

THE EFFECT OF ANGER RUMINATION ON CARDIOVASCULAR RESPONSES  
DURING SELF-ANGER AND OTHER-ANGER

by

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## ABSTRACT

CHARLSIE ANN MYERS. The effect of anger rumination on cardiovascular responses during self-anger and other-anger. (Under direction of DR. SUSAN K. JOHNSON).

The high morbidity and mortality associated with cardiovascular (CV) disease have led to a profusion of research into its etiology. With only 50% of the variance in risk associated with traditional risk factors, research has begun to focus more on psychological and behavioral risk factors to improve treatment and prevention options for CV disease. CV reactivity and recovery following an emotional stressor such as anger has been proposed as possible explanations for the relationship between negative emotional stressors and CV health through prolonged activation of the autonomic stress response. One proposed factor that may contribute to prolonged autonomic activation following anger includes anger rumination, which is the tendency to have unintentional and recurrent thoughts about anger experiences after the anger experience has ended. While previous research has shown that engaging in anger rumination following anger is associated with longer recovery time and continued experiences of anger (e.g., Gerin et al., 2006), no research has focused on CV responses following anger in which no one was to blame, such as being angry with one's self. The present study examined the role of state and trait anger rumination and state and trait negative affect on CV recovery time after having 75 healthy undergraduate students (ages 18-44) write about two anger experiences in a repeated-measures design: one in which they had been angry with someone else (other-anger) and another in which they had been angry with themselves (self-anger). Path analysis results revealed that trait anger rumination, but not state anger rumination, was a significant direct predictor of longer CV recovery time following the

other-anger writing task, but the same pattern was not observed following the self-anger writing task. Furthermore, trait negative affect was significantly but negatively associated with CV recovery time for both self-anger and other-anger, indicating that higher trait negative affect was associated with faster CV recovery time. The findings from the present study suggest that although both writing tasks were associated with significant changes in CV responses and self-reported state negative affect, the influence of trait anger rumination on CV responses may only operate when the anger was caused by someone else. While the proposed model was not supported in the present study, findings do suggest self-anger is common and is associated with similar CV responses to those seen in other-anger. Thus, further research is warranted to examine potential psychological factors that may underlie self-anger and its concomitant CV responses.

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## TABLE OF CONTENTS

LIST OF FIGURES	vi
LIST OF TABLES	vii
LIST OF ABBREVIATIONS	viii
CHAPTER 1: INTRODUCTION	1
1.1    Anger Constructs	2
1.2    Cardiovascular Responses and Health	6
1.2.1    Cardiovascular Reactivity	7
1.2.2    Cardiovascular Recovery Time	8
1.3    Anger and Cardiovascular Responses	10
1.3.1    Anger Rumination	11
1.4    Statement of the Problem and Hypotheses	16
CHAPTER 2: MATERIALS AND METHODS	19
2.1    Participants	19
2.2    Materials	19
2.3    Procedure	23
2.4    Design	27
2.5    Plan of Analysis	27
CHAPTER 3: RESULTS	32
3.1    Preliminary Analyses	32
3.2    Path Analyses	33
3.2.1    Other-anger path analysis: Mean arterial pressure	33

3.2.2	Other-anger path analysis: Heart rate	37
3.2.3	Self-anger path analysis: Mean arterial pressure	40
3.2.4	Self-anger path analysis: Heart rate	43
3.3	Comparison of self-anger and other-anger models	46
3.3.1	Models including mean arterial pressure as a predictor	46
3.3.2	Models including heart rate as a predictor	49
CHAPTER 4: DISCUSSION		54
4.1	Comparison of overall model fit for self-anger and other-anger	55
4.2	Comparisons of analogous paths	56
4.3	Limitations	63
4.4	Contributions and conclusion	65
FIGURES		68
TABLES		76
REFERENCES		83
APPENDIX A: MEASURES		91

## LIST OF FIGURES

FIGURE 1. The rumination-arousal model	68
FIGURE 2. Diagram of hypothesized relations (all paths predicted to be positive).	69
FIGURE 3. Flowchart of procedures for current study	70
FIGURE 4. Model to be tested after adding path modification between reactivity and state anger rumination	71
FIGURE 5. Standardized path coefficients for state and trait predictors with MAP reactivity of CV recovery time for other-anger (with modification)	72
FIGURE 6. Standardized path coefficients for state and trait predictors with HR reactivity of CV recovery time for other-anger (with modification)	73
FIGURE 7. Standardized path coefficients for state and trait predictors with MAP reactivity of CV recovery time for self-anger (with modification)	74
FIGURE 8. Standardized path coefficients for state and trait predictors with HR reactivity of CV recovery time for self-anger (with modification)	75



## LIST OF TABLES

TABLE 1. Mean (standard deviation) state negative affect and CV responses across tasks	76
TABLE 2. Descriptive statistics and zero-order correlations for other-anger variables	77
TABLE 3. Descriptive statistics and zero-order correlations for self-anger variables	78
TABLE 4. Standardized direct, indirect, total, and spurious effects (with 90% confidence intervals) for other-anger MAP reactivity from model	79
TABLE 5. Standardized direct, indirect, total, and spurious effects (with 90% confidence intervals) for other-anger HR reactivity from model	80
TABLE 6. Standardized direct, indirect, total, and spurious effects (with 90% confidence intervals) for self-anger MAP reactivity from model	81
TABLE 7. Standardized direct, indirect, total, and spurious effects (with 90% confidence intervals) for self-anger HR reactivity from model	82

## LIST OF ABBREVIATIONS

AR	anger rumination
ARS	anger rumination scale
BMI	body mass index
BP	blood pressure
bpm	beats per minute
CFI	comparative fit index
CI	confidence intervals
cm	centimeters
CV	cardiovascular
HR	heart rate
MAP	mean arterial pressure
mm Hg	millimeters mercury
NA	negative affect
PANAS	positive and negative affect schedule
RMSEA	root mean square error of approximation
VAS	visual analog scale

## CHAPTER 1: INTRODUCTION

Research in behavioral medicine has shown a consistent link between health and negative emotional states, such that negative emotions are typically associated with negative health behaviors and poor health outcomes (e.g., Kubzansky, Davidson, & Rozanski, 2005). For example, much of the research examining negative psychological states has focused on the impact anger can have on health, particularly health related to the cardiovascular (CV) system. While the anger and CV health relationship has been demonstrated in both healthy (e.g., Dorr, Brosschot, Sollers, & Thayer, 2007; Hogan & Linden, 2004; Labouvie-Vief, Lumley, Jain, & Heinze, 2003; Lai & Linden, 1992) and hypertensive (e.g., Hogan & Linden, 2004) populations, the mechanism(s) by which anger influences the CV system remain unknown (Linden, Hogan, Rutledge, Chawla, Lenz, & Leung, 2003).

One mechanism that has received a great deal of support in the anger literature involves the duration of one's CV responses following a negative emotional stressor, which is known as CV recovery (Trivedi, Sherwood, Strauman, & Blumenthal, 2008). It is posited that persons who are unable to return to resting levels of blood pressure and heart rate quickly after an anger event are at greater risk for development of hypertension and/or CV complications (Glynn, Christenfeld, & Gerin, 2002). One reason that this is particularly true for negative emotions, such as anger, is that negative emotions often involve a cognitive component. One example of a cognitive response

during and following anger includes ruminating about the event. Such anger rumination leads to unintentional and recurrent thoughts about anger experiences that have been hypothesized to perpetuate one's anger as well as the physiological responses to anger (Anderson, Linden, & Habra, 2005).

Based on the posited relationship between anger rumination and delayed CV recovery, the major purpose of this study was to determine whether the tendency to ruminate about anger influenced the degree of CV recovery when participants recalled two types of anger events: one event when they were angry with someone else and one when they were angry with themselves. The following introduction outlines the background literature related to anger constructs and the mechanisms by which anger is posited to influence CV health that were used to develop the underlying research questions and hypotheses for this study.

### 1.1 Anger Constructs

Anger, hostility, and aggression represent separate constructs, but are often used interchangeably in the emotion literature; therefore, it is important to differentiate these constructs and provide an operational definition of anger as used in this study. Hostility is typically described as a negative cognitive trait and attitudes that motivate aggressive behaviors toward other people or objects (Chida & Steptoe, 2009). Aggression implies intentions or actual verbal or physical behaviors that are destructive or punitive toward other people or objects (Chida & Steptoe, 2009; Spielberger, Jacobs, Russell, & Crane, 1983). Anger is defined as an emotional state that can range from mild irritation to rage in intensity that typically arises in response to perceived mistreatment or provocation (Harburg, Julius, Kaciroti, Gleiberman, & Schork, 2003; Linden et al., 2003).

Not surprisingly, anger overlaps with hostility and aggression, in that aspects of anger are present in both hostile cognitions and aggression. However, anger can occur in individuals low in trait hostility (Linden, Hogan, Rutledge, Chawla, Lenz, & Leung, 2003) and without resulting in aggressive behaviors. In addition, anger is a frequently experienced emotion, with previous research showing that mild to moderate anger is experienced daily to several times a week (Averill, 1983) and typically lasts for half an hour at a time (Kassinove, Sukhodolsky, Tsytsarev, & Soloveyva, 1997). These characteristics of anger demonstrate its uniqueness from hostility and aggression, as well as the utility of studying anger to understand how negative emotions may influence CV health. The operational definition of anger used in this study refers to a transient negative state that arises when a person appraises a situation as unpleasant due to events that lead to feelings associated with loss of control, unfairness, intentional harm, or goal blockage (Ellsworth & Tong, 2006).

Anger consists of specific action tendencies, physiological changes, and appraisals (Ellsworth & Tong, 2006; Sukhodolsky, Golub, & Cromwell, 2001). Action tendencies associated with anger typically revolve around anger response styles such as anger expression (e.g., attack, revenge, harm) (Ellsworth & Tong, 2006), anger suppression (e.g., withdrawal, avoidance) (Brosschot & Thayer, 1998), or assertion (e.g., constructively resolving conflict) (Lowenstein, 2004; Thomas, 2002). Action tendencies were not examined in the present study; instead the focus was on physiological responses and anger appraisals. Some of the physiological changes associated with anger are discussed in detail in a subsequent section on anger and CV responses.

Appraisals related to emotion involve judgments of the perceived or real environment. Accordingly, these judgments are what ultimately influence the emotions experienced, such that changes in how one perceives a situation produce changes in how one feels about the situation (Ellsworth & Tong, 2006). Several appraisal components have been identified for anger, such as blockage of a goal, perception of unfairness, loss of control, and a belief that incurred harm was intentional. Perhaps one of the most salient and supported appraisals involved in negative emotional experiences is the appraisal of who is responsible for what happened, or agency appraisal. One of the defining features of anger includes blaming others for responsibility (Berkowitz & Harmon-Jones, 2004; Ellsworth & Smith, 1988).

More recently, however, researchers have suggested that anger can occur when no one else is to blame. Such situations may lead one to be angry at an inanimate object or at themselves, which may indicate that other-blame is not necessary for the experience of anger. Anger at inanimate objects, such as an object that you trip over because it was left on the floor, can often be explained as anger at someone else (e.g., a child who left the object in the floor) or at oneself (e.g., not watching where you were going, or if you left the object in the floor). When participants have been asked to describe their anger, self-anger has been reported, although it is less common than other-anger (Averill, 1982, as cited in Ellsworth & Tong, 2006), with self-anger mostly reported when anger is perceived as unreasonable (Parkinson, 1999).

Ellsworth and Tong (2006) examined whether other-agency is necessary for anger by comparing anger, self-anger, shame, and guilt in having participants recall and relive an experience in which they were angry with someone else (other-anger) or angry

at themselves (self-anger). The results indicated that people do indeed get angry at themselves, and this self-anger resulted in markedly different appraisals, action tendencies, and associated emotions when compared to other-anger, guilt, and shame. For example, self-anger was significantly less likely to involve perceived unfairness or moral wrongness, was characterized by a strong desire to withdraw from the situation, and was strongly associated with shame, guilt, regret, embarrassment, and sadness. There were also similarities to other-anger, such that when participants were angry with themselves they reported feeling negative emotions, experiencing goal obstacles, and “boiling inwardly.”

The findings from Ellsworth and Tong’s (2006) study indicate that self-anger does overlap with other-anger and guilt/shame in some appraisals, action tendencies, and related emotions in some ways, but self-anger also has distinct characteristics that demonstrate it is not simply a form of other-anger with a different agent or simply another name for guilt/shame. However, it is unknown whether self-anger is related to CV health as research with other-anger has indicated. Furthermore, if self-anger is related to CV health, it is unclear whether it influences CV variables in a similar manner as other-anger. This study thus examined CV factors thought to be related to CV disease risk while having participants recall and write about an event in which they were angry with themselves and an event when they were angry at someone else. The following section discusses some of the proposed mechanisms whereby anger may influence CV health.

## 1.2 Cardiovascular Responses and Health

With the exception of the year 1918, since 1900, CV disease has been the leading cause of mortality among men and women in industrialized countries. The most recent mortality data from the United States showed that CV disease was the underlying cause for 33.6% of all deaths in 2007 (Roger et al., 2011). Furthermore, 82.6 million American adults (one in three) are estimated to have one or more types of CV disease, including high blood pressure or hypertension (76.4 million), coronary heart disease (including heart attack and chest pain; 16.3 million), heart failure (5.7 million), and stroke (7 million) (Roger et al., 2011). The high prevalence of CV disease also leads to high economic burden, with direct and indirect costs estimated to be \$286.6 billion in 2007 (Roger et al., 2011). These statistics and associated costs illustrate the importance of investigating the causes and course of CV disease to establish more effective methods of treatment and prevention.

The high morbidity and mortality associated with CV disease have led to a profusion of research into its etiology. Much of the focus was initially on biological risk factors, such as cigarette smoking, obesity, and high cholesterol levels. While these factors do account for 50% of the variance in CV disease risk (Brand, Rosenman, Sholtz, & Friedman, 1976), researchers have begun to focus on identifying psychological and behavioral risk factors that may account for a substantial proportion of the unexplained variance.

One area of research that has shown promise in understanding CV disease risk includes studying individual differences in the physiological stress response. The acute stress response, often called the fight-or-flight response, results in mobilization of energy



resources to deal with a stressor via increased activation of the sympathetic branch of the autonomic nervous system. This sympathetic nervous system activation leads to increased heart rate (HR), constricted blood vessels (which leads to increased blood pressure; BP), increased blood flow to the limbs, and increased rate of respiration. The other branch of the autonomic nervous system, the parasympathetic branch, normally helps to maintain homeostasis with the sympathetic branch, and is used to return the body to resting levels following a stressor, as well as in digestive and other restorative functions (Lovallo, 2004). The following section includes a discussion of proposed mechanisms of how sympathetic arousal may lead to increased risk for CV disease.

#### 1.2.1 Cardiovascular Reactivity

Several mechanisms have been proposed to explain how the physiological stress response might be implicated in CV health. For the past 25 years, the predominant biopsychosocial model of the relationship between stress and CV disease has been the CV reactivity hypothesis (Krantz & Manuck, 1984). CV reactivity is defined as the changes in the heart and blood vessels that occur while a stressor is present. Increases in HR and BP are normal and adaptive for dealing with stressors when the level of activity is proportional to the demands of the stressor (Key, Campbell, Bacon, & Gerin, 2008). However, when the stress response stays active for too long or is activated too frequently, it can become maladaptive. The CV reactivity hypothesis posits that exaggerated CV responses during stressors, when experienced often, can lead to hypertension or other pathogenic adaptations of the CV system (Anderson et al., 2005; Gerin et al, 2006; Trivedi, Sherwood, Strauman, & Blumenthal, 2008).

The CV reactivity hypothesis has received some support in stress and emotion research, such that diagnosed hypertensives (Fredrikson, Tuomisto, & Bergman-Losman, 1991) as well as normotensives (i.e., individuals with normal blood pressure) with a positive family history of hypertension (Light, Girdler, Sherwood, Bragdon, Brownley, West et al., 1999) show exaggerated CV responses during laboratory stressors. However, the strength of prospective associations of CV reactivity to hypertension and other forms of CV disease are modest (Carroll, Smith, Shipley, Steptoe, Brunner, & Marmot, 2001). Thus, displaying high levels of CV reactivity relative to the type of stressor may be a risk factor for hypertension (Trieber et al., 2003), but the predictive ability of the magnitude of an acute response appears limited.

#### 1.2.2 Cardiovascular Recovery Time

Another proposed mechanism for how stress can influence CV health, the CV recovery time hypothesis, suggests that it is not the magnitude of the response to a stressor that is harmful for health; rather, the duration of the response is posited to contribute to CV disease risk (Glynn, Christenfeld, & Gerin, 2002). CV recovery time is defined as the amount of time it takes for elevated CV measures, such as BP and HR, to return to resting levels after a stressor has ended (Trivedi et al., 2008). It has been hypothesized that CV recovery may be a marker of chronic sympathetic nervous system activation and low parasympathetic tone (Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006), and this chronic activation may lead to CV tissue damage and system dysregulation (Key, Campbell, Bacon, & Gerin, 2008).

CV recovery time may provide greater external validity than CV reactivity, as demonstrated by Trivedi and colleagues (2008), who found that CV recovery time was an

independent predictor of ambulatory BP after controlling for resting BP and BP reactivity. Borghi, Costa, Boschi, Mussi, and Ambrosioni (1986) also found that borderline hypertensive participants who had poorer diastolic BP recovery after a mental stressor significantly predicted the development of hypertension at a five-year follow up, which indicated greater predictive power for CV recovery compared to reactivity.

Furthermore, CV recovery may be more relevant when examining the health outcomes of psychological stressors that have an emotional component. For example, Glynn and colleagues (2002) compared CV responses during and after four types of laboratory stressors varying in emotional content (emotional vs. non-emotional) and CV reactivity magnitudes (low vs. high). Their results showed that emotional tasks were associated with delayed recovery, whereas non-emotional tasks were associated with quick CV recovery. It is important to note that both positive and negative emotions result in CV reactivity, and in approximately equal amounts (Jacob et al., 1999); however, only negative emotions are associated with prolonged CV recovery (Brosschot & Thayer, 2003). Thus, determining the factors that lead to prolonged CV recovery may shed light on why negative emotions are associated with CV disease.

While CV recovery time appears to better explain the relationship between CV responses during stress and factors related to CV disease risk, researchers have shown that the examination of both CV reactivity and CV recovery time accounted for the greatest variance in predicting ambulatory BP, indicating that the two are not independent from one another and should both be measured in emotional stress research (e.g., Anderson, Linden, & Habra, 2005; Brosschot & Thayer, 1998; Trivedi et al., 2008). The next section provides a discussion of CV responses in anger research.

### 1.3 Anger and Cardiovascular Responses

Acute emotional stress is thought to be particularly relevant for the study of CV disease development (Anderson, Linden, & Habra, 2005). In particular, negative valence emotions have received a lot of attention since they tend to induce greater autonomic and neuroendocrine arousal and typically involve prolonged recovery from that arousal (Anderson et al., 2005; Suchday, Carter, Ewart, Larkin, & Desiderato, 2004). In particular, risk for hypertension and other forms of CV disease have been linked to the experience and expression of anger. Studies that have examined anger and CV health factors typically assessed changes in BP and HR following some sort of anger induction or provocation, with anger showing effects on the CV system that are similar to those of any other emotional stressor (Anderson, Linden, & Habra, 2005). A recent meta-analysis of prospective studies examining anger and coronary heart disease risk demonstrated anger is significantly associated with increased risk among initially healthy populations as well as poor prognosis in patients with existing coronary heart disease (Chida & Steptoe, 2009). A brief review of some of the research on anger and CV reactivity and CV recovery time are provided below.

Much of the earlier anger research related to CV reactivity and CV disease risk attempted to determine whether specific anger expression styles, such as overt anger expression or anger suppression, were associated with greater CV reactivity. Most researchers have abandoned this narrow view of studying anger and CV health risks and have accepted that extreme forms of either suppression or expression can be detrimental to one's health and a person's anger expression style is likely to change depending upon the situation (Hogan & Linden, 2004). Regardless of anger response style, exaggerated

CV reactivity has only shown modest associations with CV disease development in prospective studies (Carroll, Smith, Shipley, Steptoe, Brunner, & Marmot, 2001; Kamarck & Lovallo, 2003; Linden, Gerin, & Davidson, 2003; Schwartz, Gerin, Davidson, Pickering, Brosschot, Thayer et al., 2003; Treiber et al., 2003), which suggests that the mechanism proposed by the CV reactivity hypothesis is not the only means by which anger can contribute to factors associated with CV disease risk.

The available literature on CV recovery time and anger supports the notion that the duration of the anger response following anger provocation can lead to CV vulnerability (e.g., Anderson et al., 2005; Brosschot & Thayer, 1998; Suchday, Carter, Ewart, Larkin, & Desiderato, 2004). Both prolonged BP (e.g., Gerin et al., 2006) and prolonged HR (e.g., McClelland, Jones, & Gregg, 2009) recovery times following anger provocation have been implicated as risk factors for the development of CV disease, again indicating CV recovery time utility in examining risk for CV disease. These studies indicate that laboratory anger provocation tasks reflect negative emotional stressors from which participants display heightened CV responses, including both reactivity and prolonged recovery. Thus, the study of anger in the laboratory by assessing CV responses appears to be a valid mechanism for examining the effects emotional stressors may have on CV variables. The following section elaborates on the role of anger rumination in eliciting anger and the subsequent physiological responses.

### 1.3.1 Anger Rumination

The findings that support prolonged CV recovery following a stressor as a potential marker for CV disease have led researchers to examine factors that contribute to the extended duration of CV responses. As previously mentioned, activating the stress

response is adaptive in the long run, as it allows organisms to have the energy to respond to a stressor. When the activation becomes chronic, however, the stress response no longer is adaptive and may actually do more harm than the stressor itself (Sapolsky, 2004, p. 13). Most of the stressors humans encounter in their daily lives in industrialized countries are psychological in nature, meaning they are not physically harmful or threatening and do not require physical activity to overcome. Regardless, the same fight-or-flight response is activated, and often too frequently, intensely, or for too long.

Another quality that differentiates humans from animals is the ability to make cognitive representations of events from the past and the anticipated future. While this is often a beneficial quality to have when it enhances survival and quality of one's life, it can also be negative if the person chooses to focus on negative events of the past or worry about future events (Brosschot, 2010). Representations and reactivation of past and future stressors can often produce similar physiological activation to those that would occur during the actual occurrence of the stressors, which makes such cognitive representations a potential underlying cause of prolonged CV responses resulting from stress (Brosschot, 2010).

As previously mentioned, researchers have recently begun to focus on one particular type of cognitive representation, known as anger rumination, and its association with CV recovery time. Anger rumination has been demonstrated as a separate construct from anger as well as anger response styles (Linden et al., 2003; Sukhodolsky et al., 2001). Similar to rumination discussed in the depression literature, anger rumination is a cognitive process that can occur following an anger experience that results in unintentional and recurrent thoughts about anger experiences (Sukhodolsky,

Golub, & Cromwell, 2001), the causes and consequences of one's anger, and self-evaluations related to the anger (Rusting & Nolen-Hoeksema, 1998). Rumination is thought to occur when there is a discrepancy between an individual's goals and what is actually occurring, and an individual may continue to ruminate until the goal has been met or disregarded (Thomsen, 2006).

Within the depression literature, several negative outcomes have been associated with depression. For example, individuals that tend to ruminate report more negative emotions in general (Segerstrom, Tsao, Alden, & Craske, 2000) and believe that they have less control over their intrusive thoughts (Watkins, 2004). Tendency to engage in rumination has also been associated with more chronic symptoms of depression (Nolen-Hoeksema, Morrow, & Fredrickson, 1993) as well as more frequent episodes of depression (Nolen-Hoeksema, 2000). A literature review by Lyubomirsky and Tkach (2003) highlights several other consequences of high levels of rumination including decreased motivation, impaired inhibition, higher levels of stress, poor health behaviors, difficulties in social relationships, as well as poor concentration, cognition, and problem solving.

Much less research has examined anger rumination compared to the number of studies that have investigated depressive rumination. However, it is important to note that these ruminative thoughts do not focus on problem solving related to one's anger; thus, these ruminative thoughts are hypothesized to reactivate anger and lead to prolonged anger responses, including prolonged negative affect and physiological activation. Furthermore, these prolonged responses may increase one's CV health risks via the mechanisms previously discussed (Gerin et al., 2006).

Research that has examined the role of rumination in prolonging CV recovery after emotional stressors has generally shown that rumination is associated with a longer recovery time in the laboratory (e.g., Glynn et al., 2002). To explain how anger rumination may influence CV responses, Gerin and colleagues (2006) developed the rumination-arousal model (shown in Figure 1). This proposed model emphasizes CV recovery time as the major mechanism that contributes to CV disease, and predicts a synergistic relationship exists among anger rumination, prolonged activation of the CV response to anger, as well as increased negative emotions, including anger (Gerin et al., 2006). In other words, engaging in rumination after an anger event leads to the experience of anger and other negative emotions, and those emotions lead to autonomic arousal, causing BP and HR to increase. This process then feeds forward, in that the cognitions, feelings, and autonomic activation continue to prolong one another until distraction occurs (Gerin et al., 2006).

Gerin and colleagues' (2006) study consisted of measuring CV responses while healthy, community-dwelling adults recalled two anger events over a one-week interval: one anger recall was followed by having participants sit quietly in a plain room (no distraction), while the other recall gave participants access to visually interesting stimuli, including posters, magazines, and small toys (distraction). When participants who scored high in trait rumination (i.e., a tendency to ruminate across a wide variety of situations) were not given the opportunity for distraction following the anger recall, they demonstrated the poorest CV recovery compared to low trait ruminators and when distraction was allowed. Furthermore, these same participants reported thinking more about the anger event during the recovery period, indicating that persons with tendencies



to ruminate about angry events will do so after the recall of such events, and will continue to do so until distraction is provided. Thus, this study showed support for the rumination-arousal model, particularly for trait anger rumination's role in prolonging CV recovery.

Other researchers that have examined anger rumination in relation to CV recovery time have found support for Gerin and colleagues' (2006) rumination-arousal model. For example, Suchday and colleagues (2004) asked male college students to role-play scenes from vignettes designed to induce anger with confederates. In one condition, participants were asked to suppress their anger toward the confederate, and in the second condition, participants were asked to express their anger toward the confederate. Participants' CV responses were recorded during and following each role play. After the recovery periods, participants completed an inventory about their anger-related thoughts that occurred during the recovery period. The results indicated that high endorsements of anger rumination were associated with prolonged CV recovery following anger provocation, regardless of the type of anger expression style participants typically preferred or were asked to use during the role plays. Thus, this study demonstrated that state anger rumination, or how much participants actually reported thinking about their anger, also influences CV recovery.

Rusting and Nolen-Hoeksema (1998) examined anger rumination and self-reported anger by having participants engage in either emotion-focused and self-focused thoughts or a distraction condition following various anger induction procedures (e.g., imagining a hypothetical event happened to them or recalling a personal anger event). Then, participants were asked to complete a story for an emotionally ambiguous

sentence, and the emotional content of their stories were coded by independent raters. Participants that were asked to focus their thoughts on themselves and their emotions reported increased feelings of anger, and this increased anger was associated with more negative beliefs, memories, and events in their story completions compared to participants that engaged in a distraction task. Rusting and Nolen-Hoeksema's (1998) results provide indirect evidence for the rumination-arousal model in that focusing on the self and one's emotions, as occurs in rumination, intensified participants' self-reported anger, and this led them to write about mood-congruent content.

#### 1.4 Statement of the Problem and Hypotheses

In general, there is support for Gerin and colleague's (2006) proposed rumination-arousal model such that following an anger event, cognition (i.e., anger rumination), affect (e.g., anger), and physiological responses (i.e., increased CV recovery time) interact in reciprocal ways that could potentially have negative CV health consequences. However, the role of anger rumination on CV responses has only been examined when the source of anger was someone else (other-anger). As previously mentioned, Ellsworth and Tong (2006) have demonstrated that anger is often focused on the self (self-anger), and that self-anger is a distinct construct from anger at others as well as from guilt or shame. To date, however, it is unknown whether self-anger has similar CV health implications as observed in research with other-anger.

Because anger rumination typically involves self-thoughts that perpetuate anger, engaging in anger rumination when anger is focused on the self may lead to other emotional problems (e.g., depression and/or anxiety). Thus, it is important to determine whether self-anger results in a similar process of responses observed with other-anger

with regard to rumination and CV recovery time, and doing so was the first goal of this dissertation project. In order to examine whether self-anger and other-anger are explained by similar models, both self-anger and other-anger were measured in the current study. The following hypothesis will be used to test whether self-anger results in a similar process as seen with other-anger:

*Hypothesis 1:* Models that include trait and state anger rumination and state and trait negative affect as predictors of CV recovery time will produce similar path coefficients and fit statistics for self-anger and other-anger.

The second goal of the current dissertation project was to examine both state and trait variables, such as negative affect and anger rumination, and their influence on CV recovery in order to test a modified version of the rumination-arousal model. As can be seen in the path diagram show in Figure 2, the path model was used to examine how CV reactivity, self-reported negative affect (state and trait), and anger rumination (state and trait) influence CV recovery following each type of anger recall and each CV response.

The current study examined the role of anger rumination and negative affect in predicting BP and HR recovery times following the recall of an event in which participants were angry with themselves (self-anger) compared to the recall of an event in which participants were angry with someone else (other-anger) using a path model based on the rumination-arousal model. The following hypotheses were predicted for both self-anger and other-anger based on previous findings regarding state and trait anger rumination and CV recovery:

*Hypothesis 2a:* CV reactivity will be positively associated with CV recovery time (path c in Figure 2).

*Hypothesis 2b:* Trait negative affect will be positively related to CV recovery, such that lower levels of trait negative affect will be associated with faster CV recovery time (path b in Figure 2).

*Hypothesis 2c:* Trait anger rumination will be positively related to CV recovery, such that lower levels of trait anger rumination will be associated with faster CV recovery time (path d in Figure 2).

*Hypothesis 2d:* State negative affect will partially mediate the relationship between trait negative affect and CV recovery time (path a + g in Figure 2).

*Hypothesis 2e:* State anger rumination will partially mediate the relationship between trait anger rumination and CV recovery time (path e + h in Figure 2).

*Hypothesis 2f:* State negative affect and state anger rumination will mediate the influence of trait anger rumination on CV recovery time (path e + f + h in Figure 2).

*Hypothesis 3a:* State negative affect (path g) will be positively related to CV recovery time, such that lower levels of state variables will be associated with faster CV recovery time.

*Hypothesis 3b:* State anger rumination (path h) will be positively related to CV recovery time, such that lower levels of state variables will be associated with faster CV recovery time.

*Hypothesis 3c:* State anger rumination will positively predict state negative affect, such that engaging in state anger rumination will increase negative affect (path f).

*Hypothesis 3d:* State negative affect will partially mediate the relationship between state anger rumination and CV recovery time (path f + g).

## CHAPTER 2: METHODS AND MATERIALS

### 2.1 Participants

Seventy-five undergraduate students (28 men and 47 women) enrolled in introductory psychology classes at The University of North Carolina at Charlotte were recruited for participation in this study. Participants received course credit in exchange for their time. Participants ranged in age from 18 to 44 years ( $M = 22.36$ ,  $SD = 5.22$ ). Fifty-eight percent of the participants self-identified as White, 19% as African American, 9% as Asian; 5% as Hispanic or Latino, and 8.0% self-identified as Multiracial/Other.

All participants met the following inclusion criteria: the ability to write in English for approximately 20 minutes, not taking medications or having a health condition known to affect blood pressure, resting systolic BP less than or equal to 140 millimeters mercury (mm Hg) and resting diastolic BP less than or equal to 90 mm Hg, and not consuming caffeine, using tobacco products, or engaging in vigorous physical activity within two hours prior to their scheduled appointment time.

### 2.2 Materials

*Eligibility Questionnaire.* Participants self-reported their dominant hand by indicating the hand used to perform a variety of activities (e.g., brushing their teeth, throwing a ball, writing a letter) on a scale of 1 (always left) to 5 (always right). Next, participants indicated whether they experience any of the following by indicating “Yes”

or “No” for each experience: chronic pain (such as migraine headaches), high or low blood pressure, presence of any type of heart disease, take medication for pain, heart problems, or high blood pressure (e.g. beta blockers, ACE Inhibitors, anti-inflammatory medications), family history of high blood pressure, or family history of heart disease. Each of the aforementioned experiences were subsequently dummy-coded as 0 for “No” responses and 1 for “Yes” responses for analysis purposes. Participants were also asked to report their current use of tobacco products on the following scale: “Yes, daily,” “Yes, occasionally,” “No, but I live with someone who does,” or “No, never.” Tobacco product use was dummy-coded as 0 for “No, never” or 1 if any of the other responses were indicated for analysis purposes. A copy of the eligibility questionnaire can be seen in Appendix A.

*Demographics Questionnaire.* A demographics questionnaire was created for this study. Participants reported basic demographic information (e.g., age, sex, race, income, year of college education, grade point average, and marital status), height and weight (used to calculate body mass index; BMI), their greatest source(s) of stress (e.g., financial, school, work, family, or personal), medications taken, whether they had consumed caffeine or used tobacco products and/or engaged in strenuous physical activity in the two hours prior to their scheduled time, and date of last menstrual cycle (if female). A copy of the demographics questionnaire can be seen in Appendix A.

*Positive and Negative Affect.* The extent to which participants feel positive and negative affect was assessed using the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a 20-item scale that is composed of two 10-item subscales designed to measure positive and negative affect by asking

participants to rate how often they experience emotions over a specified time period on a five-point Likert scale ranging from 1 (very slightly or not at all) to 5 (extremely). The time frame of the scale can be modified, such that affective rating can be related to persistent feelings (affective trait) or across shorter time-frames to detect changes over short periods of time (affective state). Thus, scores on each scale can range from 10 to 50, with higher scores indicating greater frequency of the affective experience being measured. Watson and colleagues (1988) reported Cronbach's alpha coefficients for the various time frames ranging from .86 to .90 for the Positive Affect scale and .84 to .87 for the Negative Affect scale. A more recent assessment (Crawford & Henry, 2004) demonstrated similar Cronbach's alpha coefficients (.89 for positive and .85 for negative). For this study, the time frame "in general" was used to assess for trait affect, and only the Negative Affect scale was to measure trait negative affect.

*Trait Anger Rumination.* Trait anger rumination, the degree to which participants generally think about their anger after the event has ended, was assessed using the Anger Rumination Scale (ARS; Sukhodolsky, Golub, & Cromwell, 2001). The ARS is a 19-item scale in which participants rate on a Likert scale of 1 (almost never) to 4 (almost always) how well items correspond to their beliefs about themselves. Scores can range from 19 to 76 on the ARS, with higher scores indicating higher trait anger rumination. The ARS has been shown to have adequate internal consistency (Cronbach's alpha for the total scale was 0.93) (Sukholdolsky et al., 2001). While the ARS provides subscale ratings for four factor structures, only the total ARS score was used to measure trait anger rumination in this study.

*State Affect Ratings.* Ten centimeter (cm) visual analogue scales (VAS) were used to measure participants' state affect changes throughout the experiment. Similar VAS measures have been used for pain ratings and have demonstrated adequate sensitivity for pain ratings (Jensen, Karoly, & Braver, 1986). Participants were instructed to mark along a 10 cm horizontal line anchored with "not at all" (0 cm) and "extremely" (10 cm) how much they were currently experiencing the following 11 emotions: happy, guilty, sad, anxious, angry, shameful, frustrated, stressed, calm, embarrassed, and regret. A blank mood with rating scale was provided in case participants wished to rate an emotion not on the list. An example VAS for the 11 moods can be seen in Appendix A. Because only trait negative affect was used in the analyses, the two positive emotions, happy and calm, were reverse scored (such that a 10 cm rating on the positive emotion would indicate a 0 cm rating when scored as a negative emotion) to provide a measure of state negative affect.

*Post-recovery State Anger and Anger Rumination Ratings.* Following the recovery period for each anger recall writing task, participants were asked to complete separate 10 cm VAS measures to assess their state anger on a scale of 0 cm ("not at all") to 10 cm ("extremely") and state anger rumination on a scale of 0 cm ("not at all") to 10 cm ("very much"). When asking for the state anger rumination ratings, participants were instructed to indicate how much they had been thinking about the anger event they just recalled. An example of these VAS measures can be seen in Appendix A.

*Cardiovascular Reactivity and Recovery Time.* Systolic BP (in mm Hg), diastolic BP (in mm Hg), mean arterial pressure (MAP; in mm Hg) and HR (in beats per minute, bpm) were measured using either a Critikon Dinamap 1846SX or a Critikon Dinamap



8100, both of which are automated blood pressure monitors (Dinamap, Inc., Tampa, FL, USA). MAP values were used to assess BP reactivity and recovery to minimize the number of models tested. MAP values are considered the average arterial pressure during a single cardiac cycle, which accounts for both resting (diastolic) and contraction (systolic) BP values. The BP cuff remained attached to the participant's non-dominant arm above the elbow during the baseline, writing, and recovery portions of the study. Critikon Dinamap BP monitors have been shown to provide accurate and reliable means for measurement of non-invasive BP (Friedman, 1997), and such monitors are recommended in research settings (Whincup, Bruce, Cook, & Shaper, 1992). For the current study, CV reactivity was calculated as the difference in MAP and HR values for each anger writing task relative to baseline values of the respective measure. CV recovery time was measured as the time in minutes and seconds taken to return to baseline levels, with measures taken immediately after the participant completed mood ratings following the anger writing task and then at two minute intervals until the participant's MAP and HR measures were at or below baseline levels.

### 2.3 Procedure

*Online screening.* After providing informed consent, participants completed the Eligibility Questionnaire, the PANAS, and the ARS online using SurveyShare. The online questionnaire took approximately 15 minutes to complete. Before exiting the survey, participants provided an email address to assign research credits and to allow each participant's survey answers to be matched to their in-laboratory data. Once a participant identification number had been assigned to link the online survey data to in-laboratory data, all identifying information was removed.

*Baseline.* The order in which participants completed the self-anger and other-anger writing tasks was predetermined using a random number generator. Participants were scheduled for the in-laboratory portion of the experiment either individually or in pairs. Participants that completed the session in pairs were assigned to the same order of anger writing tasks to ensure standardization of instructions for both participants and to ensure participants were not aware of the upcoming condition instructions. Once participants signed a second informed consent form for the in-laboratory session, participants were then asked to complete the Demographics Questionnaire. After eligibility was confirmed, a blood pressure cuff was fitted to the participant's non-dominant arm above the elbow. The cuff remained attached during the baseline, writing, and recovery portions of the study. First, a trial inflation was performed to ensure a comfortable fit of the cuff; if needed, adjustments were made and a second trial inflation was performed. Once the cuff was placed properly, the participant's resting MAP and HR measures were determined by taking three measurements at two-minute intervals. Averages across these three assessments were used as the participant's baseline MAP and HR measures. Next, the participants were asked to rate how much they were currently feeling 11 emotions using a VAS.

*Writing Tasks.* Immediately following the baseline CV and mood ratings, the participants were given instructions for the first writing task (self-anger or other-anger). All participants completed both the self-anger and other-anger writing tasks in counterbalanced order. Participants were given paper, pen, and the following instructions for the writing tasks (adapted from Ellsworth & Tong, 2006, p. 574):

*“Think of a time when you felt angry (with yourself (SELF)/ with someone (OTHER)). Try to recall as many details of the incident as you can. Picture this situation in your mind. Try and remember as vividly as you can what this anger situation was like. Think of what happened to make you feel angry (with yourself), and what it felt like to be feeling angry (with yourself) in this particular situation. When you have this memory clearly in mind, answer the following questions: When did this event happen? Tell us in detail what happened to cause you to feel angry (with yourself). Tell us in as much detail as you can what you were feeling and thinking. Tell us about what you did and what you said. As much as possible, write your descriptions so that someone reading it would feel the anger you felt from reading your description.”*

Participants were asked to write for a minimum of six minutes for both the self-anger and other-anger writing tasks, with an unspoken time limit of 10 minutes, and the duration of the writing task was recorded. If the participant was writing after 10 minutes had passed, they were asked to finish the current sentence and then stop writing. To assess CV reactivity, the participant's MAP and HR measures were assessed after five minutes had elapsed for each writing task. After the first writing task was completed, the instructions and participants' writings were moved out of sight from participants and a timer was started to assess recovery time. Participants were asked to complete a second set of 11 emotion ratings corresponding to their current emotions related to the first anger writing task. Participants were told they could sit quietly and rest for a few minutes before they would move on to the next writing task. Approximately two minutes after the writing task had ended, the first CV recovery measures were taken. Subsequent recovery

measures were taken every two minutes until BP and HR measures returned to mean baseline levels (or below) and the time to recover was recorded. Next, participants provided VAS ratings for their current anger and state anger rumination during the recovery period.

Before beginning the next anger writing task, participants were asked to copy a list of states and state capitals for two minutes while one BP and HR measurement was obtained approximately 30 seconds into the task. Writing states and state capitals served as a neutral writing task, which allowed for the measurement and control of the effect of writing on BP and HR. Following the neutral writing task, participants completed a third set of VAS emotion ratings for the 11 emotions previously described. An additional BP and HR measurement was taken following the emotion ratings to ensure the participants' CV measures were at or below mean baseline levels before moving on to the next anger writing task.

Following the neutral writing task, participants completed the remaining anger writing task as described above. Once the second anger writing task was completed, the participant was asked to provide a fourth set of VAS ratings for the 11 emotions, and the second recovery period begun. Once the participant recovered to resting CV levels and the time for recovery had been recorded, participants completed state anger rumination and state anger ratings. Then, the participants were debriefed and course credit was given in exchange for their participation. A flowchart of the aforementioned procedures is shown in Figure 3.

## 2.4 Design

The overall design of this study was a repeated measures design, whereby participants completed a baseline period, two types of anger recall writing tasks (self and other) separated by a neutral writing task, and recovery periods for each writing task. As previously mentioned, the order of the writing tasks was counterbalanced to ensure there were no order effects of writing task. The dependent variables of interest included changes in negative affect (using VAS ratings), BP and HR changes from baseline during each type of anger writing task (reactivity, measured five minutes into each writing task), the amount of time taken to return to resting levels on CV measures (i.e., CV recovery time) following each type of writing task, and state anger rumination (using VAS ratings) following each writing task. Trait variables that were examined included trait anger rumination (using the ARS) and negative affect (using the negative affect score from the PANAS).

## 2.5 Plan of Analysis

According to an *a priori* power analysis, a sample size of 24 was recommended in order to detect a medium effect with 80% power at an alpha level of 0.05. Furthermore, the sample size was doubled to be able to test for any order effects among the two anger tasks, with 37 completing the self-anger writing task first and 38 completing the other-anger writing task first.

To ascertain whether order effects were present, an independent t-test was performed for each of the two possible orders (self/other; other/self) for each of the following variables: MAP reactivity, HR reactivity, state anger rumination, state negative affect ratings, and recovery times for self-anger and other-anger writing tasks. Next,

means and standard deviations for the continuous variables were calculated to ensure the means and standard deviations appeared within a normal range of possible scores for each variable.

Within-subjects analysis of variance (ANOVA) tests were conducted as manipulation checks to ascertain that the anger recall tasks resulted in significant changes of state anger ratings, MAP, and HR values across each of the following tasks: baseline, self-anger recall task, self-anger recovery period, other-anger recall task, other-anger recovery period, and the neutral writing task. When a significant omnibus ANOVA was present, pair-wise comparisons of meaningful mean differences were calculated and significance levels were adjusted using Bonferroni corrections (i.e., .05 divided by the number of comparisons) to avoid making a Type I error. Bivariate correlations were also performed to ensure key variables in the proposed model were associated in the expected directions.

A state negative affect variable was created by reverse scoring the Happy and Calm VAS ratings (i.e., subtracting actual score from 10cm) for self-anger and other-anger tasks. Then, all VAS ratings for self-anger were averaged to provide a self-anger state negative affect variable and the same was then done for other-anger to provide a state negative affect variable following the other-anger recall task. State negative affect was examined rather than only state anger since the rumination-arousal model posits negative affect may influence CV recovery, rather than just state anger (Gerin et al., 2006). Furthermore, when people are asked to rate their emotions, they often report feeling more than one (Ellsworth & Tong, 2006). Because it remains unclear whether people actually feel several discrete emotions at once or the labels provided do not fully

capture their actual, complex emotional experience, participants' composite state negative affect scores were used as an overall measure of changes in their emotional experiences during the experiment.

A residual variable score was created for CV recovery time by removing the influence of the following variables: age, sex (dummy-coded), body mass index, family history of hypertension or CV disease (dummy-coded), and smoking status (dummy-coded). Creating residual variable scores for CV recovery time measures allowed for the control of the influence of variables known to influence CV responses, thereby equating participants' CV recovery time to see the unique effects of CV reactivity, state and trait anger rumination, and state and trait negative affect.

Path analysis using the maximum likelihood procedure (AMOS Student Version 5.0) addressed whether state and trait variables were differentially associated with CV recovery time for self-anger and other-anger tasks. To examine these differences, four models were tested: self-anger MAP, self-anger HR, other-anger MAP, and other-anger HR. Model fit was assessed using the following goodness of fit indicators: the Chi square statistic ( $\chi^2$ ), the Root Mean Square Error of Approximation (RMSEA) statistic, and the comparative fit index (CFI). If modification indices were recommended to improve the fit for the models tested, then the model was modified if the modification made theoretical sense to do so.

Suggestions from Hu and Bentler (1999) were used to guide cutoff scores for all indices. Namely, a non-significant Chi-square ( $\chi^2$ ) statistic represents a close fit between the hypothesized model and the data (Hu & Bentler, 1999). For the RMSEA, a cutoff score close to or less than 0.06 is indicative of good fit, while a cutoff score close to or

less than 0.08 is indicative of acceptable goodness of fit (Hu & Bentler, 1999). CFI values range from .00 to 1.00, with those greater than .90 represent an acceptable fit, while scores equal to or greater than .95 indicates a well-fitting model (Hu & Bentler, 1999). For ease of interpretation, standardized regression coefficients were reported and interpreted. Direct path coefficients were generated from AMOS output when the path models were tested. Indirect path coefficients were calculated as the cumulative product of the coefficients leading from the causal variable (e.g., trait anger rumination) through other causes (e.g., state anger rumination and state negative affect) on the criterion variable (e.g., CV recovery time). The total effects are the sum of the causal variable's direct and indirect effects. The differences between the total effect and the zero-order effect for all endogenous variables were also calculated to determine whether there were any spurious or suppression effects.

To examine whether indirect effects were significant, nonparametric bootstrapping was performed using AMOS (Student Version 5.0). Bootstrapping is a technique used to simulate drawing numerous random samples of the same size as the actual sample, allowing standard errors to be estimated as the standard deviation in the sampling distribution that was simulated (Kline, 2011; p. 42). For the present study, 2000 bootstrapping iterations were used to be able to calculate standard errors and confidence intervals. In particular, bootstrapping was used to calculate biased-corrected 90% confidence intervals (CIs) and probability statistics for the indirect effects in the present study. Finally, to compare the self-anger and other-anger models to one another, confidence intervals were compared for the analogous direct paths in the models as well



as for the indirect paths using the confidence intervals provided by the aforementioned bootstrapping technique.

## CHAPTER 3: RESULTS

### 3.1 Preliminary analyses

Before conducting any analyses, independent t-tests were conducted to determine whether the order in which participants completed the self-anger and other-anger writing tasks influenced any of the following variables: MAP reactivity, HR reactivity, recovery time, state anger rumination ratings, or state negative affect ratings. No significant differences were detected for the two counterbalanced orders, indicating no order effects were present and analyses could proceed without controlling for order of task completion.

Next, three repeated-measures ANOVAs were conducted for state anger ratings, MAP, and HR to ascertain the anger recall tasks used in this study were successful for inducing anger and physiological reactivity. The mean values were compared across baseline, both anger writing tasks and the respective recovery period, and the neutral writing task, and individual comparisons were made using pair-wise comparisons with Bonferroni corrections. There were significant omnibus ANOVAs for MAP ( $F(5, 370)=46.67, p<.001$ ), HR ( $F(5, 370)=30.15, p<.001$ ), and state anger ratings ( $F(5, 370)=46.67, p<.001$ ). Table 1 shows the mean MAP, HR, and state anger values for the six different tasks, with significant increases from baseline indicated where appropriate. In general, the anger writing tasks produced statistically significant increases in self-reported anger, MAP, and HR relative to baseline. Furthermore, these differences in CV responses can be attributed to the anger induction and not activity level, as indicated by the non-significant

change from baseline to the neutral writing task for state anger ratings, MAP, and HR measures ( $ps > .05$ ).

Next, descriptive statistics and zero-order correlations were calculated to ensure the CV variables and scores on the self-report measures fell within a reasonable range and were related to one another in the expected ways. As can be seen in Tables 2 and 3, the means and standard deviations for the variables of interest in the present study were within a reasonable range and the standard deviations indicated enough meaningful variation existed to justify further analyses. The means and standard deviations of the CV recovery times reported in Tables 2 and 3 are those in which the effects of variables known to influence CV recovery time were partialled out. For the non-residualized CV recovery times, there was a slightly longer recovery time (in minutes) following the other-anger writing task ( $M = 3.21$ ,  $SD = 1.88$ ) compared to the self-anger writing task ( $M = 2.83$ ,  $SD = 1.67$ ), but this was not a significant difference,  $t(74) = 1.64$ ,  $p = .11$ .

Pearson product moment correlations were then computed among all of the variables, and these results can also be seen in Tables 2 and 3. As predicted, trait negative affect was significantly and positively related to state negative affect for both self-anger and other-anger. Also, trait anger rumination was significantly and positively related to state anger rumination for both self-anger and other-anger. Thus, endorsing higher trait negative affect and anger rumination resulted in higher self-reported state negative affect and state anger rumination, respectively. State negative affect and state anger rumination were also strongly positively related to one another for both self-anger and other-anger. Trait negative affect was significantly but negatively related to self-anger CV recovery time, and the same pattern was observed with other-anger CV

recovery time. Furthermore, neither state negative affect nor state anger rumination was significantly related to CV recovery time for either self-anger or other-anger. Finally, only MAP (but not HR reactivity) measures were significantly and positively related to CV recovery time for both self-anger and other-anger, suggesting that greater MAP reactivity was associated with a longer recovery time.

### 3.2 Path Analysis

To examine the influence of state and trait variables on CV recovery time, path analysis using the maximum likelihood procedure (AMOS Student Version 5.0) was conducted. As previously mentioned, CV recovery time was assessed as a residual in which the effect of variables known to influence CV recovery time were partialled out. To examine CV recovery time for self-anger and other-anger, four models were tested to account for the differences in BP and HR responses: self-anger MAP, self-anger HR, other-anger MAP, and other-anger HR. The path model tested (see Figure 2) was as follows: trait negative affect was hypothesized to influence CV recovery both directly and indirectly through state negative affect; CV reactivity was expected to only influence CV recovery directly; trait anger rumination was expected to influence CV recovery directly and indirectly through state anger rumination as well as through state anger rumination's influence on state negative affect.

#### 3.2.1 Other-anger path analysis: Mean arterial pressure

The path model for other-anger using MAP reactivity as a predictor was originally tested as previously described and as shown in Figure 2. Overall, the original model was not a good fit to the data for the current study's sample,  $\chi^2(4, N=75)=11.57, p=.02$ , CFI=.91, and RMSEA=.16 (90% confidence interval=.06-.27). The modification indices

provided by AMOS suggested a path be modeled between MAP reactivity and state anger rumination (see Figure 4 for modified model and renamed paths), which made theoretical sense because displaying physiological responses during one's anger is likely to be associated with subsequent anger rumination immediately after asking one to write about that angry event. The model was thus modified as suggested and provided a good fit to the data for the current sample,  $\chi^2(3, N=75)= 2.58, p=.46$ , CFI= 1.00, and RMSEA= .00 (90% confidence interval= .00-.19).

Table 4 shows the total, direct, indirect, and spurious effects for other-anger with MAP reactivity as a predictor. MAP reactivity significantly predicted CV recovery time (hypothesis 2a) for other-anger ( $\beta=.34, p=.04$ ), indicating that for every one standard deviation increase above the mean in MAP reactivity, an increase in CV recovery time during other-anger of approximately one-third of one standard deviation around the mean was predicted. A significant portion of this effect was direct, ( $\beta=.39, p=.02$ ), with only 15% of the effect ( $\beta= -.05, p= .10$ ) occurring indirectly via state anger rumination (paths d+ i and d + g + h). Furthermore, the indirect effect observed was not in the direction predicted; for a one standard deviation increase in MAP reactivity above the mean, a one-third of a standard deviation increase above the mean for state anger rumination was expected.

Trait negative affect had a significant total effect on CV recovery time ( $\beta= -.29, p=.04$ , 90% CIs [= -.52- -.06]), as tested in hypothesis 2b, but in the opposite direction predicted. There was a non-significant trend for trait negative affect to directly predict CV recovery time ( $\beta= -.26, p=.07$ ), accounting for 90% of the total effect observed. Furthermore, trait negative affect was predicted to have an indirect effect on CV recovery

via state negative affect (hypothesis 2d; see paths a + g in Figure 4); however, the indirect effect was calculated to be weak and negative ( $\beta = -.03$ ), accounting for only about 10% of the total effect (90% CIs [-.11-.01]). This indicated that the negative association observed for trait negative affect on CV recovery time was 90% direct. Overall, each one standard deviation increase in trait negative affect above the mean was non-significantly associated with just over one-quarter of a standard deviation change in the mean recovery time following the other-anger writing task.

The total effect for trait anger rumination to predict CV recovery time (hypothesis 2c) was not significant ( $\beta = .20$ ,  $p = .06$ ), although there was a trend in the predicted direction for hypothesis 2c. There was a significant direct effect for trait anger rumination on CV recovery time ( $\beta = .25$ ,  $p = .04$ ), with the positive association accounting for more than 100% of the total effect. Trait anger rumination also indirectly influenced CV recovery time by two pathways: through state anger rumination only (hypothesis 2e; paths f + i in Figure 4) and through state anger rumination and state negative affect (hypothesis 2f; paths f + g + h in Figure 4). According to the effects observed, these two indirect pathways accounted for approximately 20% of the influence of trait anger rumination on CV recovery time, but did not reach statistical significance (90% CIs [-.12-.00]).

The indirect effects of the state variables on CV recovery time were negative, unlike the direct effect of trait anger rumination on CV recovery time, suggesting a case of inconsistent mediation (i.e., suppression). This was further confirmed since the total effect ( $\beta = .21$ ) was greater than the zero-order effect ( $\beta = .02$ ) (MacKinnon, Krull, & Lockwood, 2000). In other words, about 18% of the effect of trait anger rumination on

CV recovery time was revealed by the current model tested. Finally, there was not a significant direct effect of trait anger rumination on state anger rumination ( $\beta = .26$ ,  $p = .06$ ), although the effect that was present indicated a trend for higher trait anger rumination to be associated with higher state anger rumination. While not specifically tested in the current study, it was noted that trait anger rumination significantly influenced state negative affect indirectly via state anger rumination ( $\beta = .11$ ,  $p = .04$ ; 90% CIs [.03-.23]). Thus, a tendency to ruminate about one's anger after it has passed was mediated by actually ruminating after the other-anger recall task and led to increased state negative affect.

State negative affect had a weak negative effect on CV recovery time ( $\beta = -.12$ ,  $p = .29$ ), which was not in the direction predicted by hypothesis 3a. This indicated that the higher one's state negative affect was following the other-anger recall, the faster his or her recovery time was. State anger rumination also had a weak negative effect on CV recovery time ( $\beta = -.06$ ,  $p = .25$ ), in the opposite direction predicted by hypothesis 3b. Neither the direct ( $\beta = -.10$ ,  $p = .45$ ) or indirect effect via state negative affect ( $\beta = -.06$ ,  $p = .25$ ; hypothesis 3d) were significant. Finally, hypothesis 3c, which predicted that state anger rumination would show a significant positive association with state negative affect, was supported by the significant direct association observed ( $\beta = .51$ ,  $p = .001$ ).

In summary, the predictors in the proposed model only explained 25% of the variance in CV recovery time. Of these predictors, MAP reactivity and trait anger rumination significantly and directly predicted CV recovery time in the directions predicted, such that higher MAP reactivity during the anger writing task and higher trait anger rumination were associated with longer CV recovery time. Trait negative affect

showed a marginally significant association with CV recovery time, but in the opposite direction predicted. Although not hypothesized, it was noted that trait anger rumination was a significant indirect predictor of state negative affect through state anger rumination. Finally, no significant indirect effects were found for predicting CV recovery time.

### 3.2.2 Other-anger path analysis: Heart rate

The path model for other-anger using HR reactivity as a predictor can be seen in Figure 5. Overall, the model was an adequate fit to the data for the current study's sample,  $\chi^2(4, N=75) = 6.35, p = .17$ , CFI = .96, and RMSEA = .09 (90% confidence interval = .00-.21). As with MAP reactivity for other-anger, the modification indices provided by AMOS suggested a path be modeled between HR reactivity and state anger rumination. The model was also modified as suggested, but it should be noted that the modifications slightly decreased the fit of the model to the data compared to the previous model,  $\chi^2(3, N=75) = 6.35, p = .10$ , CFI = .95, and RMSEA = .12 (90% confidence interval = .00-.26).

Table 5 shows the direct, indirect, total, and spurious effects for other-anger with HR reactivity as a predictor. Unlike the significant effect observed for MAP reactivity, HR reactivity did not significantly predict CV recovery time (hypothesis 2a) for other-anger ( $\beta = .14, p = .29$ ). Most of this non-significant effect was direct ( $\beta = .17, p = .29$ ), with only 21% of the effect ( $\beta = -.03, p = .93$ ) occurring indirectly via state anger rumination (paths d + i and d + g + h). Overall, HR reactivity was not a significant predictor of CV recovery time following the other-anger writing task.



Trait negative affect had a significant total effect on CV recovery time ( $\beta = -.40$ ,  $p = .005$ , 90% CIs  $[-.59, -.15]$ ), as tested in hypothesis 2b, but in the opposite direction predicted. There was a significant direct effect of trait negative affect on CV recovery time ( $\beta = -.38$ ,  $p = .008$ ), accounting for 95% of the total effect observed. Furthermore, trait negative affect was predicted to have an indirect effect on CV recovery via state negative affect (hypothesis 2d; see paths a + g in Figure 4); however, the indirect effect was calculated to be weak and negative ( $\beta = -.02$ ,  $p = .51$ ), accounting for only about 5% of the total effect (90% CIs  $[-.09, .03]$ ). Overall, each one standard deviation increase in trait negative affect above the mean was associated with just over one-third of a standard deviation change in the mean recovery time following the other-anger writing task for the current model.

The total effect for trait anger rumination to predict CV recovery time (hypothesis 2c) was not significant ( $\beta = .21$ ,  $p = .05$ ), although there was a trend in the predicted direction. There was a trend for a significant direct effect for trait anger rumination on CV recovery time ( $\beta = .23$ ,  $p = .09$ ), with the positive association accounting for more than 100% of the total effect. Trait anger rumination also indirectly influenced CV recovery time by two pathways: through state anger rumination only (hypothesis 2e; paths f + i in Figure 4) and through state anger rumination and state negative affect (hypothesis 2f; paths f + g + h in Figure 4). According to the effects observed, these two indirect pathways accounted for approximately 9% of the influence of trait anger rumination on CV recovery time, but did not reach statistical significance (90% CIs  $[-.07, .04]$ ).

The indirect effects of the state variables on CV recovery time were negative, unlike the direct effect of trait anger rumination on CV recovery time, suggesting a case

of inconsistent mediation (i.e., suppression) as seen in the MAP reactivity for other-anger model. However, this case of suppression was only associated with about 3% of the effect of trait anger rumination on CV recovery time being revealed by the current model tested. Finally, there was not a significant direct effect of trait anger rumination on state anger rumination ( $\beta = .22, p = .12$ ), although the effect that was present indicated a trend for higher trait anger rumination to be associated with higher state anger rumination. While not a specific hypothesis tested in the current study, it was noted that trait anger rumination showed a trend for influencing state negative affect indirectly via state anger rumination ( $\beta = .11, p = .09$ ; 90% CIs [.00-.20]). Thus, a tendency to ruminate about one's anger after it has passed was mediated by actually ruminating after the other-anger recall task and led to increased state negative affect.

State negative affect had a weak negative effect on CV recovery time ( $\beta = -.06, p = .65$ ), which was not in the direction predicted by hypothesis 3a. This indicated that the higher one's state negative affect was following the other-anger recall, the faster his or her recovery time was. State anger rumination also had a weak negative effect on CV recovery time ( $\beta = -.04, p = .81$ ), again in the opposite direction predicted by hypothesis 3b. Neither the direct ( $\beta = -.01, p = .99$ ) or indirect effects via state negative affect ( $\beta = -.03, p = .62$ ; hypothesis 3d) were significant. Finally, hypothesis 3c, which predicted that state anger rumination would show a significant positive association with state negative affect, was supported by the significant direct association observed ( $\beta = .51, p = .001$ ).

In summary, the predictors in the proposed model only explained 15% of the variance in CV recovery time. Of these predictors, only trait NA significantly directly

predicted CV recovery time, but not in the direction predicted. Therefore, HR reactivity, trait anger rumination, state anger rumination, and state negative affect did not have significant direct or indirect effects on CV recovery time. Trait anger rumination showed a trend for predicting state negative affect through state anger rumination, but this indirect effect did not reach statistical significance. Finally, no significant indirect effects were found for predicting CV recovery time.

### 3.2.3 Self-anger path analysis: Mean arterial pressure

The path model for self-anger using MAP reactivity as a predictor can be seen in Figure 6. Overall, the model was a good fit to the data for the current study's sample,  $\chi^2(4, N=75)= 1.45, p=.84$ , CFI= 1.00, and RMSEA= .00 (90% confidence interval= .00-.10). No modifications were suggested for this model, but the same modification was performed so that the self-anger MAP model could be compared directly to the other-anger MAP model. Therefore, a path predicting MAP reactivity to have a direct effect on state anger rumination was added to the model. The modified model was also a good fit to the data,  $\chi^2(3, N=75)= 1.14, p=.77$ , CFI= 1.00, and RMSEA= .00 (90% confidence interval= .00-.13). The direct, indirect, total, and spurious effects for all variables are shown in Table 6.

MAP reactivity significantly predicted CV recovery time (hypothesis 2a) for other-anger ( $\beta=.28, p=.02$ ), indicating that for every one standard deviation increase above the mean in MAP reactivity, an increase in CV recovery time during self-anger of approximately one-quarter of one standard deviation around the mean was predicted. Essentially all of the observed effect was direct, ( $\beta=.28, p=.03$ ), with less than 1% of the

effect ( $\beta = .00$ ,  $p = .54$ ) occurring indirectly via state anger rumination (paths  $d + i$  and  $d + g + h$ ).

Trait negative affect had a non-significant total effect on CV recovery time ( $\beta = -.26$ ,  $p = .05$ , 90% CIs  $[-.45, -.05]$ ), as tested in hypothesis 2b, but in the opposite direction predicted. Trait negative affect showed a marginally significant direct effect on CV recovery time ( $\beta = -.26$ ,  $p = .05$ ), accounting for 100% of the total effect observed. Furthermore, trait negative affect was predicted to have an indirect effect on CV recovery via state negative affect (hypothesis 2d; see paths  $a + g$  in Figure 4); however, the indirect effect was calculated to be weak and negative ( $\beta = .00$ ), accounting for none of the total effect (90% CIs  $[-.05, .01]$ ). Overall, each one standard deviation increase in trait negative affect above the mean was non-significantly associated with just over one-quarter of a standard deviation change in the mean recovery time following the self-anger writing task.

The total effect for trait anger rumination to predict CV recovery time (hypothesis 2c) was not significant ( $\beta = .03$ ,  $p = .90$ ). Furthermore, there was no support for hypotheses 2e or 2f, which predicted that trait anger rumination would indirectly influence CV recovery time by two pathways: through state anger rumination only (hypothesis 2e; paths  $f + i$  in Figure 4) and through state anger rumination and state negative affect (hypothesis 2f; paths  $f + g + h$  in Figure 4).

The indirect effects of the state variables on CV recovery time were negative, unlike the direct effect of trait anger rumination on CV recovery time, suggesting a case of inconsistent mediation (i.e., suppression) as seen in the previously tested models. Furthermore, there was a significant direct effect of trait anger rumination on state anger

rumination ( $\beta = .30, p = .04$ ), although the effect that was present indicated that higher trait anger rumination was associated with higher state anger rumination. While not a specific hypothesis tested in the current study, it was noted that trait anger rumination significantly influenced state negative affect indirectly via state anger rumination ( $\beta = .17, p = .03$ ; 90% CIs [.04-.29]). Thus, a tendency to ruminate about one's anger after it has passed was mediated by actually ruminating after the self-anger recall task and led to increased state negative affect.

State negative affect showed a weak negative effect on CV recovery time ( $\beta = -.04, p = .75$ ), which was not in the direction predicted by hypothesis 3a. This weak relationship indicated that state negative affect had essentially no relationship with CV recovery time in the current model. State anger rumination also showed a weak negative effect on CV recovery time ( $\beta = -.05, p = .64$ ), again in the opposite direction predicted by hypothesis 3b. Neither the direct effect ( $\beta = -.03, p = .77$ ) nor indirect ( $\beta = -.02, p = .74$ ; hypothesis 3d) effect via state negative affect were significant. Finally, hypothesis 3c, which predicted that state anger rumination would show a significant positive association with state negative affect, was supported by the significant direct association observed ( $\beta = .51, p = .001$ ).

In summary, the predictors in the proposed model only explained 14% of the variance in CV recovery time. Of these predictors, MAP reactivity was the only significant direct predictor of CV recovery time and this association was in the direction predicted, such that higher MAP reactivity during the self-anger writing task was associated with longer CV recovery time. Trait negative affect showed a trend for a significant negative association with CV recovery time, but this was in the opposite

direction predicted. Trait anger rumination significantly predicted state anger rumination, and state anger rumination significantly predicted state negative affect. Trait anger rumination was also a significant indirect predictor of state negative affect through state anger rumination. Finally, no significant indirect effects were found for predicting CV recovery time.

### 3.2.4 Self-anger path analysis: Heart rate

The path model for self-anger using HR reactivity as a predictor can be seen in Figure 7. Overall, the model was a good fit to the data for the current study's sample,  $\chi^2(3, N=75) = 2.05, p = .73$ , CFI = 1.00, and RMSEA = .00 (90% CIs [.00-.23]). No modifications were suggested for this model, but the same modification was performed so that the self-anger HR model could be compared directly to the other-anger HR model. Therefore, a path predicting HR reactivity to have a direct effect on state anger rumination was added to the model. The modified model was also a good fit to the data,  $\chi^2(4, N=75) = 1.14, p = .70$ , CFI = 1.00, and RMSEA = .00 (90% confidence interval = .00-.15). The direct, indirect, total, and spurious effects for all variables are shown in Table 7.

HR reactivity did not significantly predict CV recovery time (hypothesis 2a) for self-anger ( $\beta = .22, p = .06$ ), but did show a trend that indicated for every one standard deviation increase above the mean in HR reactivity, a change in CV recovery time during self-anger of approximately one-quarter of one standard deviation around the mean was predicted. Essentially all of the observed effect was direct, ( $\beta = .22, p = .07$ ), with less than 1% of the effect ( $\beta = .00, p = .69$ ) occurring indirectly via state anger rumination (paths d + i and d + g + h).

Trait negative affect had a marginally significant total effect on CV recovery time ( $\beta = -.29, p = .05$ , 90% CIs  $[-.48, -.06]$ ), as tested in hypothesis 2b, but in the opposite direction predicted. However, there was significant direct effect for trait negative affect on CV recovery time ( $\beta = -.29, p = .04$ ), accounting for 100% of the total effect observed. Furthermore, trait negative affect was predicted to have an indirect effect on CV recovery via state negative affect (hypothesis 2d; see paths a + g in Figure 4); however, the indirect effect was calculated to be weak and negative ( $\beta = .00, p = .57$ ), accounting for none of the total effect (90% CIs  $[-.05, .01]$ ). Overall, each one standard deviation increase in trait negative affect above the mean was significantly associated with just over one-quarter of a standard deviation change in the mean recovery time following the self-anger writing task.

The total effect for trait anger rumination to predict CV recovery time (hypothesis 2c) was not significant ( $\beta = .02, p = .99$ ). Furthermore, there was no support for hypotheses 2e or 2f, which predicted that trait anger rumination would indirectly influence CV recovery time by two pathways: through state anger rumination only (hypothesis 2e; paths f + i in Figure 4) and through state anger rumination and state negative affect (hypothesis 2f; paths f + g + h in Figure 4).

The indirect effects of the state variables on CV recovery time were negative, unlike the direct effect of trait anger rumination on CV recovery time, suggesting a case of inconsistent mediation (i.e., suppression) as seen in the previously tested models. Furthermore, there was a significant direct effect of trait anger rumination on state anger rumination ( $\beta = .30, p = .04$ ), although the effect that was present indicated that higher trait anger rumination was associated with higher state anger rumination. While not a specific

hypothesis tested in the current study, it was noted that trait anger rumination significantly influenced state negative affect indirectly via state anger rumination ( $\beta = .17$ ,  $p = .03$ ; 90% CIs [.05- .29]). Thus, a tendency to ruminate about one's anger after it has passed was mediated by actually ruminating after the self-anger recall task and led to increased state negative affect.

State negative affect showed a weak negative effect on CV recovery time ( $\beta = -.03$ ,  $p = .85$ ), which was not in the direction predicted by hypothesis 3a. This weak relationship indicated that state negative affect had essentially no relationship with CV recovery time in the current model. State anger rumination also showed a weak negative effect on CV recovery time ( $\beta = -.01$ ,  $p = .94$ ), again in the opposite direction predicted by hypothesis 3b. Neither the direct ( $\beta = .01$ ,  $p = .99$ ) or indirect ( $\beta = -.02$ ,  $p = .84$ ; hypothesis 3d) effect via state negative affect were significant. Finally, hypothesis 3c, which predicted that state anger rumination would show a significant positive association with state negative affect, was supported by the significant direct association observed ( $\beta = .58$ ,  $p < .001$ ).

To summarize, the predictors in the proposed model only explained 11% of the variance in CV recovery time. Unlike MAP reactivity, HR reactivity was not a significant direct predictor of CV recovery time, although it showed a trend in the predicted direction. Trait negative affect was a significant direct predictor of CV recovery time, but in the opposite direction predicted. Also, trait anger rumination was a significant direct predictor of CV recovery time, which was not observed in the MAP self-anger model. Trait anger rumination was a significant indirect predictor of state negative affect through state anger rumination. No significant indirect effects were found for predicting



CV recovery time. Finally, state variables did not have a significant direct effect on CV recovery time.

### 3.3 Comparison of self-anger and other-anger models

Comparisons were made between the self-anger and other-anger models to gauge how well the models explained CV recovery time by comparing the overall fit of the model using confidence intervals associated with the RMSEA model fit indices. For the RMSEA confidence intervals, a smaller confidence interval was indicative of a better-fitting overall model. Next, using the 90% biased-corrected confidence intervals that were calculated from the bootstrapping technique previously described, comparisons were also made for the direct and indirect path coefficients for the models (i.e., those shown in Tables 4 through 7). Comparisons were based on two characteristics of the confidence intervals. First, whether the confidence interval includes zero was noted for each model. If zero is included in the range of the confidence intervals, this implies that the observed standardized path coefficients do not explain a significant portion of variance in the criterion. Second, comparisons were based on whether the 90% biased-corrected confidence intervals between the two models overlapped with one another. If the models' confidence intervals overlapped, then the self-anger and other-anger models were assumed to account for variability in the criterion variable being compared in a similar manner.

#### 3.3.1 Models including mean arterial pressure as a predictor

First, self-anger and other-anger models that used MAP reactivity as a predictor in the models were compared. As previously discussed, the other-anger RMSEA value equaled zero (90% CIs [.00-.19]), as did the self-anger RMSEA (90% CIs [.00-.13]).

According to the cut-off criteria of Hu and Bentler (1999), an RMSEA value less than or equal to .06 indicates a good fit of the hypothesized model and the observed data. Thus, both the self-anger and other-anger models fit the current data well. The range of confidence interval values was slightly smaller for the self-anger model compared to the other-anger model, however, which indicated that the proposed model may have been better for predicting CV recovery time following the self-anger writing task relative to following the other-anger writing task in the current sample.

Next, the confidence intervals associated with the direct and indirect paths for the self-anger and other-anger models that included MAP reactivity as predictors were compared based on the two criteria previously described. The values that were compared can be seen in Tables 4 and 6 for other-anger and self-anger with MAP reactivity included as a predictor, respectively. Confidence intervals that included zero for both the self-anger and other-anger paths were not discussed, since the inclusion of zero indicated the models were not accounting for a significant portion of the variance in the criterion of interest.

The present data produced overlapping confidence intervals for the following direct/total paths for both models: CV recovery time regressed onto trait negative affect, CV recovery time regressed onto MAP reactivity, state negative affect regressed onto state anger rumination, and state anger rumination regressed onto trait anger rumination. The indirect path between state negative affect and trait anger rumination also had overlapping confidence intervals for self-anger and other-anger tasks. Therefore, the aforementioned direct and indirect paths, while accounting for a significant proportion of

the variance in their respective criterion variables, did not differ among the two models, indicating approximately equal effects for the self-anger and other-anger models.

The next comparisons were made between the analogous paths in which only either self-anger or other-anger produced confidence intervals that did not include zero. The direct path in which trait anger rumination predicted CV recovery time contained zero for self-anger (90% CIs [-.20-.30]) but did not for other-anger (90% CIs [.05-.43]). A similar pattern was observed for the total effects between the two models for trait anger rumination and CV recovery time. Thus, in the tested model, trait anger rumination accounted for a significant proportion of the variance in CV recovery time following the other-anger writing task for the present sample, but this was not the case following the self-anger writing task.

The confidence intervals for the direct and total effects for state negative affect regressed onto trait negative affect also differed for self-anger (90% CIs [-.06-.26]) and other-anger (90% CIs [.08-.42]). This difference indicated that trait negative affect predicted state negative affect following the other-anger writing task but did not predict state negative affect following the self-anger writing task. A similar pattern was also seen for the path in which state anger rumination was regressed onto MAP reactivity, such that the confidence interval for the self-anger model contained zero, but the other-anger model did not (90% CIs [.13-.50]). Thus, MAP reactivity during the other-anger writing task significantly predicted anger rumination following the other-anger writing task, but the pattern for MAP reactivity during the self-anger writing task to influence state anger rumination during recovery was not observed.

The indirect path between MAP reactivity and state negative affect via state anger rumination also showed differences in 90% confidence intervals between the two models. For self-anger, the confidence intervals included zero, 90% CIs [-.09-.16], whereas the other-anger model's confidence intervals did not, 90% CIs [.03-.23]. This difference indicated that the amount of physiological arousal during the other-anger writing task was associated with greater state negative affect when participants also engaged in anger rumination following the writing task. This indirect effect was not significant for the self-anger writing task.

Overall, the comparisons between these two models that included MAP reactivity as a predictor indicated that the other-anger model was better for accounting for the influence of trait anger rumination on CV recovery time, trait negative affect on state negative affect, MAP reactivity onto state anger rumination, and MAP reactivity's indirect influence on state negative affect (via state anger rumination). Furthermore, several paths shared 90% confidence intervals, such as trait negative affect's direct influence on CV recovery time, MAP reactivity's direct influence on CV recovery time, trait anger rumination's direct influence on state anger rumination, and state anger rumination's direct influence on state negative affect, and the indirect influence of trait anger rumination on state negative affect (via state anger rumination).

### 3.3.2 Models including heart rate as a predictor

First, self-anger and other-anger models that used HR reactivity as a predictor in the models were compared. As previously discussed, the other-anger RMSEA value equaled .12 (90% CIs [.00-.26]), while the self-anger RMSEA was .00 (90% CIs [.00-.15]). According to the cut-off criteria of Hu and Bentler (1999), an RMSEA value less

than or equal to .06 indicates a good fit of the hypothesized model and the observed data. Thus, only the self-anger models fit the current data well when HR reactivity was included as a predictor. As seen in the models including MAP reactivity, it appeared the proposed model may have been better for predicting CV recovery time following the self-anger writing task relative to following the other-anger writing task in the current sample.

Next, the confidence intervals associated with the direct and indirect paths for the self-anger and other-anger models with HR reactivity as a predictor were compared based on the two criteria previously described. The values that were compared can be seen in Tables 5 and 7 for other-anger and self-anger with HR reactivity included as a predictor, respectively. As for the MAP reactivity models, confidence intervals that included zero for both the self-anger and other-anger paths were not discussed, since the inclusion of zero indicated the models were not accounting for a significant portion of the variance in the criterion of interest.

The present data produced overlapping confidence intervals for the following direct/total paths for both models: CV recovery time regressed onto trait negative affect, state negative affect regressed onto state anger rumination and state anger rumination regressed onto trait anger rumination. Therefore, the aforementioned direct paths, while accounting for a significant proportion of the variance in their respective criterion variables, did not differ among the two models, indicating approximately equal effects for the self-anger and other-anger models. Fewer confidence intervals for the path coefficients in the HR reactivity models overlapped relative to the MAP reactivity models previously discussed. Of these, only one path (the direct path between HR reactivity and

CV recovery time) included zero for both models, indicating that HR reactivity did not significantly predict CV recovery time.

The next comparisons were made between the paths in which only either self-anger or other-anger produced confidence intervals that did not include zero while the remaining model did. The direct path for CV recovery time regressed onto HR reactivity did not contain zero for the self-anger model, 90% CIs [.04-.40], but did for the other-anger model, 90% CIs [-.09-.39]. This indicated that HR reactivity during the self-anger writing task was a significant predictor of CV recovery time following the self-anger writing task, but the analogous association for the other-anger model was not observed.

For the direct path between trait anger rumination and CV recovery time, only the other-anger model had a 90% confidence interval that did not contain zero, 90% CIs [.01-.38]. Thus, for the current data, a tendency to ruminate about anger after it has passed was associated with a longer recovery time following the other-anger writing task, but this effect was not significant for the self-anger writing task. Another case where the other-anger model better accounted for an observed association was for the direct path of trait negative affect onto state negative affect, 90% CIs [.08-.41], whereas the analogous path for the self-anger model included zero, 90% CIs [-.06-.26]. Thus, trait negative affect was a significant predictor of state negative affect, but only for the other-anger writing task.

The direct path confidence intervals for trait anger rumination to predict state anger rumination in the self-anger model did not contain zero, 90% CIs [.07-.47], but the other-anger model did contain zero, 90% CIs [-.01-.38]. Thus, trait anger rumination significantly predicted state anger rumination following the self-anger writing task, but the analogous path did not reach statistical significance for the other-anger writing task.

Another advantage for the self-anger model was observed for the indirect influence of trait anger rumination on state negative affect (via state anger rumination), 90% CIs [.05-.29]. The analogous path for the other-anger model contained zero, 90% CIs [.00-.20], although there was a trend for a significant indirect effect in this model. The indirect effect indicated that lower trait anger rumination was associated with lower negative affect ratings when participants reported lower negative state rumination following the self-anger writing task.

In summary, the models in which HR reactivity were included as a predictor were equal in the ability to account for the direct influence of the following paths: between trait negative affect and CV recovery time and between state anger rumination and state negative affect. The self-anger model was better than the other-anger model at accounting for the following paths: the total effect of HR reactivity on CV recovery time, the direct effect of trait anger rumination on state anger rumination, and the indirect path in which trait anger rumination influenced state negative affect via state anger rumination. The other-anger model was better than the self-anger model at accounting for the following paths: the total effect of trait anger rumination on CV recovery time and the direct effect of trait negative affect on state negative affect.

When comparing the overall trends noted between MAP reactivity and HR reactivity models, there appears to be less overlap in the HR reactivity models of self-anger and other-anger. Furthermore, for MAP reactivity models, the other-anger model always showed advantage over the self-anger model when there was not overlap in confidence intervals. This was not always the case for the HR reactivity models, in which self-anger showed advantage for three of the five non-overlapping confidence intervals.

Thus, it appears that self-anger may have a greater influence on variables in the tested model when considering HR reactivity, whereas other-anger may have a greater influence on variables in the tested model when considering MAP reactivity.



## CHAPTER 4: DISCUSSION

This study examined the influence of trait and state levels of negative affect and anger rumination on the amount of time it took for CV responses to return to resting levels following the written recall of two anger events: one in which participants were angry with someone else (other-anger) and another in which participants were angry with themselves (self-anger). This was the first study to examine the affective, cognitive, and physiological aspects of self-anger and compare them to other-anger variables. Furthermore, this was the first study to attempt to directly test the rumination-arousal model proposed by Gerin and colleagues (2006) using path analysis. By examining whether similar responses arise when the target of one's anger is self-focused or other-focused, a better understanding of the mechanisms that prolong CV responses following anger can be gained. Furthermore, examining differences among cognitive and affective responses among self-anger and other-anger can inform future research to understand factors that underlie self-blame and any associated health consequences.

The overall findings of the current study indicated that the anger recall tasks used in the present study were sufficient to produce significant changes in MAP and HR and self-reported negative affect relative to baseline. Furthermore, these overall changes were similar for both self-anger and other-anger recall tasks, which confirms that self-anger was commonly experienced by the present sample and that this self-anger results in

similar CV and affective changes commonly reported in previous studies and observed in the present study. An elaboration of the specific findings from this study is provided below in subsequent sections.

#### 4.1 Comparison of overall model fit for self-anger and other-anger

The first hypothesis included a prediction that the proposed model that included trait and state variables would similarly predict CV recovery time for both self-anger and other-anger models. A total of four models were tested: self-anger (1) and other-anger (2) in which MAP reactivity was used as a predictor and self-anger (3) and other-anger (4) in which HR reactivity was used as a predictor. For the models in which MAP reactivity was included as a predictor, both self-anger and other-anger models had similar model fit statistics, indicating that the proposed model fit the current data well for both self-anger and other-anger recall tasks.

It is important to note, however, that the predictors in the current model only accounted for 14% and 25% of the variance in CV recovery time for self-anger and other-anger, respectively. For the remaining models in which HR reactivity was included as a predictor, only the self-anger model produced adequate fit statistics for the current sample, accounting for 11% of the variance in CV recovery time. While the other-anger model did not fit the data well, the predictors accounted for 15% of the variance in CV recovery time. Overall, the low amount of variance accounted for in CV recovery time for all of the models tested indicated that modifications to the proposed model are warranted. Some potential explanations and recommendations will be discussed in the following sections.

#### 4.2 Comparisons of analogous paths in self-anger and other-anger models

Hypothesis 2a posited that CV reactivity measures (MAP and HR) would be associated with longer CV recovery time. This hypothesis was partially supported for the analogous direct effects among the four models tested. While MAP reactivity for the self-anger and other-anger models significantly predicted CV recovery time in the predicted direction, the predicted associations did not reach statistical significance for HR reactivity in either the self-anger or other-anger models. These findings indicated that both the self-anger and other-anger resulted in significant increases in MAP, with higher MAP reactivity being predictive of longer CV recovery time. Thus, the self-anger recall task used in the present study was successful in producing MAP reactivity at a comparable level to that observed in the other-anger writing task, and the reactivity during both tasks was predictive of the CV recovery time following each task. It is important to note that most studies that have examined the influence of anger rumination on CV recovery also only found effects for BP recovery and not HR recovery (e.g., Gerin et al., 2006; Glynn et al., 2002). Overall, these findings suggested that examining CV reactivity and recovery time is important in the study of self-anger, as has been reported for other-anger in previous studies (Anderson et al., 2005).

Hypothesis 2b predicted that higher levels of trait negative affect would be directly associated with longer CV recovery time (path b in Figure 4). Significant associations were found for the four models tested, but in the opposite direction of what was predicted. In other words, for the current sample, higher levels of trait negative affect were associated with a faster CV recovery time. These findings were surprising since they do not align with previous research that has implicated trait negative affect as

detrimental to stress-related health outcomes (e.g., Mayne, 1999; Polk, Cohen, Doyle, Skoner, & Kirschbaum, 2005). However, some empirical studies have failed to find relationships between NA and objective measures of health and health-related behaviors (e.g., Costa & McCrae, 1985, 1988; Watson & Pennebaker, 1989).

Furthermore, most studies that have examined CV outcomes associated with trait negative affect have focused on CV reactivity, so it is unclear whether the negative association observed between trait negative affect and CV recovery was specific to this sample or actually reflects the relationship among these variables. Thus, while trait negative affect may be associated with having heightened CV reactivity during the experience of a negative emotion (such as the anger-recall tasks employed in the current study), it may not actually prolong the CV responses after the immediate emotional stressor has passed. While this explanation is merely speculation, the current results indicate trait negative affect may serve an adaptive function when considering CV recovery time.

While future research is warranted to examine the relationship among trait negative affect and CV recovery, there are some possible explanations for the observed findings. First, participants in the present study did not report very high levels of negative affect on the PANAS, so the association that was noted should be interpreted with caution. Second, endorsing higher negative affect implies subjective distress and unpleasurable engagement while low negative affect implies the absence of these experiences (Watson & Clark, 1984). Thus, taking the opposite perspective of the aforementioned interpretation of the association between negative affect and CV recovery time, lower trait negative affect was associated with longer CV recovery time, indicating

prolonged autonomic activation relative to those with higher endorsement of trait negative affect. Asking participants that are low in trait negative affect to recall an angry experience they have had may have disrupted their normally low levels of distress and higher levels of calmness (Pettit, Kline, Gencoz, Gencoz, & Joiner, 2001), which may have accounted for the observed changes in both CV reactivity and an increased CV recovery time.

Another possible explanation includes the role of trait positive affect in influencing CV responses. Like much of the emotion literature, the models tested in the current study focused specifically on the role of negative affect in CV responses. However, the increased focus on factors that are protective and promote health, including positive affect, have led researchers to focus on the beneficial aspects of both positive and negative affect. For example, high levels of trait positive affect are positively related to learning, creativity, problem solving, relationship formation (e.g., Carnevale & Isen, 1986; Isen, 1987; Isen, Daubman, & Norwicki, 1987), and self-reported health (Pettit et al., 2001).

Although not tested in the current study, participants' levels of trait positive affect may have influenced CV recovery time. For example, the undoing hypothesis proposes that positive emotions may serve to restore homeostasis following negative emotional experiences (Frederickson & Levenson, 1998), thereby speeding CV recovery time. A negative association between negative affect and CV responses was also recently reported by Dowd, Zautra, and Hogan (2010), in which participants reporting higher negative affect before undergoing a speech task (a social stressor) actually showed improved CV recovery measures compared to participants with lower negative affect

before the task. Furthermore, they also found that those endorsing higher positive affect before the stressor also had better CV recovery, indicating that positive affect may provide a protective effect on CV health. It is recommended that future studies that examine the role of trait negative affect on CV recovery time also include a measure of trait positive affect to examine the influence of both positive and negative affect on CV responses.

Hypothesis 2c predicted a significant association between trait anger rumination and CV recovery time, such that higher trait negative association would be associated with longer CV recovery time (path e in Figure 4). There was no support for the two models with CV recovery time following the self-anger writing task. However, both models for other-anger showed significant direct effects for trait anger rumination on CV recovery time in the predicted direction. Thus, based on the present sample, it appears that trait anger rumination only influences CV recovery following the other-anger recall and not the self-anger recall, which is in line with previous research with anger and CV responses (e.g., Gerin et al., 2006; Glynn, Christenfeld, & Gerin, 2002). While most questions on the Anger Rumination Scale (Sukhodolsky, Golub, & Cromwell, 2001; also see Appendix A) are neutral in the target of one's anger, there are several questions that do specifically focus on other-anger (e.g., "I have difficulty forgiving people who have hurt me;" "When someone makes me angry, I can't stop thinking about how to get back at that person."), which may account the non-significant findings in the self-anger models. It is recommended for future studies examining anger rumination in self-anger to include a measure of anger rumination that excludes language that implies someone else was the cause of the anger.

Hypothesis 2d predicted a significant indirect effect of trait negative affect on CV recovery time through state negative affect (paths a+ h in Figure 4). For the four models, none of the indirect effects for this path were significant. The direct effect of trait negative affect on state negative affect (path a in Figure 4) were only significant for other-anger models, indicating that trait negative affect did not significantly predict state negative affect following the self-anger writing task. Furthermore, hypothesis 3a which predicted state negative affect would be significantly associated with CV recovery time, was not supported for any of the four models tested. Repeated-measures t-tests revealed that state negative affect did increase significantly from baseline for both the self-anger and other-anger writing tasks. Interestingly, this effect was actually greater for self-anger, which resulted in significantly higher state negative affect than the other-anger writing task.

Based on the current sample, it appears that state negative affect does not mediate the relationship observed with trait negative affect on CV recovery time, nor does it directly influence CV recovery time on its own. While the VAS scales used in the present study have been used in pain research (Jensen et al., 1986) and in emotion research (e.g., Key et al., 2008), future studies might explore using the negative affect subscale of the PANAS as a measure of state negative affect since it has demonstrated good reliability (Watson et al., 1988). Further testing of VAS ratings for measures of state affect is warranted. As previously mentioned in discussing the results of trait negative affect, it is also recommended to examine the role of state positive affect in the affect-CV recovery time association.

Hypothesis 2e predicted a significant indirect effect of trait anger rumination on CV recovery time through state anger rumination (paths  $f + i$  in Figure 4). There was no support for this hypothesis for any of the models tested. Similarly, there was no support for hypothesis 2f, which predicted a significant indirect effect of trait anger rumination on CV recovery time through both state anger rumination and state negative affect (paths  $f + g + h$  in Figure 4). Thus, the only effect of trait anger rumination on CV recovery time was the direct effect observed for other-anger that was previously discussed.

Trait anger rumination did significantly predict state anger rumination for all models except for the other-anger model that included HR reactivity. Hypothesis 3b, which predicted that state anger rumination would significantly predict CV recovery time was not supported. Not surprisingly, hypothesis 3d (paths  $g + h$ ), which predicted an indirect effect of state anger rumination on CV recovery time via state negative affect was also not supported. The state anger rumination measure was a VAS rating in which participants reported how much of the recovery period following each anger event they had spent thinking about the event they had just written about. Thus, a limitation of using this measure of state rumination was that it could not account for whether participants were actually ruminating or if they were reappraising the event they wrote about, which is defined as the process of reinterpreting the meaning of an upsetting event (Ray, Wilhelm, & Gross, 2008).

Reappraisal of emotional events has been associated with reduced anger and faster CV recovery (Ray et al., 2008), and the finding that the state anger rumination rating used in the present study showed a strong positive association with state negative affect (hypothesis 3c; path  $g$  in Figure 4) indirectly implied that participants were



ruminating and not reappraising their anger events. Future studies examining state anger rumination are recommended to ask participants what they were thinking about during the recovery period (i.e., thought sampling) in addition to assessing anger rumination to ensure the observed findings were actually due to rumination.

As previously mentioned, hypothesis 3c was supported, indicating state anger rumination was positively associated state negative affect (path g in Figure 4). This finding suggested that when participants were engaging in anger rumination during the recovery period following the self-anger and other-anger writing tasks, this rumination also predicted higher state negative affect. Thus, the precipitating effect of state anger rumination that was proposed in the rumination-arousal model (Gerin et al., 2006) was partially supported. It is unclear why the state negative affect and state anger rumination measures utilized in the present study did not significantly predict CV recovery time.

Some possible explanations of the lack of an association among state variables and CV recovery time include measurement issues, such as the state measures used and the use of CV recovery time as the assessment of CV recovery. Christenfeld, Glynn, and Gerin (2000) compared several techniques for assessing CV recovery and found that a curve-fitting technique provides the most reliable assessment of CV recovery. While the current study was not designed to have a long enough recovery period nor was the BP machine used designed to give continuous assessment of CV responses (both of which are necessary to test CV recovery with a curve-fitting technique), post-hoc models were tested using an excursion equation as described by Neumann and colleagues (2004). No significant differences in path estimates were found by using this technique for the

current sample's data. Therefore, a similar model that was proposed for this study should be tested using curve-fitting techniques of CV recovery in future studies.

#### 4.3 Limitations

There were several limitations in this study that may have contributed to the failure to support many of the proposed hypotheses. One limitation of this study is the small sample size. A typical sample size in studies using structural equation modeling techniques such as path analysis is 200, and models with less than 100 participants may be unsound (Kline, 2011; p. 12). A related limitation arises from the use of path analysis in the present study. Path analysis is used to test hypothesized causal pathways between observed variables and is typically used based on theory. While the proposed model was based on the rumination-arousal model proposed by Gerin and colleagues (2006), the proposed model was still quite exploratory in nature. Thus, the findings of the present study should be interpreted with caution.

As previously mentioned, the state measures of negative affect and anger rumination were not associated with CV recovery time. While the state variables were strongly associated with one another, the lack of association with CV recovery time was contradictory to the rumination-arousal model and the models tested in this study. This lack of finding significant association may be associated with the use of VAS ratings for state anger rumination and state negative affect, or the use of CV recovery time to assess the duration of autonomic activation, or a combination of both. The small range of CV recovery time also could contribute to the limited findings in the present study. Participants were young and healthy, and all possible factors that might have contributed to longer recovery time, such as family history of hypertension or CV disease, were

controlled for statistically. Furthermore, physical activity has also been shown to contribute to faster CV recovery (e.g., Glynn et al., 2002), but was not assessed in the current study.

Other limitations associated with the current study include the use of a writing technique as an emotion induction technique. There were two reasons that writing was used rather than speaking about angry experiences. First, the instructions were adapted from Ellsworth and Tong's (2006) study in which self-anger and other-anger were compared. Because self-anger has not been examined otherwise in the literature, it seemed reasonable to include the same instructions to add to the findings of Ellsworth and Tong (2006). Second, self-anger does not have the same criterion of having a social component as that typically seen with other-anger; thus, the writing task was chosen in order to examine self-anger without a social component. By allowing participants to choose their own events, there was a great amount of variability in the types of events participants recalled. Furthermore, there was not a measure of whether participants had forgiven the person that made them angry for the other-anger writing task, or themselves for the self-anger writing task. Self-forgiveness has been studied in the context of anger and anger rumination, such that individuals with an inability to forgive themselves are more likely to ruminate about events and do so in a self-focused way (Barber, Maltby, & Macaskill, 2005). Furthermore, having the trait to forgive was associated with lower hostility ratings, higher positive affect ratings, and faster CV recovery after verbal harassment (Whited, Wheat, & Larkin, 2010). Thus, whether forgiveness or resolution of an anger event should be examined in future studies of anger rumination and CV responses.

One aspect of the writing task that was not considered when designing the present study was the notion that expressive writing can actually have a beneficial impact on health outcomes (e.g., Baikié & Wilhelm, 2005). Expressive writing, in which participants are asked to relive a personal stressor by writing about the event and their feelings surrounding the event, typically results in increased negative affect immediately following the writing task, but has been shown to have many long-term benefits for both clinical (e.g., Frisina, Borod, & Lepore, 2004) and non-clinical (e.g., Smyth, 1998) populations. The effect of expressive writing on CV responses during and immediately following the writing has not been examined to date. Thus, it remains unclear if the task that was chosen for the present study could have been responsible for the relatively quick CV recovery time observed among all participants. Furthermore, expressive writing has been demonstrated as a potential intervention for maladaptive ruminators to confront their negative thoughts and feelings, reappraise the events, and provide an opportunity for constructive problem solving (Sloan, Marz, Epstein, & Dobbs, 2008). It is highly recommended that other types of anger induction techniques for both self-anger and other-anger be tested and compared to the writing tasks used for the current study to examine whether expressive writing can have immediate CV benefits.

#### 4.4 Contributions and future directions

This was the first study to examine CV responses associated with self-anger. This study confirmed Ellsworth and Tong's (2006) finding that self-anger does result in different state affective ratings from those observed in after the other-anger writing task. For example, self-anger resulted in significantly greater self-reported guilt, shame, regret, and embarrassment ( $ps < .001$ ) compared to other-anger ratings of the same emotions.

Self-anger did result in significantly lower anger ratings relative to other-anger ( $p < .001$ ), but both self-anger and other-anger resulted in similar self-reported sadness ( $p = .18$ ). Self-anger resulted in significant increases in MAP and HR relative to baseline, as did other-anger. The construct of self-anger demonstrated some differences in associated emotions from other-anger, distinguishing it from other-anger. However, based on the findings in the present study that suggest self-anger results in significant increases in MAP, HR, and state negative affect ratings, it is evident that the study of self-anger is warranted, as self-anger may influence CV health like those observed in other-anger studies.

This study was also the first to examine the role of trait and state anger rumination and trait and state affect to predict CV recovery time using path analysis. Path analysis allows for the simultaneous testing of multiple predicted associations, such that paths can be interpreted as the effect of the predictor on the criterion after controlling for the effect of all other included variables. By having similar writing tasks for the self-anger and other-anger condition, comparisons of the analogous paths in the predicted model could be made. The main finding for the present sample demonstrated that trait anger rumination was predictive of CV recovery time following the other-anger writing tasks. Also, trait negative affect was significantly and negatively associated with CV recovery time for both self-anger and other-anger. While the sample size was small for the present study, these findings do indicate that there is some utility to the models tested.

Overall, despite many non-significant effects and methodological issues for the current study, it appears self-anger is commonly experienced and is associated with similar CV responses to those seen in other-anger. Anger is a complex emotion with a well-established link to CV health. Because the present sample included young, healthy

college students, it may be that these effects were not readily apparent in this sample. The findings from this study call for further study of the developmental trajectory of anger's effects on CV variables. Therefore, future studies are recommended to examine self-anger in a variety of populations. Furthermore, it is well established that a history of depression is associated with poor CV outcomes, and the relationship among different types of rumination in self-anger and depression should be considered in future research.

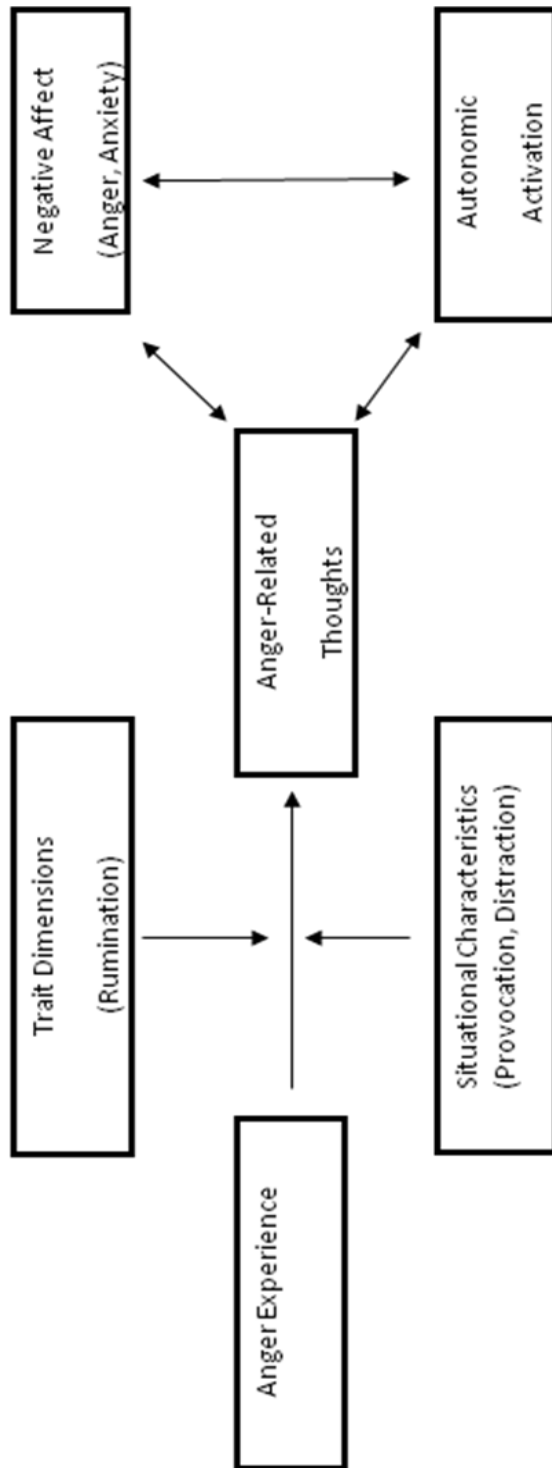


Figure 1. The rumination-arousal model (adapted from Gerin et al., 2006).

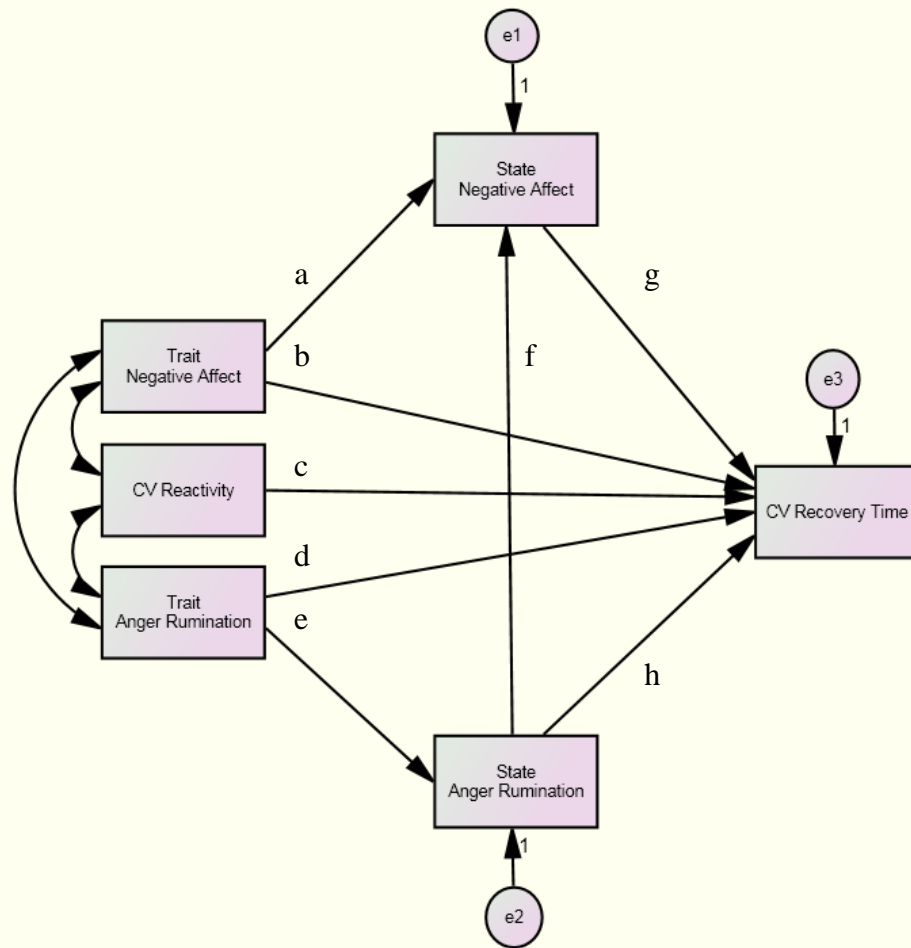


Figure 2. Diagram of hypothesized relations (all paths predicted to be positive).  
*Note:* e's indicate error measurement.



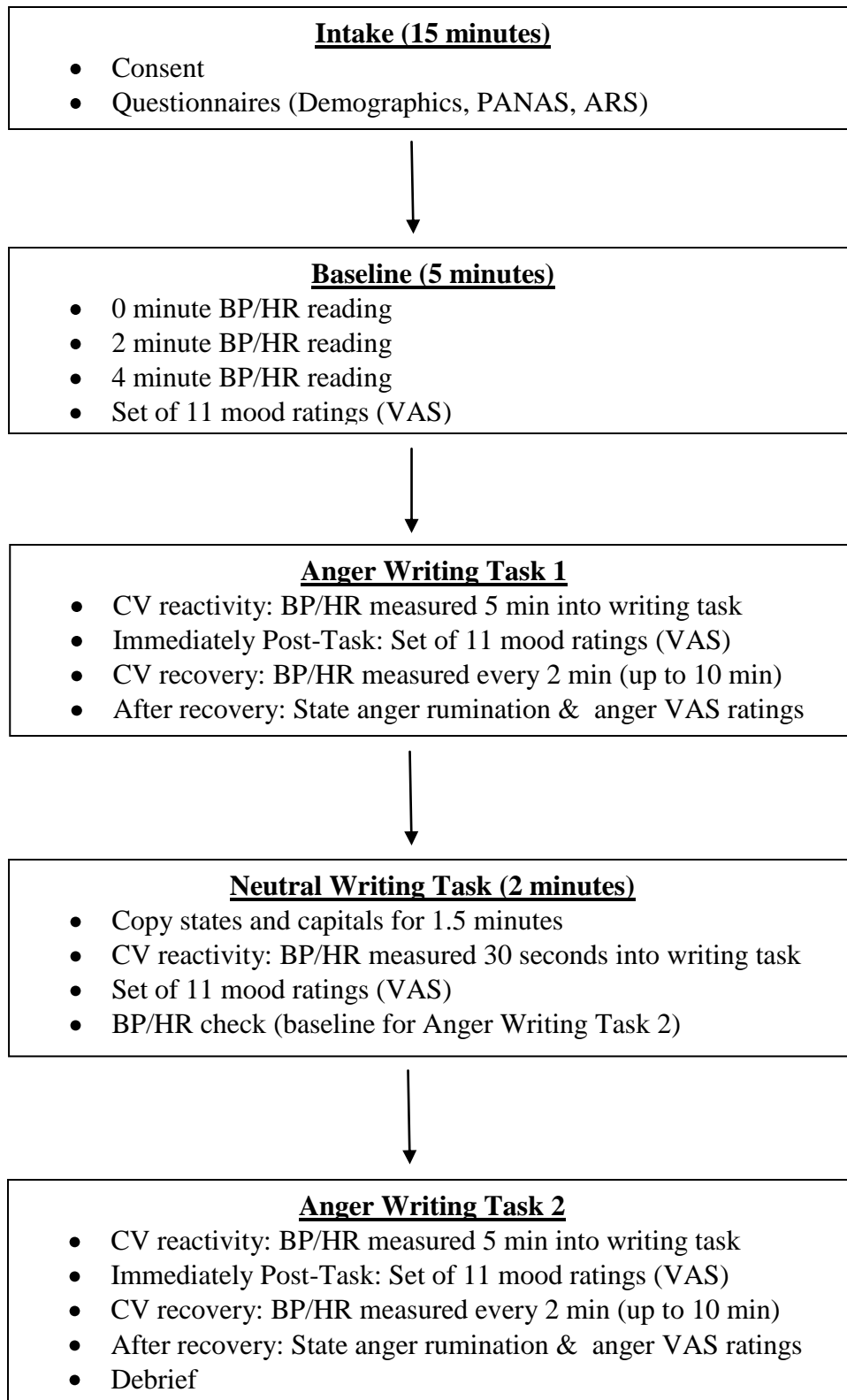


Figure 3. Flowchart of procedures for current study

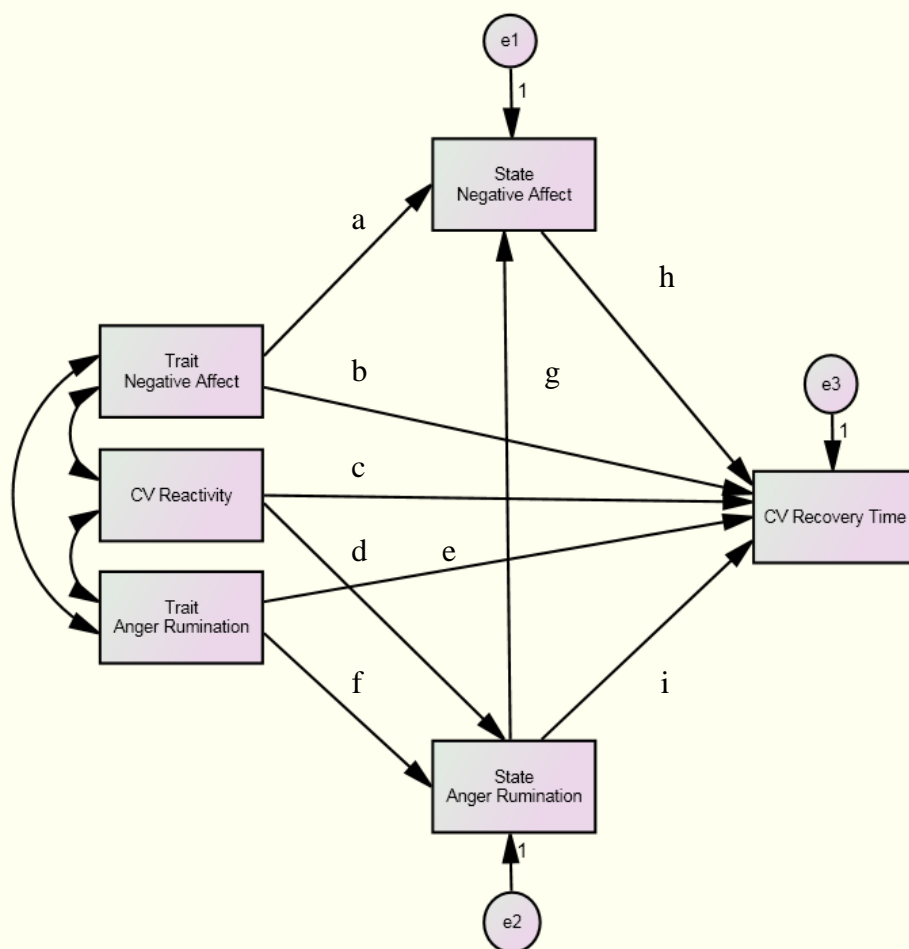


Figure 4. Model to be tested after adding path modification between reactivity and state anger rumination.

*Note:* e<sub>i</sub>s indicate error measurement.

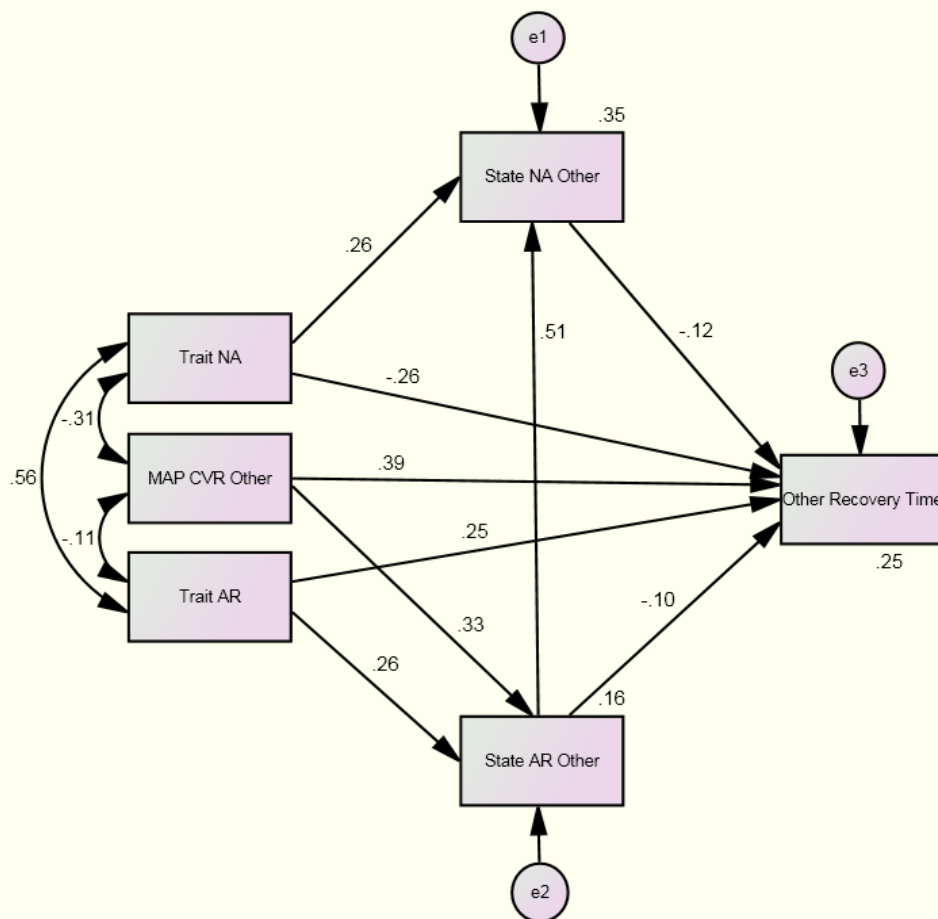


Figure 5. Standardized path coefficients for state and trait predictors with MAP reactivity of CV recovery time for other-anger (with modification).

*Note:* e<sub>i</sub>s indicate error measurement.

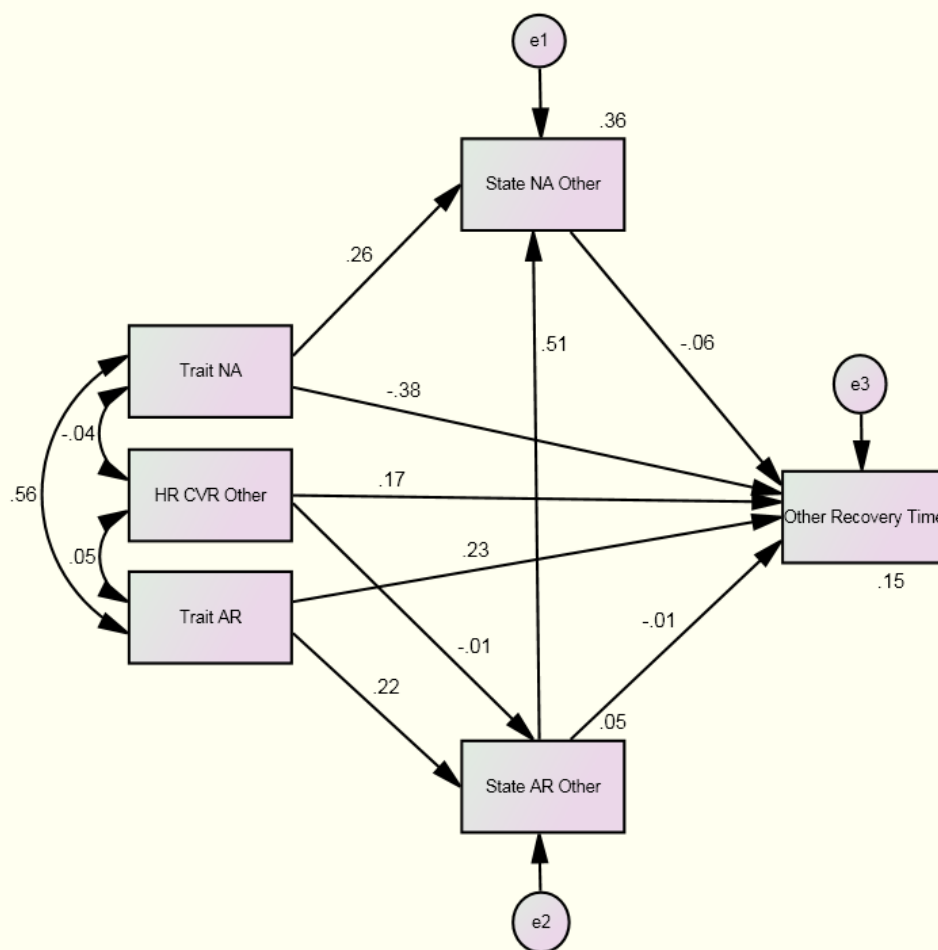


Figure 6. Standardized path coefficients for state and trait predictors with HR reactivity of CV recovery time for other-anger (with modification).

*Note:* e<sub>i</sub>s indicate error measurement.

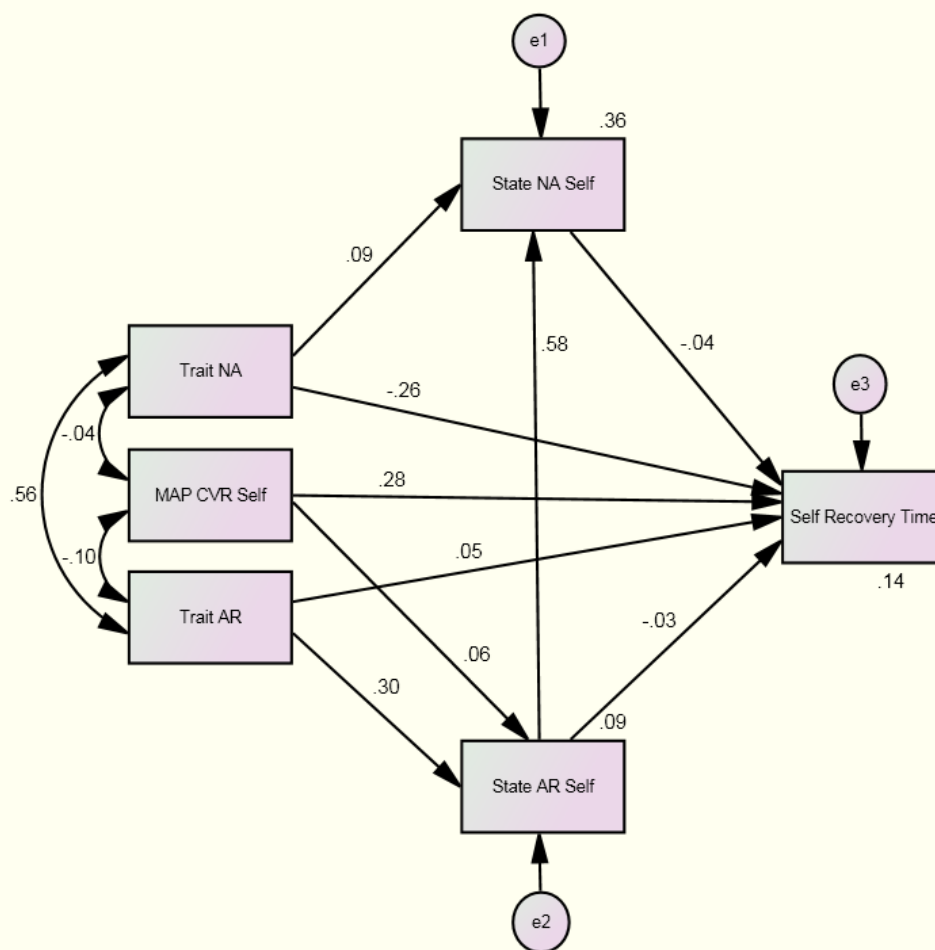


Figure 7. Standardized path coefficients for state and trait predictors with MAP reactivity of CV recovery time for self-anger (with modification).

*Note:* e<sub>i</sub>s indicate error measurement.

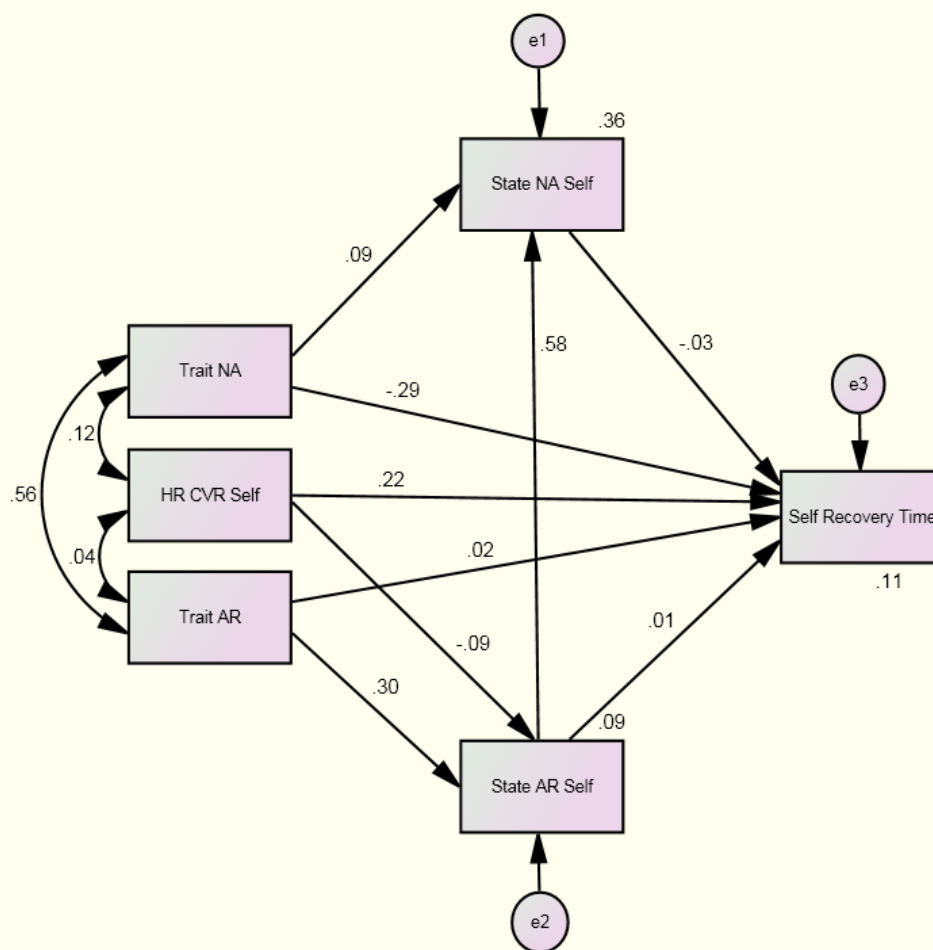


Figure 8. Standardized path coefficients for state and trait predictors with HR reactivity of CV recovery time for self-anger (with modification). *Note:*  $e_i$ s indicate error measurement.

Table 1. Mean (standard deviation) state negative affect and CV responses across tasks

Variables	Task			Self-anger		Other-Anger	
	Baseline	Self-Anger	Recovery	Recovery	Other-Anger	Recovery	Neutral
MAP	82.30 (8.51)	86.96 (9.23)**	83.10 (7.43)	86.83 (9.12)**	72.21 (11.23)**	82.55 (8.90)	
HR	74.32 (11.73)	79.21 (12.04)**	72.11 (10.07)*	79.00 (11.55)**	83.26 (7.65)**	74.04 (11.01)	
State anger	0.98 (1.22)	3.75 (2.56)**	2.90 (2.22)**	4.82 (2.69)**	3.60 (2.49)**	1.71 (2.16)	

Note:  $N=75$ . Significant Bonferroni –corrected pair-wise comparisons between baseline and all other tasks are flagged such that \*indicates  $p<.01$  and \*\*indicates  $p<.001$ .

Table 2. Descriptive statistics and zero-order correlations for other-anger variables

Variables	<i>M</i>	<i>SD</i>	1	2	3	4	5	6
1. Trait NA	18.68	5.72						
2. Trait AR	34.08	10.53	.56***					
3. State NA	3.41	1.53	.25*	.17				
4. State AR	5.40	2.93	-.03	.22*	.51***			
5. Recovery Time (residual)	0.00	1.79	-.27*	.02	-.15	.02		
6. MAP Reactivity	4.53	8.11	-.31**	-.11	.11	.30**	.40**	
7. HR Reactivity	4.68	9.09	.04	.06	-.15	.01	.26*	.21

Note: *N*= 75. \*indicates  $p<.05$ , \*\* indicates  $p<.01$ , and \*\*\* indicates  $p<.001$ . NA stands for negative affect; AR stands for anger rumination, MAP stands for mean arterial pressure, and HR stands for heart rate.



Table 3. Descriptive statistics and zero-order correlations for self-anger variables

Variables	<i>M</i>	<i>SD</i>	1	2	3	4	5	6
1. Trait NA	18.68	5.72						
2. Trait AR	34.08	10.53	.56***					
3. State NA	3.81	1.80	.22*	.16				
4. State AR	4.69	2.93	.23*	.30**	.60***			
5. Recovery Time (residual)	0.00	1.57	-.25*	-.13	-.10	-.08		
6. MAP Reactivity	4.66	8.19	-.04	-.10	-.00	.03	.28*	
7. HR Reactivity	4.89	7.60	.12	.04	-.07	-.07	.25*	.19

Note: *N*= 75. \*indicates  $p<.05$ , \*\* indicates  $p<.01$ , and \*\*\* indicates  $p<.001$ . NA stands for negative affect; AR stands for anger rumination, MAP stands for mean arterial pressure, and HR stands for heart rate.

Table 4. Standardized direct, indirect, total, and spurious effects (with 90% confidence intervals) for other-anger MAP reactivity from model

Predictors		State NA	State AR	CV Recovery Time
Effects				
Total $R^2$		.35	.16	.25
Trait NA				
Direct		.26 (.08- .42)	--	-.26 (-.51- -.03)
Indirect		.00	--	-.03 (-.11- .01)
Total		.26	--	-.29 (-.52- -.06)
Spurious		-.01	--	.01
MAP Reactivity				
Direct		.00	.33 (.13-.50)	.39 (.13- .59)
Indirect		.00 (.07- .28)	.00	-.05 (-.13- .00)
Total		.00	.33	.34 (.08- .54)
Spurious		.00	.03	.06
Trait AR				
Direct		.00	.26 (.05-.43)	.24 (.05- .43)
Indirect		.11 (.03- .23)	.00	-.04 (-.12- .00)
Total		.11	.26	.20 (.03- .37)
Spurious		.00	.00	-.18
State NA				
Direct		--	.52 (.38-.63)	-.12 (-.32- .07)
Total		--	.52	-.12
Spurious		--	.00	-.02
State AR				
Direct		--	--	-.10 (-.33- .12)
Indirect		--	--	-.06 (-.18- .03)
Total		--	--	-.16 (-.33- .02)
Spurious		--	--	-.14

Note.  $N=75$ . NA = Negative Affect; AR= Anger Rumination; MAP= mean arterial pressure, CV= cardiovascular. Spurious effect reflects difference between the variable's zero-order effect and its estimated total effect in the model.

Table 5. Standardized direct, indirect, total, and spurious effects (with 90% confidence intervals) for other-anger HR reactivity from model

Predictors	Effects	State NA	State AR	CV Recovery Time
Total $R^2$		.36	.05	.15
Trait NA				
Direct		.26 (.08- .41)	--	-.38 (-.59- -.15)
Indirect		.00	--	-.02 (-.09- .03)
Total		.26	--	-.40 (-.61- -.17)
Spurious		-.01	--	.13
HR Reactivity				
Direct		.00	-.01 (-.19-.20)	.17 (-.11- .40)
Indirect		.00 (-.10- .10)	.00	-.03 (-.02- .03)
Total		.00	-.01	.14 (-.09- .39)
Spurious		.00	.00	.07
Trait AR				
Direct		.00	.22 (-.01-.38)	.23 ( .01- .38)
Indirect		.11 (.00- .20)	.00	-.02 (-.07- .04)
Total		.11	.22	.21 ( .03- .40)
Spurious		.00	.00	-.03
State NA				
Direct		--	.51 (.38-.62)	-.06 (-.27- .16)
Total		--	.51	-.06
Spurious		--	.00	-.20
State AR				
Direct		--	--	-.01 (-.23- .26)
Indirect		--	--	-.03 (-.14- .08)
Total		--	--	-.04 (-.24- .18)
Spurious		--	--	-.02

Note.  $N=75$ . NA = Negative Affect; AR= Anger Rumination; HR= heart rate, CV= cardiovascular. Spurious effect reflects difference between the variable's zero-order effect and its estimated total effect in the model.

Table 6. Standardized direct, indirect, total, and spurious effects (with 90% confidence intervals) for self-anger MAP reactivity from model

Predictors	Effects	State NA	State AR	CV Recovery Time
Total $R^2$		.36	.09	.14
Trait NA				
Direct		.09 (-.06- .26)	--	-.26 (-.45- -.05)
Indirect		.00	--	-.00 (-.05- .01)
Total		.09	--	-.26 (-.45- -.05)
Spurious		.13	--	-.01
MAP Reactivity				
Direct		.00	.06 (-.15- .28)	.28 (.07- .43)
Indirect		.04 (-.09- .16)	.00	.00 (-.06- .01)
Total		.04	.06	.28 (.08- .42)
Spurious		.00	.03	.00
Trait AR				
Direct		.00	.30 ( .06- .47)	.05 (-.20- .30)
Indirect		.17 (.04- .29)	.00	-.02 (-.10- .03)
Total		.17	.30	.03 (-.21- .27)
Spurious		.00	.00	-.10
State NA				
Direct		--	.58 ( .44- .68)	-.04 (-.23- .18)
Total		--	.58	-.04
Spurious		--	.02	-.06
State AR				
Direct		--	--	-.03 (-.26- .17)
Indirect		--	--	-.02 (-.13- .11)
Total		--	--	-.05 (-.24- .13)
Spurious		--	--	.03

Note.  $N=75$ . NA = Negative Affect; AR= Anger Rumination; MAP= mean arterial pressure, CV= cardiovascular. Spurious effect reflects difference between the variable's zero-order effect and its estimated total effect in the model.

Table 7. Standardized direct, indirect, total, and spurious effects (with 90% confidence intervals) for self-anger HR reactivity from model

Predictors		State NA	State AR	CV Recovery Time
Effects				
Total $R^2$		.36	.09	.11
Trait NA				
Direct		.09 (-.06- .26)	--	-.29 (-.48- -.06)
Indirect		.00	--	-.00 (-.05- .01)
Total		.09	--	-.29 (-.48- -.06)
Spurious		.13	--	.04
HR Reactivity				
Direct		.00	-.09 (-.35- .14)	.22 (-.02- .39)
Indirect		-.05 (-.22- .08)	.00	.00 (-.02- .05)
Total		-.05	-.09	.22 (-.04- .40)
Spurious		.00	.00	-.03
Trait AR				
Direct		.00	.30 (.07- .47)	.02 (-.24- .26)
Indirect		.17 (.05- .29)	.00	.00 (-.06- .06)
Total		.17	.30	.02 (-.24- .25)
Spurious		.00	.00	-.15
State NA				
Direct		--	.58 (.44- .69)	-.03 (-.24- .20)
Total		--	.58	-.03
Spurious		--	.02	-.07
State AR				
Direct		--	--	.01 (-.22- .20)
Indirect		--	--	-.02 (-.14- .12)
Total		--	--	-.01 (-.20- .18)
Spurious		--	--	-.07

Note.  $N=75$ . NA = Negative Affect; AR= Anger Rumination; HR= heart rate, CV= cardiovascular. Spurious effect reflects difference between the variable's zero-order effect and its estimated total effect in the model.

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## APPENDIX A: MEASURES

## Demographics Questionnaire

1. Date of Birth (MM/DD/YYYY) \_\_\_\_\_
2. Sex     \_\_\_ (male)     \_\_\_ (female)
3. Race     \_\_\_ African-American     \_\_\_ American Indian/Alaska Native  
               \_\_\_ Asian     \_\_\_ White     \_\_\_ Hispanic/Latino (non-white)  
               \_\_\_ Native Hawaiian/ Pacific Islander     \_\_\_ Other
4. What is your height? \_\_\_ feet \_\_\_ inches
5. What is your weight? \_\_\_\_\_ pounds
6. What is your best estimate of your annual family income (including parents' if you receive support from them)? \_\_\_\_\_
7. What is your current Marital Status?  
               Single \_\_\_     Married \_\_\_     Long-term Relationship \_\_\_     Divorced \_\_\_
8. Do you have children? \_\_\_ Yes     \_\_\_ No     If yes, how many? \_\_\_\_\_
9. Please describe your current education level by circling the response below.  
       HS diploma   GED   1 yr college   2 yr college   3 yr college   4+ yr college
10. Please estimate your GPA by circling the response below:  
       4.0-3.5       3.49-3.0       2.9-2.0       <2.0
11. Please indicate the average number of hours you work per week: \_\_\_\_\_
12. Please indicate your greatest source(s) of stressors over the past year from the list below.  
       School \_\_\_     Work \_\_\_     Financial \_\_\_     Personal \_\_\_     Family \_\_\_  
       Other \_\_\_ (please explain) \_\_\_\_\_
13. Because different levels of hormones can affect how one perceives pain, if you are a woman, please indicate the date that your most recent menstrual period began:  
       \_\_\_\_ / \_\_\_\_ / \_\_\_\_  
       (MM)   (DD)   (YYYY)
14. Have you consumed caffeine, alcohol, smoke cigarette(s), or exercised vigorously (e.g., running, lifting heavy objects, aerobic exercise) in the past 2 hours?  
       \_\_\_ (yes)   \_\_\_ (no)
15. Please list any medications you are currently taking:  
       \_\_\_\_\_  
       \_\_\_\_\_

**ARS**

Please write the number that indicates how well the each of the following items corresponds to your beliefs about yourself.

1= Almost Never      2= Sometimes      3= Often      4= Almost Always

1. I ruminate about my past anger experiences. \_\_\_\_\_
2. I ponder about the injustices that have been done to me. \_\_\_\_\_
3. I keep thinking about events that angered me for a long time. \_\_\_\_\_
4. I have long living fantasies of revenge after the conflict is over. \_\_\_\_\_
5. I think about certain events from a long time ago and they still make me angry. \_\_\_\_\_
6. I have difficulty forgiving people who have hurt me. \_\_\_\_\_
7. After an argument is over, I keep fighting with this person in my imagination. \_\_\_\_\_
8. Memories of being aggravated pop up into my mind before I fall asleep. \_\_\_\_\_
9. Whenever I experience anger, I keep thinking about it for a while. \_\_\_\_\_
10. I have had times when I could not stop being preoccupied with a particular conflict. \_\_\_\_\_
11. I analyze events that make me angry. \_\_\_\_\_
12. I think about the reasons people treat me badly. \_\_\_\_\_
13. I have day dreams and fantasies of violent nature. \_\_\_\_\_
14. I re-enact the anger episode in my mind after it has happened. \_\_\_\_\_
15. I feel angry about certain things in my life. \_\_\_\_\_
16. When someone makes me angry, I can't stop thinking about how to get back at that person. \_\_\_\_\_
17. When someone provokes me, I keep wondering why this should have happened to me. \_\_\_\_\_
18. Memories of even minor annoyances bother me for a while. \_\_\_\_\_
19. When something makes me angry, I turn this matter over and over again in my mind. \_\_\_\_\_

### PANAS

Directions: This scale consists of a number of words that describe different feelings and emotions. Read each item and then circle the appropriate answer next to that word.

**Please indicate the extent you feel each feeling in general.**

	<b>Very slightly or not at all</b>	<b>A little</b>	<b>Moderately</b>	<b>Quite a bit</b>	<b>Extremely</b>
1. Interested	1	2	3	4	5
2. Distressed	1	2	3	4	5
3. Excited	1	2	3	4	5
4. Upset	1	2	3	4	5
5. Strong	1	2	3	4	5
6. Guilty	1	2	3	4	5
7. Scared	1	2	3	4	5
8. Hostile	1	2	3	4	5
9. Enthusiastic	1	2	3	4	5
10. Proud	1	2	3	4	5
11. Irritable	1	2	3	4	5
12. Alert	1	2	3	4	5
13. Ashamed	1	2	3	4	5
14. Inspired	1	2	3	4	5
15. Nervous	1	2	3	4	5
16. Determined	1	2	3	4	5
17. Attentive	1	2	3	4	5
18. Jittery	1	2	3	4	5
19. Active	1	2	3	4	5
20. Afraid	1	2	3	4	5



## Visual Analog Scales (11 moods)

Please indicate how much you are currently feeling each of the following moods by making a dash through the solid line.

Happy	_____
	Not at all <span style="float: right;">Extremely</span>
Guilty	_____
	Not at all <span style="float: right;">Extremely</span>
Sad	_____
	Not at all <span style="float: right;">Extremely</span>
Anxious	_____
	Not at all <span style="float: right;">Extremely</span>
Angry	_____
	Not at all <span style="float: right;">Extremely</span>
Shameful	_____
	Not at all <span style="float: right;">Extremely</span>
Frustrated	_____
	Not at all <span style="float: right;">Extremely</span>
Stressed	_____
	Not at all <span style="float: right;">Extremely</span>
Calm	_____
	Not at all <span style="float: right;">Extremely</span>
Embarrassed	_____
	Not at all <span style="float: right;">Extremely</span>
Regret	_____
	Not at all <span style="float: right;">Extremely</span>

### Visual Analog Scales (Post-Recovery)

Angry

Not at all Extremely

How much were you thinking about the event(s) that you wrote about?

Not at all Very Much