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## **Mechanisms Driving Suicidal Ideation to Action: The Impact of Rumination and Cardiovascular Reactivity on Momentary Fluctuations in Pain Tolerance and Persistence**

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Mechanisms Driving Suicidal Ideation to Action: The Impact of Rumination and  
Cardiovascular Reactivity on Momentary Fluctuations in Pain Tolerance and Persistence.

by

Keyne Catherine Law

A Dissertation  
Submitted to the Graduate School,  
the College of Education and Psychology  
and the Department of Psychology  
at The University of Southern Mississippi  
in Partial Fulfillment of the Requirements  
for the Degree of Doctor of Philosophy

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## ABSTRACT

To prevent suicide, it is crucial to understand the mechanisms and processes associated with deaths by suicide. The capability for suicide is a critical factor that enables an individual to endure the physical pain necessary to make a lethal suicide attempt (Joiner, 2005; Klonsky & May, 2015). Few studies have examined whether the ability to tolerate and persist through pain are subject to momentary fluctuations during different emotional contexts. This study sought to directly compare the effects of sadness rumination and anger rumination on pain tolerance and pain persistence. Furthermore, this study aimed to examine the effect of heart rate on the aforementioned relationships. Specifically, it was hypothesized that rumination, particularly anger rumination, will elevate pain tolerance and pain persistence indirectly through increased heart rate. A sample of 82 undergraduate students were randomly assigned into one of four conditions: control, anger, sadness, or anger with sadness and underwent an idiographic emotion (Pitman et al., 1987) and rumination induction (Nolen-Hoeksema & Morrow, 1993). They completed subjective and behavioral measures assessing emotion, impulsivity, and pain tolerance. Heart rate was measured at baseline, during cold pressor tests, following the cold pressor tests, and during both the emotion and rumination induction tasks. The results of this study suggest that only pain threshold may be subject to momentary fluctuations. The emotions on which participants were asked to ruminate also did not influence changes in their pain responses or heart rate throughout the experiment.

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## CHAPTER I - INTRODUCTION

### Suicide

Suicide is a worldwide public health issue that claims the lives of approximately 800,000 individuals annually (WHO, 2014) and demands our attention. Despite the last 50 years of suicide research that has aimed to answer the questions of who, why, and what causes people to die by suicide, our attempts to predict and prevent suicide have been unfruitful (Franklin et al., 2017). Over the past 13 years, suicide rates in the United States (US) have not decreased. In 2008, suicide became the 10th leading cause of death in the US (Centers for Disease Control and Prevention [CDC], 2012a) and it has maintained this position with increasing rates of death each year (CDC, 2012b; 2013; 2015a; 2015b; 2016). While there has been a recent shift in researchers' interest in the use of large scale pattern recognition and predictive analytics to predict suicide (Walsh, Ribeiro, & Franklin, 2017), understanding the mechanisms and processes associated with suicide deaths is crucial in the prevention of suicide. According to two prominent theories of suicide, the Interpersonal Theory of Suicide (ITS; Joiner, 2005) and Three-Step Theory of Suicide (3ST; Klonsky & May, 2015), a critical factor that enables an individual to make a lethal suicide attempt is the capability to endure the physical pain necessary to make a suicide attempt.

### Pain and Suicide

Suicide researchers have consistently demonstrated that an elevated risk for suicide is associated with pain tolerance, the maximum level of pain an individual is able to tolerate (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006; Franklin, Hessel, & Prinstein, 2011, Pennings & Anestis, 2013). Specifically, the ability to tolerate more

pain has been found to differentiate individuals who have made a suicide attempt from their counterparts who only thought about suicide (Smith, Edwards, Robinson, & Dworkin, 2010). More recent research has also suggested that pain persistence – the difference between the point at which pain is first detected and the point at which an individual can no longer tolerate pain - may also be essential in determining the capability for suicide (Law, Khazem, Jin, & Anestis, 2018). Further clarity is needed, however, to fully understand the relationship between the aforementioned pain variables and the capability for suicide.

The majority of existing research on pain tolerance has conceptualized this variable as relatively stable and increasing in a linear manner in response to painful and provocative experiences (Franklin et al., 2011), yet the trajectory of suicide risk seems to be non-linear and fluctuating depending on changes in risk factors. The Fluid Vulnerability Theory of suicide (Rudd, 2006) posits that suicide risk fluctuates based on the interaction between baseline and acute risk factors. Baseline risk factors involve predisposing vulnerabilities that elevate suicide risk while acute risk factors involve short-term fluctuations in context, both external and internal to an individual, that lead to temporal increases in suicide risk. Pain tolerance and pain persistence have been consistently researched as baseline risk factors for suicide, yet minimal research has examined the role of pain tolerance and persistence as acute risk factors for suicide. Indeed, recent studies have found pain tolerance and pain persistence are susceptible to momentary changes (Ludascher et al., 2009, Law & Anestis, in preparation), a finding with potentially substantial implications for the manner in which aspects of suicide risk emerge across time.

## Emotions and Emotion Regulation

In the context of suicide, emotions may be a particularly relevant variable contributing to momentary fluctuations in the ability to tolerate and persist through pain in order to make a suicide attempt. Notably, Chapman and Dixon-Gordon (2007) found that a greater percentage of psychiatric inpatients who attempted suicide in their study (40.9%) reported feeling angry immediately before making a suicide attempt. Emotions have often been posited to have two qualities: valence and arousal. Valence is defined as the perception of an emotion as being pleasant or unpleasant while arousal is defined as the state of being physiologically activated or deactivated (Barrett, 1998). Although emotions of negative valence are often attributed to the psychological pain and hopelessness associated with the development of suicidal ideation (Klonsky & May, 2015), the arousal quality of an emotion may contribute to the ability to make a suicide attempt. Indeed, past studies have found heightened states of arousal to contribute to increases in suicide risk, particularly among individuals with high capability for suicide (Ribeiro, Silva, & Joiner, 2014; Ribeiro, Yen, Joiner, & Siegler, 2015).

The relationship between arousal and pain sensitivity may be impacted by changes in psychophysiological responses. Past studies have supported the theory that a common mechanism exists between pain sensitivity and cardiovascular responses (Vassend & Knardahl, 2004). Particularly, changes in blood pressure and heart rate have been consistently demonstrated to be associated with pain threshold and pain tolerance (Campbell, Holder, & France, 2006; Duschek, Heiss, Buchner, & Schandry, 2009). As such, the physiological differences that occur with low (e.g. sadness) and high (e.g. anger) arousal states (Marci, Glick, Loh, Dougherty, 2007) may have varying effects on

the ability to tolerate and persist through physical pain. Particularly, acute experiences of emotions that are of negative valence and high arousal (e.g. anger) have been found to have analgesic effects (Burns et al., 2009; Rhudy & Meagher, 2001).

Although all individuals experience a range of emotions, the experience of negative emotions may not necessarily increase the risk of suicide. Furthermore, the acute analgesic effect of emotion may not necessarily be sustained long enough for an individual to engage in suicidal behavior. Thus, the regulation of negative emotional experiences, related but distinct from the emotional experience itself, may be a crucial factor in increasing suicidality. Indeed, past studies have found emotion regulation to increase the desire and, when paired with elevations in painful and/or provocative experiences (e.g., nonsuicidal self-injury), it has also been shown to be associated with the capability for suicide (Law, Khazem, & Anestis, 2015). Rumination, the repetitive fixation on the experience, causes, and consequences of a negative emotion (Nolen-Hoeksema, 1991), is a maladaptive emotion regulation strategy that has been consistently found to exacerbate and sustain the processing of negative emotion (McLaughlin, Borkovec, & Sibrava, 2007; Selby & Joiner, 2013). Furthermore, rumination has been associated with increases in both suicidal ideation and suicide attempts (Morrison & O'Connor, 2008). As such, it is plausible that rumination may sustain the analgesic effect of emotion, thereby creating a momentary increase in the ability to tolerate and persist through pain.

Indeed, experimental and correlational studies alike have found rumination to be associated with increased blood pressure and heart rate (Ottaviani et al., 2016) and a delayed recovery following cardiovascular reactivity (Glynn, Christenfeld, & Gerin,

2002). Moreover, the delayed recovery for cardiovascular reactivity can extend past 24 hours following the onset of rumination (Ottaviani, Shapiro, & Fitzgerald, 2011). Given the association between cardiovascular reactivity, emotion, and decreased pain sensitivity (Appelhans, & Luecken, 2008), it is reasonable to anticipate that rumination may increase pain tolerance and persistence through cardiovascular reactivity. Particularly, rumination on high arousal emotions may be especially pernicious to the development of state capability for suicide by enabling momentary increases in cardiovascular reactivity that persist for an extended period of time.

### Present Study

Although past studies have investigated the role of rumination on pain tolerance (Stimmel, Crayton, Rice, & Raffeld, 2006) and cardiovascular reactivity (Ottaviani et al., 2011; Ottaviano et al., 2016), few studies have directly compared the effects of rumination on pain tolerance in the context of low and high arousal emotions. Furthermore, no known studies have examined the effects of rumination on pain persistence. As such, the present study sought to directly compare the differential effects of sadness rumination, the fixation on sad experiences and their implications (Nolen-Hoeksema, 1991), and anger rumination, the recurrent processing of anger experiences and their implications (Sukhodolsky, Golub, & Cromwell, 1999) on pain tolerance and pain persistence. Past studies have found both sadness and anger to be linked to elevated levels of pain tolerance (Carter et al., 2002; Stimmel et al., 2006) with anger producing greater levels of pain tolerance (van Middendorp, Lumley, Jacobs, Bijlsma, & Geenen, 2010). As such, it is anticipated that anger rumination will lead to heightened levels of pain tolerance and pain persistence compared to sadness rumination. Furthermore, this

study aimed to examine the role of cardiovascular reactivity on influencing the aforementioned relationships. In addition to research that has found negatively valenced, high arousal emotions to have analgesic effects (Burns et al., 2009; Rhudy & Meagher, 2001), past findings have also demonstrated greater cardiac responses following anger compared with sadness (Schwartz, Weinberger, & Singer, 1981; Deichert, Flack, & Craig, 2005) with greater cardiac reactivity yielding higher thresholds for pain (Appelhans & Luecken, 2008). Thus, it is hypothesized that rumination, particularly anger rumination, will elevate pain tolerance and pain persistence indirectly through increased cardiovascular activity. Results supportive of these hypotheses would suggest that the transition from suicidal ideation to behavior may be malleable and shifting in response to the ruminative processing of high arousal affective states such as anger. Furthermore, results from this study may provide important clinical implications by supporting the use of coping strategies that decrease, and not increase, arousal in patients who are at risk for self-injurious and/or suicidal behaviors.

## CHAPTER II – METHODS

### Participants

Participants for this study include 175 undergraduate students who were enrolled in psychology courses and recruited through SONA systems (See Table 1 for more demographic information). Upon registration for the study, a secure link was sent to the participants directing them to the online phase of the study where they completed a battery of questionnaires focused on demographic variables and trait measurements of psychiatric variables such as their history of suicidal ideation and suicide attempts. During the online phase of the study, participants were also randomized into one of four Conditions: Control (n=44), Anger Only (n=46), Sadness Only (n=55), and Anger and Sadness (n=39) and were asked to provide a narrative involving a personal experience where an interaction with another person made them feel the emotion(s) to which they were assigned.

Following the online phase of the study, participants (n=126) who provided appropriate and detailed narratives were then invited to participate in the laboratory phase of the study. To minimize potential of third variable influences on pain tolerance and persistence, participants who were invited to the laboratory phase of the study were asked to refrain from ingesting sugared foods and alcoholic beverages for at least one hour prior to their scheduled appointment (Mercer & Holder, 1997). Furthermore, they were asked to refrain from taking analgesics (e.g., aspirin, acetaminophen) and other pain suppressants for at least eight hours prior to participation (Bender, Anestis, Anestis, Gordon, & Joiner, 2012).

Of the participants who were invited, 82 participants completed the study ( $M_{\text{age}}=20.87$ ,  $SD=5.51$ ; 77.6% female; 67.1% White; See Table 2 for additional demographic information) and 44 participants were lost to follow up. A total of 47 participants were excluded from the laboratory phase of the study. Specifically, 21 participants were excluded because they failed to answer quality assurance questions correctly and 26 participants were excluded for providing narratives that were inappropriate or did not contain enough detail ( $< 150$  words) to be used for the emotion induction procedures. Given that the laboratory phase of the study involved a cold pressor test, 1 participant with Reynauld's Disease was excluded from the study. See Table 2 for additional demographic information for participants who did not complete the laboratory phase of the study.

Past literature examining the role of emotion and rumination on cardiovascular activity had yielded effect sizes in the large range (Deichert et al., 2005; Ottaviani et al., 2011; Ottaviani et al, 2016; Vassend & Knardahl, 2007). A sensitivity power analysis conducted using G\*Power 3 (Faul, Erdfelder, Lang, & Buchner, 2007), suggested that a sample size of 82 allowed us to detect moderate to large effect sizes ( $f^2=.17$ ) with adequate power (.95) while holding type one error at  $\alpha = .05$ .

Table 1 *Demographic Characteristics of the Full Sample*

	<b>Full Sample</b>	<b>Completed Study</b>	<b>Failed Quality Assurance</b>	<b>Poor Narrative</b>	<b>Lost to Follow up</b>
N	175.00	84.00	21.00	26.00	44.00
Age (Mean, SD)	21.50 (5.50)	20.87	21.48 (4.73)	21.60 (4.86)	22.50 (5.94)
% Female	80.60	78.60	63.30	69.00	81.80
Race					
% White	60.60	63.10	43.30	55.20	54.50
% African American	29.40	27.40	23.30	27.60	27.30
% Asian	1.80	1.20	0.00	0.00	4.50
% Hispanic/Latino	5.90	3.60	3.30	5.10	9.10
% Biracial	2.40	4.80	0.00	3.40	4.50
Condition					
# Control	44.00	25.00	11.00	3.00	5.00
# Anger	46.00	18.00	6.00	9.00	13.00
# Sadness	55.00	24.00	8.00	7.00	16.00
# Anger and Sadness	39.00	17.00	5.00	7.00	10.00

Table 2 *Demographic Characteristics of Participants Who Completed the Study*

	<b>Control</b>	<b>Anger Only</b>	<b>Sadness Only</b>	<b>Anger and Sadness</b>	<b>Full Sample</b>
N	25.00	18.00	24.00	17.00	84.00
Age (Mean, SD)	19.84 (2.41)	22 (9.99)	20.67 (3.84)	21.47 (4.22)	20.87
% Female	80.00	72.20	91.70	64.70	78.60
Race					
% White	56.00	72.20	58.30	70.60	63.10
% African American	32.00	27.80	29.20	17.60	27.40
% Asian	0.00	0.00	0.00	5.90	1.20
% Hispanic/Latino	4.00	0.00	8.30	0.00	3.60
% Biracial	8.00	0.00	4.20	5.90	4.80

## Experimental Manipulations

### *Emotion induction*

An adapted version of the Pitman Protocol (Pitman, Orr, Forgue, & de Jong, 1987) was used to induce the emotional contexts in which participants were to ruminate. In the online phase of the study, participants were asked to write for 10 minutes about a situation in which they felt sad or angry and to include specific details about the sequence of events, people involved, context, descriptions of thoughts, feelings, and physical reactions that were experienced. They were then asked to select the bodily sensations and emotions they experienced during the event from two separate lists. Finally, they listed the thoughts that they were experiencing during the situation they described. The information acquired from the participant were combined and written into scripts between 350 and 550 words in length and subsequently recorded into two-minute audio files using simple, direct language in the active voice and in the second person. The audio file was presented to the participant in the experimental session. Participants who did not provide enough detail (e.g. less than 250 words) in their narratives to elicit emotion as part of the emotion induction procedures were excluded from participation in the laboratory phase of the study.

### *Rumination induction*

To induce rumination, the original rumination induction developed by Nolen-Hoeksema & Morrow (1993) was adapted, in terms of verb tense, to guide participants to

think about their emotional state, within the context of the event that they had been presented in the emotion induction. All participants were guided through a series of 45 items (e.g., "think about why people treated you the way they did", "think about why you reacted the way you did") by an audio recording presented simultaneously with corresponding text in visual slides to simulate thoughts that often arise during rumination.

## Measures

### *Subjective emotional state*

The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) was used to assess the subjective emotional state of participants at baseline, after the emotion induction procedure, and after the rumination induction procedure. Participants provided ratings on 10 positive emotion items and 10 negative emotion items which represented how they were feeling "right now, at the present moment" using a 5-point scale where 1= not at all or very slightly and 5= very much. Individual items on the PANAS indicating anger and sadness were also used to determine whether or not the emotion induction procedures elicited the intended effect. The PANAS has shown good test-retest reliability in past studies using a sample of students (Watson et al., 1988) as well as good convergent validity (MacKinnon et al., 1999).

### *Baseline and state pain tolerance*

The Anova A-40 Refrigerated Circulator System was used to administer a cold pressor test (CPT) to examine participants' pain threshold, pain tolerance, and ability to persist through pain past the pain threshold (pain persistence). The cold pressor task is a frequently used pain induction procedure in studies examining self-injurious behaviors

(e.g., Bohus, Limberger, Ebner, Glocker, Schwarz, Wernz, et al., 2000; Russ, Roth, Lerman, Kakuma, Karrison, Shindlecker, Hull et al., 1992; Gratz, Hepworth, Tull, Paulson, Clarke, Remington, et al., 2011). Participants were asked to submerge their hand, up to their wrist, into a water bath maintained at 2°C with a circulator that prevents the water surrounding the participant's hand from warming. They were also asked to alternate hands (dominant/non-dominant) between the first trial (baseline) and the second trial (post-experimental manipulation); hand order was counterbalanced across both trials.

Pain tolerance was operationalized as the time elapsed until the participants pull their hand out of the water and indicate that they can no longer tolerate the pain. A two-minute time limit was used for the task to reduce outliers as past studies have found that participants seldom continue past two minutes and those that do often continue due to a numbed sensation in their hand (Franklin, Aaron, Arthur, Shorkey, & Prinstein, 2012). Pain persistence was operationalized as the time elapsed between the participant's pain threshold, the time elapsed until participants indicate that they first feel pain, and pain tolerance. Time elapsed was measured and recorded using two timers which both began when the participant's hand was submerged and stopped at pain threshold and pain tolerance, respectively. Participants were also asked to indicate their subjective level of pain on a scale of 1 (barely perceptible pain) to 10 (most intense pain imaginable) at the moment they reach pain threshold and pain tolerance. Due to the nature of this task, individuals with Reynaud's syndrome were excluded from participation in the laboratory session.

*Cardiovascular reactivity.*

Cardiovascular reactivity was indicated by changes in Heart Rate (HR) derived from electrocardiogram (ECG) acquired using the Biopac MP150 Data Acquisition System and the BN-RSPEC wireless transmitters and receivers. Data was recorded through Acqknowledge 4.4.2 using a sampling rate of 1,000 samples per second. Pre-jelled electrodes were placed in a lead (III) configuration below the participants' right and left clavicles and on the left iliac fossa (See Figure 1). Measurements were taken at ten time points including baseline, during both sets of experimental manipulations and both cold pressor tasks, and after a 20 minute follow-up recovery period. Physiological measurements that were not task-related (e.g. baseline, post-recovery) were measured using 300 second periods. In preparation for data analysis, all ECG waveforms were visually inspected for noise and heart beats were identified using QRS peak detection.

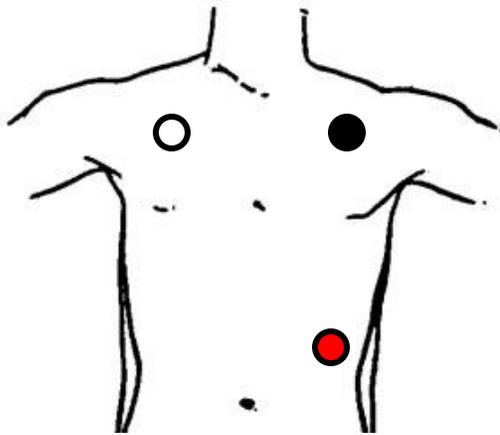


Figure 1. ECG Lead III Configuration.

### Procedures

The current study protocol's was approved by The University of Southern Mississippi's Institutional Review Board. Once participants reviewed the informed consent form and consented to participate in the study, they were directed to the first

phase of the study where they were randomly assigned to receive instructions to provide a narrative of an event that made them feel a) angry but not sad, b) sad but not angry, c) angry and sad, or d) neutral using the Pitman Protocol (Pitman et al., 1987) on an online form. Eligible participants were then invited to schedule an appointment at the Suicide and Emotion Dysregulation laboratory at the University of Southern Mississippi for the second phase of the study. Their narratives were then written into scripts to emphasize the emotional experience and recorded into an audio file prior to the participant's scheduled laboratory session to be used for the emotion induction procedure.

In the laboratory session, participants, again, reviewed the informed consent form and consented to participate in the study. Participants were connected to the BN-RSPEC wireless transmitters and receivers and the Biopac MP150 Data Acquisition System. The pre-jelled electrodes were then allowed to warm on the participants' skin while an initial risk assessment was administered to improve the integrity of the acquired physiological data. After the initial visual inspection of the participants' physiological data and necessary adjustments were made, baseline measurements of the participants' emotional state (PANAS; Watson et al., 1988) and resting heart rate were taken. The CPT was administered to measure baseline levels of pain tolerance, persistence. Heart rate was recorded during the CPT. Following the first CPT, Participants received an idiographic emotion induction, based on the narrative they provided in the online stage of the study using the Pitman Protocol (Pitman et al., 1987), in the form of an audio recording. They were then asked to rate their subjective emotional state using the PANAS (Watson et al., 1988) following the emotion induction procedure. Subsequently, participants were guided through the rumination induction procedure (Nolen-Hoeksema & Morrow, 1993)

followed, again, by the PANAS (Watson et al., 1988) to measure subjective emotional state after the rumination induction procedure. Heart rate was measured during both the emotion induction and rumination induction tasks. Subsequently, participants completed the CPT again to test for changes in pain tolerance, persistence following the experimental manipulations. Heart rate was, again, recorded during the CPT. Finally, after a recovery period of approximately 20 minutes, the participants' heart rate, followed by a final measurement of subjective emotional state (PANAS; Watson et al., 1988), were taken. A final risk assessment was administered and participants were debriefed before their participation in the study was complete. All self-report questionnaires and experimental manipulations in the laboratory session were delivered using laboratory computers. Behavioral (CPT) and physiological (HR) measurements (HR) were recorded by trained research assistants.

#### Data Analytical Approach

To select the appropriate demographic covariates, bivariate correlations were used to test if there was a significant effect of age on changes on pain responses and heart rate. One-way ANOVAs were then used to determine if there was a significant effect of gender and race on pain responses and heart rate. To determine if the emotion and rumination inductions had the intended effect on the participants, two repeated measure ANOVAs (RM-ANOVAs) and subsequent Bonferroni-corrected pairwise comparisons were used to test for main and interaction effects of Time and Condition on subjective emotional state (positive affect subscale, negative affect subscale, specific items relevant to sadness and/or anger) and heart rate. Based on previous studies using similar forms of

experimental manipulations (e.g., Rusting & Nolen-Hoeksema, 1998; Wisco & Nolen-Hoeksema, 2009; Ciesla & Roberts, 2007), it was expected that there will be a significant increase in negative affect and items relevant to the assigned Condition (anger and sadness) between baseline and post-emotion induction, another significant increase between post-emotion induction and post-rumination induction. Finally, it was expected that negative affect and items that are relevant to the Conditions will decrease and return to baseline between post-rumination induction and at the end of the laboratory session. The opposite effects are anticipated for positive affect. Given past findings on emotional states and cardiac reactivity (Schwartz et al., 1981; Deichert et al., 2005), it was expected that heart rate will demonstrate a significant increase between baseline and baseline CPT, become further elevated following the emotion induction and rumination induction tasks, and peak at the post-experimental manipulation CPT. It was then anticipated that heart rate will decrease following the 20-minute recovery period.

To test our hypothesis that anger rumination will lead to heightened levels of pain tolerance and pain persistence compared to sadness rumination, two repeated measure ANOVAs (RM-ANOVAs) with subsequent Bonferroni-corrected pairwise comparisons were used to test for differences between the four conditions on changes in pain tolerance and pain persistence from baseline to post-experimental manipulation. Consistent with existing research on the effects of high arousal emotions on inhibiting pain (Burns et al., 2009; Rhudy & Meagher, 2001) it was expected that there will be a significant Time x Condition interaction where pain tolerance and pain persistence will be greatest following the experimental manipulation in the Anger Only condition, followed by the Anger and Sadness, then Sadness Only conditions.

## CHAPTER III - RESULTS

### Selection of Covariates

No significant effect of Age was found on changes in pain threshold ( $r=.077$ ,  $p=.497$ ), tolerance ( $r=-.010$ ,  $p=.931$ ), and persistence ( $r=-.036$ ,  $p=.751$ ). There was no significant effect of Gender (all  $ps>.142$ ). There was, however a significant effect of Race on pain tolerance ( $F(4,80)=3.007$ ,  $p=.023$ ) and pain persistence ( $F(4,79)=4.714$ ,  $p=.002$ ) such that the four individuals who identified as Hispanic/Latino reported greater increases in pain tolerance ( $M=45.250$ ,  $SD=131.129$ ) and pain persistence ( $M=56.250$ ,  $SD=124.653$ ) than their counterparts. As such, Race was included as a covariate in the primary analyses examining pain tolerance and persistence.

### Manipulation Check

#### *Positive Affect*

There was a significant effect of Time but not Condition ( $F(3,77)=1.796$ ,  $p=.155$ ) on positive affect ( $F(3,231)=23.314$ ,  $p<.001$ ; See Figure 2). Specifically, for all four conditions, positive affect significantly decreased from Baseline ( $M=2.641$ ,  $SD=.987$ ) to Post-Emotion Induction ( $M=2.109$ ,  $SD=.902$ , all  $ps <.056$ ). There was also no significant interaction effect of Time and Condition on positive affect ( $F(9,231)=1.783$ ,  $p=.072$ ).

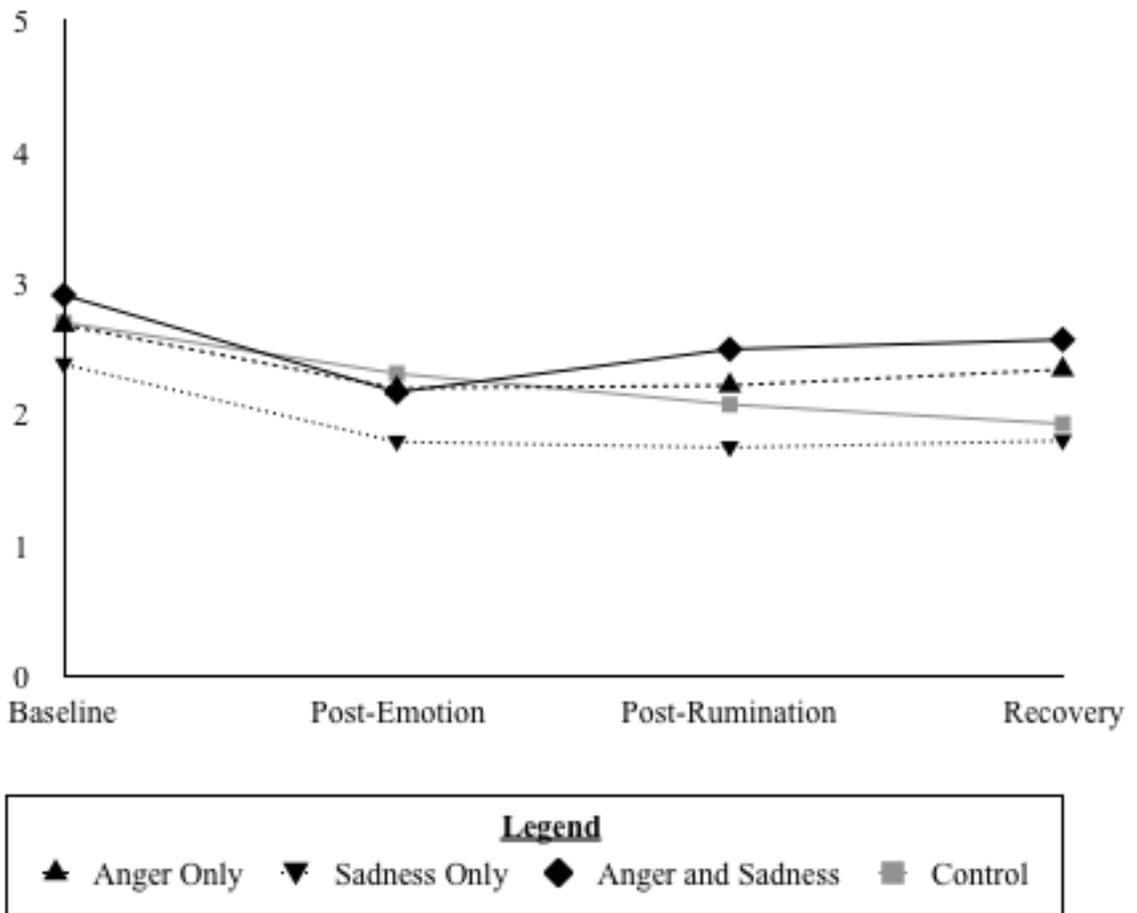


Figure 2. Changes in Positive Affect

### *Negative Affect*

There was a significant effect of Time ( $F(3,231)=35.452, p<.001$ ) but not Condition ( $F(1,77)=1.313, p=.276$ ) on negative affect (See Figure 3). There was also a significant interaction effect between Time and Condition on negative affect ( $F(9,231)=8.039, p<.001$ ). Specifically, in the Control condition, there was a significant increase in negative affect between Baseline ( $M=1.616, SD=.746$ ) and Recovery ( $M=1.288, SD=.504, p=.008$ ). In the Anger condition, there was not a significant increase in negative affect between Baseline ( $M=1.728, SD=.821$ ) and Post-Emotion Induction ( $M=2.061, SD=.758, p=.416$ ), but there was a significant decrease in negative affect between Post-Emotion Induction and Post-Rumination Induction ( $M=1.483, SD=.453, p<.001$ ). In the Sadness condition, there was a significant increase in negative affect between Baseline ( $M=1.313, SD=.368$ ) and Post-Emotion Induction ( $M=1.983, SD=.928, p<.001$ ), as well as a significant decrease in negative affect between Post-Emotion Induction and Post-Rumination Induction ( $M=1.583, SD=.624, p=.002$ ). There was also a significant decrease in negative emotion between Post-Rumination Induction and Recovery ( $M=1.365, SD=.431, p=.005$ ). In the Anger and Sadness condition there was a significant increase in negative affect between Baseline ( $M=1.307, SD=.291$ ) and Post-Emotion Induction ( $M=2.460, SD=.811, p<.001$ ) as well as a significant decrease in negative affect between Post-Emotion Induction and Post-Rumination Induction ( $M=1.660, SD=.606, p<.001$ ). Finally, there was a significant decrease in negative affect from Post-Rumination to Recovery ( $M=1.367, SD=.440, p=.002$ ).

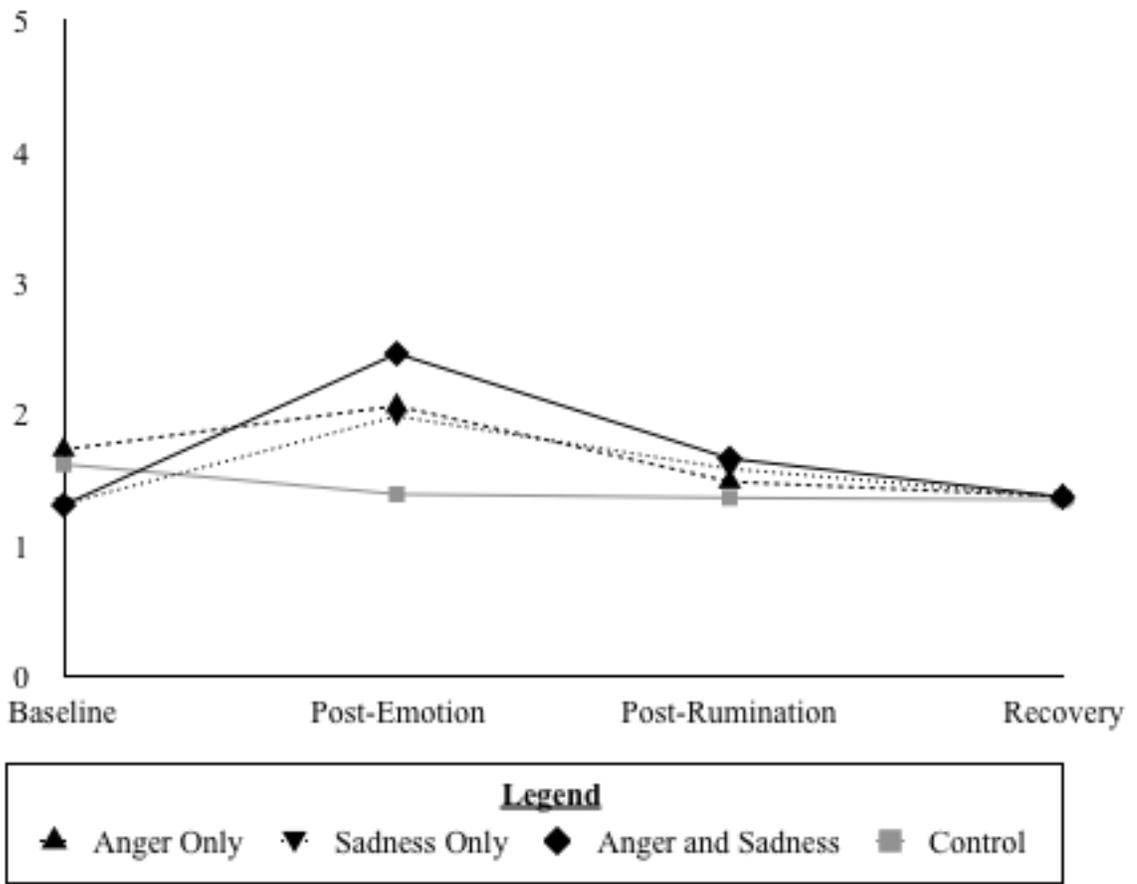


Figure 3. Changes in Negative Affect.

### *Sadness*

There was a significant main effect of Time ( $F(3,231)=24.974, p<.001, \eta^2=.245$ ) but not Condition ( $F(3,77)=1.978, p=.124$ ) on subjective ratings of sadness (See Figure 4). Additionally, there was a significant interaction effect of Time and Condition on feelings of sadness ( $F(9,231)=7.089, p<.001, \eta^2=.216$ ). As expected, individuals in the Sadness Only (Baseline:  $M=1.250, SD=.554$ ; Post-Emotion Induction:  $M=2.174, SD=1.223; p<.001$ ) and Anger and Sadness (Baseline:  $M=1.283, SD=.248$ ; Post-Emotion Induction:  $M=2.567, SD=.961; p<.001$ ) conditions reported significant increases in feelings of sadness after the emotion induction. Individuals in both aforementioned conditions, however, also reported a significant decrease in feelings of sadness after the rumination induction (Sadness Only: Post-Rumination Induction:  $M=1.707, SD=.852, p=.003$ ; Anger and Sadness: Post-Rumination Induction:  $M=1.783, SD=.801, p<.001$ ).

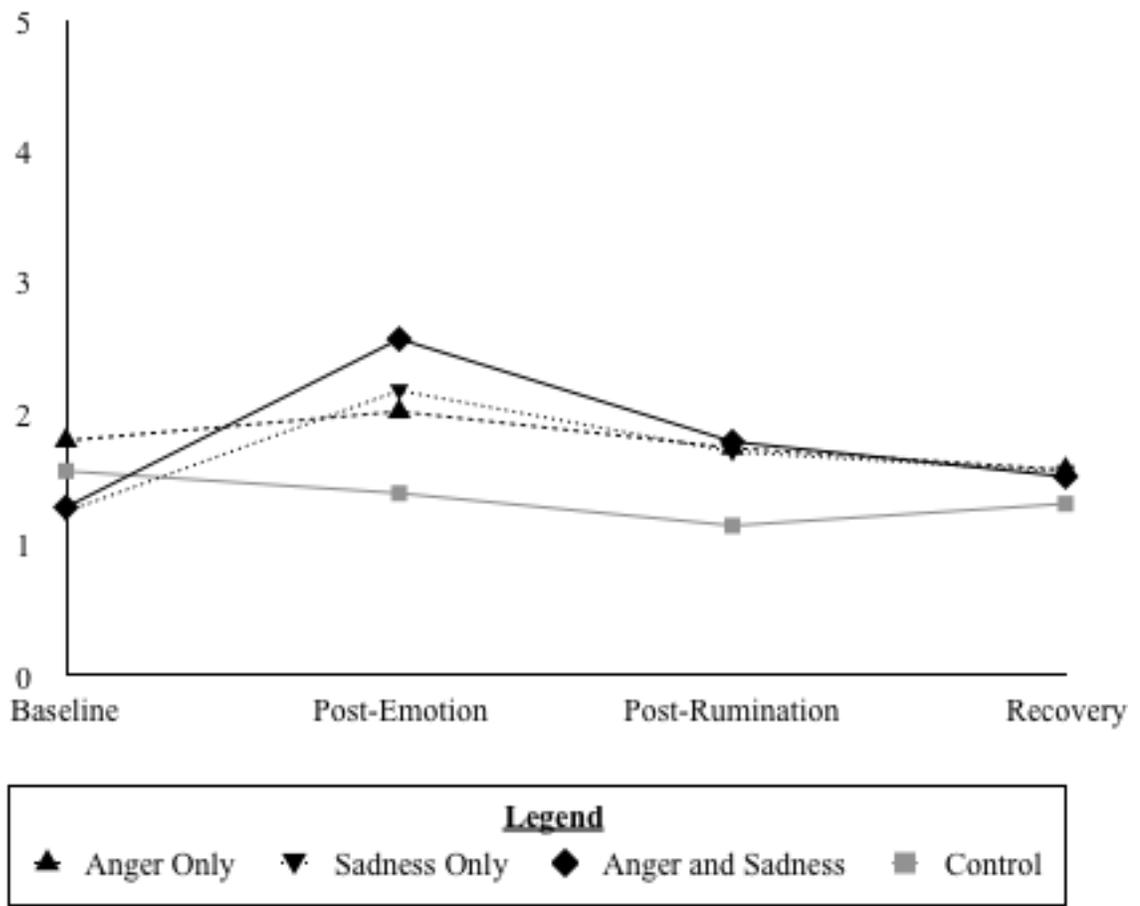


Figure 4. Changes in Sadness

### *Anger*

Similarly, there was a significant main effect of Time ( $F(3,231)=33.040$ ,  $p<.001$ ,  $\eta^2=.300$ ) but not Condition ( $F(3,77)=.983$ ,  $p=.405$ ) on subjective ratings of anger (See Figure 5). Additionally, there was a significant interaction effect of Time and Condition on feelings of anger ( $F(9,231)=7.983$ ,  $p<.001$ ,  $\eta^2=.237$ ). As expected, individuals in the Anger Only (Baseline:  $M=1.431$ ,  $SD=.865$ ; Post-Emotion Induction:  $M=2.333$ ,  $SD=1.298$ ;  $p<.001$ ) and Anger and Sadness (Baseline:  $M=1.200$ ,  $SD=.254$ ; Post-Emotion Induction:  $M=2.483$ ,  $SD=.858$ ;  $p<.001$ ) conditions reported significant increases in feelings of anger after the emotion induction. At the same time, however, individuals in both of the aforementioned conditions also reported a significant decrease in feelings of sadness after the rumination induction (Anger Only: Post-Rumination Induction:  $M=1.478$ ,  $SD=.661$ ,  $p<.001$ ; Anger and Sadness: Post-Rumination Induction:  $M=1.433$ ,  $SD=.458$ ,  $p<.001$ ).

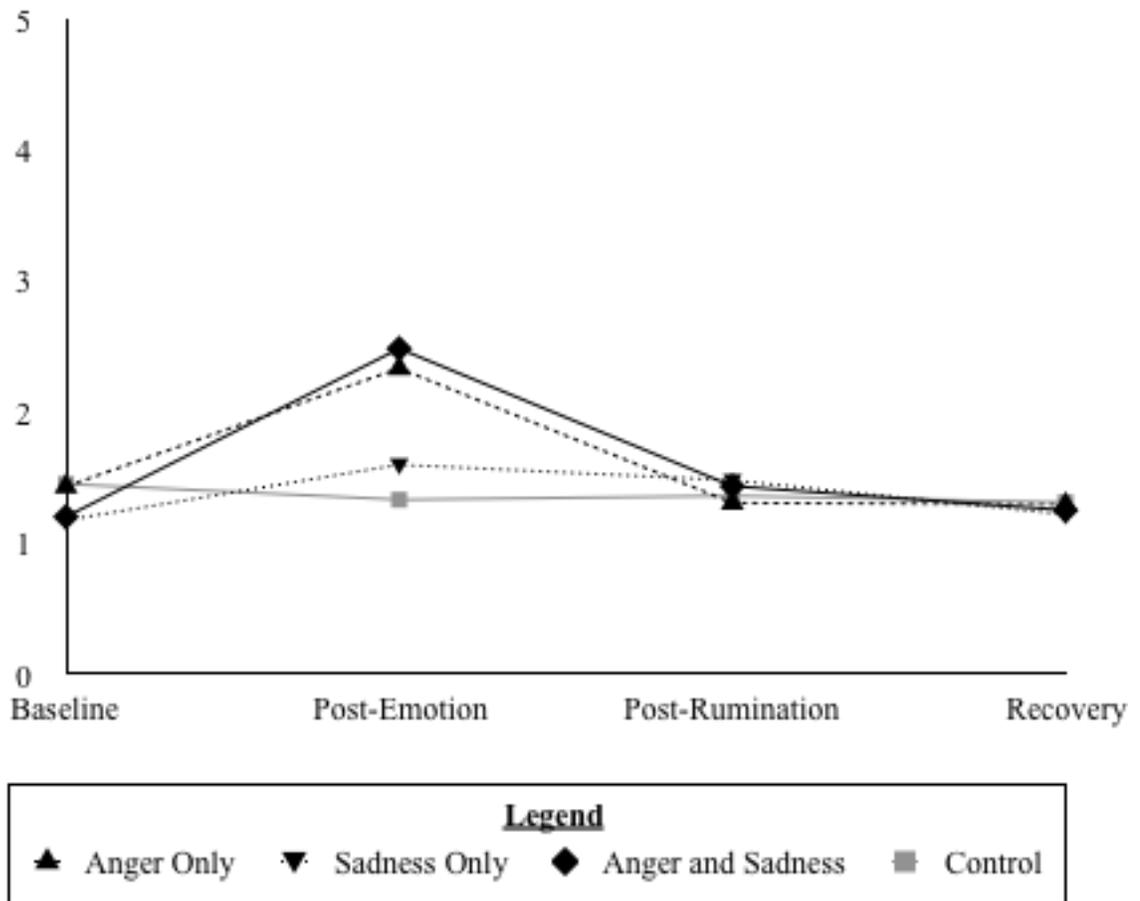


Figure 5. Changes in Anger.

### Changes in Pain Responses

In regards to changes in pain threshold, a significant effect of Time was found ( $F(1,77)=6.617$ ,  $p=.012$ ,  $\eta^2=.079$ ; See Figure 6). Specifically, participants' threshold for pain detection decreased between the baseline measurement of their pain threshold and the post-manipulation measurement of their pain threshold. There was no significant main effect of Condition ( $F(3,77)=1.227$ ,  $p=.306$ ) and no significant interaction effect between Time and Condition ( $F(3,77)=2.668$ ,  $p=.094$ ).

There were no significant main effects of Time ( $F(1,76)=1.156$ ,  $p=.286$ ) or Condition ( $F(3,76)=.014$ ,  $p=.998$ ) on changes in participants' ability to tolerate pain (See Figure 7). The interaction between Time and Condition also did not have a significant effect on pain tolerance ( $F(3,76)=1.527$ ,  $p=.214$ ). Similarly, there were no significant main effects of Time ( $F(1,75)=.808$ ,  $p=.372$ ) or Condition ( $F(3,75)=.078$ ,  $p=.972$ ) on changes in the participants' ability to persist through pain (See Figure 8). We also did not find a significant interaction effect of Time and Condition on pain persistence ( $F(3,75)=.857$ ,  $p=.468$ ).

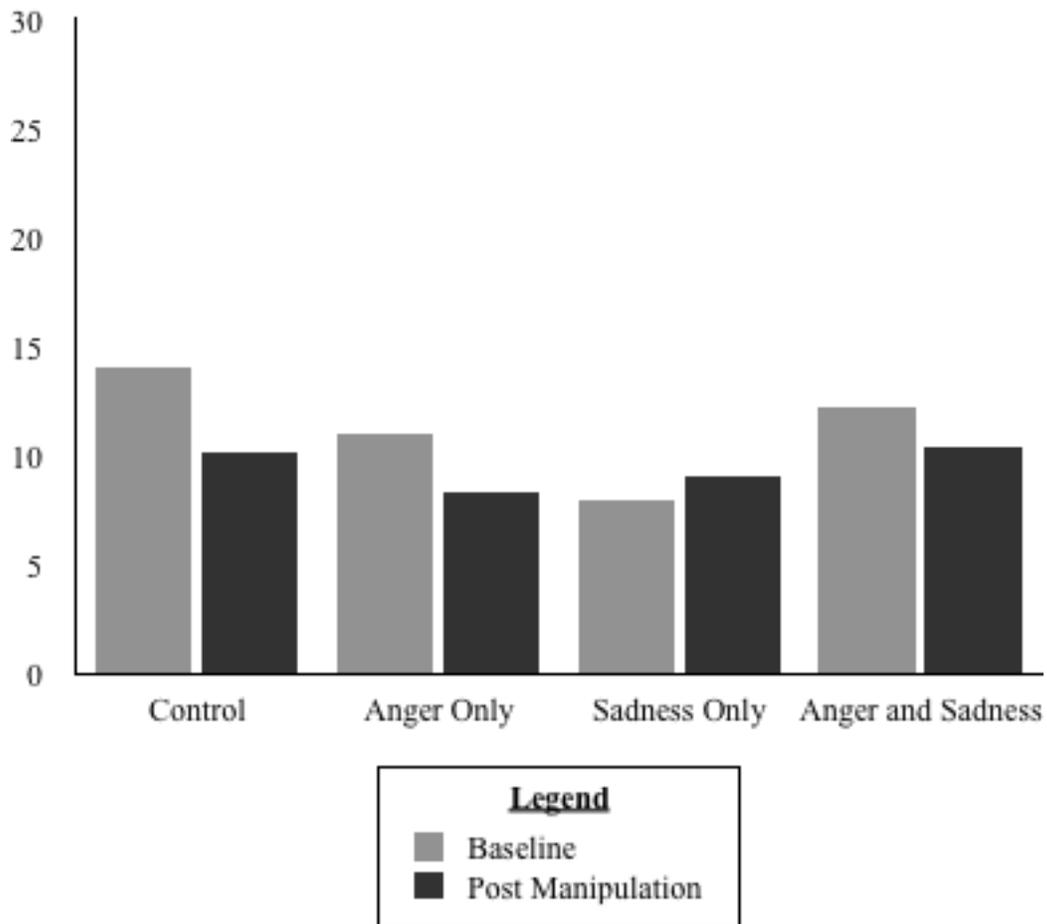


Figure 6. Changes in Pain Threshold.

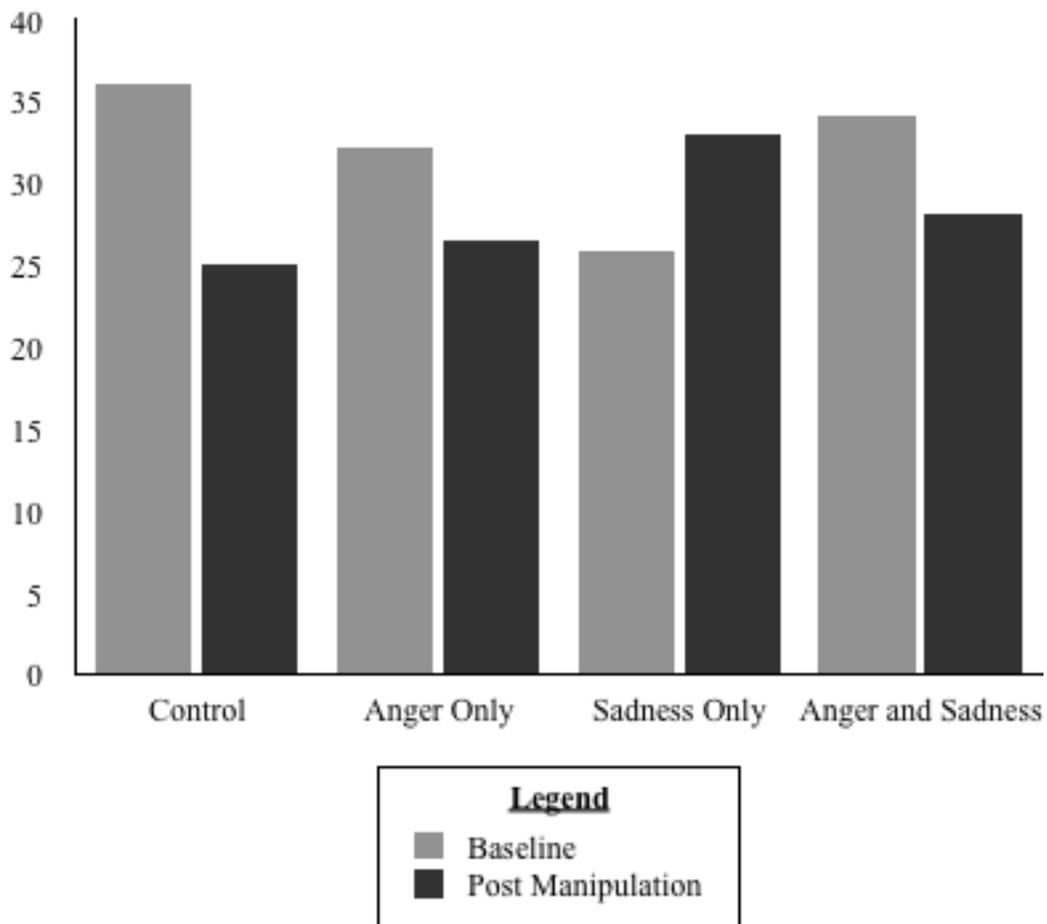


Figure 7. Changes in Pain Tolerance.

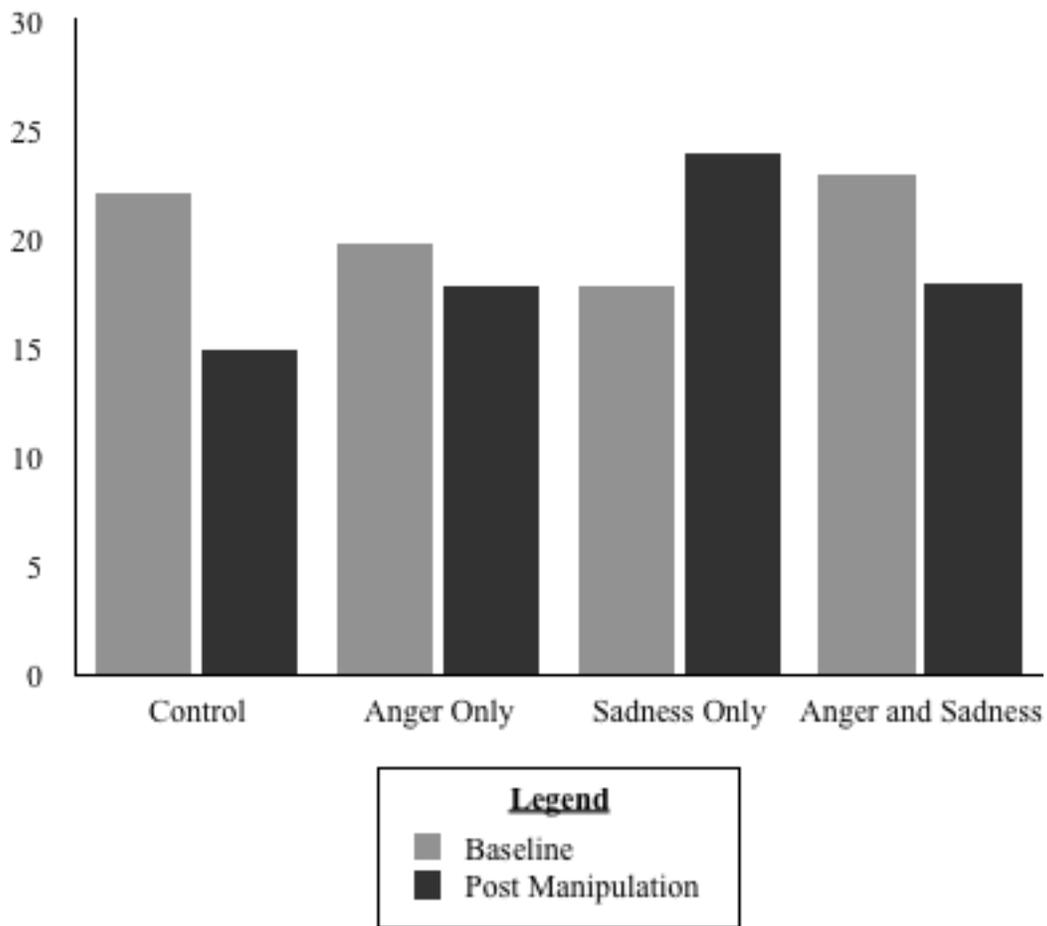


Figure 8. Changes in Pain Persistence

## Changes in Heart Rate

### *Average Heart Rate*

There was a significant effect of Time on participants' average heart rate ( $F(9,486)=79.383, p<.001, \eta^2=.595$ ; See Figure 9). There was, however, no significant main effect of Condition ( $F(3,54)=.157, p=.925$ ) or a significant interaction effect of Time and Condition ( $F(27,486)=1.102, p=.331$ ) on average heart rate. Compared to Baseline ( $M=80.326, SD=10.709$ ), there was a significant increase in heart rate at Pain Threshold 1 ( $M=92.365, SD=11.788, p<.001$ ) and Pain Tolerance 1 ( $M=94.364, SD=12.108, p<.001$ ) during the first cold pressor test, but no significant change in average heart rate between Pain Threshold 1 and Pain Tolerance 1 ( $p=1.000$ ). This was followed by a subsequent significant decrease between Pain Tolerance 1 and the Post-Pain Recovery 1 ( $M=78.005, SD=11.884, p<.001$ ). There were no changes in heart rate between Post-Pain Recovery 1 and the emotion induction ( $M=77.702, SD=10.432, p=1.000$ ). There were also no changes in heart rate between the emotion induction and rumination induction tasks ( $M=79.367, SD=9.828, p=.912$ ). There was, again, a significant increase at Pain Threshold 2 ( $M=88.445, SD=10.982, p<.001$ ) and Pain Tolerance 2 ( $M=89.705, SD=11.333, p<.001$ ) but no significant change in average heart rate between Pain Threshold 2 and Pain Tolerance 2 ( $p=1.000$ ). Finally, there was a significant decrease in average heart rate from Pain Tolerance 2 to Post-Pain Recovery 2 ( $M=73.540, SD=9.703, p<.001$ ). There were no significant changes between Post-Pain Recovery 2 and Recovery ( $M=73.540, SD=9.703, p=.860$ ).

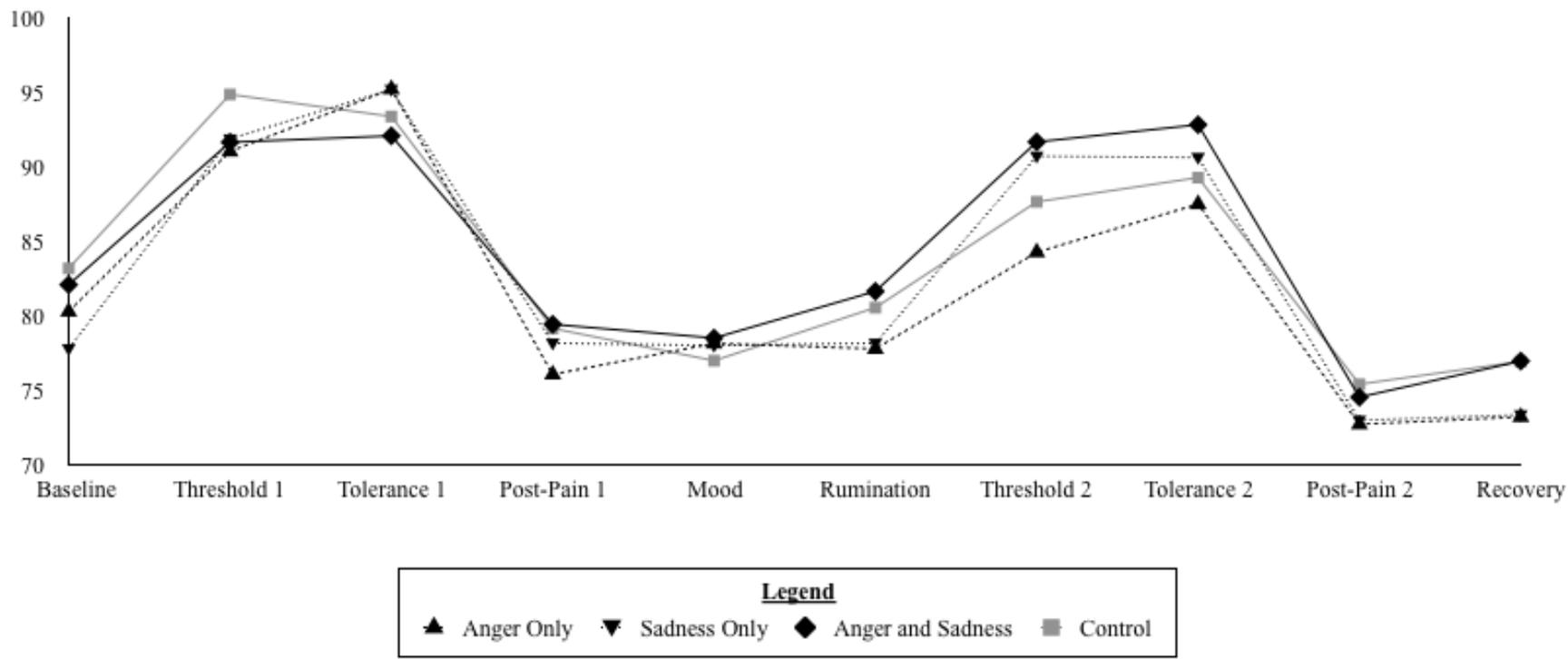


Figure 9. Changes in Average Heart Rate.

### *Maximum Heart Rate*

There was a significant effect of Time on participants' maximum heart rate ( $F(9,486)=10.704, p<.001, \eta^2=.165$ ; See Figure 10). There was, however, no significant main effect of Condition ( $F(3,54)=.927, p=.434$ ) or a significant interaction effect of Time and Condition ( $F(27,486)=1.031, p=.423$ ) on maximum heart rate. Unlike average heart rate, there was not a significant increase in maximum heart rate between Baseline ( $M=98.324, SD=15.427$ ) and Pain Threshold 1 ( $M=, p<.001$ ) but there was a significant increase in maximum heart rate between Baseline and Pain Tolerance 1 ( $M=94.364, SD=12.108, p=.008$ ). There was not a significant increase between Pain Threshold 1 and Pain Tolerance 1 ( $p=.091$ ). There was a significant decrease in maximum heart rate between Pain Tolerance 1 and Post-Pain Recovery 1 ( $M=97.701, SD=16.017, p<.001$ ). There were, however no changes in heart rate between Post-Pain Recovery 1 and the emotion induction task ( $M=99.266, SD=15.323, p=1.000$ ) but there was a significant increase in maximum heart rate between Post-Pain Recovery and the rumination induction task ( $M=110.99, SD=20.789, p=.010$ ). Maximum heart rate did not significantly decrease between the rumination induction task and Pain Threshold 2 ( $M=104.200, SD=13.009, p=1.000$ ) and Pain Tolerance 2 ( $M=103.681, SD=14.441, p=1.000$ ). Maximum heart rate did, however, significant decrease between Pain Tolerance 2 and Post-Pain Recovery 2 ( $M=96.508, SD=15.555, p=.024$ ) and did not have any significant changes between Post-Pain Recovery 2 to Recovery ( $SD=93.331, SD=11.568, p=1.000$ ).

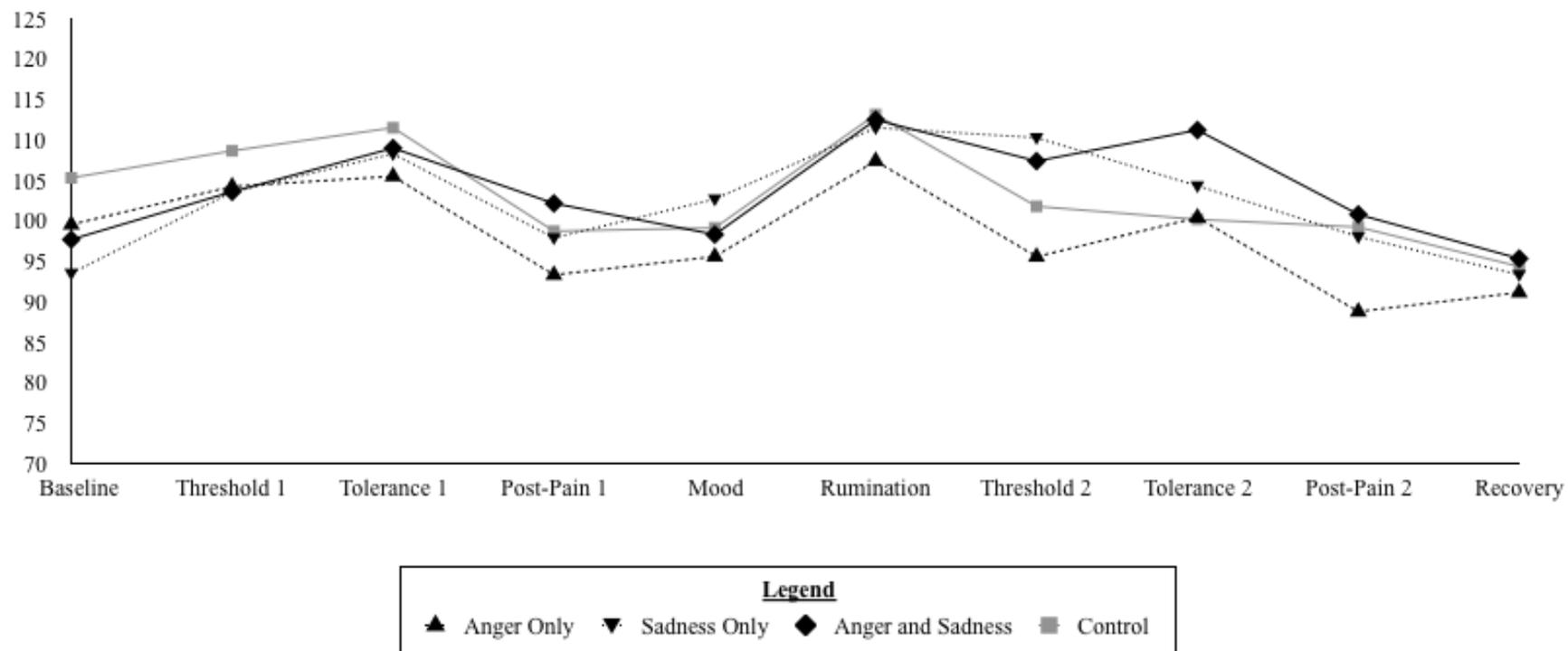


Figure 10. Changes in Maximum Heart Rate

### *Indirect Effects*

It was originally proposed that the PROCESS macro, following the guidelines detailed by Hayes and Preacher (2013), would be used to test if heart rate mediates the relationship between Condition and pain tolerance. Specifically, the PROCESS macro was to be executed 3 separate times using 10,000 bootstrapped samples with one of the three, dummy coded, experimental Condition variables alternating as the independent variable while the remaining Condition variables are entered as covariates at each execution. Given that Condition had no effect on change in pain responses or heart rate, however, the proposed indirect effects analyses were not conducted.

## CHAPTER IV – DISCUSSION

Contrary to our expectations, the results of this study suggest that only pain threshold, and not pain tolerance or persistence, may be subject to momentary fluctuations. The emotions on which participants were asked to ruminate also did not influence changes in their pain responses. Furthermore, participants in the four separate conditions did not significantly differ in their changes in heart rate throughout the experiment. As such, it is unlikely that an indirect effect of heart rate would be found on condition and changes in pain responses in this sample.

There is a possibility that changes in pain tolerance and persistence are, in fact, not dynamic. There is, however, some research that supports this claim. Extant research and theories examining pain emphasize the role of neuronal activity and pain transmission in influencing the perception of pain (Moayedi & Davis, 2013). Biologically, nociceptive pathways associated with pain transmission are dynamic in

their ability to modulate the awareness and perception of pain (See Urch, 2007 for a review). Specifically, the release of inhibitory neurotransmitters can attenuate the signaling and response to painful stimuli to influence the activation of the conscious awareness of pain. Past research has also found that cognitive state can modify the perception of pain and contribute to pain analgesia (Calloca & Benedetti, 2005; Salomons, Johnstone, Backonja, Shackman, & Davidson, 2007). As such, it is likely that pain tolerance and persistence are malleable through various mechanisms acting on nociceptive pathways. Furthermore, in the context of Borderline Personality Disorder, which is often associated with the use of self-injurious and other painful/provocative behaviors to regulate emotion, a pilot study has found that induced states of dissociation can temporarily reduce the sensitivity to pain (Ludäscher et al., 2010).

Several limitations of this study may have impacted our ability to effectively manipulate and measure the changes in pain tolerance and persistence. Firstly, the nature of the sample may not have been conducive to detecting the anticipated effect. A preceding study that had found changes in pain responses over time using a similar paradigm had used a larger sample ( $n=120$ , Law & Anestis, in preparation). Furthermore, the participants in this study may not have been sufficiently motivated to tolerate and persist through pain. Given that the laboratory protocol can last up to 3 hours, participants may have experienced fatigue and been more motivated to end the study early than to fully engage in the cold pressor tests. This problem is more likely to have influenced their performance in the second cold pressor test at the end of the experiment. Indeed, a previous study using the same experimental paradigm has found significant decreases in pain tolerance and persistence during the second cold pressor test (Law & Anestis, in

preparation). As such, future studies should consider providing a source of motivation to engage in the cold pressor test that can emulate an individual's motivation for suicidal behavior and self-injury. The non-clinical student sample used in this study may also be a poor representation of the population of individuals who may be at risk for suicide. Specifically, students who have the privilege of attending university, compared to their counterparts, may experience less exposure to painful and provocative experiences given that they more often raised in a more protective western, educated, industrialized, rich, and democratic culture (Henrich, Heine, & Norenzayan, 2010). Indeed, a two-part study examining distress and pain tolerance in unmatched student and community samples suggests that, on average, college student participants may have a lower pain tolerance than community participants (Law, Khazem, Jin & Anestis, 2016). As such, shifts in their pain responses may be less prominent than their counterparts who have had more experience and practice tolerating and persisting through distress and pain in their daily lives. Furthermore, given the demographics of the sample, the experiences that were provided for emotion induction task may be not produced the level of intensity necessary to induce changes arousal and subsequent pain responses. The experiences that were used to induce emotion included themes such as betrayal by a partner, car accidents, conflict with parents, physical altercations, and verbal altercations. At the same time, while these experiences may not be intense enough to produce a significant change in capability for suicide in adults, they may be like the experiences that contribute to self-injurious and suicidal behavior in adolescents and young adults. As such, despite the lack of findings in this sample, it would be important to continue replicating this experiment across diverse

samples to gain insight into the nuances of how emotional, cognitive, and physiological perturbations contribute to changes in pain responses.

As demonstrated by the manipulation check, participants' experience of negative emotions regardless of condition significantly decreased between the emotion induction and rumination induction tasks. This demonstrates that the rumination induction task did not provide the intended effect of exacerbating negative emotions that has been consistently demonstrated through tests of the Emotional Cascade Model (Selby & Joiner, 2009; Selby, Kranzler, Panza, & Fehling, 2016). As such, the intensity and effect of the emotions induced by the emotion induction task was neither exacerbated or sustained long enough to influence participants' responses to the cold pressor test. Alternatively, anger and sadness may not be particularly salient emotions that motivate an individual to tolerate or persist through pain. While anger and sadness may be relevant to suicidal ideation and intent, perhaps another high arousal emotion, such as fear, may be more activating and motivating towards a suicide attempt. In past studies examining decision making in the context of survival, avoidance and escape decisions have been found to involve both a fast reactive-fear circuit and a slow cognitive-fear circuit (Qi, Hassabis, Sun, Guo, Daw, & Mobbs, 2018). In the context of suicide, fear may motivate individual to engage in fast, proximal fearful/painful experiences (e.g. suicide, self-injury) to avoid a more slow, distal fear (e.g. living in pain/hopelessness, being a burden to others). Furthermore, recent studies have also suggested that physiological changes in response to emotion differ from individual to individual (Siegel et al., 2018). In other words, across all individuals, there is no single physiological pattern that corresponds to specific emotions like anger.

Physiologically, heart rate may not have been the best indicator of arousal to use in the context of experiment due to its quick return to baseline and sensitivity to movement. Electrodermal activity, a more stable indicator of physiological arousal with a slower return to baseline, may have been a more appropriate. Furthermore, electrodermal activity might be more relevant to the sensation of pain given its proximity to sensory receptors. Indeed, skin conductance has been found to be sensitive to self-report ratings of pain in past studies using postoperative patients in recovery (Ledowsky, Bromilow, Wu, Paech, Storm, & Schug, 2007). Alternatively, blood pressure may have also be a more suitable indicator of physiological arousal associated with changes in pain responses given its role in managing blood flow and circulation through the body. Indeed, past research has found increases in blood pressure to be associated with greater relief from pain (Pickering, 2007).

While we did attempt to manipulate the regulation of negative emotional experiences by inducing rumination, an individual's pre-existing ability to cope with distress and pain may have also impacted their performance in the cold pressor test regardless of the experimental manipulations. Specifically, some individuals may have engaged in various strategies to decrease their arousal during the rumination induction task and during the cold pressor tests. Thus, it would be interesting to examine whether self-report (e.g. trait level rumination, distress tolerance) and physiological (e.g. heart rate variability) indicators of effective/ineffective emotion regulation may have impacted participants' performance on the cold pressor test. Despite the lack of significant findings and the limitations of this study, however, this study is one of the first experiments aimed to test the processes and mechanisms that lead to changes in pain responses and

capability for suicide. Much of the research examining pain responses in the context of difficult emotions and suicide has relied on cross-sectional designs. As such, this study provides a blueprint for future experimental research that seeks to test the effects of various experiences and emotions that can influence dynamic changes in the capability for suicide. Gaining a more in depth understanding of these dynamic changes will then allow us to develop and refine the delivery of interventions by providing us with the ability to determine when and how to effectively thwart a suicide attempt.

## APPENDIX A – IRB Approval Letter



**INSTITUTIONAL REVIEW BOARD**  
118 College Drive #5147 | Hattiesburg, MS 39406-0001  
Phone: 601.266.5997 | Fax: 601.266.4377 | [www.usm.edu/research/institutional.review.board](http://www.usm.edu/research/institutional.review.board)

### NOTICE OF COMMITTEE ACTION

The project has been reviewed by The University of Southern Mississippi Institutional Review Board in accordance with Federal Drug Administration regulations (21 CFR 26, 111), Department of Health and Human Services (45 CFR Part 46), and university guidelines to ensure adherence to the following criteria:

- The risks to subjects are minimized.
- The risks to subjects are reasonable in relation to the anticipated benefits.
- The selection of subjects is equitable.
- Informed consent is adequate and appropriately documented.
- Where appropriate, the research plan makes adequate provisions for monitoring the data collected to ensure the safety of the subjects.
- Where appropriate, there are adequate provisions to protect the privacy of subjects and to maintain the confidentiality of all data.
- Appropriate additional safeguards have been included to protect vulnerable subjects.
- Any unanticipated, serious, or continuing problems encountered regarding risks to subjects must be reported immediately, but not later than 10 days following the event. This should be reported to the IRB Office via the "Adverse Effect Report Form".
- If approved, the maximum period of approval is limited to twelve months.  
Projects that exceed this period must submit an application for renewal or continuation.

PROTOCOL NUMBER: 16101802  
PROJECT TITLE: Cognition, Arousal and Pain Processing  
PROJECT TYPE: New Project  
RESEARCHER(S): Keyne Law  
COLLEGE/DIVISION: College of Education and Psychology  
DEPARTMENT: Psychology  
FUNDING AGENCY/SPONSOR: N/A  
IRB COMMITTEE ACTION: Expedited Review Approval  
PERIOD OF APPROVAL: 10/28/2016 to 10/27/2017

**Lawrence A. Hosman, Ph.D.**  
**Institutional Review Board**

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