., 2010).

To date, however, the effects of CRA on psychological health have only been examined cross-sectionally. Understanding the long-term relationship between CRA and psychological health is an important next step in building a causal model of CRA and long-term adjustment to stress. In addition, although most previous research on emotion regulation has focused on intraindividual processes, many theoretical accounts suggest that no psychological process is inherently adaptive in all contexts (Folkman & Moskowitz, 2000b; Mischel, 1968). This theoretical perspective suggests that CRA may not act as a protective factor in all contexts, and may even be associated with negative outcomes in some situations. Although previous research on CRA has not examined

whether the observed protective effects are context specific, theoretical considerations suggest that one aspect of stressful situations may be especially important: stressor controllability. Specifically, CRA may be a particularly adaptive process in uncontrollable situations: if very little can be done to change the situation itself, it may be better to change one's emotions instead. In more controllable situations, however, where direct action can and should be taken, CRA may be less adaptive. Thus, the present research extends upon previous research on CRA by examining two open questions: 1) Does CRA have long-term, prospective effects on psychological health? and 2) Do the protective effects of CRA differ depending on how controllable the context is?

The Prospective Effects of CRA

Previous research has found that CRA interacts with life stress to predict depression, such that those who are highly stressed and high in CRA are significantly less depressed than those who are highly stressed and low in CRA (Troy et al., 2010). Because these findings are cross-sectional, however, it remains unclear whether CRA is implicated in long-term adjustment to stress, and whether or not CRA plays a causal role in psychological health. That is, it may be that the effects of CRA on psychological health are short-lived, and therefore, not important for long-term outcomes. In addition, it is possible that the previously reported relationship between CRA and depression is driven by third variable confounds, or the directionality of the relationship between CRA and depression is reversed.

Therefore, in the first part of the present study, I examined the prospective effects of CRA on depressive symptoms. As hypothesized, CRA-SAD (the ability to down-

regulate feelings of sadness) interacted with changes in stress over six months to predict changes in depressive symptoms over six months. Specifically, individuals with large increases in stress and high CRA-SAD reported significantly smaller increases in depressive symptoms over a six month time period, relative to individuals with large increases in stress and low CRA-SAD. Thus, CRA appears to be an important protective factor against increases in depression for individuals who are highly stressed.

Theoretical implications. The observed prospective findings lend support to a theoretical model in which cognitive reappraisal ability is critically implicated in long-term adjustment to stress. These findings advance our understanding of how emotion regulation ability contributes to long-term risk and resilience in highly stressed populations. In line with previous research (Troy et al., 2010), CRA interacted with stress to predict depression, which supports the hypothesis that CRA is particularly important for psychological health amongst highly stressed individuals, perhaps because these individuals have elevated levels of negative emotions that need to be regulated.

The fact that individual differences in CRA-SAD *preceded* the observed changes in stress and depressive symptoms strengthens the argument that CRA causally contributes to psychological health in highly stressful contexts. That is, the prospective design used in this study allows us to rule out the alternative explanations that CRA may simply be a side effect of someone's current life stress, or an indicator of current depression. In addition, given that this relationship held when controlling for potential key confounds, it is unlikely that the present findings were simply driven by a third variable such as trait reappraisal use, sadness reactivity, age, or sex. However, while the

present study is an important step in the direction of a causal model of CRA, it does not allow for causal conclusions because it did not experimentally manipulate CRA.

In addition, these results enhance our understanding of what constitutes “successful” cognitive reappraisal. The present results support the hypothesis that, in addition to simply *using* an adaptive strategy like cognitive reappraisal, it is also critically important to be *able* to use this strategy to change one’s emotional states. Importantly, in line with previous research (Troy et al., 2010), the laboratory measure of CRA was unrelated to trait reappraisal use. In addition, the prospective findings remained significant when controlling for trait reappraisal, supporting the hypothesis that CRA predicts variance in psychological health over and above the effects of trait reappraisal use. These results are consistent with the idea that the frequency of reappraisal use is distinct from the ability to use reappraisal, and that these two constructs predict psychological health and adjustment to stress differently. It is also important to note that the laboratory measure of CRA was unrelated to several potential confounds, including cumulative stress, stressor controllability, age, education, self-efficacy, and verbal intelligence.

Notably, CRA-SAD was positively related to self-reported reappraisal ability, suggesting that this laboratory challenge measure is valid. The fact that CRA-SAD and CRA-SCL did not always predict depression in the same way and, (as shown in Appendix A, Table 3) did not always correlate with other measures in the same way suggests that these two indices of CRA may be capturing separable constructs, rather than two measures of the same thing. It will be important for future research to further

examine the regulation of feelings versus the regulation of arousal in order to better understand how these potentially independent abilities may differentially relate to psychological health.

Clinical implications. These findings also have several important clinical implications. First, these findings support a model of depression in which deficits in the ability to effectively regulate negative emotions play a key role in the development and maintenance of depression in highly stressed populations. In this way, these findings may help to elucidate the etiology of depression and other forms of psychopathology. Second, these results have the potential to inform clinical interventions for depression. Notably, many existing forms of treatment, especially cognitive therapies, already include large components that focus on identifying overly negative appraisals and replacing them with more realistic or positive reinterpretations (Campbell-Sils & Barlow, 2007). This practice clearly overlaps with the definition of cognitive reappraisal. In light of the current findings, it may be that cognitive therapies that strengthen individuals' ability to use reappraisal will lead to positive clinical outcomes. In particular, interventions that target negative appraisals may be particularly beneficial for highly stressed individuals, as well as other populations at risk for depression. Importantly, laboratory paradigms like the one used in the present study could potentially be used to identify people who are low in CRA, and thus, may be at particular risk for developing depression in the face of high stress. In this way, at risk individuals could potentially be identified and targeted for intervention before psychopathology has developed.

Discussion of null effects. It is important to note that the prospective effects that were observed for CRA-SAD (based on changes in self-reported sadness) were not present when using the second index of CRA, CRA-SCL (changes in skin conductance level). It is always difficult to determine why null results are obtained. For instance, if the effect sizes are smaller when using CRA-SCL as the index of CRA, the null results may have simply been due to a lack of power. Indeed, when examining the significant three way interactions between cumulative stress, CRA, and stressor controllability (reported in Appendix A, Tables 8 and 9), the effect size associated with the three-way interaction using CRA-SAD (change in $r^2=.04$, $p=.001$) was stronger than the effect size for the three-way interaction using CRA-SCL (change in $r^2=.02$, $p=.02$). Thus, even though both interactions were statistically significant, the effect size for the interaction with CRA-SAD was twice as strong as the interaction with CRA-SCL.

Additionally, it may be that the ability to change one's subjective experience of sadness is more important for psychological health than one's ability to change physiological arousal. That is, in the long-term, individuals who are only able to change their level of physiological arousal, but who are not able to feel subjectively "better" after reappraisal may not be protected against increased depression. It will be important for future research to better determine whether the present null results are simply due to a lack of power, or if physiological indicators of CRA are simply not associated with long-term psychological health.

In addition, the present study did not replicate the previously reported *cross-sectional* interaction between CRA and cumulative stress in predicting depressive

symptoms (Troy et al., 2010). As mentioned above, it is always difficult to interpret null effects, and there may be several reasons for this failure to replicate. First, the present study recruited individuals who had experienced a stressful life event in the past 8 weeks, while the previously reported study recruited individuals who had experienced a stressful life event in the past 12 weeks. Therefore, the time period between Time 1 and Time 2 captured in the present study (between 8 weeks and 6 months after the stressor) partially overlaps with the time period captured at Time 1 in the previous study (12 weeks after the stressor). Given that reappraisal is thought to be an adaptive strategy because it allows individuals to engage with negative events, and to find positive meaning in these events, it may be that it takes time after exposure to a stressor for the relationship between CRA and depression to emerge. If this is the case, the present sample may not have been far enough removed from the initial stress exposure to experience the benefits of CRA. Future research could examine this hypothesis by examining several windows of time since exposure to stress to better determine when the relationship between CRA and depression emerges. If the hypothesis is true, the significant interaction between CRA and stress in predicting depression would not be present until 12 weeks or more have elapsed since stress exposure.

Another potential explanation for the lack of cross-sectional effects observed in the present study may be the levels of cumulative stress observed at Time 1. Although the mean levels of cumulative stress observed in the present study ($M=15.9$, $SD=10.9$) are comparable to those observed in the previous study ($M=15.9$, $SD=11.5$; Troy et al., 2010), the observed range of scores was narrower in the present study (Range: 1-46)

relative to the previous study (Range: 2-60). Thus, the present sample included fewer people with very high levels of cumulative stress. Given that predicted differences in depression due to CRA were hypothesized (and previously reported) to be at high levels of stress, this restriction of range may have been responsible for the null cross-sectional results.

It should be noted, however, that the prospective relationship was significant in the current study, even though Time 2 stress levels were not as high as Time 1 stress levels (Time 2 cumulative stress $M=9.8$, $SD=9.3$, Range: 0-40). It may be that the relationship between CRA and depression in the immediate aftermath of stress exposure is particularly driven by individuals with very high levels of stress (and presumably the highest levels of negative emotions), while the long-term relationship between CRA and depression may hold for a wider range of stress levels, as people have more time to use reappraisal to make meaning of the stressors they have encountered. Future studies could test this hypothesis by recruiting participants with a wider range of stress levels, a wider range of time since stress exposure, and more than two data collection points.

The Protective Effects of CRA in Controllable Versus Uncontrollable Contexts

Although previous research (and the prospective results reported above) suggest that CRA is an adaptive process that protects against negative psychological health outcomes, theoretical models across the social sciences have emphasized that few, if any, psychological processes are adaptive in all contexts (Endler & Magnusson, 1976; Folkman & Moskowitz, 2000b). This raises the important question of whether there may be contexts in which CRA is maladaptive. I hypothesized that CRA is maladaptive (i.e.,

associated with increased depression) in controllable stressful contexts, where the most adaptive coping strategy likely involves taking direct action to decrease the impact of the stressor. In uncontrollable stressful situations, however, where more active coping is likely not possible, CRA should be an adaptive process because it allows individuals to manage the one thing they do have control over: their emotions.

In the second part of the present research, I found support for the hypothesis that the protective effects of CRA would be present in uncontrollable stressful contexts and absent in controllable stressful contexts. Specifically, in the context of relatively *uncontrollable stress*, CRA interacted with stress to predict depression, such that those who were highly stressed and high in CRA were significantly less depressed than those who were highly stressed and low in CRA. That is, for uncontrollable stress, the protective effects of CRA that have been reported in past research (Troy et al., 2010) were present. In the context of relatively *controllable stress*, however, this same pattern of results was not present. Instead, CRA interacted with stress to predict depressive symptoms such that those who were highly stressed and high in CRA were significantly more depressed than those who were highly stressed and low in CRA. Thus, the protective effects of CRA in the context of controllable stress were reversed, such that high CRA was associated with worse psychological health.

These results support a novel theoretical model in which the protective effects of CRA depend upon the context. In uncontrollable stressful contexts, in which an individual is unlikely to be able to diminish the negative effects of a stressor by taking direct action, CRA may be a particularly adaptive process because it allows the individual

to manage his or her negative emotions and help come to terms with the reality of the negative situation. In situations in which nothing else can be done, effective emotion regulation should be a particularly adaptive way to arm oneself against negative psychological health outcomes.

In controllable contexts, however, where it is possible for the individual to use more direct forms of coping such as active problem solving, CRA may be a maladaptive process. From a functionalist perspective, negative emotions can be adaptive, because they allow us to respond appropriately to the environment. For example, feelings of anxiety may motivate us to work harder if we are worried about an important deadline at work. In the context of controllable stress, the presence of negative emotions may be particularly adaptive because they motivate individuals to take direct action to cope with the stressor at hand. Importantly, however, individuals who successfully down-regulate their negative emotions in controllable contexts may not be motivated to take direct action, because they are no longer experiencing high levels of negative emotions. That is, effective emotion regulation may lead to negative outcomes in the context of controllable stress because it leads to lower levels of active coping. The present study does not allow for the examination of active coping. Therefore, future research should examine the hypothesis that high CRA may lead to lower levels of active coping in controllable stressful contexts.

Theoretical implications. The present study is one of the first to suggest that CRA may be maladaptive in certain contexts. Given that the relationship between emotion regulation ability and psychological health appears to be context dependent,

these results have very important implications for what “adaptive” emotion regulation is. That is, rather than simply considering which emotion regulation strategy is being used, and how effectively an individual can use a particular strategy, we also need to consider the context in which the strategy is being used. In this way, as others have suggested, the most adaptive emotion regulation may involve the flexible deployment of lots of different strategies, depending upon the context an individual finds him or herself in (Kashdan & Rottenberg, 2010). On a general level, these results suggest that it is important to consider the fit between persons (in terms of their ability to use different emotion regulation strategies, and the frequency with which they use these strategies) and situations (in terms of which strategies are generally adaptive in the specific context that the individual is in).

In the future, it will be important to continue testing this general theoretical model by examining other types of situational factors that may moderate the protective effects of CRA. Specifically, other factors related to the nature of stressful life events such as stressor predictability, normativeness, and acuteness may all also serve as important moderators. For example, it may be that CRA is more adaptive in the context of predictable (i.e., stressors that individuals know will happen ahead of time, for instance, retirement) stressors because individuals have lots of time to use reappraisal to come to terms with the reality of the event before it has even happened, which may enhance the protective effects of CRA. Relatedly, CRA may be maladaptive in the context of very acute (i.e., very short-lasting) stressors because there may not be an adequate amount of

time for the protective effects of CRA to emerge before the negative effects of the stressor have diminished simply as a function of time.

Although it is likely that numerous contextual factors serve as moderators of the effects of CRA, theoretical considerations suggest that the controllability of a stressful situation is a *key* moderator of these effects. That is, uncontrollable stressful situations appear to be particularly well-suited for CRA – when you cannot do anything to change the stressor itself, it is adaptive to be able to change your negative emotions. More controllable stressful situations appear to be more poorly suited for CRA, when direct action and problem solving would be more appropriate forms of coping.

It bears noting that the two broad forms of coping that have been discussed in the present study, emotion focused coping and problem focused coping, are by no means mutually exclusive. Indeed, Folkman and Lazarus (1980) have found that most people report using a combination of both types of coping when experiencing stress. Therefore, the results of the present study do not imply that an individual should never use reappraisal in a situation that is controllable. Rather, it appears important to consider *the degree to which reappraisal is being used*, relative to other active forms of coping. Future studies that examine both emotion focused coping and active coping are needed to better understand how these two forms of coping could be combined in potentially adaptive ways.

Future research should also examine other emotion regulation strategies to examine whether the context specificity observed in the present study would extend to strategies besides reappraisal. For instance, although suppression has been consistently

linked with negative psychological health outcomes (Ehring et al., 2008; Gross & John, 2003), there may be some situations in which the use of suppression is actually adaptive. For example, expressive suppression may be adaptive (or at least, less maladaptive) in the context of controllable stress, where not showing how one is feeling may better allow an individual to engage in more active coping processes (relative to a strategy like reappraisal, which may hinder active coping). On a more general level, it may be that emotion regulation strategies that target emotion experience are less adaptive in controllable circumstances, while strategies that target emotion expression are less adaptive in uncontrollable circumstances.

It is important to note that the vast majority of research on cognitive reappraisal to date has found a positive relationship between the use of this strategy and psychological health outcomes (cf. Garenfski et al., 2001; Gross & John, 2003; Kraaij et al., 2002). The present study is one of the first to suggest that reappraisal may lead to worse psychological health outcomes in certain contexts. This apparent contradiction is likely due to a number of factors. First, most research has not examined reappraisal in different contexts. The fact that many studies have found significant main effects of reappraisal on psychological health while collapsing across contexts, however, suggests that CRA is an adaptive process in many different situations. Second, most research has not examined reappraisal specifically in the context of high stress. Given that the context specific effects of CRA in the present study were only observed among highly stressed individuals, it may be that the context specific effects of CRA are not present in less

stressful situations. Future research can further test this hypothesis by examining other types of stressful contexts and a wide range of stress levels.

Clinical implications. While many existing clinical interventions may specifically target and strengthen CRA (Campbell-Sils & Barlow, 2007), these results suggest that individuals should be taught that reappraisal should be used in context appropriate ways, rather than simply using it in any emotional situation that an individual encounters. Specifically, clinicians could include psycho-educational components in their interventions that help clients understand when reappraisal would be a helpful strategy to use (i.e., uncontrollable situations), and when it wouldn't be helpful (i.e., controllable situations). In addition, it may be useful to help clients identify alternate emotion regulation strategies besides reappraisal that could be used in controllable contexts, such as distraction, problem-solving, or situation selection. The current study did not examine other regulatory abilities – future research should measure the ability to use other strategies like distraction, suppression, and acceptance to try to identify specific strategy/context combinations that appear to be adaptive.

Discussion of null effects. It bears noting that my hypotheses for Question 2 were only supported cross-sectionally. When examining the three-way interaction between CRA, cumulative stress, and stressor controllability prospectively, the results were not significant for either index of CRA. As with other null results in this study, this may have been due to a lack of power -- the regression models used to test these hypotheses required the presence of a large number of variables in the models, which may have resulted in these complex models being underpowered. Indeed, a post-hoc power

analysis, based on the size of the longitudinal regression models and the current sample size, estimates the minimum detectable β to be 0.3 assuming that power was 80% (Lenth, 2006-9). Given that the significant three-way interactions between CRA (both indices), stressor controllability, and cumulative stress at Time 1 both had β s less than 0.3, it seems likely that the Time 2 regression models in question were indeed underpowered.

In addition, because the present study design was not experimental, it is possible that there could have been an unmeasured third variable that accounted for the observed cross-sectional effects. This possibility seems unlikely, given that the results remained significant when controlling for key confounds such as reactivity, trait reappraisal, sex, and age. In addition, the observed discriminant validity of the CRA measures (see Appendix A, Table 2) and the measure of stressor controllability (see Appendix A, Table 3) was quite high.. In the future, however, it will be important to conduct high-powered longitudinal and experimental studies in order to test the claim that the observed relationship between CRA, cumulative stress, and stressor controllability predicts long-term changes in psychological health.

Limitations and Future Directions

The current study had several important methodological strengths that allowed me to build upon previous research. First, I tested my hypotheses using a large community sample of both men and women who represented a wide range of ages, ethnicities, and socioeconomic backgrounds. Thus, the results of the present study are highly generalizable. Second, I used a multi-method laboratory challenge measure of CRA, rather than self-reported ability. Thus, my estimates of CRA are unlikely to be

contaminated by retrospective biases, self-presentational biases, or a lack of insight into one's regulatory ability. Third, in addition to cross-sectional methods, I employed a longitudinal design, which allowed me to assess change in cumulative stress, stressor controllability, and depressive symptoms over time. This prospective design allowed me to better rule out third variable confounds and to make advances toward a causal model of CRA. Fourth, rather than using a self-report measure of stressor controllability, which may be confounded with an individual's current levels of life stress, depression, or coping ability, I recruited a separate sample of coders who provided more objective ratings of stressor controllability. There are four important limitations to the current study, however, that warrant further research.

First, the present study only examined individuals' ability to use reappraisal, which is just one of many different strategies that individuals can use to regulate their emotions. Indeed, the relationship between other types of regulatory abilities and psychological health has not been heavily studied, and remains poorly understood. In light of the finding that CRA is not adaptive in all contexts, it will be important for future research to examine individual differences in the ability to use other strategies such as suppression, acceptance, distraction, and situation selection in order to better understand how these abilities predict psychological health, and in which contexts these abilities may be adaptive.

Similarly, I only measured individuals' ability to use one very specific type of cognitive reappraisal: positive reappraisal. Recent research has shown that there are many different sub-types of reappraisal, including reappraisals related to self-distancing,

challenging reality, or agency (McRae, Ciesielski, & Gross, 2012; Ochsner et al., 2004)). It may be that individuals have different abilities to use different sub-types of reappraisal, and that different types of reappraisal predict psychological health and adjustment to stress in different ways. It will be important for future research to examine these important hypotheses. With particular regard to intervention and prevention programs, it may be helpful to know if an individual is low in the ability to use one type of reappraisal but high in the ability to use other types of reappraisal. In this way, unique strengths and weaknesses could be identified and targeted for improvement. It will also be important for future research to examine the context specific effects of other sub-types of reappraisal. For instance, reappraisals targeting an individual's agency may be particularly adaptive in controllable contexts.

Second, the present study only examined depression as the psychological health outcome of interest. This is because depression is one of the most common and one of the most debilitating outcomes associated with exposure to stressful life events (Greenberg et al., 2003). From a theoretical standpoint, I would predict that CRA is broadly implicated in psychological health, which would include other negative outcomes like anxiety symptoms and PTSD symptoms, as well as positive outcomes like psychological well-being and satisfaction with life. The present study, however, did not allow me to examine these other outcomes. In addition, the present sample was not a clinical sample but a community sample of highly stressed individuals who were at risk for increased depression. It remains unknown, therefore, whether the present results would extend to clinical populations that have been diagnosed with Major Depressive Disorder. From a

theoretical perspective, it seems likely that CRA is an important contributor to psychological health in both clinical and non-clinical populations. However, future studies are needed to formally test this hypothesis.

Third, I assume that CRA is a fairly stable ability that individuals possess, similar to intelligence, and is thus relatively unaffected by stress, depression, or socioeconomic conditions. The fact that neither index of CRA was significantly correlated with Time 1 cumulative stress, Time 1 depressive symptoms, stressor controllability, years of education, or verbal intelligence lends partial support to this hypothesis. However, the present study design did not allow me to assess test-retest reliability of the CRA measure. Therefore, it remains unknown whether an individual's performance on the CRA task is relatively stable over time.

Fourth, future research should continue to examine the validity of the CRA task. Although we avoid many important confounds that may be inherent in self-reported CRA, it is difficult to know if everyone in our sample was truly using reappraisal during the task, and thus, whether or not the CRA scores are truly tapping reappraisal ability and not another construct. The fact that CRA was not related to constructs like current depressive symptoms, cumulative stress, stressor controllability, age, or verbal intelligence, and that it was related to self-reported reappraisal ability certainly lends support to the hypothesis that we are measuring CRA with this task. It is still possible, however, that some participants did not understand the instructions, or used a different strategy during the task. This possibility was minimized by encouraging participants to ask questions if they did not understand the instructions, and the participants' responses

to the manipulation check questions indicated that they tried very hard to use reappraisal when instructed. However, future research can further explore this question by collecting more detailed data from the participants after the task to gain a better understanding of what participants are truly doing when they are instructed to reappraise. The fact that we still found significant results without being able to examine or remove individuals who were not following the instructions suggests that the present results may have been even stronger if we had been able to identify such individuals.

Concluding Comment

In sum, the results of the present study show that the ability to use cognitive reappraisal ability (CRA) serves as an important protective factor against long-term increases in depression. Specifically, CRA interacted with changes in life stress to predict changes in depressive symptoms, such that those individuals with large increases in stress and high levels of CRA reported significantly smaller increases in depressive symptoms over a six-month period, relative to individuals with large increases in stress and low levels of CRA. These results support the hypothesis that CRA is a critical contributor to psychological health in highly stressful contexts. However, part two of the present study highlighted the idea that the *type of stress* that individuals encounter matters. Results indicated that the protective effects of CRA are present in the context of uncontrollable stress, but the protective effects of CRA were reversed in the context of controllable stress. That is, in controllable stressful contexts, CRA was associated with increased depressive symptoms. Importantly, this is one of the first studies to show that cognitive reappraisal can be maladaptive in some contexts, which suggests that the protective

effects of specific emotion regulation strategies are context dependent. These results have important implications for understanding how emotion regulation ability contributes to risk and resilience in the face of stress, for clinical interventions and prevention programs, and for understanding what constitutes adaptive emotion regulation across contexts.

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Appendix A

Table 1
Participant Characteristics for Original Sample and Matched Coders

Variable	Original Sample (<i>n</i> =164)	Matched Coders (<i>n</i> =22)	Statistic	<i>p</i>
Mean age (SD)	40.4 (11.5)	38.3 (10.8)	<i>t</i> (184)=0.79	.43
Sex (% female)	50.6	45.5	$\chi^2(1)=0.21$.65
Race (%)			$\chi^2(4)=1.80$.77
White	85.3	81.8		
American Indian/Alaskan Native	1.2	0		
Asian	1.2	4.5		
Black	5.5	4.5		
Multiple Races	6.7	9.1		
Mean education (SD; 1-7; <7 th grade-graduate training)	5.6 (1.0)	5.5 (1.0)	<i>t</i> (184)=0.38	.71
Income (1-8; ≤10,000-≥100,000)	5.0 (2.1)	4.2 (1.8)	<i>t</i> (166)=1.56	.12
Mean depressive symptoms (SD; 0-60)	12.1 _a (9.9)	8.6 _b (7.0)	<i>t</i> (184)=2.06	.05

Note. Means with differing subscripts within rows are significantly different at the $p < .05$ level. Specific *ns* for each comparison may slightly differ due to different numbers of missing values across variables.

Table 2.

Correlations of Key Measures with Both Indices of Cognitive Reappraisal Ability (CRA)

	CRA-SAD ^a	CRA-SCL ^b
Variables in the regression model		
Time One Depressive Symptoms	-.14 [†]	.09
Time One Cumulative Stress	-.12	-.03
Time One Stressor Controllability	.01	-.06
Time Two Depressive Symptoms	-.22 ^{*c}	.18 ^{† d}
Time Two Cumulative Stress	-.20 ^{* c}	.15 ^d
Time Two Stressor Controllability	.14	-.11
Demographics		
Years of Education	-.09	-.04
Family Income	.26 [*]	.01
Age	-.05	-.08
Emotion Regulation and Personality Variables		
Trait Reappraisal Use	-.01	-.09
Self-Reported Reappraisal Ability	.18 [*]	-.13
Self-Efficacy	.12	-.11
Social Desirability	.22 [*]	-.03
Neuroticism	-.09	.15
Extraversion	.14 [†]	-.14
Emotional Reactivity		
Sadness Reactivity	.20 [*]	.01
SCL Reactivity	.10 ^e	.36 ^{* f}
Verbal Intelligence	-.01	-.02

^an = 164, except for Time 2 Variables and SCL Reactivity. ^bn = 127, except for Time Two variables and SCL Reactivity. ^cn = 136. ^dn = 107. ^en = 125. ^fn = 118. Specific *ns* for each cell differ due to different numbers of missing values across variables.

† $p < .10$, * $p < .05$

Table 3.
Correlations of Key Measures with Time 1 and Time 2 Stressor Controllability

	T1 Controllability ^a	T2 Controllability ^b
Variables in the regression model		
Time One Depressive Symptoms	.08	-.06
Time One Cumulative Stress	.21 [*]	.03
Time Two Depressive Symptoms	.03 ^c	-.07
Time Two Cumulative Stress	.15 ^{†c}	-.04
CRA-SAD (changes in sadness)	.01	.14
CRA-SCL (changes in skin conductance level)	-.06 ^d	-.11 ^f
Demographics		
Years of Education	-.07	.04
Family Income	-.13	.01
Age	-.11	-.08
Emotion Regulation and Personality Variables		
Trait Reappraisal Use	-.04	.08
Self-Reported Reappraisal Ability	-.05	-.03
Self-Efficacy	-.08	.08
Social Desirability	-.07	.07
Neuroticism	.10	-.03
Extraversion	-.06	.24 [*]
Emotional Reactivity		
Sadness Reactivity	-.05	.03
SCL Reactivity	.11 ^e	-.03 ^f
Verbal Intelligence	-.04	-.04

^an = 161, except for Time 2 Variables, CRA-SCL, and SCL reactivity. ^bn = 117, except for CRA-SCL and SCL reactivity. ^cn = 134. ^dn = 126. ^en = 123. ^fn = 93. Specific *ns* for each cell differ due to different numbers of missing values across variables.

† $p < .10$, * $p < .05$

Table 4
Current Depressive Symptoms as Predicted by Cognitive Reappraisal Ability (CRA-SAD: Changes in Sadness) and Cumulative Stress.

	<i>B</i>	<i>t</i>	<i>p</i>
Cumulative stress	.61	9.72	.001
Cognitive reappraisal ability (CRA-SAD)	-.07	-1.09	.28
Cumulative stress x CRA-SAD	.05	0.87	.39

Note. R^2 for the model = .39. *B* = *Betas* (standardized), n = 164

Table 5
Current Depressive Symptoms as Predicted by Cognitive Reappraisal Ability (CRA-SCL: Changes in Skin Conductance Level) and Cumulative Stress.

	<i>B</i>	<i>t</i>	<i>p</i>
Cumulative stress	.62	9.34	.001
Cognitive reappraisal ability (CRA-SCL)	.10	1.44	.15
Cumulative stress x CRA-SAD	.01	0.20	.85

Note. R^2 for the model = .41. *B* = *Betas* (standardized), n = 141

Table 6
Time 2 Depressive Symptoms as Predicted by Time 1 Depressive Symptoms, Time 1 Cumulative Stress, Time 2 Cumulative Stress, and Cognitive Reappraisal Ability (CRA-SAD: Changes in Sadness).

	<i>B</i>	<i>T</i>	<i>p</i>
Time 1 Depressive symptoms	.53	8.57	.001
Time 1 Cumulative stress	-.07	-1.05	.30
Time 2 Cumulative stress	.48	7.68	.001
Cognitive reappraisal ability (CRA-SAD)	-.06	-1.18	.24
Time 2 Cumulative stress x CRA-SAD	-.10	-2.01	.05

Note. R^2 for the model = .68. *B* = *Betas* (standardized), n = 136

Table 7
Time 2 Depressive Symptoms as Predicted by Time 1 Depressive Symptoms, Time 1 Cumulative Stress, Time 2 Cumulative Stress, and Cognitive Reappraisal Ability (CRA-SCL: Changes in Skin Conductance Level).

	<i>B</i>	<i>T</i>	<i>p</i>
Time 1 Depressive symptoms	.59	8.78	.001
Time 1 Cumulative stress	-.14	-1.89	.06
Time 2 Cumulative stress	.49	7.29	.001
Cognitive reappraisal ability (CRA-SCL)	.03	0.51	.62
Time 2 Cumulative stress x CRA-SCL	.02	0.33	.74

Note. R^2 for the model = .70. *B* = *Betas* (standardized), n = 119

Table 8
Current Depressive Symptoms as Predicted by Cumulative Stress, Cognitive Reappraisal Ability (CRA-SAD; changes in sadness), and Stressor Controllability

	<i>B</i>	<i>T</i>	<i>p</i>
Cumulative stress (Stress)	.69	10.55	.001
Cognitive reappraisal ability (CRA-SAD)	-.15	-2.34	.02
Stressor controllability (Control)	-.05	-0.66	.51
Stress x CRA-SAD	-.09	-1.21	.23
Stress x Control	-.07	-0.85	.40
CRA-SAD x Control	.26	3.33	.001
Stress x CRA-SAD x Control	.29	3.47	.001

Note. R^2 for the model = .45. *B* = *Betas* (standardized), n = 161

Table 9
Current Depressive Symptoms as Predicted by Cumulative Stress, Cognitive Reappraisal Ability (CRA-SCL; changes in skin conductance level), and Stressor Controllability

	<i>B</i>	<i>T</i>	<i>p</i>
Cumulative stress (Stress)	.65	9.65	.001
Cognitive reappraisal ability (CRA-SCL)	.02	0.31	.76
Stressor controllability (Control)	-.12	-1.55	.12
Stress x CRA-SCL	.05	0.76	.45
Stress x Control	-.13	-1.73	.09
CRA-SCL x Control	.13	1.57	.12
Stress x CRA-SCL x Control	.21	2.35	.02

Note. R^2 for the model = .45. *B* = *Betas* (standardized), *n* = 139

Table 10

Time 2 Depressive Symptoms as Predicted by Time 1 Depressive Symptoms, Time 1 Cumulative Stress, Time 1 Stressor Controllability, Time 2 Cumulative Stress, Cognitive Reappraisal Ability (CRA-SAD; changes in sadness), and Time 2 Stressor Controllability

	<i>B</i>	<i>T</i>	<i>p</i>
Time 1 Depressive Symptoms	.49	6.95	.001
Time 1 Cumulative Stress (T1 Stress)	-.05	-0.57	.57
Time 1 Stressor Controllability	-.14	-2.38	.02
Time 2 Cumulative Stress (T2 Stress)	.49	7.17	.001
Cognitive reappraisal ability (CRA-SAD)	-.07	-1.33	.19
Time 2 Stressor controllability (T2 Control)	.03	0.36	.72
T2 Stress x CRA-SAD	-.07	-1.18	.24
T2 Stress x T2 Control	-.05	-0.74	.46
CRA-SAD x T2 Control	.05	0.84	.41
T2 Stress x CRA-SAD x T2 Control	.06	0.88	.38

Note. R^2 for the model = .71. *B* = *Betas* (standardized), n = 115

Table 11

Time 2 Depressive Symptoms as Predicted by Time 1 Depressive Symptoms, Time 1 Cumulative Stress, Time 1 Stressor Controllability, Time 2 Cumulative Stress, Cognitive Reappraisal Ability (CRA-SCL; changes in skin conductance level), and Time 2 Stressor Controllability

	<i>B</i>	<i>T</i>	<i>p</i>
Time 1 Depressive Symptoms	.54	7.17	.001
Time 1 Cumulative Stress	-.11	-1.28	.20
Time 1 Stressor Controllability	-.09	-1.41	.16
Time 2 Cumulative stress (T2 Stress)	.48	6.56	.001
Cognitive reappraisal ability (CRA-SCL)	-.01	-0.18	.86
Time 2 Stressor controllability (T2 Control)	-.03	-0.40	.69
T2 Stress x CRA-SCL	-.05	-0.82	.41
T2 Stress x T2 Control	-.12	-1.68	.10
CRA-SCL x T2 Control	-.11	-1.80	.08
T2 Stress x CRA-SCL x T2 Control	-.09	-1.36	.18

Note. R^2 for the model = .73. *B* = Betas (standardized), n = 103

Appendix B

Figure 1. Schematic of procedures for measuring cognitive reappraisal ability. All participants were randomly assigned to either Group 1 or Group 2. As shown below, the only thing that differed between groups was the order of reappraisal instructions.

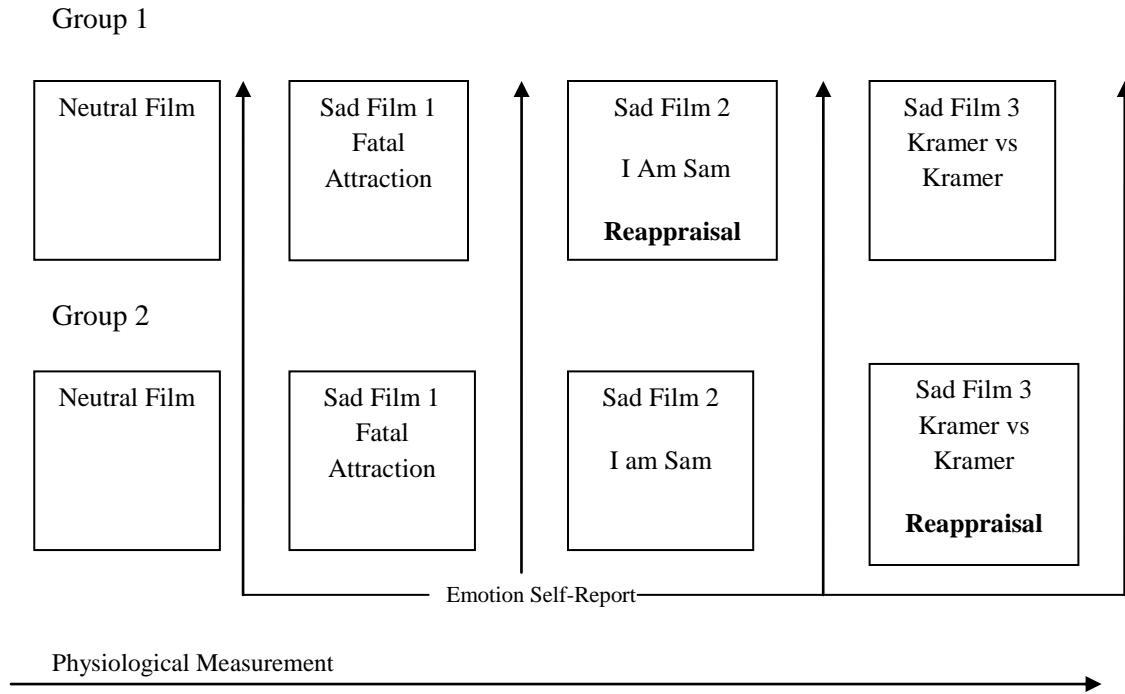


Figure 2. Sadness ratings (change from baseline sad film clip) during the cognitive reappraisal task for each experimental group. The Y-axis represents the z-scored sadness ratings during either Sad Film 2 or Sad Film 3, minus z-scored sadness ratings during the baseline sad film (Film 1). Thus, more negative scores mean greater decrease in self-reported sadness relative to the baseline sad film. R's indicate which experimental group was instructed to use cognitive reappraisal during each film clip.

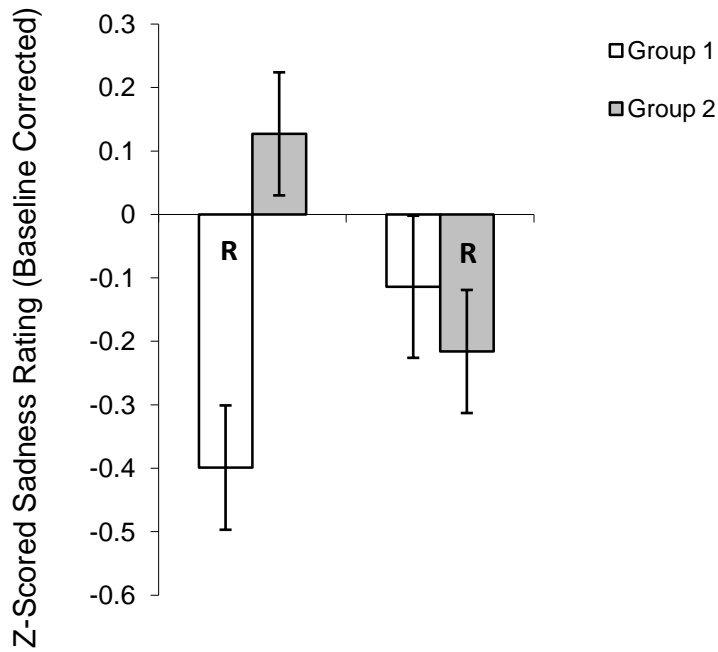


Figure 3. The interaction of Time 2 cumulative stress and cognitive reappraisal ability (CRA-SAD) on Time 2 depressive symptoms (BDI scores), controlling for Time 1 depressive symptoms and Time 1 cumulative stress. Values depict estimates at ± 1 SD for Time 2 cumulative stress and CRA-SAD. Error bars represent one standard error of the mean.

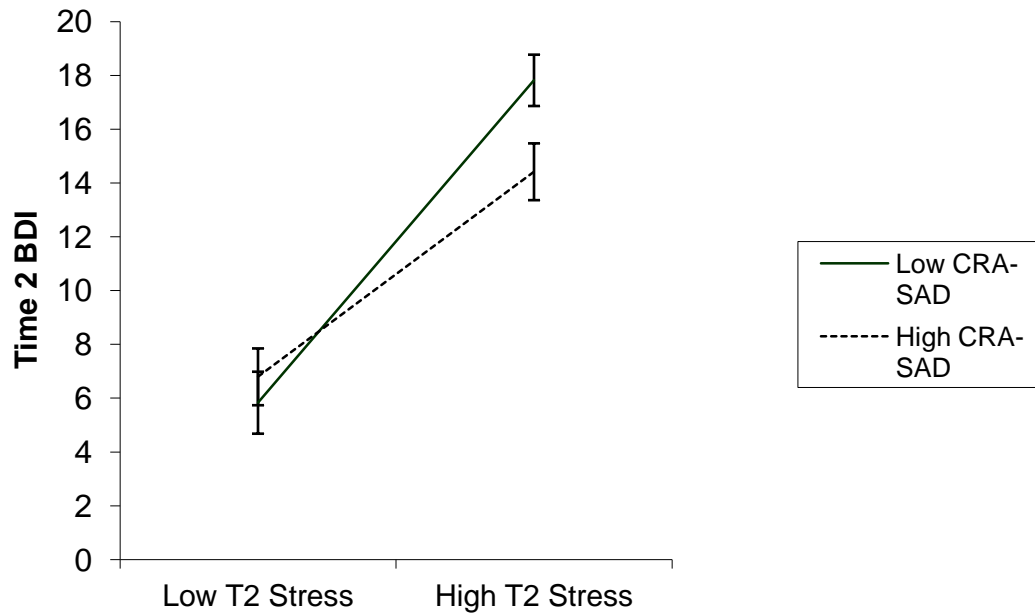
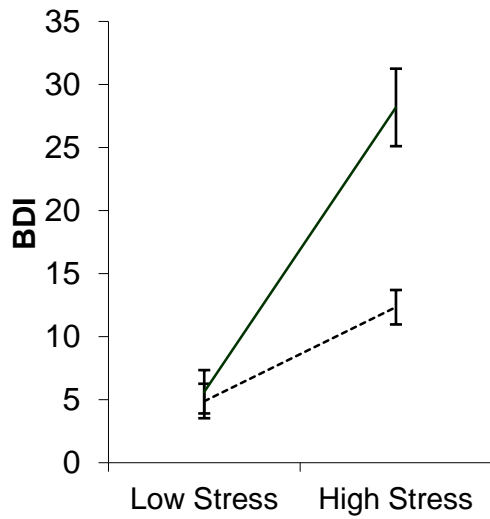


Figure 4. The three-way interaction of cumulative stress, cognitive reappraisal ability (CRA-SAD), and stressor controllability on current depressive symptoms (BDI scores). Values depict estimates at ± 1 SD for cumulative stress, CRA-SAD, and stressor controllability. Panel A depicts the interaction between cumulative stress and CRA-SAD in the context of more *uncontrollable* stress. Panel B depicts the context of more *controllable* stress. Error bars represent one standard error of the mean.

Panel A



Panel B

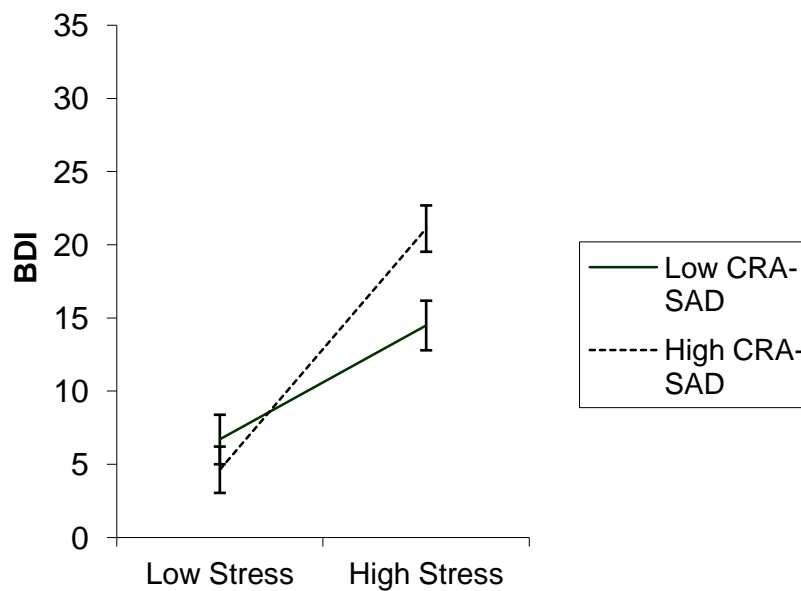
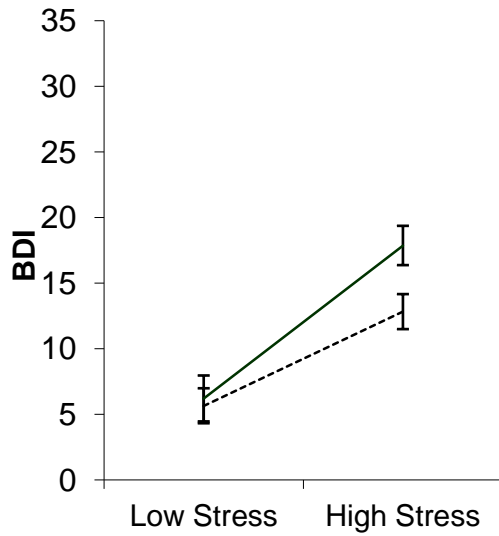


Figure 5. The three-way interaction of cumulative stress, cognitive reappraisal ability (CRA-SCL), and stressor controllability on current depressive symptoms (BDI scores). Values depict estimates at ± 1 SD for cumulative stress, CRA-SCL, and stressor controllability. Panel A depicts the interaction between cumulative stress and CRA-SCL in the context of more *uncontrollable* stress. Panel B depicts the context of more *controllable* stress. Error bars represent one standard of the mean.

Panel A



Panel B

