The Moderating Role of Emotional Regulation Regarding the Predictive Relationship Between Two Forms of Stress and Depressive Symptoms Among a

College Sample

A Thesis

Submitted to the Faculty

of

Drexel University

by

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in partial fulfillment of the

requirements for the degree

of

Master of Science in Clinical Psychology

December, 2014



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Dedication

I would like to dedicate this thesis to all of my grandparents for instilling in me the value

of hard work and perseverance.

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Acknowledgements

I would like to thank everyone who provided me with encouragement and support throughout this entire process. Firstly I would like to thank the members of my committee for all of their feedback throughout the course of this project. Specifically, I would like to thank my advisors Arthur Nezu, Ph.D., ABPP and Christine Maguth Nezu, Ph.D., ABPP for sharing their expertise, offering guidance, and providing inspiration for this project and all other endeavors, and my committee member, Kelly McClure, Ph.D. for sharing her valuable insight and feedback on this project.

I would also like to extend my immense gratitude to my family and friends for their continued encouragement, without which this thesis could not have been possible. Mom, Dad, and Michelle, you encourage me every step of the way and provide me with incredible support, for which I will be forever grateful. Lastly, I would like to thank all of my junior colleagues, faculty, and labmates for their help and support along the way.

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Abstract

The Moderating Role of Emotional Regulation Regarding the Predictive Relationship Between Two Forms of Stress and Depressive Symptoms Among a College Sample Jessica B. Stern, B.A. Arthur M. Nezu, Ph.D., ABPP

It has long been shown that varying types of stress can predict and precipitate the onset of a depressive episode. Much research has illustrated that there is an increased frequency and severity of premorbid stressors in depressed individuals as compared with controls (Hammen, 2005). However, not all individuals who experience stressful life events, irrespective of how severe they may be, experience psychopathological symptomology (Monroe & Simons, 1991). It is for this reason that the diathesis-stress model, in which certain factors, such as premorbid vulnerabilities, interact with stress and trigger depressive symptoms, has been studied and expanded upon extensively. Contrary to the earlier biological perspective of the model, researchers are increasingly recognizing psychological factors such as cognitive and behavioral styles and personality traits, as diatheses (Monroe & Simons, 1991).

While cognitive, behavioral and personality factors may be important, few investigators have studied the moderating role of emotional regulation. In Gross and Munoz's (1995) model of emotion, emotions are a response to, or interpretation of, events or intrapsychic processes. Furthermore, Gross (1998) described emotion regulation as "*the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions.*" Not surprisingly, it has been shown that there are many negative implications regarding the inability to successfully regulate emotions such as the increased risk for developing depression.

Whereas there is growing literature on the effects of emotion dysregulation on depression, there has been little research regarding how emotion regulatory processes fit into the stress-depression relationship. The aim of this study is to test whether emotion regulatory styles serve to moderate stress in predicting depressive symptoms. More specifically, 160 undergraduate students were recruited to complete the following measures: *Difficulties in Emotion Regulation Scale*, the *Center for Epidemiologic Studies* – *Depression-10*, the *Perceived Stress Scale* and the *Survey of Recent Life Experiences*. In analyzing the data, two hierarchical linear regressions were conducted to test the hypotheses that two measures of stress, perceived stress and experience life stress will each be moderated by emotion regulation. In the present study, experience life stress (as measured by the SRLE) was significantly moderated by emotion regulation while perceived stress was not. This suggests that stress measurements differentially interact with emotion regulation to predict depressive symptoms.

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INTRODUCTION

It has long been recognized that life stress of varying types can precipitate the onset of a diagnostic psychopathological episode, such as a depressive episode. However, scholars and clinicians alike have come to the realization that, interestingly so, not all individuals who experience stressful life events, regardless of their severity will experience psychopathological symptomology (Monroe & Simons, 1991). Conversely, individuals who experience what many would consider the fairly benign minutia of everyday life as particularly distressing, often times and seemingly unexplainably so, experience clinically significant, and perhaps even drastic, disturbances in mental health. Why might this be? Perhaps there are predispositions and/or protective factors that may dictate how an individual's stress and life experiences predicate and perhaps, cause depressive symptoms. In an attempt to assess this theory, we must examine the relationship between stress and depressive as well as other factors, such as emotion regulation that have predictive effects on depressive.

1.1 Stress and Depression

Diathesis-stress model

A vast body of literature has illustrated that stressors predict and increase the development and severity of depressive episodes (Hammen, 2005; Hammen, et al., 1992; Mazure, 1998). By nature of the fact that individuals struggle with stressors, such as illness, professional instability, and interpersonal conflict on a regular basis, it could be assumed that most, if not all, individuals would and should experience depressive symptoms at one point or another throughout their lives. However, although the

prevalence of depression is high and of clinically significant concern at a rate of approximately 10% in the United States (CDC, 2010), this occurrence would suggest that there must be a reason, if not a combination of reasons, that the remaining 90% of the population does not express, or at least report, depressive symptoms or meet criteria for a depressive disorder, other than the fact that they simply don't encounter stress in their lives. It is for this reason that over the past half-a-century scholars and clinicians have studied a model in which a variety and perhaps, amalgamation, of factors, such as premorbid vulnerabilities, interact with stress and lead to the development of depressive symptoms. This model, known as the "diathesis-stress model", synthesizes the predictive and causal effects of stress and premorbid vulnerabilities, recognizing that not only do they both play a role, but they are in fact linked. In this model, first conceptualized in the context of schizophrenia, the diathesis is the premorbid predisposition. In the presence of stress (whether episodic or chronic), the diathesis is triggered, potentially resulting in the development of a psychopathological episode (Monroe & Simmons, 1991). This model has been embraced by many, as it is intuitive, and incorporates and synthesizes the factors that present in the clinical scenario. A study that utilized a simulation model, suggested that not only is this model easy to understand and interpret, but it is an appropriate conceptual and theoretical framework to process the relationships between stress and depression (Patten, 2013).

Traditionally, as was proposed in light of the early medical model of mental illness, such premorbid dispositions were rooted in biological and genetic factors. Such a perspective, however, deemed insufficient as researchers and practitioners recognized the

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significance of psychological premorbid factors, such as cognitive and emotional styles and personality traits (Monroe & Simons, 1991). It is for this reason that we must examine which psychological factors may contribute to this model, and how our ability to assess and understand such factors may aid in our ability to determine, and perhaps even prevent, the development of depressive symptomatology. First, let us examine the relationship between stress and depression.

Stress as a predictor and cause of depression

The diathesis-stress model highlights the crucial impact stress can have on the development of psychopathology. Though the diathesis is the "foundational" factor in this chain, the chain is primarily worth investigation if the stress that is activated leads to distress, such as depression. As such we will first focus on the nature of the relationship between stress and depression in this model. A large body of research has illustrated that there is an increased frequency and severity of stressors in individuals who experience major depressive episodes prior to episode onset, as compared to individuals with no such episodes (Hammen, 2005). Furthermore, Mazure (1998) reported that studies have shown that depressed individuals are 2.5 times more likely than controls to have experienced a stressful life event prior to the onset of their depressive episode (Shrout, Link, Dohrenwend, Skodol, Stueve, & Mirotznik, 1989). Additionally, she highlights that in a group of studies conducted in the U.K. with a community sample of women, most studies showed that at least 80% of depressed individuals had experienced a major negative life event.

Although prevalence rates and statistical observations allow us to appreciate the fact that stress is an important predictor and perhaps causal factor in the development of depressive symptoms, we must take a step back and consider what is meant by "stress." There are in fact many types of stress: daily hassles such as traffic or a flat tire, social stress such as parental or spousal conflict, or severe stress such as chronic illness or loss. Additionally, stress can be acute or episodic, such as a fight with a boss, or it can be chronic, such as being a caretaker for an ailing family member. As such, it is likely that not all stressors, regardless of their temporal nature, content and severity, are equal in their propensity to lead to depression. Hammen (2005) delineates the difference between independent and dependent stress; independent stress, or "fateful" stress, for which one does not have a hand in causing or perpetuating, is less likely to predict depression than dependent stress, or those types of stress to which one has contributed. Additionally, she explains that chronic stressors are more predictive of depressive symptoms than are acute stressors. In a later study, however, Hammen and colleagues showed that both chronic and acute stressors predicted depression, and that there was a trend such that that greater acute stress was more strongly associated with depression in individuals with high chronic stress as compared to low chronic stress (Hammen, Kim, Eberhart, & Brennan, 2009). Unfortunately, however, individual researchers' theoretical understanding and operational definitions of chronic stress vary, and there has not been a thorough enough investigation into the differing effects of acute vs. chronic stressors (Hammen, 2005). Additionally, as it would seem to make sense that chronic stress plays a more salient role in depression, which many would consider to be a chronic illness, an important question arises: at what point does a chronic stressor trigger the onset of a depressive episode

(Monroe & Simons, 1991)? Unlike the effects of an acute stressor, such as sudden loss, from which the trigger point can be identified, the specific role of chronic stress is less clear. While the biological implications (e.g., immunological and genetic effects) of chronic stress have been studied in more recent years, the literature on psychological effects is lacking.

In addition to the temporal aspects of a stressor that may predict whether or not a depressive episode is impending, Monroe and Simons (1991) postulate that the particular qualities of a stressful event may be crucial. For instance, as they describe, a loss may be more likely to predict depressive symptoms than would danger, which would more likely predict anxiety symptoms. The severity and implications of both types of stress may be of equal value, however, the differentiating nature of the two problems cause them to translate into differing types of distress.

Lastly, it is incredibly important to recognize the nature in which a stressful event is assessed, such as whether a stressor is objectively or subjectively measured and deemed stressful. Some would say that the best way to assess the distress caused by a stressor is with a quantitative and/or qualitative subjective measure by the individual experiencing the stress. Others would argue, however, that individuals may differ in their appraisal of a stressful situation, particularly those who are depressed, such that the experience of an event may not in-and-of-itself predict a depressive onset for a given individual. Whereas this clash between the value of subjective measure and the subjectivity between individuals may seem like a conflict, it may actually serve great

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purpose in the understanding of how and why individuals appraise differently, how that difference may serve a clinical utility, and in light of the diathesis-stress model, what diatheses may lead to these differences.

Psychological diatheses

Whereas earlier versions of the diathesis-stress model focus on biological factors as the diatheses that activate a stress response, in more recent decades, particularly with the growth of cognitive and personality perspectives of mental health, psychological factors have been suggested to be key diatheses in this model (Monroe & Simons, 1991). One such type of factor, as is discussed by Beck and Rush (1978) are cognitive schemas, which are present premorbidly. Though schemas may not materialize into a problem or distress in the absence of an event that is dissonant or supportive of it's negative aspects, a stressor of some sort may activate the schema and lead to a depression. Furthermore, some have suggested that certain cognitive constructs, such as hopelessness, can serve as a diathesis to activate stress and to develop a specific type of depression: hopelessness depression (Spangler, Simons, Monroe & Thase, 1993). Moreover, it has been shown that cognitive organization can be seen as a vulnerability for depressive symptoms in light of stress (Seeds & Dozois, 2010). In addition to a cognitive framework of the diathesisstress model, others propose that personality factors too are important diatheses that can contribute to depressive symptomatology. Although there has not been a significant amount of work in this domain, Coyne and Whiffen (1995) discuss psychoanalytic perspectives of personality traits and styles that model the cognitive approaches to the diathesis stress model. Another variable that has been shown to be an important

moderator in the stress-depression relationship, social problem solving, is one that combines cognitive, behavioral, emotive, and personality constructs. Social problem solving is the cognitive-behavioral process by which individuals identify problems in their lives and effective ways to solve those problems. According to research by Nezu and colleagues (Nezu & Ronan, 1985; Nezu, Nezu, Saraydarian, Kalmar & Ronan, 1986), an individual's problem solving ability and style can, and likely will, moderate the predictive effects of his or her stress on the potential development of depressive symptomology.

Though the literature on psychological diatheses is limited (with the exception of the fair body of work on cognitive factors by Beck), Robbins and Block (1988) aptly state that much of the research on the relationship between stress and depression fails to recognize the importance of individual factors; only a small portion of depression scores is representative of life stressors, as some individuals are more vulnerable than others to particular life events and subsequently depression. Whether such individuals are more vulnerable due to cognitive schemas and attribution styles, personality traits or emotional reactivity, further work need be conducted in order to round out our understanding of the relationship between personal factors, stress and depression.

Moderators and mediators in the stress-depression relationship

Although the literature on psychological vulnerabilities to the manifestation of depression in response to stress is limited, there is a growing body of literature on what types of factors may potentially affect this relationship. This may provide an appropriate

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segue into further investigation on aforementioned psychological diatheses. In a thorough summary of potential moderators and mediators, Hammen (2012) presents research that has suggested that there are biological, developmental, and sociodemographic moderators and mediators. Additionally, she shows that there are key psychological moderators and mediators in this relationship such as attribution styles (Lewinsohn, Joiner & Rohde, 2001), self-esteem vulnerability (Bifulco, Brown, Moran, Ball & Campbell, 1998; Brown, Andrews, Bifulco & Veiel, 1990; Brown and Harris, 1978) and personality constructs such as neuroticism (Kendler, Kuhn, & Prescott, 2004; Kendler, Gardner, & Prescott, 2003; Poulton & Andrews, 1992). What is interesting, however, is that there has been limited research conducted to investigate how emotion, emotional processing, and emotion regulation factor into this relationship. One isolated recent study showed that emotion regulation is a mediator in the prediction of depression from interpersonal stress (Moriya & Takahashi, 2013), however further study on the relationship between these factors does not exist.

Stress and depression in college students

While the literature previously explored in this paper has applied to the general adult population, we must examine whether or not general adult trends in stress and depression apply to college students who will be the sample of this study. Depression is an ever-present clinical concern among college students, with approximately 15% of students being depressed (American College Health Association, 2008). Furthermore, research conducted in the past decade has suggested that approximately 18-70% of undergraduate college students have considered committing suicide or have had suicidal

ideations (Drum, Brownson, Burton, Denmark, & Smith, 2009; Westefeld et al, 2005; Gutierrez, Osman, Kopper, Barrios, & Sacks, 2000). Although suicidality is not necessarily directly linked to or perfectly correlated with depression, the high rates for depression likely contribute to the suicidality, and conversely the even higher rates of suicidality suggest that perhaps depression amongst college students is a larger problem than may be explicitly measured. For this reason, it is important to further investigate this subset of the population to determine if the stress trends found in the general adult

population can be seen here as well, possibly aiding in the predictability of depression in college populations.

In light of the discussion earlier about the different types of stressors and how they may predict depression differentially, it is firstly important to note that college students are less likely to experience major life stressors and are far more likely to experience "daily hassles" (Felsten, 2004). As Felsten explains, in college samples, the use of hassles inventories has been a useful measure of stress, and that even when measures of hassles were used along with measures of stressful life events, the cumulative effect of increased minor stressors was more predictive of physical and psychological distress than major life events (Felsten, 2004). This is interesting as it presents an opposite trend to that described earlier whereby chronic, major stressors are more predictive of depressive symptoms. Perhaps somewhat conversely, in a study conducted at a university in the UK, researchers examined the effects of five categories of adverse life experiences on depression and anxiety (financial difficulties, personal physical suffering, physical suffering of a close other, relationship difficulties, and valued

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items lost or stolen). The researchers found that the two types of stressors to significantly predict depression were financial difficulties and personal illness and injury (Andrews & Wilding, 2004). Although it is not clear whether or not those stressors were more chronic or episodic in nature, it would appear that they are more "severe" and perhaps more influential temporally than the daily hassles discussed by Felsten. Similarly, a study conducted among Puerto Rican college freshman found that the stressors most likely to predict depressive symptoms were stressors subjectively larger than daily hassles: relocation, break-up of a significant relationship, and illness (Reyes-Rodríguez, Rivera-Medina, Cámara-Fuentes, Suárez-Torres & Bernal, 2013).

Although there has been limited work done on the mediating and moderating effects of cognitive factors in the prediction of depression from stress among college students, Hammen and Cochran (1981) have shown that as has been seen in general adult samples, there are significant cognitive correlates, namely cognitions about consequences and uncertainty of life. This calls to question whether certain psychological variables (such as the tendency for one to view the world with a hopeless appraisal style) may serve as a diathesis in college students similar to the general adult population suggested earlier.

1.2 Emotion and Emotion Regulation

The construct of emotion

Emotion is perhaps one of the key constructs that separates human beings from other species. As humans, we have the ability to feel happiness, sadness, rage, and fear.

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But what exactly are emotions? This question may not be as easy to answer as one would think and the consensus on the definition of the construct is weak. According to Gross and Munoz (1995), emotions are reactions that are biologically rooted and serve as an adaptive response. In Gross and Munoz's model of emotion, our emotions are a response to our interpretation of external or internal events through the process of appraisal. In consequence, we respond to those events through the direction of those emotions. Additionally, before the emotions are expressed through behavior or physiology, they have the capacity to be modulated by psychological, environmental or physiological factors (Gross & Munoz, 1995).

Emotion regulation processes

Now that we have an understanding of what emotions are, let us examine what emotional regulation is. Gross (1998, pg. 275) described it as "the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions. Emotion regulatory processes may be automatic or controlled, conscious or unconscious, and may have their effects at one or more points in the emotion generative process." Though through this definition Gross emphasizes one's role, either consciously or unconsciously, in controlling his or her emotions, there is question and debate amongst psychologists as to whether emotion regulation is primarily a process in which one controls his or her emotions through cognitive processes (such as repression, distraction and reappraisal), or rather, if the conceptualization of emotion regulation should include an emphasis on one's ability to experience a wide range of emotion and more importantly, understand and appreciate those emotions. What

is most likely is that emotion regulation at its core is actually a combination of these two processes occurring in tandem. In a model presented by Gross and Munoz (1995), a distinction between antecedent-focused and response-focused emotion regulation is crucial. In this linear process, we behave in such a way so as to regulate the creation of an emotional response before it happens as part of antecedent focused emotion regulation, and modulate an already existing response in response-focused emotion regulation.

Consequences of emotion dysregulation

Whereas the definitions and conceptualizations of emotion regulation are variable, what it well accepted and established is that there are likely many negative implications of emotion dysregulation, or the inability to successfully regulate emotions, regardless of whether emotion regulation refers to a control process or an internalization of emotion. It had been suggested that emotion regulatory processes are a key component in healthy psychological living. Is has also been suggested that increased emotion regulation, as assessed through a measure of emotional intelligence, is linked to increased emotional sensitivity and prosocial behavior, suggesting that those who employ emotion regulation techniques are more likely to have meaningful interpersonal relationships (Lopes, Salovey, Cote & Beers, 2005). Additionally, evidence has shown that those with poor regulatory processes are at risk for developing depression, and perhaps cyclically those with depression are at risk for developing poor regulatory processes (Gross, 2013). Furthermore, as Gross (1997) shows through a comprehensive literature search, emotion dysregulation has been implicated in binge eating (Lingswiler, Crowther, & Stephens, 1989) and alcohol abuse (M.L. Cooper, Froner, Russell, & Mudar, 1995; Marlatt, 1985;

Sayette, 1993), as well as over half of Axis I and all of Axis II disorders of the DSM-IV (APA, 1994; Gross & Levenson, 1997; Thoits, 1985). In a meta-analytic view on the relationship between emotion regulation and psychopathology, Aldao, Nolen-Hoeksema and Schweizer (2010) showed that emotion regulation strategies that are generally maladaptive, such as avoidance, rumination and suppression, were positively correlated with psychopathology, although strategies that are generally considered positive, such as reappraisal, were negatively correlated with psychopathology. This evidence linking emotion dysregulation and maladaptive emotion regulation with psychopathology, such

as depression, strongly suggests that emotion regulation should be a large component in the assessment and treatment of clinical disorders. Though the relationship between emotion regulation and depression has been generally studied across genders, there is evidence to suggest that (a) males and females use different emotion regulation strategies, and (b) those differences may be reflected in, or causal to, gender differences in psychopathology (Nolen-Hoeksema, 2012).

1.3 Emotion Regulation and the Diathesis-Stress Model

Though further research need be conducted on the relationship between emotion regulation and depression to further dissect the nature of the relationship, as has been discussed above (see also Joormann & Gotlib, 2010) it has been well documented that emotion regulation, and more importantly, emotion *dysregulation*, is a major predictor of depression. There has been limited work, however, on how emotion regulatory processes fit into the stress-depression relationship. As Joormann and Gotlib state, "*theorists have suggested that depression vulnerable and non- vulnerable people do not differ primarily*

in their initial response to a negative event, but in their ability to recover from the ensuing negative affect" (Joormann & Gotlib, 2010, pg. 1). If we were to consider emotion regulatory styles, namely those that are ineffective, or non-existent, as a vulnerability, or a diathesis, that may provide useful information as to why certain people are more likely to appraise negative life events and stressors as stressful, such that they ultimately develop depressive symptomatology. Presumably, emotion regulation could follow a similar structure of cognitive schemas as a diathesis, as discussed earlier. Just as one may have the tendency to view their life as hopeless in the face of stress thereby developing depressive symptoms, so to may an individual who is unable to reappraise or suppress his or her sadness in the eyes of stress, similarly developing depressive symptoms.

1.4 The Present Study

Rationale

Although there has been previous work on the effect of emotion regulation on depression, as well as extensive literature that has examined the relationship between stress and depression, there is no literature on whether the effect of stress on depression is moderated by emotion regulation. As has been discussed previously, whereas there is evidence to suggest that such a model exists, there has been no research conducted to address this question or test this model. As such, the purpose of this study is to examine whether or not there is in fact an interaction effect between stress and emotion regulation in the prediction of depressive symptoms.

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Implications

Because stress is a high predictor of depressive symptoms in a relationship that is pervasive throughout the general population, having a better understanding of a model in which a moderator may strengthen or weaken this relationship can provide tremendous insight to a clinician looking to treat an individual with high life stress and either a susceptibility to or an already existing depressive disorder. Being able to identify emotion dysregulation as a significant factor in the development of an individual's depressive symptoms as a reaction to everyday life stress may further aid a clinician to target emotive and cognitive processes, as well as behaviors that need to be altered for a healthier lifestyle, either as part of a treatment modality for someone presenting with psychopathology or as a preemptive measure for someone at risk. Additionally, should there be a significant interaction effect, treatment can be better tailored for diagnostic populations with known deficiencies in emotion regulation, such as individuals with depression.

Aims and Hypotheses

The primary aim of the current study is to determine whether emotion regulation moderates stress as a predictor of depression.

- Specific aim 1. To determine if emotion regulation moderates actual experienced life stress as a predictor of depression.
 - Hypothesis 1. It was hypothesized that there would be an interaction effect between emotion regulation and actual experienced life stress on depression.

- Specific aim 2. To determine if emotion regulation moderates perceived stress as a predictor of depression.
 - Hypothesis 2. It was hypothesized that there would be an interaction effect between emotion regulation and perceived stress on depression.

METHODS

2.1 Recruitment

For this study, 160 individuals from the undergraduate community at Drexel University were enrolled to partake in an online survey. Students taking psychology classes during the duration of the recruitment phase were provided the opportunity to enroll and participate in this study, which was listed on the university Sona system, for extra credit for participating psychology courses. In addition to the study listing on Sona for all those who have access to the system to see, IRB approved flyers were distributed on psychology listserves and to students in undergraduate psychology courses via course Teaching Assistants.

The study was advertised to interested participants as a one-session, online participation, in which they would complete a series of questionnaires about their mood, the way they handled their emotions, and the stress in their lives. Furthermore, it was advertised to take approximately 30-40 minutes and would involve no expected risks, including anticipated discomfort from answering questions, as all questionnaires are

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rather benign. Participants who completed the study were compensated with two credits of extra credit on Sona towards one of their psychology courses.

2.2 Sample

The sample included undergraduate students taking psychology courses who were eligible for extra credit on Sona. Participants were required to be enrolled at the university and fluent English speakers as either a first or second language. No screening procedures were used for exclusion of participants.

2.3 Measures

Emotion Regulation. In order to assess emotion regulation, the *Difficulties in Emotion Regulation Scale (DERS;* Gratz and Roemer, 2004) was administered. The DERS was developed to assess clinically relevant difficulties that individuals may have in the following dimensions of emotion regulation: "(a) awareness and understanding of emotions; (b) acceptance of emotions; (c) the ability to engage in goal-directed behavior, and refrain from impulsive behavior, when experiencing negative emotions; and (d) access to emotion regulation strategies perceived as effective" (Gratz and Roemer, 2004). More specifically, this self-report questionnaire includes 36 items designed to reflect the four aforementioned constructs of emotion regulation. Participants are presented with statements intended to capture how certain feelings lead to other feelings, whether or not they can control their feelings and how those feelings were to affect them if they were to feel them. They are then asked to rate on a scale of one to five how often those statements apply to them. In addition to producing an overall total item score, six subscales reflective of the six primary factors are produced: nonacceptance, goals, impulse, awareness, strategies, and clarity. In order to assess the initial psychometric properties, this measure was administered to undergraduate psychology students at the University of Massachusetts Boston by the scale's developers. In addition to the administration of the DERS, scales on the expectancies for the self-regulation of negative moods (Generalized Expectancy for Negative Mood Regulation Scale; Catanzaro & Mearns, 1990), experiential avoidance (*The Acceptance and Action Ouestionnaire*; Haves et al., 2004), emotional expressivity (The Emotional Expressivity Scale; Kring, Smith, & Neale, 1994), deliberate self-harm (The Deliberate Self-Harm Inventory; Gratz, 2001) and childhood physical and sexual abuse (The Abuse-Perpetration Inventory; Lisak, Conklin, Hopper, Miller, Altschuler, & Smith, 2000) were administered and contrasted with the DERS. Data collected from 373 participants who completed these questionnaires indicated that the DERS had strong internal consistency with item-total correlations ranging from r =.16 to r = .69. Similarly, the DERS subscales representative of the six primary factors exhibited adequate internal consistency, with Cronbach's $\alpha > .80$ for each subscale (Gratz and Roemer, 2004). Additionally, the DERS presented with adequate construct and predictive validity good test-retest reliability, and adequate construct and predictive validity (Gratz and Roemer, 2004).

Depressive symptoms. The Center for Epidemiologic Studies – Depression, Revised (CES-D-10, also known as the CES-D-R) was used for assessment of depressive symptomology. The CES-D-10 is an abbreviated 10-item self-report scale adapted from the 20-item depression assessment developed by the Center for Epidemiologic Studies. The scale was designed as a short self-report used to assess depressive symptomatology in the general, non-clinical population, (Radloff, 1977). In the factor analysis used to design the full 20-item scale, four factors were found to be underlying the items of this scale: depressed affect, positive affect, somatic and retarded activity, and an interpersonal factor. Although there are items on this scale that assess all four of these depressive constructs, the scale focuses on depressive affect (Radloff, 1977). Participants are instructed to indicate the frequency at which they experienced each symptom in the past week on a scale of zero to three. Though the original full-item scale is widely used as a diagnostic and screening tool, the CES-D-10 was developed to increase feasibility and reduce stress in participants completing the scale as compared with the full-item version (Irwin, Artin, & Oxman, 1999). The structure and use of the abbreviated models that of the full-item version, while focusing primarily on the items related to emotion. Additionally, the abbreviated version demonstrates the same four-construct model as the full-item version without a sizable compromise in validity and reliability. The CES-D-10 has exhibited high internal consistency ($\alpha = .92$) and test-retest reliability (r = 0.83) as well as good specificity (84%), sensitivity (97%) and positive predictive value (85%) when using a cut-off score of \geq 4(Irwin et al., 1999).

Stress. In order to evaluate stress, both perceived stress and actual experienced stress were measured. As previous research has shown, individuals employ various different adaptive and maladaptive coping styles in response to stressors. Though certain styles such as acceptance and reappraisal may help diminish the negative perception of a stressor, certain coping styles very commonly seen in individuals who present with

depressive symptoms, such as catastrophizing, amplify an individuals perception of their stressors. Furthermore, these maladaptive internalizations of stress may play a role in such an individual's negative emotions (Martin & Dahlen, 2005; Garnefski, Kraaij, Spinhoven, 2001). For this reason, it was both helpful and beneficial to capture how an individual interprets the stress in his or her life (i.e. "perceived stress") as well as actually experienced stress. Collection of both types of information allowed for the assessment of (a) whether or not there are differences in the way individuals experiencing the same types of stress perceive and internalize that stress, and, (b) whether individuals' emotion regulation skills affect the "differential" between perceived and actual stress, and (c) whether the potential "differential" affects the predictive value of stress on depression.

For the measurement of perceived stress, the *Perceived Stress Scale* (PSS; Cohen, Kamarck & Mermelstein, 1983) was used. This 10-item self-report questionnaire was designed to measure the degree to which situations in individuals' lives are appraised as stressful, while specifically reflecting the extent to which individuals find their lives to be unpredictable, uncontrollable and overloading. This scale was developed to be used with individuals of community samples with a minimum of a middle school education (Cohen, Kamarck & Mermelstein, 1983). Participants asked to complete this scale are instructed to rate, on a scale of one to five, how often they experience a variety of statements that capture the notion of feeling stressed. The initial psychometric properties of the PSS were evaluated across two college samples with a cumulative sample size of 446 individuals. Participants also completed the College Student Life-Event Scale (CSLES; Levine & Perkins, 1980), the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff,

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1977), the Cohen-Hoberman Inventory of Physical Symptoms (CHIPS; Cohen & Hoberman, 1983) and the Social Avoidance and Stress Scale (Watson & Friend, 1969) for comparison with the PSS. In addition to the college population, the PSS was also evaluated in contrast with the Cohen-Hoberman Inventory of Physical Symptoms in a sample of 54 individuals in a smoking-cessation study. The PSS demonstrated good internal consistency among all three samples ($\alpha = 0.84$, 0.85, 0.86, respectively) and adequate test-retest reliability in the follow up college and smoking cessation samples (.85 and .55, respectively; Cohen, Kamarck & Mermelstein, 1983).

To measure actual experienced stress, the *Survey of Recent Life Experiences* (SRLE; Kohn & Macdonald, 1991) was used. This assessment is a 51-item questionnaire, developed as an alternative to other commonly used scales to assess stress associated with daily hassles. The questionnaire was designed as an alternative to "daily hassles" questionnaires as these scales were thought to be contaminated such that they would "reflect the disturbance...they were intended to predict" (Kohn and Macdonald, 1991, pg. 222). This scale represents six factors: social and cultural difficulties, work, time pressure, finances, social acceptability and social victimization. In this scale, participants are presented with a list of stressors (both episodic and chronic) and are asked to rate on a scale of one to four how much such stressors have been a part of their lives in the past month. The psychometric properties of the scale were assessed with two adult community groups, the item-selection subsample (n=100) and the cross-replication subsample (n=136) and items overall were correlated with the PSS. The internal consistencies of both subsamples were strong ($\alpha = .92$ and .91, respectively) with adequate correlation

against the PSS (.57 and .60, respectively and p < .01) (Kohn and Macdonald, 1992). Although this scale does not include many of the major life stressors or significant chronic stressors that several of the researchers discussed previously believe are most likely to be implicated and predictive of depression, it captures the stressors that are most likely to be experienced by the general population, particularly those who are university students.

2.4 Procedure

Interested participants registered for the study on Sona and were then granted access to an online link where they completed the agreement to participate and complete the study measures. As the study was eligible for an IRB waiver of consent, and Sona does not have the platform for a formal online consent process, there was no official consent process. However, due to the ethical importance for prospective participants to understand their rights, a summary of the necessary and relevant components of a standard IRB consent form was included. The following language was presented at the beginning study of the study to resemble a consent form:

"As part of this research study, you will be asked about your basic demographic information, as well as questions about stress and sadness you may have recently experienced and how you experience and handle your emotions. We don't anticipate that there are any risks associated with participation; however, in the event that participation leads to some distress, you may, at any time, stop filling out the questionnaires without any adverse effects from the investigators.

All of your information will be kept confidential and only trained and approved study personnel will have access to it. Additionally, once you begin the study, you will be assigned a study ID number, and your name will not be associated with any of your responses. If you wish to withdraw from the study at any time, you are welcome to do so at no penalty to you. If you withdraw from the study, the research team will have no record of you signing up to and withdrawing from the study, and as such your professors will not be privy to this. However, if you do

withdraw from the study, you will not receive credit. In order to receive credit, please follow through the entirety of the survey until the end.

In order to ensure that your effort it reflective of true and meaningful responses, we ask that you please complete this survey at a time when you can devote 30-40 minutes of your time with no distraction. Once you start the survey you will not be able to pause and come back without losing your data. If you cannot complete the survey in one sitting but would like to participate again, you may withdraw from the study and sign up again at a later time."

Individuals were then presented with two questions:

- 1. Do you understand the above information?
 - Yes
 - *No*
- 2. Based on the above information, do you agree to participate in this study?
 - If yes, please click here, and continue onto the next page.
 - If no, please click with the withdraw button on the top right hand corner.

Following the second question, individuals who selected the former statement were then presented with the first set of questionnaires on the next screen. The first set of information that was asked of participants was basic demographic information, which included inquires about age, sex, race, educational study and standing at Drexel and course of study/major. Participants then completed the primary questionnaires in the following order: DERS, PSS, SRLE and CES-D. Time of participation lasted on average approximately 12 minutes (range = 4 - 43 minutes). After participants completed the presented questionnaires, they were presented with a screen with the following statement: "I agree that I completed the previous questionnaires to the best of my ability". In response, individuals were required to select a "Yes" or "No" box. Additionally, as the study did not allow for debriefing, there was a text box with the follow optional prompt:

"If you had any trouble completing the previous questionnaires (e.g., there was something you didn't understand, had to guess on, etc.), please list them below. This will help us interpret your data with the utmost accuracy".

Those who completed participation were awarded two extra credit points on Sona.

2.5 Statistical analysis

All data analysis was conducted using SPSS 20. Firstly, descriptive statistics were run to assess demographic information. Additionally, a correlation matrix was produced in order to examine correlations between all of the key variables (DERS, PSS, SRLE, and CESD). In order to assess the primary hypothesis, two hierarchical linear regressions were performed, whereby, stress, emotion regulation, and the interaction of stress and emotion regulation were entered as the predictor variables and depressive symptoms was entered as the outcome variable. In the first regression, PSS was used as the measure of stress and in the second regression SRLE was used as the measure of stress. In order to determine whether or not the previously discussed potential gender effects in the relationship between emotion regulation and depression were present, gender differences among variables were assessed. Such differences did not exist, and therefore, gender was not necessary to use as a covariate.

Complete data sets for all participants were obtained as the online platform was designed such that individuals could not proceed onto questions without completing previous questions. As such, no data was missing and all measurements for all participants are included.

RESULTS

3.1 Sample characteristics

Demographic characteristics of the sample are summarized in Table 1. The sample consisted of 46 males and 114 females between the ages of 17 and 38, with a mean age of 20.3 years (SD = .21). The majority of participants self-identified as Caucasian (51.2%) and Asian (30.6%), while the rest identified as Hispanic/Latino (1.3%), Other (6.9%) and Multiple Race (5.0%). Of the participants 73 (45.6%) were freshmen, 25 (15.6%) were sophomores, 34 (21.3%) were juniors, and 28 (17.5%) were seniors. The overwhelming majority of individuals (n = 133; 83.1%) who participated identified as English as their first or native language.

3.2 Assumption testing

Descriptive statistics were conducted to check the major assumptions of a multiple regression: linearity, normality, homoscedasticity, and independence of residuals. Testing revealed that all assumptions were met, and as such, transformation of any of the variables was not necessary.

3.3 Preliminary analyses

Descriptive statistics of the independent and dependent variables, including means, standard deviations, minima, and maxima, were computed for all continuous variables, and are summarized in Table 2. Of note, perceived stress in this sample (M = 19.5, SD = 7.2) was slightly elevated as compared to the age norm for the PSS (M = 14.2,

SD = 6.2). Additionally, the mean depressive symptom score on the CESD-10 (M = 9.89) was just below the scale cutoff score of 10 for depression.

A correlation matrix was produced in order to assess the relationship between all of the key variables: DERS, PSS, SRLE, and CESD. Significant correlations were found between all pairs of the variables at the <.01 level, and can be seen in Table 3. Additionally, independent samples t-tests were conducted on each of the independent and dependent variables between genders. Significant differences were not found between males and females on perceived stress (t(158) = -.69, p = .49), actual experiences stress (t(158) = -1.03, p = .30), emotion regulation (t(158) = -36, p = .72), or depressive symptoms (t(158) = -1.37, p = .17). Additionally, multiple independent samples t-tests were used to determine if there were significant differences in the variables of race or class on any of the independent and dependent variables. Significant differences on any such variables were not found.

3.4 Primary analyses

The primary aim of this study was to determine if emotion regulation moderated the predictive effects of stress, both perceived and actual experienced stress, on the manifestation of depressive symptoms. It was hypothesized that emotion regulation would moderate the relationship between depression and each measurement of stress. To test this hypothesis, two hierarchical linear regressions with centered predictor variables were used. In the first regression, PSS, DERS, and a PSSxDERS interaction term were used as predictor variables, and CES was imputed as the dependent variable (Table 4). In

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this regression, perceived stress and emotion regulation both significantly predicted depressive symptoms (b = .48, t(156) = 7.93, p < .001; b = .07, t(156) = 2.65, p < .001; respectively), however, contrary to the hypothesis, the interaction term was not significant (b = .00, t(156) = .14, p = .89).

A second regression was conducted in which, SRLE, DERS, and an SRLExDERS interaction term were used as predictor variables, and depression was entered as the dependent variable (Table 5). In this regression, actual experienced stress and emotion regulation both significantly predicted depressive symptoms (b = .12, t(156) = 7.12, p < .001; b = .11, t(156) = 4.55, p < .001; respectively). Additionally, while the R² Change of the interaction term was small (R² Change = .02), it was significant as hypothesized (b = -.00, t(156) = -70, p < .001). The negative direction of this interaction effect reflects the hypothesized relationship between the variables. Specifically, due to the fact that higher scores on the DERS reflect poorer emotion regulation, or emotion *dysregulation*, lower scores on this scale would be expected to be, and in fact were, moderators of higher actual experienced stress. A plot of the interaction effect at high and low values of emotion regulation (at one standard deviation above and below, respectively) can be seen in Figure 1.

DISCUSSION

4.1 Review and Conclusions

Stress in all manifestations, chronic and episodic, perceived and experienced, is known to be a strong predictor of depressive symptoms (Grant et al., 2013; Hammen, 2005). Although the temporal nature, severity, content and chronicity of stress can aid in

the prediction of depressive symptoms, these characteristics of a stressful experience or sustained problem are not enough to provide insight as to who and why will develop depressive symptoms, and furthermore, a diagnosable depressive disorder in the face of stress. The diathesis-stress model has allowed researchers and clinicians to recognize that additional factors may play a role in this relationship. Although psychologists have been increasing their study of the role of several psychological factors in this model, the study of emotional regulation is sparse. This study aimed to assess whether or not emotion regulation (or here measured as, *dysregulation*) moderates the predictive effects of stress on depressive symptoms. Stress was assessed in two demonstrations: perceived stress and actual experienced stress.

Emotion regulation was found to be a significant moderator in the prediction of depressive symptoms from stress when the stress being assessed was actual experienced stress. While the R^2 Change for the interaction term in this model was not large (R^2 Change = .02), the addition of interaction term was significant. This is contrasted with the perceived stress model in which the interaction term was in fact not significant. The difference likely lies in the process by which a person perceives a problem and reacts to it. In the model of experienced stress, an objective stressor is presented to an individual and he or she is required to process it, both cognitively and emotionally. In addition to the cognitive appraisal aspect of the process (e.g., "this stressor is unfortunate", or "this stressor is a challenge I can learn from"), the process also involves emotion regulation, by which the individual will respond with emotion (e.g., fear, anger, excitement). In this model, emotion regulation likely presents as a moderator due to the fact that the way in

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which an individual responds to the objective stressor can determine the outcome of both the stressor and the individual's wellbeing following it. This conception is supported by work regarding resilience in the face of stress. Troy and Mauss (2011) discuss the notion in which two individuals who experience the same stressor may be unequally likely to react negatively to the stressor due to individual personal differences, such as emotional regulatory styles. While they have not tested this model, they propose that emotion regulation is a moderator in the stress-resilience relationship such that the way an individual appraises and internalizes a stressor he or she may encounter may affect his or her resilience. This conceptualization can be extrapolated to depressive symptoms whereby it can be assumed that should emotional regulation interact with stress to predict resilience, by extension depressive symptoms resulting from poor resilience can be further predicted.

Contrastingly, in the model of perceived stress, the appraisal process has already occurred; an individual has already interpreted the stress that surrounds him or her nearly sealing the fate of their ultimate response. The appraisal that has already occurred suggests that perhaps the cognitive appraisal that promotes the individual to perceive stress as high enough to induce depressive symptoms already includes an element of emotional processing that either stumps emotion regulatory processes from moderating the relationship, or is encompassing this process enough for interaction of stress and emotion regulation to add nothing new to the model. Furthermore, as compared to the course in which an individual processes, both cognitively and emotionally, his or her actual experienced stress in generally isolated scenarios, perceived stress can be viewed

as a generally more pervasive state in which an individual views stressors in his or her life and sees a "cloud of distress". This more global perception eliminates, or muddles,

the linear process be which an individual's underlying emotional regulatory styles can mitigate the stressor-to-depression pathway.

The role of cognitive appraisal and emotional regulation, both individually and in synchrony, as described above, seems to be supported by literature. Previous studies have suggested that not only is the cognition-emotion relationship strong, but they are pieces to the same puzzle, or process. One study showed that cognitive reappraisal, conceptualized and measured as an emotion regulation technique, was a moderator in the stress-depression relationship (Troy, Wilhelm, Shallcross, & Mauss, 2010). Additionally, another study showed that higher appraisals of the negative impact of life events is a predisposing factor for, and predictor of, depressive and anxiety disorders (Espejo, Hammen, & Brennan, 2012). These two studies demonstrate the importance of appraisal, whether viewed as a predominantly cognitive or emotional process, as a key piece of the stress-depression pathway. Similar to the concept of cognitive appraisal and it's role in the stress-depression relationship, cognitive bias has been explored as a predictor of depressive symptoms. In a review conducted by Joormann and Vanderlind (2010), data was presented that suggests that individuals with depression may be more likely to interpret emotionally ambiguous stimuli with mood-congruent biases. While the data on this topic is conflicted, should this conclusion be accurate it can only be assumed that if an emotionally ambiguous stimuli is interpreted with negative emotion, how much more so would a stressor be interpreted as such? Siemer and Reisenzein (2007) postulate that

interpretation biases, particularly those that are automatic, may hinder emotional control techniques such as reappraisal, thereby leading to "inflexible and inappropriate responding" (Joormann & Vanderlind, 2010, pg. 410). Based on this principle, when measuring perceived stress as compared to more objectively measured stress, should individuals have a bias to perceive stressors as stressful (perhaps even more stressful than others would perceive it), their emotional regulation processes may be muted or inhibited. This may then lead to the development of depressive symptoms that are congruent with their stress, and their attempted regulation (whether up or down regulation) will be meaningless, particularly in interacting with the stressor as a positive or negative predictor for the depressive symptoms.

In the current study, the perceived stress model used likely presented a redundancy that impeded the expected results from presenting; perceived stress, as a predictor variable, already includes partial or complete elements of emotional regulation, the moderator variable; as such, adding the emotion regulation by perceived stress interaction term does not add anything new to the theoretical model.

4.2 Limitations

Although any anticipated factors and variables that may potentially bias participant responses or compound the hypothesized model were controlled for as best as possible, several limitations to this study should be noted. Firstly, this study was a crosssectional study investigating the relationship between stress, emotion regulation, and depressive symptoms. Due to the fact this it was not longitudinal, the linearity or

causality of this relationship cannot be determined. Additionally, there was no screening for any psychological or psychiatric disorders that may introduce bias, such as mood, anxiety and personality disorders. It is anticipated, however, that all necessary and relevant information was captured by the aforementioned measures, for the general picture to be well understood and qualified. Additionally, due to the fact that the hypotheses and aims are not specific to a particular population (such as a clinically depressed population, or a specific sub-threshold population), the results reflect a general, non-clinical population, which was the intention of the study.

The method of measurement of emotion regulation may pose another limitation. Some believe that self-report measurements are not as reflective of emotion regulation as should be due to the fact that such measures require insight and meta-cognition that participants may not possess (Aldao, Nolen-Hoeksema, Schweizer, 2010), especially in an online platform. However, at this point in time, self-report measures are the most commonly used and feasible assessments, and in order to produce literature that is comparable with other studies being conducting, this type of assessment seems most appropriate. Additionally, and perhaps more importantly, it can be argued that there is no better way to capture an individual's emotion regulatory processes than by asking him or her to assess by self-report how they process and deal with their emotions.

Additionally, some may view the measure of only "hassle-like" stress as a limitation. Although there are other measures that incorporate larger and/or more chronic stressors, those are likely not going to be stressors that the majority of the population is

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likely to experience. Furthermore, due to the fact the emotion regulatory processes are continuously occurring, being able to assess them next to continuously occurring (or at least fairly common) stressors would likely yield the most meaningful results.

Furthermore, there may be a limitation in the sample distribution, as the majority of the psychology undergraduates at the research institutions, and therefore participants of this study, are female. This may cause a problem, as there has been research to show gender differences in several of the current study research constructs. However, in order to assess the acceptability of this limitation, gender analyses were conducted on all study variables and did not yield any significant or trending results. As such, it is not anticipated that gender confounds of the presented data.

Lastly, the online nature of this study may pose as a limitation. Due to the fact that individuals could participate in any environmental context they chose, there was a lack of controlled supervision of participation. Participants may have potentially participated in this study at times when their attention was not fully on the study itself, and without the presence of a researcher in their midst, individuals may have been potentially more careless or distracted while completing the survey. While this may have been the case, investigation of the data suggests that individuals may have been careful and honest in their responses; specifically, the moderately high depressive symptoms scores suggest that individuals may have been more thoughtful and realistic in their responses.

4.3 Implications and Future Directions

This study yielded results that beg for future elaborated replication studies. As this study was completed in a college sample, an identical study in other samples, both general community samples and specific samples with presenting problems different than those of college individuals (such as business executives, or terminally ill patients), could be very insightful. Results from such studies would suggest that there is a certain moderator effect that is either pervasive throughout society, or population specific. Results from other studies conducted in differing population that would differ may suggest that in college students, emotional regulatory processes play a specific role in the stress-depression relationship that need to be better understood. Additionally, although there were no significant differences in outcome variables between ethnic groups in this study, further studies in cultures where stress, emotion, and pathology are viewed differently should also be conducted. Specifically, studies in Near Eastern cultures when stress is minimalized and emotion is intellectualized could provide important commentary onto effects of interpretation styles of such factors.

As this study used a "daily hassles" questionnaire to assess actual experienced stress, a similar study using a measurement of chronic and more severe stress would be important to conduct. As it has been suggested that there may be differences in the prediction of depressive symptoms based on the content, chronicity, and severity of stress, a replication study measuring more chronic and severe stress would enhance researchers' and clinicians' understanding of how emotional regulation style affect the outcome of differing types of stress.

Stern

The results of this study suggest that the identification of effective emotion regulation as a protective factor or dysregulation as a risk factor in the development of depressive symptoms as a reaction to stress may be a useful process in a clinical treatment environment. Based on this knowledge, clinicians would be able to use assessment techniques to measure emotion regulation in individuals early on in therapy, and can subsequently use appropriate emotion focused intervention techniques with individuals who are at risk for or present with depression based on the aforementioned assessment measures. In this light, future studies investigating the application of this model to a treatment study would be an enhancement to the field. More specifically, a study examining the presented moderator effect as a potential predictor of treatment outcome may shed light on how the interaction between stress and emotion regulation can be utilized by clinicians. Additionally, first studying the model hypothesized in this study in a clinical population, such as individuals with an already presenting clinical depression or anxiety disorder, may provide better insight into this relationship and may lessen the potential concerns from the limitations presented earlier. Furthermore, should results similar to those found in this study be found among clinical samples, study of intervention techniques could have potentially meaningful implications for treatment of such individuals.

An additional implication and future direction for this study is the potential relationship between emotion (dys)regulation, social problem solving, stress, and depression. As has been discussed earlier, social problem solving, which is the cognitivebehavioral process by which individuals identify life problems and explore solutions, is a moderator in the stress-depression relationship. As such, individuals who have more effective social problem solving styles are less likely to develop depressive symptoms from stressful life events (Nezu & Ronan, 1985; Nezu et al., 1986). Problem-Solving Therapy, a therapeutic approach that aids individuals in learning how to better cope with the problems in their lives has been commonly used with individuals with depression. Contemporary Problem-Solving Therapy adds a component of emotional control, such that individuals learn not only cognitive and behavioral skills for problem solutions, but also how to deal with emotions that are interfering with everyday life and effective problem solving. It is hypothesized that the synthesis of social problem solving and emotional regulation may together further aid in the prediction of depressive symptoms from stress. Furthermore, problem-solving therapy with an emphasis on emotional regulation may increase positive treatment outcomes in individuals who are depressed or at risk for depression. Studies examining the relationship between social problem solving and emotion regulation should therefore be conducted in the future.

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Table 1

Demographic characteristics of the sample

Variable	Mean	SD
AGE	20.2	0.2
Variable	Frequency	Percent
GENDER		
Males	46	28.7
Females	114	71.3
RACE		
Asian	49	30.6
Black	8	5.0
Hispanic/Latino	2	1.3
White/Caucasian	82	51.2
Other	11	6.9
Multiple Race	8	5.0
CLASS		
Freshman	73	45.6
Sophomore	25	15.6
Junior	34	21.3
Senior	28	17.5
NATIVE ENGLISH SPEAKER		- / ••
No	27	169
Yes	133	83 1

Table 2

Descriptive statistics of continuous variables

Variable	Min	Max	Mean	SD
PSS	3	40	19.53	7.19
CESD	0	30	9.89	5.97
DERS	73	176	103.55	18.92
DERS_nonaccept	6	29	13.73	5.56
DERS_goals	9	25	16.49	4.12
DERS_impulse	8	30	14.07	4.43
DERS_aware	20	30	24.47	2.33
DERS_strategies	10	40	20.42	6.69
DERS_clarity	10	24	14.38	2.505
SRLE	62	183	103.56	25.86

Table 3

Correlation matrix of key variables

	1	2	3	4
1. PSS	-	.72**	.66**	.62**
2. CESD	.72**	-	.59**	.67**
3. DERS	.66**	.59**	-	.63**
4. SRLE	.62**	.67**	.63**	-

**. Correlation is significant at the 0.01 level (2-tailed).

Table 4

Hierarchical Regression Analysis for PSSxDERS

						Change Stati	stics	
Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	R Square Change	F Change	df1	df2
1 ^a	.72 *	.51	.51	4.18	.51	166.28	1	158
2 ^b	.73 *	.54	.53	4.08	.03	8.69	1	157
3°	.73	.54	.53	4.10	.00	.02	1	156

^a Predictor: PSS

^b Predictors: PSS and DERS

° Predictors: PSS, DERS, and PSSxDERS interaction term

* significant at the < .01 level

Table 5

Hierarchical Regression Analysis for SRLExDERS

						Change Stati	stics	
Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	R Square Change	F Change	df1	df2
1 ^a	.67 *	.45	.45	4.43	.45	131.28	1	158
2 ^b	.71 *	.50	.49	4.25	.05	14.15	1	157
3°	.72 *	.52	.51	4.17	.02	7.27	1	156

^a Predictor: SRLE

^b Predictors: SRLE and DERS

° Predictors: SRLE, DERS, and SRLExDERS interaction term

* significant at the < .01 level

Figure 1

Plot of Interaction at high and low levels of ER



APPENDIX

Subject ID:		Date of Part	icipation:	
	How	do you experience y	our emotions?	
Instructions:				
Please indicate l number from the	how often the follo e scale below (1 –	owing 36 statements app 5) in the box alongside of	oly to you by write each item.	ing the appropriat
1 Almost never (0-10%)	2 Sometimes (11-35%)	3 About half the time (36-65%)	4 Most of the time (66-90%)	5 Almost always (91-100%)
1. I am clear at	bout my feelings (l	R)		
)		
2. I pay attention	on to how I feel (R	<pre>}</pre>		
 I pay attention I experience 	on to how I feel (R e my emotions as o	R) overwhelming and out of	control	
 I pay attention I experience I have no identified 	on to how I feel (R e my emotions as o ea how I am feelin	R) overwhelming and out of g	control	
 I pay attention I experience I have no ide I have diffic 	on to how I feel (R e my emotions as o ea how I am feelin sulty making sense	R) overwhelming and out of g out of my feelings	control	
 I pay attention I experience I have no identified I have diffied I am attentive 	on to how I feel (R e my emotions as o ea how I am feelin sulty making sense ve to my feelings (R) overwhelming and out of g out of my feelings R)	control	
 I pay attention I experience I have no identified I have diffied I am attentive I know exact 	on to how I feel (R e my emotions as o ea how I am feelin culty making sense ve to my feelings (ctly how I am feelin	R) overwhelming and out of g out of my feelings R) ng (R)	control	
 I pay attention I experience I have no idensity I have diffice I am attentive I know exact I care about 	on to how I feel (R e my emotions as o ea how I am feelin sulty making sense ve to my feelings (etly how I am feeling what I am feeling	R) overwhelming and out of g out of my feelings R) ng (R) (R)	control	
 I pay attention I experience I experience I have no ide I have diffic I am attentive I know exact I care about I am confuse 	on to how I feel (R my emotions as o ea how I am feelin sulty making sense ve to my feelings (utly how I am feelin what I am feeling ed about how I fee	R) overwhelming and out of g out of my feelings R) ng (R) (R)	control	
 I pay attention I experience I have no ide I have diffice I have diffice I am attentive I know exact I care about I am confuse When I'm up 	on to how I feel (R my emotions as o ea how I am feelin sulty making sense ve to my feelings (thy how I am feeling what I am feeling ed about how I fee pset, I acknowledg	R) overwhelming and out of g out of my feelings R) ng (R) (R) (R) d ge my emotions (R)	control	
 I pay attention I experience I experience I have no ide I have diffic I have diffic I am attentive I know exact I care about I am confuse When I'm up When I'm up 	on to how I feel (R my emotions as o ea how I am feelin sulty making sense ve to my feelings (utly how I am feeling what I am feeling ed about how I fee pset, I acknowledg pset, I become ang	 R) overwhelming and out of g out of my feelings R) ng (R) (R) d ge my emotions (R) gry with myself for feeling 	control g that way	
 I pay attention I experience I have no ided I have no ided I have difficed I am attentive I know exact I care about I am confused When I'm up When I'm up When I'm up 	on to how I feel (R my emotions as o ea how I am feelin sulty making sense ve to my feelings (ty how I am feeling what I am feeling ed about how I fee pset, I acknowledg pset, I become ang pset, I become em	R) overwhelming and out of g out of my feelings R) ng (R) (R) d ge my emotions (R) gry with myself for feeling barrassed for feeling that	control ng that way	
 I pay attention I experience I have no ide I have no ide I have diffice I am attentive I know exace I care about I am confuse I am confuse When I'm up When I'm up When I'm up When I'm up 	on to how I feel (R my emotions as o ea how I am feelin sulty making sense ve to my feelings (otly how I am feeling what I am feeling ed about how I fee pset, I acknowledg pset, I become ang pset, I become em	 R) overwhelming and out of g out of my feelings R) ng (R) (R) d ge my emotions (R) gry with myself for feeling barrassed for feeling that alty getting work done 	control ng that way t way	

Subject ID:		Date of Participation:					
1 Almost never (0-10%)	2 Sometimes (11-35%)	3 About half the time (36-65%)	4 Most of the time (66-90%)	5 Almost alway: (91-100%)			
15. When I'm up	oset, I believe that	I will remain that way fo	r a long time				
16. When I'm up	oset, I believe that	I'll end up feeling very d	epressed				
17. When I'm up	oset, I believe that	my feelings are valid and	l important (R)				
18. When I'm up	oset, I have difficu	lty focusing on other thin	gs				
19. When I'm up	oset, I feel out of c	ontrol					
20. When I'm up	oset, I can still get	things done (R)					
21. When I'm up	oset, I feel ashame	d with myself for feeling	that way				
22. When I'm up	oset, I know that I	can find a way to eventua	ally feel better (R)				
23. When I'm up	oset, I feel like I ar	n weak					
24. When I'm up	oset, I feel like I ca	n remain in control of m	y behaviours (R)				
25. When I'm up	oset, I feel guilty fo	or feeling that way					
26. When I'm up	oset, I have difficu	lty concentrating					
27. When I'm up	oset, I have difficu	lty controlling my behavi	iours				
28. When I'm up	oset, I believe that	there is nothing I can do	to make myself feel b	etter			
29. When I'm up	oset, I become irrit	ated with myself for feel	ing that way				
30. When I'm up	oset, I start to feel	very bad about myself					
31. When I'm up	oset, I believe that	wallowing in it is all I ca	n do				
32. When I'm up	oset, I lose control	over my behaviours					

Stern

Subject ID:		_ Date of Partie	cipation:	
1	2	3	4	5
Almost never	Sometimes	About half the time	Most of the time	Almost alway

34. When I'm upset, I take time to figure out what I'm really feeling (R)

35. When I'm upset, it takes me a long time to feel better

36. When I'm upset, my emotions feel overwhelming

Subject ID: _____ Date of Participation: How have you felt in the past week? Instructions: For each statement, please circle the number in the column that best describes how you have been feeling *in the past week*. Rarely or none Some or a little Occasionally Most or all of of the time of the time or a moderate the time amount of the (less than 1 (1-2 days) (5-7 days) time day) (3-4 days) 1 2 3 I felt depressed. 0 1. I felt that everything I did was an 2. 0 1 2 3 effort. My sleep was restless. 0 2 3 3. 1 I was happy. * 3 2 4. 1 0 I felt lonely. 5. 0 3 1 2 2 3 6. People were unfriendly. 0 1 7. I enjoyed life. * 3 2 1 0 8. I felt sad. 0 1 2 3 9. I felt that people dislike me. 0 1 2 3 10. I could not get "going". 0 2 3 1

ach	n case, you will b	e asked to inc	dicate t	by circ	ling how o	often you	u felt or t	hou	ght a ce	ertain	way.	I
lame								Date				
lge	Geno	der (<i>Circle</i>):	ΜF		Other			<u></u>				
	0 = Never	1 = Almost N	lever	2 = S	ometimes	3 = F	airly Ofte	en	4 = Ve	ry Ofte	en	
1. 	In the last month, because of somet	how often have hing that happe	e you be ened un	en up expec	set tedly?			0	1	2	3	4
2. 	In the last month, to control the impo	how often have ortant things in	e you fe your life	It that e?	you were u	nable		0	1	2	3	2
3.	In the last month,	how often have	e you fe	lt nerv	ous and "st	ressed"	?	0	1	2	3	4
4. 1	In the last month, to handle your per	how often have sonal problem	e you fe s?	lt conf	ident about	your ab	ility	0	1	2	3	2
5. I	In the last month, were going your w	how often have /ay?	e you fe	It that	things			0	1	2	3	4
6. I	In the last month, with all the things	how often have that you had to	e you fo do?	und th	at you coul	d not co	pe	0	1	2	3	4
7. 1	In the last month, to control irritation	how often have s in your life?	e you be	en ab	le			0	1	2	3	4
8.	In the last month,	how often have	e you fe	lt that	you were o	n top of	things?	0	1	2	3	4
9. I I	In the last month, because of things	how often have that were outs	e you be ide of y	een an our co	gered ntrol?			0	1	2	3	4
0.	In the last month,	how often have	e you fe	It diffic	culties)		0	1	2	3	4

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The PSS Scale is reprinted with permission of the American Sociological Association, from Cohen, S., Kamarck, T., and Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24,* 386-396.
Cohen, S. and Williamson, G. Perceived Stress in a Probability Sample of the United States. Spacapan, S. and Oskamp, S. (Eds.) *The Social Psychology of Health.* Newbury Park, CA: Sage, 1988.

A4. Survey of Recent Lift Experiences

	SURVEY OF RECENT LIFE EXPERIENCES	
	(SRLE)	
indi Put ove was part past	Following is a list of experiences which many people have some time or other. cate for each experience how much it has been a part of your life over the past a "1" in the space provided next to an experience if it was not at all part of your r the past month (e.g., "trouble with mother in law -1 "); "2" for an experience only slightly part of your life over that time; "3" for an experience which was d t of your life; and "4" for an experience which was very much part of your life of t month.	Please month. our life e which <i>istinctly</i> ver the
Inte	ensity of Experience over Past Month	
1 2 3 4	= not at all part of my life = only slightly part of my life = distinctly part of my life = very much part of my life	
1.	Disliking your daily activities	
*2.	Lack of privacy	
3.	Disliking your work Ethnic or racial conflict	
4. 5.	Conflicts with in-laws or boyfriend's/girlfriend's family	
6.	Being let down or disappointed by friends	
7.	Conflict with supervisor(s) at work	
8. 9	Social rejection Too many things to do at once	
10.	Being taken for granted	
11.	Financial conflicts with family members	
12.	Having your trust betrayed by a triend	
13.	Having your contributions overlooked	
15.	Struggling to meet your own standards of performance and accomplishment	
16.	Being taken advantage of	
17. *18	Not enough lessure time Financial conflicts with friends or fellow workers	
*19.	Struggling to meet other people's standards of performance and accomplishmen	t
*20.	Having your actions misunderstood by others	
21.	Cash-flow difficulties	
22.	A lot of responsibilities Dissetisfaction with work	
23. 24.	Decisions about intimate relationship(s)	
25.	Not enough time to meet your obligations	
*26.	Dissatisfaction with your mathematical ability	
27.	Financial burdens	
28. 29	Experiencing high levels of noise	
*30.	Adjustments to living with unrelated person(s) (e.g., roommate)	
31.	Lower evaluation of your work than you hoped for	
32.	Conflicts with family member(s) Finding your work too demanding	<u> </u>
	Finding your work too demanding	
36.	Trying to secure loan(s)	
37.	Getting "ripped off" or cheated in the purchase of goods	
*38.	Dissatisfaction with your ability at written expression	
39. 40.	Social isolation	
41.	Being ignored	
42.	Dissatisfaction with your physical appearance	
43	Unsatisfactory housing conditions	
44. 45	Failing to get money you expected	
46.	Gossip about someone you care about	
47.	Dissatisfaction with your physical fitness	
48.	Cossip about yourself Difficulty dealing with modern technology (e.g. computers)	
49. *50.	Car problems	
51.	Hard work to look after and maintain home	<u></u>
Not	Asterisks identify items to be omitted from the 11 item short form	
INOIE	a received menting nems to be omnied from the 41-field Shoft form,	