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Modeling the Parasympathetic Nervous Response to an Emotional Task: The Interaction of Heart Rate Variability, Personality and Emotion Regulation

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MODELING THE PARASYMPATHETIC NERVOUS SYSTEM RESPONSE TO AN EMOTIONAL TASK: THE INTERACTION OF HEART RATE VARIABILITY, PERSONALITY AND EMOTION REGULATION

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Heart rate variability (HRV), a physiological marker of autonomic nervous system (ANS) engagement, has been associated with a wide variety of clinical and psychological processes. High frequency (HF) HRV power, specifically, has been linked with the parasympathetic nervous system (PNS) and self-regulation. The current inquiry used a random effects growth model to study the HF HRV response to an emotional task and to predict individual differences in HF HRV as a function of trait hostility, neuroticism, and emotion regulation strategies (e.g., positive reappraisal, positive refocusing). Results indicated that the task engaged both branches of the ANS. HF HRV was not related to either hostility or neuroticism. However, positive reappraisal was associated with both high baseline values of HF HRV (i.e., greater initial parasympathetic activation) and lower rates of reactivity (i.e., less parasympathetic withdrawal). Overall, these results add to the evidence that positive reappraisal is a powerful component of emotion regulation and may be an important intervention target.

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Chapter 1

Introduction

The relationship of cardiovascular disease and cardiovascular mortality with autonomic nervous system functioning has prompted researchers to develop quantitative markers that can isolate the separate contributions of the sympathetic and parasympathetic branches of the nervous system to cardiac control (Task Force, 1996). Heart rate variability (HRV) is one promising and frequently used marker. Measurements of HRV have been used in a variety of areas of inquiry (Berntson et al., 1997), from basic investigations of autonomic regulation (e.g., Grossman, 1992; Porges, 1995) to studies of risk for physical illness (e.g., Stein &Kleiger, 1999; Thayer & Lane, 2007), to studies of autonomic relationships with psychological processes (e.g., Demaree& Everhart, 2004; Friedman & Thayer, 1998; McCraty, Atkinson, Tomasino&Stuppy, 2001; Ramaekers, Ector, Demyttenaere, Rubens & Van de Werf, 1998; Udupa, et al., 2007). Recently, HRV has been used as an indicator of self-regulation, as self-regulation and autonomic regulation occur within the same structures in the brain (Segerstrom& Solberg Nes, 2007; Thayer & Lane, 2000).

The Components and Measurement of Heart Rate Variability

Simply stated, HRV is the measurement of the variation present in the intervals between heartbeats in a consecutive series of beats. These beat-to-beat variations in heart rate are the product of multiple physiological systems whose influences upon the heart muscle oscillate with differing periods. Two important oscillating systems that cause beat-to-beat variations in heart rate are the sympathetic and parasympathetic branches of the autonomic nervous system. By assessing the variation in the intervals between

consecutive heartbeats (interbeat intervals, or IBIs) caused by the input of these discrete physiological systems, heart rate variability indicates the relative strength with which these neural systems influence the heart's operation. Thus, this noninvasive measure can index several physiological processes, which themselves have been linked to several psychological processes (Task Force, 1996; Berntson et al., 1997).

Researchers have developed a variety of methods to quantify heart rate variability. More than a decade ago, a group of prominent psychophysiology researchers recommended a set of best practices for measuring HRV in efforts to standardize its use in psychophysiological research (Task Force, 1996). As a result of the Task Force's report, two major approaches for investigating heart rate variability are now frequently used: (a) time domain methods that use statistical analyses to summarize variability using the normal-to-normal interval (NN; the length of IBIs) and (b) frequency domain methods that decompose an ECG signal into frequency components and quantify the power (i.e. variance) of the signal within discrete frequency bands. Both approaches can be used to generate global descriptive statistics to describe overall HRV, or model periodic patterns to estimate long- and short-term components.

Time domain measurement of HRV. Two time domain methods estimate overall variability in HRV: the standard deviation of the NN interval (SDNN) and the HRV triangular index. The SDNN, which is the square root of the variance, reflects all the components responsible for variability in heart rate. However, SDNN varies as a function of the length of the overall recording, so it is not acceptable to compare SDNN values obtained from recordings of different lengths. Despite this limitation, SDNN is a relatively simple variable to calculate and provides a good approximation of overall HRV

with a record of IBIs. Alternatively, theHRV triangular index, the integral of the density distribution divided by the maximum of the density distribution (i.e. the ratio of all NN intervals to the number of NN intervals within the modal bin of the interval histogram) provides a summary statistic for long-term (e.g., 24-hr) recordings (Task Force, 1996).

Additional time domain measures can be used to approximate long- and short-term variability: SDANN, the standard deviation of the of the average NN interval calculated over short intervals (e.g., 5 min) and the rMSSD, the square root of the mean squared differences of successive NN intervals. The SDANN estimates change in heart rate due to cycles longer than 5 min, whereas the rMSSD estimates high-frequency variations in heart rate. Similarly, Berntson et al., (1997) discussed an additional time domain estimate of variability that separates the oscillations occurring due to respiratory frequency (also known as respiratory sinus arrhythmia, or RSA) from other components. This statistic is a breath-by-breath measure of heart rate variability, which represents the difference between the longest and shortest heart period within the respiratory cycle. Porges and colleges (Porges& Byrne, 1992; Porges, 1995; Porges, 1991; Umhau et al., 2002) have used RSA as a measure of vagal tone. This is because respiration gates parasympathetic input to the heart during respiration; vagal efferent traffic is blocked during inspiration, resulting in a heart rate increase, and is reinstated during expiration, resulting in a heart rate decrease(Grossman, 1992; Porges, 1995). The simple range statistic described above thereby provides an index of parasympathetic control of the heart.

Frequency domain measurement of HRV. Because the periodic components of heart rate variability aggregate within several frequency ranges (Berntson et al., 1997),

frequency domain methods have also become popular (Task Force, 1996; Berntson et al., 1997). As mentioned above, frequency domain methods, power spectral analysis being the most common, decompose the total variance in a set of IBIs into frequency components, expressing variance as a function of frequency. The area under that function within a given frequency band is then quantified as power. The periodic variations in heart rate that are the focus of HRV measures accumulate within several specific frequency ranges. These include very low frequency (VLF), low frequency (LF), and high frequency (HF) within short recordings (e.g. 5 min) and an additional ultra-low frequency (ULF) component in longer recordings (e.g. 24 h; Task Force, 1996).

Oscillation of heart rate in these frequency domains has been attributed to specific components of the autonomic nervous system. The HF band (.15-.40 Hz), which has been shown to correlate with vagally mediated heart activity and is almost completely eliminated by cholinergic blockade, is a widely accepted index of parasympathetic activity. The LF range (.04-.15 Hz) reflects a more complex interplay of autonomic influences. Whereas this frequency band has been interpreted as a measure influenced mostly by sympathetic nervous system activity (Cohen, Matar, Kaplan, &Kotler, 1999; Malliani, Lombardi, &Pagani, 1994; Pagani et al., 1986) others consider it a parameter that includes both sympathetic and parasympathetic influences (Berntson et al., 1997; Eckberg, 1997; Houle&Billman, 1999; Kop, Krantz, & Baker, 2001; Polanczyk et al., 1998; Skyschally, Breuer, &Heusch, 1996). Therefore, several researchers have used LF/HF as an index of sympathovagal balance (Demaree& Everhart, 2004; Malliani et al., 1994; Lombardi, Malliani, Pagani, &Cerutti, 1996; McCraty, Atkinson, Tiller, Rein, & Watkins, 1995; Rossy& Thayer, 1998; Sloan et al., 2001). However, Berntson et al.

(1997) noted that neither beta-adrenergic blockade nor blocking sympathetic outflow by high spinal anesthesia reliably reduce LF power, consequently arguing that LF is not a proper measure of sympathetic input. Additionally, Eckberg (1997) argued vigorously against the use of the LF/HF proportion as an index of sympathovagal balance.

Despite disagreement regarding the interpretation of these measures, they have been widely used. As mentioned above, measures reflecting parasympathetic input to the heart, which is mediated by the vagus nerve, are the most widely accepted. Researchers have commonly used HF power, RSA, and rMSSD as indicators of parasympathetic nervous system activation (Berntson et al., 1997; Cacioppo et al., 1995; Ingjaldsson, Laberg, & Thayer, 2003; McCraty, et al., 1995; Task Force, 1996).

The Clinical and Psychological Correlates of Heart Rate Variability

Researchers have linked heart rate variability with a wide variety of medical conditions and psychological dysfunctions and risk factors. Decreased HRV, particularly decreased variability associated with vagal or parasympathetic control, is associated with poorer overall heart health, higher risk for heart disease, and higher risk for cardiac mortality (Kautzner&Camm, 1997; Stein &Kleiger, 1999; Thayer & Lane, 2007). Panic disorder is also associated with lower levels of overall HRV (Friedman & Thayer, 1998; McCraty et al., 2001),whereas low HF power is associated with generalized anxiety disorder and worry (Thayer, Friedman, &Borkovec, 1996). Studies of the HF power's associations with depression and bipolar disorder have produced mixed results (e.g., O'Connor, Allen, &Kaszniak, 2002; for a review, see Voss, Baier, Schultz, & Bar, 2006). Coping (O'Connor et al., 2002; Ramaekers et al., 1998), emotional states (McCraty et al., 1995; Sakuragi, Sugiyama, & Takeuchi, 2002) and hostility (Demaree& Everhart, 2004;

Sloan et al., 1994; Sloan et al., 2001) are also correlated with HRV. Finally, Thayer and Lane's (2000, in press) model of emotion regulation and dysregulation associates lower HRV with poor attention, ineffective mood regulation, and behavioral inflexibility (see also Porges, 1992).

The Links of Heart Rate Variability to Stress and Induced Emotion

Given the potential of this easily assessed, noninvasive measure to shed light on clinically important influences of the autonomic nervous system on cardiovascular functioning, several studies have examined the response of HRV to laboratory stressors. Shively et al. (2007) used a stress paradigm to examine the effect of alcohol consumption on heart rate variability in an acute stress condition in a sample of long-tailed macaques (*Macacafascicularis*). Regardless of alcohol consumption, moving the monkeys to a novel environment (acute stress) resulted in decreased overall HRV and decreased HF power. Isowa, Ohira, and Murashima (2006) also examined the effect of stress on HRV in humans, using aversive noise as a stimulus. For these participants, HF power decreased during the stressful event, whether the noise was controllable (i.e., linked to their task performance) or uncontrollable (i.e., unrelated to their task performance).

Researchers have also examined HRV responses to stressful mental tasks (e.g., mental arithmetic). Cacioppo et al. (1995) found that RSA decreased in response to such stress tasks, which suggests that parasympathetic input to the heart decreased. In a similar study, Wright, O'Donnell, Brydon, Wardle, & Steptoe (2007) measured HF HRV using rMSSD and found that HRV decreased during the mental task (i.e., Stroop task) and was the lowest during the emotional stress task (i.e., participants defended themselves in a

hypothetical scenario in which they were accused of shoplifting). This study too, then, suggested that the stressful task reduced parasympathetic input to the heart.

Other research has also examined the effect of emotional tasks on the parasympathetic nervous system using measures of HRV. Demaree, Schmeichel, Robinson, & Everhart (2004) found that people watching emotion-eliciting (i.e., positive and negative affect) video clips experienced a shift in sympathovagal balance (LF/HF power) although they did not experience a change in HF power alone. Participants who were asked to exaggerate their emotional response experienced the largest increases. Based on these results, Demaree et al. suggested that emotional expression and experience might be more closely tied to sympathetic arousal than has been previously assumed, creating a complex effect involving the balance of sympathetic and parasympathetic influences. However, Neumann, Sollers, Thayer, &Waldstein (2004) found that an anger recall task dampened high frequency power (although this finding was slightly too small to cross the conventional threshold for statistical significance). Sakuragi et al. (2002) examined the effects of laughing and weeping on HRV. HF power decreased while watching both comedy and tragedy videos and remained low during the recovery period after the tragedy videos, suggesting that arousing negative states, such as sadness, might result in more lasting withdrawal of parasympathetic modulation of the heart. The majority of these tasks appeared to cause the withdrawal of the parasympathetic nervous system, suggesting that emotional tasks may impact HRV through parasympathetic withdrawal.

Cognitive Emotion Regulation and HRV

The impact of emotion on HRV may be modulated, however, by the use of emotion regulation strategies. Thayer and Lane (2000, in press) reviewed a variety of studies and put forth a cohesive theory, which describes how the processes of cognitive, emotional, and physiological regulation may work together in goal-directed behavior; thus, people with mental health disorders (e.g., anxiety, depression) have both lower HRV and less effective emotion regulation than do individuals without clinical symptoms (Thayer &Brosschot, 2005).

Gross, Richards, and John (2006) defined emotion regulation as "attempts individuals make to influence which emotions they have, when they have them, and how these emotions are experienced and expressed" (p. 14). Generally speaking, humans often experience emotions that must be managed if they expect to navigate their lives. Gross and colleagues (Gross, 1998, 2002; Gross et al.) draw a broad distinction between antecedent-focused and response-focused strategies. Antecedent-focused strategies are attempts made to change our potential experience of emotion, before those emotions' response tendencies are fully activated and have changed our behavior or physiology. Alternatively, response-focused strategies are attempts to change our emotional experience once response tendencies have already initiated.

Several studies by Gross and colleagues (for a review, see Gross, 2002; Gross & John, 2003; Gross et al., 2006, andJohn & Gross, 2004) have compared reappraisal (an antecedent-focused strategy) to suppression (a response-focused strategy). These studies have shown cognitive reappraisal effectively influences the experience of emotion, decreasing emotional experience and expression in negative contexts without negatively

impacting cognition, physiology or interpersonal relationships. Individual difference studies have also found that people who more frequently employ reappraisal in their everyday lives show enhanced emotional and interpersonal functioning (Gross et al). Recently, Siemer, Mauss& Gross (2007) systematically tested appraisal theory, which suggests that it is the way a person interprets a situation, instead of the reality of the situation, that facilitates particular emotional experiences. They found that appraisals influenced both the type and intensity of emotional experience in response to identical feedback regarding performance on a counting task. This finding suggests that appraisals are necessary and sufficient for differing emotional responses.

One might safely presume, therefore, that positive reappraisal of a situation would impact emotional experience in a positive direction. In support of this idea, Lepore, Greenburg, Bruno and Smyth (2002) posited that expressive writing facilitates positive mental and physical health outcomes because it induces cognitive restructuring, which is a regulatory process. Garnefski et al. (2002) likewise found that people from a nonclinical sample reported that they used positive reappraisal significantly more often than those from a clinical sample. Additionally, several studies (Garnefski, Kraaij, &Spinohoven, 2001; Martin &Dahlen, 2005; Schroevers, Kraaij&Garnefski, 2007) have found that the use of positive reappraisal was negatively related to symptoms of both depression and anxiety. These findings indicate that the ability to reappraise may be an important component of self-regulation and mental health.

The Association of HRV with Self-Regulation

Self-regulation has also become a topic of interest for researchers studying HRV. For example, several research groups have tied higher HRV to more effective self-

regulation (e.g., Appelhans&Luecken, 2006; Porges, 1991; Segerstrom& Solberg Nes, 2007; Thayer & Lane, 2000, in press). Porges (1991) proposed that high vagal tone allows the autonomic nervous system to react and to return the system to homeostasis. As noted above, Thayer and colleagues (Thayer & Lane, 2000, 2002, in press) have similarly linked low vagal control of the heart with impaired ability to respond to environmental changes based on three major deficits. Specifically, low parasympathetic activity is associated with poor attention control (i.e., monitoring one's environment and processing relevant information), ineffective emotion regulation (i.e., affective information processing and expression), and behavioral inflexibility (i.e., adaptability to changing environmental and task demands).

Ingjaldsson et al. (2003) examined the relationship of HRV with self-regulation among alcoholics and nonalcoholics. They exposed participants to an imaginary alcohol scenario and found that alcoholics had lower tonic HRV levels. Additionally, HRV was negatively correlated with the reported urge to drink during the alcohol script exposure among alcoholic participants, suggesting that they may have had poorer emotion regulation and impulse inhibition. In a similar study, Segerstrom and Solberg Nes (2007) found that hungry participants who were asked to exert self-regulatory effort (i.e., eating carrots, but resisting cookies and chocolates) experienced greater HRV elevation (rMSSD) compared to participants who exerted less effort (i.e., eat cookies, resist carrots). The experimental manipulation and higher baseline HRV levels then predicted persistence at a subsequent anagram task, suggesting that HRV is in an indicator of both self-regulatory effort and self-regulatory strength.

The model put forth by Thayer and Lane (2000, 2008) of neurovisceral integration describes the connections between autonomic, attentional, and affective systems. The central autonomic network (CAN), which consists of prefrontal cortices, anterior cingulate, insula, amygdala, hypothalamus, and periaqueductal gray, is in part responsible for parasympathetic influence to the heart. Simultaneously, they control behavioral responses that are necessary for goal-directed behavior and adaptability (Thayer, 2007). These common brain structures and the associations between them may explain the link between HRV and self-regulation.

The Association of HRV with Personality Traits

Investigators have also examined the associations of HRV with personality variables such as hostility and neuroticism. In an early effort by Muranaka et al. (1988), participants completed a mental arithmetic task and cold face stimulus stressor (i.e., plastic bags of water and ice were applied to the participants' foreheads) while experimenters measured their forearm vasoconstriction (another indicator of vagal activity). Type B participants, as compared to Type A participants, had greater forearm vasoconstriction during the cold face stimulus, indicating that those participants (i.e., presumably the less hostile group) had a stronger vagal response to the stressor.

Using more contemporary methods for assessing HRV, Demaree and Everhart (2004) found that hostile participants had low parasympathetic activity at baseline relative to participants low in hostility. Similarly, Neumann, Waldstein, Sollers, Thayer, and Sorkin (2004) found that low-hostile women experienced an increase in HF power when distracted after an anger-recall task whereas hostile women did not. In addition,

Riese et al. (2007) examined HRV among female twin pairs and found that neuroticism was negatively correlated with HRV (HF power).

Research relating personality to HRV has not been entirely consistent, however. For example, Virtanen et al. (2003) failed to find a relationship between HRV and personality measures (i.e., hostility, trait anxiety, trait anger), as did Takahashi et al. (2005; i.e., novelty seeking, harm avoidance, reward dependence, and persistence). Similarly, Schweiger, Wittling, Genzel, and Block (1998) found no correlation between HF power and the subscales of the Freiburger Personality Inventory, which measures 12 subscales (i.e., life satisfaction, social orientation, achievement orientation, inhibition, excitability, aggressiveness, subjective feelings of strain, physical complaints, worries concerning physical health, frankness, extraversion, and emotionality).

Possible Relationships Among HRV, Emotion Regulation and Personality

Studies that suggest a link between personality traits and HRV (e.g., Demaree& Everhart, 2004; Neumann et al., 2004; Riese et al., 2007) indicate that both neuroticism and hostility may be negatively related to HRV, particularly parasympathetic activity as measured by HF power. Work by other researchers in the field of emotion regulation has suggested a negative relationship between reappraisal and neuroticism (Gross & John, 2005). Martin and Dahlen (2005) also found significant positive correlations between trait anger and the cognitive emotion regulation strategies of self-blame, blaming others, rumination, and catastrophizing. In contrast, they found a negative correlation between trait anger and putting things into perspective, positive refocusing, and positive reappraisal. Given the research that demonstrates the impact of emotion, and emotion regulation, on HRV (e.g., Demaree et al, 2004; Neumann et al., 2004; Sakuragi et al.,

2002), it seems worthwhile to explore the possibility that some emotion regulation strategies mediate the relationship between personality traits and HRV.

Limitations of the Existing Studies on HRV Correlates of Psychological Traits

Previous studies attempting to link HRV with psychological traits have been, at times, inconsistent. Several factors may be a work. First, individual studies have used a wide variety of instruments to measure personality, which may produce null findings in some studies, and significant relationships in others. For example, Demaree and Everhart (2004) used the Cook-Medley Hostility Scale as an indicator of hostility, whereas Virtanen et al. (2003) used a Brief Symptom Inventory (BSI-37). These instruments may not operationally define hostility in the same way, so it not surprising that these studies produced conflicting results.

Second, several studies have looked only at tonic HRV levels, rather than reactivity in response to a stressor (e.g., Schweiger et al., 1998; Virtanen et al., 2003). Furthermore, even when researchers have evaluated HRV as "reactivity" in response to an emotional or mental task, they have used simple pre-post scores, rather than using a specific model of change (e.g., Cacioppo et al., 1995; Demaree& Everhart, 2004; Segerstrom& Solberg Nes, 2007; Wright et al., 2006). A multilevel modeling approach would not only allow for the interpretation of a response pattern but also, for the first time, allow for the separation of true change from measurement error. Introducing personality variables as predictors in this kind of model would also test the hypothesis that personality affects the response pattern of vagal influence to the heart instead of, or in addition to, the magnitude of response.

A third limitation is that all of the studies that have been conducted to date have focused on either (a) measurements of tonic HRV; or (b) task-induced HRV reactivity. Without doubt, tonic levels of HRV and HRV reactivity in response to stressors are important, but it seems equally likely to understand the processes that influence the rates at which HRV recovers to its tonic levels after the offset of a stressor. Given enough measurements before the onset of a stressor, after the onset of a stressor, and after the offset of a stressor, it is feasible to model reactivity and recovery in HRV within a single statistical model.

The Present Study

The goals of the proposed study were (a) to use a random effects growth model to examine the response of the parasympathetic nervous system to an emotional task; and (b) to predict individual differences in baseline, reactivity, and recovery to the task using emotion regulation strategies and personality variables that have been previously implicated as predictors of baseline HRV and HRV reactivity. I modeled HF HRV power before, during, and after an emotional task to determine how the parasympathetic nervous system responds to the task. I then predicted individual differences in tonic HRV functioning, HRV reactivity, and HRV recovery using trait hostility, Neuroticism, and an extensive set of emotion regulation strategies.

I hypothesized that hostility and Neuroticism would be related to lower HF power during the baseline task, greater reductions in HF power during the emotional task, and lower rates of recovery in HF power after the emotional task had ended. I also planned to evaluate whether any such personality-HRV relationships were mediated by cognitive emotion regulation strategies. Specifically, I hypothesized that more hostile individuals

would be more likely to blame others, ruminate, and engage in less positive refocusing, positive reappraisal and putting things into perspective, which would in turn affect their baseline levels of HRV (indicative of self-regulatory strength; Segerstrom& Solberg Nes, 2007) and reactivity (indicative of self-regulatory effort; Segerstrom& Solberg Nes). I also expected that the associations of hostility and HRV recovery would be mediated by the use of rumination as an emotion regulation strategy.

Similarly, I anticipated that people high in neuroticism would be more likely to use the emotion regulation strategies of self-blame, rumination, catastrophizing, and less likely to use positive reappraisal. I expected that these associations between neuroticism and emotion regulation strategies would furthermore mediate the associations of neuroticism with baseline, reactivity, and recovery in HRV.

Finally, Lepore et al. (2002) suggested that expressive writing facilitates cognitive restructuring, which in turn impacts physiology and health. Therefore, on an exploratory basis I tested whether the proportion of words related to cognitive processing and positive and negative affect that participants used when describing their thoughts during the emotional task predicted individual differences in the growth components underlying participants HF HRV responses to the task. Trait positive affect and negative affect were also tested as predictors to distinguish between the tendency to experience particular emotions and the use of specific emotion regulation strategies.

Chapter 2

Method

Participants

Undergraduate psychology students at the University of Miami (N=164, 54 males, 111 females) voluntarily participated in this study as one of several options to fulfill a course requirement. Participants received course credit and $20 after completing the study. Participants were 17 to 46 years of age, although one participant did not report his or her age (N= 163, *M* = 19.6, *SD* = 3.82). Each participant indicated having experienced an interpersonal transgression (i.e., a negative life event in which someone whom they knew committed an act against them that they judged as both painful and morally wrong) within the last 9 days (Range = 0-9 days, *M* = 4.37, *SD* = 1.85).

Self-Report Measures

The self-report measures used in this study are found in Appendix A.

Positive Affect and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS consists of 20 items designed to measure positive and negative affect. Participants indicated how often they experienced different emotions (e.g., upset, proud)in the last 2 weeks on a 5-point Likert scale. Internal consistency estimates were high for both positive and negative PANAS subscales (alphas were 0.86 and 0.71, respectively).

The Aggression Questionnaire (Buss& Perry, 1992). The Aggression Questionnaire consists of 29 items designed to measure physical aggression, verbal aggression, anger and hostility.Each item is rated on a 5-point Likert-type scale (1 = *not at all like me*, 5 = *very much like me*). The internal consistency for the entire scale is good (α = 0.82).

Big-Five Inventory (BFI; John, Donahue, &Kentle, 1991).The Big Five Inventory consists of 44 self-report items intended to measure the five higher-order dimensions of personality that are typically recovered from factor analyses of personality ratings (Openness, Conscientiousness, Extraversion, Agreeableness, and Neuroticism). The items on the BFI were rated on 5-point Likert-type scale (1 = *strongly disagree*, 5 = *strongly agree*) and were then averaged to yield a composite score for each of the five scales. All five subscales have high convergent and discriminant validity. Internal consistency for Neuroticism was $\alpha = 0.84$ in this study.

Cognitive Emotion Regulation Questionnaire (CERQ; Garnefski, et al., 2001).The CERQ is a 36-item scale with 9 subscales to measure discrete emotion regulation strategies for responding to life events. Participants rated each item on a 5-point Likert-type scale (1 = *strongly disagree*, 5 = *strongly agree*) to indicate whether they used each strategy when they thought about their recent transgressions. The items from each subscale were then averaged for a composite score. The following subscales were used: (a) self-blame (e.g., "I felt that I am the one to blame for it"), (b) acceptance (e.g., "I thought that I had to accept the situation"), (c) rumination (e.g., "I was preoccupied with what I think and feel about what I have experienced"), (d) positive refocusing (e.g., "I thought of nicer things than what I have experienced"), (e) refocus on planning (e.g., "I thought about how to change the situation"), (f) positive reappraisal (e.g., "I thought that I can learn something from the situation"), (g) putting into perspective (e.g., "I thought that it could have been much worse"), (h) blaming other (e.g., "I felt that others are to blame for it") and (i) catastrophizing (e.g., "I continually thought about how horrible the situation has been"). Internal consistencies for these 9 scales ranged from 0.64 to 0.89.

Forty-four additional items were added to the CERQ to create 8 additional subscales that were designed by the research team as potential indicators of forgiveness as an additional area of inquiry: (a) humble self appraisals (e.g., "I thought about the fact that I'm not perfect either"), (b) perspective taking (e.g., "I tried to imagine the position/state they were in at the time"), (c) benefits to the relationship (e.g., "I tried to see that our relationship could grow from this"), (d) benign causal attributions (e.g., "I realized that he/she would not normally behave this way"), (e) focus on positives of the relationship (e.g., "I focused on the positives in our relationship"), (f) mental simulation of positive outcomes (e.g., "I thought about ways to strengthen our relationship"), (g) emulation of moral exemplars (e.g., "I imagined how a good person would respond to this situation") and (h) focus on positive aspects of transgressor (e.g., "I thought about his/her strong points"). Internal consistencies ranged from 0.65 and 0.92.

Post-Imagery Emotions (Root, McCullough, Berry, & Bono, 2007). After completing the imagery task described below, participants rated 33 emotion words(e.g., "angry," "grateful," "empathetic") to indicate "how you are feeling right now regarding the person whom you just imagined yourself interacting with" on a 7-point Likert scale (1 = *not at all*, 7 = *extremely*). The majority of these emotion words loaded onto 3 oblique principal components: (a) positive affect (e.g., *happy*, *content*; 15 total items), (b) negative affect (e.g., *afraid*, *anxious*, 6 total items), and (c) angry affect (e.g., *angry*, *hostile*; 5 total items). We created linear composites of these items for a previous study, using the same sample, αs = 0.97, 0.83, and 0.90, respectively.

Linguistic Inquiry and Word Count (LIWC; Pennebaker, Francis, & Booth, 2001). The LIWC is a software program designed to analyze the grammatical, linguistic, and

psychological features of text. After the task, as described below, participants described their thoughts during each component of the protocol (i.e., baseline, task, and recovery). I ran each written description through the LIWC program, which counted the total number of words and then compared each word to its internal dictionary of over 2000 words. Each word is assigned to one or more grammatical, linguistic and psychological categories. The number of words in each category (e.g., cognitive mechanisms) was then divided by the total number of words, which resulted in the percentage of total words. I focused on affective and cognitive predictors for this study, given that emotion and cognition are the focus of this inquiry. Specifically, I focused on the descriptions participants wrote about the reactivity portion of the task, and the percentage of words that were categorized as: (a) cognitive processes (e.g., cause, know, ought), (b) positive emotion (e.g., happy, good, love), and (c) negative emotion (e.g., hate, afraid, sad).

Physiological Apparatus and Data Acquisition

Participants sat alone in a laboratory room adjacent to an equipment room from which experimenters ran the procedure and collectedphysiological data. We useda PC built by the Fulton Computer Co. (Gloversville, NY) and the STIM software (James Long Company, Caroga Lake, NY) to collect online physiology data and to time the presentation of tones. These tones signaled the end and beginning of each condition (i.e., relaxation, imagery, post-imagery), were 5s long and 1 kHz, and were generated by the computer's speaker. The participants wore Phillips HN100 noise-canceling headphones to hear the tones and the music presented during the baseline period ("Blue Pool;" McCarty &Cennamo, 1988). We used three disposable, self-adhesive Unitrace electrodes placed axially on the left and right rib at approximately the same elevation as the heart,

with the ground electrode placed just below the sternum to collect electrocardiogram data. Diastolic and systolic blood pressure were measured every 60 s using an IBS automated SD700A Blood Pressure/Pulse Monitor (IBS Corporation, Waltham, MA). A standard occluding cuff was placed on the participant's nondominant arm, with measurements occurring every 60 seconds.

Signal Processing. A custom bioamplifier from SA Instruments (San Diego, CA), amplified the ECG signals and Snap-Master Data Acquisition System (HEM Data Corp., Southfield, MI) digitized the signal at 1000 Hz. The IBI Analysis System from the James Long Company (Caroga Lake, NY) was used for all of the remaining processing and analysis of the ECG signal. R-wave detection occurred offline using a 4-pass self-scaling peak detection algorithm, resulting in a file containing the onset times of each detected R-wave. A graphical representation of the signal with tick marks indicating the start of each R-wave was used for artifact editing. I corrected undetected R-waves or incorrect tick marks manually.

Procedure

Throughout the semester, research assistants visited undergraduate psychology courses to describe the study. As prospective participants encountered significant transgressions in their daily lives (i.e., someone in their life had to do something to him or her that was both hurtful and morally wrong), they became eligible to enroll. Interested participants completed an initial screening packet. This questionnaire solicited a brief description of the offense, the measures of personality as described above, and several other measures that are not relevant to the current inquiry. Participants then returned the initial packet to the laboratory and began completing a booklet consisting of 21 copies of

a questionnaire that is not relevant for the current study. Once they returned the initial packet, participants were contacted to schedule a laboratory appointment approximately 21 days after their enrollment in the study.

Each participant completed the laboratory visit individually in an hour-long session consisting of an imagery task and a final questionnaire. The imagery task consisted of three sections; the start of each section was signaled using a tone in participants' headphones and by brief instructions on the computer monitor. First, participants engaged in a relaxation task (the *baseline period*) during which they sat quietly and thought about the most pleasant and peaceful place that came to mind for four minutes while listening to the song "Blue Pool" (McCarty &Cennamo, 1988). At the end of the four-minute period, a tone signaled participants to begin thinking about their transgressor and to "bring to mind what they [the transgressor] did to you…and imagine what you would say to them and how you might act toward them if you could say and do anything you wanted" for three minutes (i.e., the *reactivity period* or *task period*). For the remaining phase of the imagery task, participants were asked to sit quietly for the final seven minutes with no specific topic to think about (i.e., the *recovery period*).

Once the reactivity task was completed, participants completed additional questionnaires that are not relevant for the present study. The experimenters then debriefed the participants and thanked them for their participation.

Data Analysis

Data Reduction. The edited R-wave data were converted to a prorated heart period series with a sampling interval of 8 s. Heart periods that spanned two sampling intervals were prorated between the two intervals using a weighted mean. The data were

then discrete Fourier transformed (DFT) on a window-by-window basis within 1-minute intervals (4 minutes baseline, 3 minutes imagery, 7 minutes post-imagery) and the resulting power data (ms^2) was be divided into frequency bins (HF = .15-.40 Hz). We likewise obtained 14 measures of systolic and diastolic blood pressure (4 baseline, 3 imagery, and 7 post-imagery).

Statistical Models. To describe the form of HF power response over the 14-minute laboratory task, I used a multilevel modeling framework. This enabled me to fit both (a) within-subject models (Level-1) and (b) between-subjects models (Level-2) that allowed for individual differences in the growth components. For example, with HF power measures from at least three time points (i.e., minutes) for a single individual, one can write:

$$HF_{ij} = \pi_{0j} + \pi_{1j}(Time_{ij}) + r_{ij} \qquad (1).$$

In Equation (1), HF_{ij} is individual j's HF power at minute i, modeled as a function of an intercept $\pi_{0j,}$ which is a theoretical construct representing person j's initial HF power at Time 0 (i.e., average HF power) , and a rate of change (π_{1j}), which is the rate that person j's rate of change in HF power as a linear function of time. The residual (r_{ij}) is the deviation of person j's HF power at time i from what would be expected based on his or her initial HF power (π_{0j}) and the rate of linear change in his or her HF power (π_{1j}). As I explain below, it is possible to extend this equation to include latent variables representing HRV reactivity and HRV recovery in a single model.

Before modeling the data, I examined the plots of HF power for each participant over time for initial guidance in model building. Then, I used a piecewise latent growth curve model (LGC), as described by Llabre, Spitzer, Saab, &Schneiderman (2001) to

estimate the parameters of the growth model. I first modeled the reactivity and recovery periods separately and then combined them using an adaptation of piecewise regression. This method uses dummy coded vectors, *D1* and *D2* to create interaction vectors, *REACT* and *RECOV*, so that two different slopes (one for reactivity and one for recovery) can be estimated simultaneously. Specifically, for this 14-minute task, minutes 1-4 are baseline values, 5-7 are reactivity values, and 8-14 are recovery values. Although multiple baseline measurements were taken, the assumption is that baseline values will be stable during that period (Llabre et al.). Therefore, I assigned all 4 baseline values a time value of zero, which is similar to averaging the baseline values, but also accounts for measurement error around the mean value. I then reassigned the remaining 10 values minute designations (1-10) and centered time around the shared time point in both the reactivity and recovery models, which is the last reactivity measurement. This approach resulted in the following basis coefficients for time for those 10 values:

-3 -2 -1 0 1 2 3 4 5 6 7.

The dummy vectors were assigned as follows:

D1 = 0, if time $\geq$ 0 (i.e., part of the recovery period)

D1 = 1, if time < 0 (i.e., part of the reactivity period)

D2 = 0, if time $\leq$ 0 (i.e., part of the reactivity period)

D2 = 1, if time > 0 (i.e., part of the recovery period).

When time was multiplied with the dummy vectors, the resulting interaction vectors were:

REACT = *D1* * time: -3 -2 -1 0 0000000 and

RECOV = *D2* * time: 0 000 1 2 3 4 5 6 7.

Because it was meaningful to consider time = 0 at the baseline point of this task, I added a value of 3 to each of the values in *REACT,* which yielded:

REACT: 0 1 2 3 3333333 and

RECOV: 0 000 1 2 3 4 5 6 7.

In this way, each vector ultimately contained appropriately varying intervals for the period it measured and constant values for the period it did not measure. The resulting equation for both the reactivity and recovery was:

$$HF_{ij} = \pi_{0j} + \pi_{1j}(REACT_{ij}) + \pi_{2j}(RECOV_{ij}) + r_{ij} \quad (2).$$

In Equation (2), when time = 0, both *REACT* and *RECOV* are 0, and the response value represents the intercept, or latent baseline value (π_{0j}). During the reactivity period, the three reactivity measurements within the *REACT* vary appropriately and are then constant for the recovery period. Similarly, during the recovery period, *RECOV* contains appropriately varying values for the recovery period and holds constant values for the reactivity period. Taken together, the two vectors express information about the common intercept and respective time values.

Whereas Equation (1) parameterizes the observations of a single individual (called a "Level-1" or "within-persons" equation), additional "between-persons" or "Level-2" equations can be used to specify the between-person variation that occurs within a sample. Level-2 equations model the parameters in a Level-1 equation as the result of (a) expected parameter estimates for the entire sample (fixed effects) and (b) person-specific deviations from the expected values (random effects). Person-specific variations in linear change, for example, can be decomposed according to the following between-persons or Level-2 model:

$$\pi_{1j} = \beta_{10} + u_{1j} \qquad (3).$$

In Equation (3), person *j*'s rate of linear change (π_{1j}) is expressed as a function of a fixed effect (β_{10}) and a random effect (u_{1j}). The fixed effect is the expected rate of linear change for the entire sample and the random effect is the deviation of person *j*'s parameter estimate for linear change π_{1j} from the fixed effect β_{10}. The fixed effect estimates the typical degree of linear change that an individual from our sample can be expected to experience. At the same time, the random effect estimates the extent that person *j*'s HF power differs from the "average" person in the sample. Person-specific predictors can then be added to the level-2 equations.

Chapter 3

Results

Descriptive Statistics

The means and standard deviations for all self-report and physiological variables are reported in Tables 1 and 2, respectively. The correlations among major self-report variables are listed in Table 3.

Factor Analysis of Emotion Regulation Strategies

To shed light on the statistical structure underlying the emotion regulation strategies that participants reported using, I factor-analyzed the 106 items administered as part of the Modified CERQ. Using a Maximum Likelihood method, five factors with eigenvalues greater than 4were extracted, which explained 32% of the total variance. An alternative solution with 27 factors with eigenvalues greater than 1 was also generated, which explained 76% of the total variance. However, neither solution produced factors that cohered in a logical or consistent way.

To simplify this data-reduction task, I submitted the 21 subscales, instead of the individual items, to a maximum likelihood factor analysis. Six factors with eigenvalues greater than 1.0 were extracted, which explained 45% of the total variance. I named these factors Relationship Benefit-Finding, Empathy/Personal Benefit-Finding, Catastrophizing, Imagining Good things, Emotional Denial, and Avoiding/Positive Planning. Generally speaking, the shared variance between the factors was low (correlations < 0.30), although factors 1 and 2 (Relationship Benefit Finding and Empathy/Personal Benefit Finding) were correlated at $r = 0.44$. The factor loadings were then extracted to be used as individual predictors in the HF power Reactivity and

Recovery models. None of these factors ultimately performed very well as predictors of individual differences in the HF HRV growth parameters, so I reverted to analyzing the individual CERQ subscales.

Characterization of the Task

Modeling Heart Rate. To characterize the effects of the imagery task on overall cardiac functioning, I specified latent growth curve models for heart rate, systolic blood pressure, and diastolic blood pressure. For example, heart rate at level 1, or the "within-persons" level, is expressed as a function of time. By examining the plots of 10 randomly selected participants, it appeared that a quadratic model would best describe the data (see Figure 1). That model may be specified as

$$HeartRate_{ij} = \pi_{0j} + \pi_{1j}(Time_{ij}) + \pi_{2j}(Time_{ij}^2) + r_{ij} \quad (4).$$

In Equation (4), $HeartRate_{ij}$ is individual *j*'s heart rate in beats per minute at minute *i*, modeled as a function of an intercept $\pi_{0j,}$ which is a theoretical construct representing person *j*'s initial heart rate at Time 0 (i.e., average heart rate), and a linear coefficient of participant *j*'s trajectory (i.e., the instantaneous slope at time = 0). The change in the slope as a function of time (i.e., curvature) is the quadratic component, π_{2j}, and contributes to the conditional slope. The residual (r_{ij}) consists of the random measurement error, or the deviation from the trajectory for person *j*.

Table 4 shows the values, standard errors, and *t*-values for the fixed effect parameters and the variance and covariance components of the random effect parameters for the quadratic heart rate model. When running models, I correlated the measurement error variances of each 1-minute epoch with the adjacent epoch because I expected the errors to be correlated in an autoregressive way. For this model, the predicted mean heart

rate was 75.27 beats per minute at baseline. During the task, the average heart rate increased by an average of 2.37 beats per minute every minute and this increase was reduced by an average of 1.08 beats per minute each minute (2 x -0.54, the first derivative of the linear coefficient, which is the conditional slope). This model was a good fit for the data, $\chi^2(19) = 126.544$, $p<0.001$. The root mean square error of approximation (RMSEA) value was 0.196, the Comparative Fit Index (CFI) = 0.958 and the Standardized Root Mean Square Residual (SRMR) value = 0.060. Estimates of the variance components showed significant variability in all three parameters (see Table 3; *t*-values >2.000 are significant), indicating significant differences between individuals in baseline values, conditional slope and curvature.

For the recovery model, I tested both quadratic and linear models. Although the quadratic recovery model was a good fit for the data, $\chi^2(27) = 85.50$, $p<0.001$, RMSEA=0.121, CFI = 0.980 and SRMR = 0.018, the fixed and random effects for the quadratic term were not significant (see Table 5). Therefore, a linear recovery model was also tested. For the linear recovery model a similar model was specified, except without a quadratic term:

$$HeartRate_{ij} = \pi_{0j} + \pi_{1j}(Time_{ij}) + r_{ij} \qquad (5).$$

In Equation (5), $HeartRate_{ij}$ is individual *j*'s heart rate in beats per minute at minute *i*, modeled as a function of the intercept $\pi_{0j,}$ representing person *j*'s initial heart rate at Time 0 (i.e., average heart rate), and a linear coefficient of participant *j*'s trajectory (i.e., the linear slope). The residual (r_{ij}) is the deviation of person *j*'s heart rate at time *i* from what would be expected based on his or her initial heart rate (π_{0j}) and the rate of linear change in his or her heart rate (π_{1j}).

For the linear model (see Table 5), the predicted mean heart rate was 76.89 beats per minute at the end of the task. Once the task was complete, the average heart rate decreased by an average of -0.172 beats per minute every minute. In this model, the latent variable for recovery was significant, and there was significant variability in both the ending task and recovery values across participants, indicating that were individual differences across participants. The correlation between baseline and reactivity was -0.256 and significant. This model was a good fit to the data with $\chi^2(31) = 91.17$, $p<0.001$,RMSEA=0.114, CFI = 0.980 and SRMR = 0.019. The chi-square difference between a quadratic model and this linear model was not significant, $\Delta \chi^2 (4) = 5.67$, $p> 0.05$, indicating that the inclusion of a quadratic term for time did not improve the fit of the model to the data. For the average person in this study, heart rate therefore apparently increased to an asymptote during the reactivity task and then fell linearly. Computations based on the fixed effects indicated that HR would have returned to baseline after about 11 minutes.

Modeling Systolic Blood Pressure. When modeling systolic blood pressure, I initially specified a quadratic model for reactivity. The fixed and random parameters of the models are shown in Table 6. For the quadratic reactivity model, the predicted mean systolic blood pressure (SBP) was 115.60 mmHg at baseline. During the task, the SBP increased by an average of 0.605 mmHg per minute and this increase was reduced by an average of -0.22 mmHg per minute (2 x -0.11, the first derivative of the coefficient, which is the conditional slope). This model was an appropriate fit for the data $\chi^2(17) = 35.516$, p =0.005, RMSEA=0.084, CFI = 0.992 and SRMR = 0.042. Although both the linear and quadratic terms were not significantly different from zero, the random effects

for both of the terms were significant, indicating that there was significant variability across the sample. However, because the terms were not significant, a linear model was also tested. Although the linear reactivity model was also an appropriate fit for the data, $\chi^2(23) = 141.99$, $p<0.001$, RMSEA=0.184, CFI = 0.947 and SRMR = 0.067, the chi-square difference test indicated that the linear model was a significantly worse fit than the quadratic model, $\Delta \chi^2 (4) = 46.275$, $p < 0.05$.

For the recovery data, I again specified a model with a quadratic term. The quadratic recovery model was an adequate fit for the data, $\chi^2(21) = 47.764$, $p = 0.007$, RMSEA=0.091, CFI = 0.990 and SRMR = 0.042. All three latent variables were significant, as were their variances. In this model, the predicted mean systolic blood pressure (SBP) was 116.45 mmHg at the end of the task. During the recovery period, the SBP decreased by an average of 0.990 mmHg per minute and this decline is reduced by an average of 0.208 mmHg per minute (2 x 0.104, the conditional slope). For the average participant, systolic blood pressure increased to a peak, and then it decreased, falling below the baseline value to about 114 mmHg and returning to the baseline value of about 115 mmHg.

Modeling Diastolic Blood Pressure. When modeling diastolic blood pressure, I initially specified a linear model for reactivity (see Table 7). For this model, the predicted mean diastolic blood pressure (DBP) was 63.38 mmHg at baseline. During the task, the DBP decreased by an average of 0.05 mmHg per minute, although this parameter was not significantly different from zero and variance for this parameter was not significant. This model was an appropriate fit for the data $\chi^2(18) = 72.496$, $p<0.001$, RMSEA=0.141, CFI = 0.967 and SRMR = 0.175. To test if there were changes in diastolic blood pressure not

solely predicted by linear change, I then specified a quadratic model. This model was also an appropriate fit for the data, $\chi^2(17) = 67.643$, $p<0.001$, RMSEA=0.140, CFI = 0.969 and SRMR = 0.134, and was a significant improvement over the linear model, $\Delta \chi^2 (1) = 4.853$, $p < 0.05$. In this model, the predicted mean DBP was 63.432 mmHg at baseline. During the reactivity period, the DBP instantaneously decreased by an average of 0.004 mmHg per minute and this decline is decreased by an average of 0.036 mmHg per minute (2 x 0.018, the conditional slope). Although the linear and quadratic fixed effects were not significantly different from zero, the random effects were both significant (see Table 4), so the quadratic term was retained in the reactivity model.

For the linear recovery model, the predicted mean diastolic blood pressure (DBP) was 62.837 mmHg at the end of the task. During the recovery period, the DBP decreased by an average of 0.09 mmHg per minute, although this parameter was not significantly different from zero and the random parameter was not significant. This model was an appropriate fit for the data $\chi^2(25) = 66.745$, $p<0.001$, RMSEA=0.104, CFI = 0.979 and SRMR = 0.066. A quadratic model was again tested to determine if there was significant nonlinear variance. The quadratic model was a good fit for the data, $\chi^2(21) = 41.252$, $p=0.005$, RMSEA=0.079, CFI = 0.990 and SRMR = 0.056. This model was a significant improvement over the linear model, $\Delta \chi^2 (4) = 25.493$, $p < 0.05$, indicating that there were significant individual differences in curvature during the recovery period. In this model, the predicted mean DBP was 63.212 mmHg at the end of the reactivity task. During the reactivity period, the DBP instantaneously decreased by an average of 0.487 mmHg per minute and this decline is decreased by an average of 0.106 mmHg per minute (2 x 0.053, the conditional slope). It appears that for the average person, there was no

significant change in diastolic blood pressure over the course of reactivity or recovery, but there were significant individual differences.

Generally speaking, it appears that this task increased sympathetic cardiac drive. This is evident from the general increase in HR and SBP to a peak during the imagery task. Both HR and SBP then fell back to baseline; HR fell in a linear fashion while SBP fell below baseline values and then rebounded. In contrast, there was no significant change in DBP, although there was significant variance for both the linear and quadratic random effects. In addition, this task appeared to initiate parasympathetic withdrawal (indicated by changes in HF HRV), as described below.

Level-1 Models for HRV Reactivity and HRV Recovery

Modeling Reactivity. Figure 2 shows the plots of the same 10 randomly selected participants as in Figure 1, which suggests a linear reactivity model for HRV, that is,

$$HF_{ij} = \pi_{0j} + \pi_{1j}(Time_{ij}) + r_{ij} \qquad (6).$$

Similar to the models specified above for the other cardiovascular parameters, in Equation (6), HF_{ij} is individual j's HF power at minute i, modeled as a function of an intercept $\pi_{0j,}$ which represents person j's initial HF power at Time 0 (i.e., average HF HRV), and a rate of change (π_{1j}), which is the rate that person j's change as a linear function of time. The residual (r_{ij}) is the deviation of person j's HF power at time i from what would be expected based on his or her initial HF power (π_{0j}) and the rate of linear change in his or her HRV (π_{1j}).

Figure 3 shows the linear latent grown curve model of HF power reactivity as a path diagram. These loadings are fixed to values that express information about the latent variables and the means, whereas the variance and covariance of the latent variables (π_{0j}

and π_{1j}) are estimated. The loadings for the latent intercept are 1, which represents the constant coefficient for the intercept in the model. The loadings for the slope are the corresponding times when the HF power was measured.

Table 8 lists the parameter estimates for the reactivity model of HF power. The HF power data was transformed using a natural log transformation. The predicted mean lnHF power was 7.063 $lnms^2$ at baseline. During the reactivity task, there was an average reduction of -0.140 $lnms^2$ per minute. There was also significant variability in both the baseline and reactivity values across participants, indicating that were individual differences in both the baseline and reactivity worth exploring at Level 2. The correlation between baseline and reactivity was -0.201 and statistically significant. This model was an adequate fit to the data, $\chi^2(20) = 56.807$, $p<0.001$, RMSEA = 0.112, CFI = 0.969, and SRMR = 0.067. I also evaluated attempted to run a quadratic model, to test if there were any additional sources of nonlinear variance, but this model would not converge because the latent variable covariance matrix was not positive definite.

Modeling Recovery. For recovery, I first tested a linear model. The parameter estimates for the linear model of recovery are shown in Table 9.For the linear recovery model, the predicted mean lnHF power was 6.796 $lnms^2$ at the end of the task. Once the task was complete, the HF power increased by an average of 0.031 $lnms^2$ per minute. In this model, the latent variable for recovery was significant, and there was significant variability in both the ending task and recovery values across participants, indicating that were individual differences worth exploring at Level 2. The correlation between reactivity and recovery was 0.012 and nonsignificant. This model was a good fit to the data with $\chi^2(25) = 39.932$, p=0.030RMSEA=0.063, CFI = 0.988 and SRMR = 0.049. To

test for nonlinear sources of variation, a quadratic model was also tested. Figure 4 shows the path diagram for recovery, with a quadratic parameter. The results of the parameter estimates for the quadratic model of recovery are also shown in Table 6.Although this model was an adequate fit to the data with $\chi^2(21) = 43.53$, p =0.023, RMSEA=0.064, CFI = 0.987 and SRMR = 0.064, the quadratic fixed effect was not significantly different from zero, and the variance terms (random effects) were not significant for either the linear or quadratic terms. The chi-square difference between the quadratic and linear models was also not significant, $\Delta \chi^2 (4) = 3.482$, $p > 0.05$, indicating that the inclusion of a quadratic term did not improve the fit of the model to the data.

Piecewise Regression of Reactivity and Recovery. Once the appropriate models for reactivity and recovery were determined, I united the reactivity and recovery data in a piecewise regression model, as described above. This model is summarized in Table 10. Indices of model fit indicated a good fit for the data, $\chi^2(83) = 149.017$, $p < 0.001$,RMSEA=0.073, CFI = 0.974 and SRMR = 0.077. In this model, the fixed effects estimated an average start value of 7.069 $lnms^2$, with an average reduction of 0.132 $lnms^2$per minute during the imagery task and an average recovery of 0.035 $lnms^2$per minute during the recovery period.

In the piecewise regression, there was significant variability in all of the random effects. The latent reactivity variable was significantly correlated with both baseline (-0.327) and recovery (-0.650), with higher baseline values associated with larger reductions in HF power during the reactivity period and a larger recovery after the task was complete. The correlation between the baseline and recovery variables was not significant.

I then restated the piecewise model to incorporate specifications of possible causal relations among the latent values. In particular, baseline values were posited to influence reactivity values, and baseline and reactivity values were both posited to influence recovery. As shown in Figure 5, baseline levels directly affected reactivity and reactivity directly affected recovery. The dashed line between baseline and recovery represents the covariance pathway that was removed from the model reported above. Therefore this model is nested within the previous model. The chi-square difference between these two models was not significant, $\Delta\chi^2$ (1) = 0.601, p> 0.05, indicating that the more restricted model was comparable to the less restrictive model. The fit of this restated model was quite good, $\chi^2(84) = 149.618$, $p<0.001$,RMSEA=0.072, CFI = 0.975 and SRMR = 0.081.

Overall, parasympathetic withdrawal is evident in this task by the linear decrease of HF HRV during the reactivity task, and the consequent linear increase in HF HRV in the recovery period, although at a much slower rate (i.e., calculations on the fixed effects implied that it would have taken 7-8 minutes HF power to return to baseline, even though the task-related declines took place over a much shorter, 4-minute period. The baseline values then predicted reactivity, and reactivity subsequently predicted recovery. There was no evidence of a direct effect of baseline values on recovery.

Level-2 Predictors of HRV Reactivity and HRV Recovery

Whereas Equations (1), (2), and (3) parameterize the observations of a single individual (called a “Level-1” or “within-persons” equation), additional “between-persons” or “Level-2” equations can be used to specify the between-person variation that occurs within a sample. Level-2 equations model the parameters in a Level-1 equation as

the result of (a) expected parameter estimates for the entire sample (fixed effects) and (b) person-specific deviations from the expected values (random effects). Person-specific variations in linear change, for example, in Equation (3) can be decomposed according to the following between-persons or Level-2 model:

$$\pi_{0j} = \beta_{00} + u_{0j} \quad (7)$$

$$\pi_{1j} = \beta_{10} + u_{1j} \quad (8).$$

In Equations (7) and (8), person *j*'s initial HF power (π_{0j}) and rate of linear change (π_{1j}) are expressed as functions of fixed effects (β_{00}, β_{10}) and random effects (u_{0j}, u_{1j}). The fixed effect (β_{00}) is the expected initial HF power for the entire same and the random effect (u_{0j}) is the deviation of person *j*'s parameter estimate (π_{0j}) from the corresponding fixed effect. Similarly, $\beta_{10}$is the expected rate of linear change for the entire sample and the random effect (u_{1j}) is the deviation of person *j*'s parameter estimate for linear change π_{1j} from the fixed effect β_{10}. The fixed effect estimates the typical initial amount of HF power or degree of linear change that an individual from our sample can be expected to experience. At the same time, the random effect estimates the extent that person *j*'s HF power differs from the "average" person in the sample.

To introduce potential predictors, each possible covariate was tested individually with the model described above. Table 11 contains the results of these models. Each variable was tested as a predictor of individual differences by adding a path from the predictor to each latent variable (see Figure 6 for an example), with the exception of those variables that resulted from the LIWC analysis, which I expected to be related only to the reactivity and recovery latent variables. All of the models were an appropriate fit for the data, with *CFI*s$\geq$ 0.970, RMSEA $\leq$ 0.079 and SRMR $\leq$ 0.074.

Contrary to my hypotheses, neuroticism and hostility were not significantly related to the baseline, reactivity, or recovery. Therefore, emotion regulation strategies were not evaluated as possible mediators for these personality and trait variables. Age was a significant predictor of baseline values, as was positive affect from the PANAS. None of the post-task emotion factors (i.e., positive affect, angry affect, negative affect) were significantly related to the piecewise model.

Of the LIWC variables, only cognitive processing was a significant predictor of reactivity. However, because only 60% of the sample completed free response descriptions of their thoughts during the task, this predictor was not investigated further or included in the final model.

Among the emotion regulation strategies, positive reappraisal predicted baseline and reactivity in the piecewise regression model. Benefits to the relationship predicted baseline values, and perspective-taking and positive refocusing was associated with recovery. I began with positive reappraisal, as it was a significant predictor of both baseline and reactivity, and then added each subsequent predictor, beginning with age. Direct effects for age on baseline, and positive reappraisal on baseline and reactivity were all significant. Model fit was good, $\chi^2(109) = 183.198$, $p<0.001$,RMSEA=0.066, CFI = 0.971 and SRMR = 0.071. Positive refocusing was then added to the model and there was again good model fit, $\chi^2(122) = 200.911$, $p<0.001$, RMSEA=0.066, CFI = 0.970 and SRMR = 0.071, and each of the direct effects remained significant. The chi-square difference between these two models was not significant, $\Delta\chi^2$ (13) = 17.713, $p> 0.05$, indicating that the two models were comparable in fit, so the direct effect of positive refocusing was retained. I then attempted to add perspective taking, benefits to the

relationship and positive affect to the model, but the direct effects were not significant and did not cause an improvement in model fit, so they were not retained. The final model (see Figure 7 for the structural model and Table 12 for effect parameters) explained 12% of the variance in baseline values, 17% of the variance in reactivity, and 46% of the variance in recovery.

Predictors of baseline HF power.Examination of the partial effect parameters revealed that for age, which was a significant predictor of baseline values, each 1-year increase in participants' ages was associated with a decrease of 0.068 $lnms^2$in baseline values. While controlling for age, each 1-unit increase in positive reappraisal was associated with a 0.153 $lnms^2$increase in baseline HF HRV.

Predictors of HF power reactivity. Baseline HF HRV values related to reactivity such that every 1.00 $lnms^2$increase in baseline value was associated with a -0.074 $lnms^2$decrease in reactivity (i.e., increased parasympathetic withdrawal). Similarly, controlling for baseline values, each unit increase in the positive reappraisal subscale was associated with 0.036 $lnms^2$higher slopes, indicating less parasympathetic withdrawal during the task.

Predictors of HF power recovery. Finally, each 1.00 $lnms^2$change in task reactivity (parasympathetic withdrawal) was associated with 0.215 $lnms^2$ higher recovery scores (indicative of parasympathetic reengagement), which suggests that people with less parasympathetic withdrawal during the task also had quicker rates of parasympathetic recover after the task ended. Also, each 1-unit increase in positive refocusing was associated with 0.010 $lnms^2$ lower recovery scores, which suggests that positive refocusing predicted slower parasympathetic recovery.

Chapter 4

Discussion

Segerstrom and Solberg Nes (2007) suggested that HRV may be viewed as both an indicator of self-regulatory strength and as an indicator of self-regulatory effort. The results of this study, which was designed to model the parasympathetic nervous system's response to an emotional task and link emotion regulation and personality variables to individual differences in HRV, corroborates their assertion.

Characterization of the Task

The analyses of the various systems monitored during the task in the study (i.e., heart rate, systolic and diastolic blood pressure, HRV) together indicated which physiological systems were engaged by this task. As heart rate increased, along with systolic blood pressure, there is evidence that the sympathetic nervous system was at work. Simultaneously, it appears that parasympathetic withdrawal was also at work during the task: HF power decreased linearly during the task and then rebounded during the recovery period, although it took more than twice as long to recover fully from the task-related reductions in HF power. The sympathetic nervous system also appeared to withdraw during the recovery, as indicated by the decline of SBP, which also contributed to the decline of heart rate during the monitored recovery period. Therefore, it seems reasonable to conclude that our laboratory task (i.e., having participants imagine what a transgressor had done to them, and to then imagine interacting with them), engaged both branches of the autonomic nervous system.

Predictors of HRV

HF power was not related to personality factors as hypothesized. Neither hostility nor neuroticism levels were significant predictors of HRV baseline, reactivity, or recovery values. Although the tendency to experience positive affect was a significant predictor of baseline HF HRV values, it did not add any value to the final model and was not a significant predictor when other variables were also included. This finding suggests that HRV is more closely tied with emotion regulation than emotional experience. In fact, two emotion regulation strategies, positive refocusing and positive reappraisal, did predict individual differences in HF power.

Positive refocusing was a negative predictor of recovery. That is, people who reported engaging in high levels of positive refocusing (e.g., thinking that one can become a stronger person as a result of what happened) experienced slower recovery in parasympathetic functioning during the recovery period. Although this finding was not expected, there is evidence in the cognitive emotion regulation literature that positive refocusing may be related to poorer self-regulation. For example, Garnefski, Kraaif, and van Etten (2005) found that the use of positive refocusing was positively correlated with externalizing problems (e.g., delinquent and aggressive behavior) in adolescents. Taken together, these findings suggest that positive refocusing, a strategy that reflects a tendency to distract oneself from a negative situation, may ultimately be an ineffective approach. More puzzling, however, is the fact that this same measure was negatively correlated with depression in adults (Garnefski et al., 1995), suggesting that positive refocusing is partially effective in regulating mood.

A more easily interpreted finding in this study was the association of positive reappraisal with both baseline and reactivity HRV values. People who used positive reappraisal as a coping strategy experienced higher initial levels of parasympathetic activation and less parasympathetic withdrawal during the reactive task. These results support Segerstrom and Solberg Nes's (2007) findings that HRV is an indicator of both self-regulatory strength (i.e., baseline) and effort (i.e., reactivity). Positive reappraisal, or the ability to reframe a negative situation and find benefits, therefore seems to capture a particularly powerful aspect of self-regulation. Similarly, Thayer and Lane (2000, in press) describe the psychological flexibility that is associated with higher vagal tone, particularly within the context of mental health. Their theoretical model highlights the correlation between high HRV and the ability to self regulate.

This study is not the first to evaluate cardiovascular measures in the context of positive reappraisal. However, the most of the previous studies examined patterns of general cardiovascular responding or sympathetic activation, rather than parasympathetic activation. For example, Mauss, Cook, Cheng and Gross (2007) induced anger in people high and low in reappraisal. Participants who were higher in reappraisal displayed a pattern of cardiac output associated with an adaptive challenge response (i.e., high cardiac output and ventricular contractility with lower total peripheral resistance), as compared to those who were low in reappraisal.

Regarding the sympathetic nervous system, positive reappraisal has often been compared to an alternative strategy of emotion regulation: suppression. Researchers have found that reappraisal has not had a discernable effect on sympathetic activation, although it effectively regulates the both the experience and expression of negative

emotion. Alternatively, suppression, which also inhibits the expression of negative emotion, has been linked with physiological costs (i.e., greater sympathetic activation; Egloff, Schmukle, Burns, &Schwerdtfeger, 2006; Gross, 1998a). Similarly, Ray, Wilhelm and Gross (2008) compared reappraisal to rumination and found that people who engaged in reappraisal experienced less anger, cognitive perseveration, and sympathetic nervous system activation than those who engaged in rumination.

Recently, researchers have also begun to examine indicators of parasympathetic activation when studying reappraisal. Among recently bereaved adults, participants with higher baseline HRV endorsed more active coping and acceptance and less passive coping (O'Connor, Allen, and Kasniak, 2002). Using an experimental design, Butler, Wilhelm & Gross (2006) found that women in a discussion who tried to regulate their emotions (using either suppression or reappraisal) showed greater increases in RSA than participants who did not try to regulate their emotions.

The connection of HRV with positive reappraisal as a specific emotion regulation strategy may indicate a potential avenue to impact problematic human responses to difficult situations. A preliminary study suggests that this may be promising route. Volokhov (2008) showed participants a total of four video clips (two positive and two negative) while recording physiological data. As expected, participants with high RSA at baseline naturally engaged in more reappraisal during the clips. However, participants with low RSA showed more improvement in implementing reappraisal after learning about reappraisal as strategy for regulating emotion.

The use of positive reappraisal, a well-documented strategy of emotion regulation, has been shown to affect the cardiovascular system and HRV in the studies described

above. This study is the first to show that positive reappraisal was simultaneously related to both baseline and change in HRV, which indicates that it is linked with both strength and effort of self-regulation. Overall, these results add to the evidence that positive reappraisal is a powerful component of emotion regulation, and may be an important intervention target.

Null Findings

Several parameters that were expected to predict HRV, particularly the personality variables of hostility and neuroticism, were not significant predictors. The lack of relationship between these variables may be due to a number of factors. First, the null findings regarding hostility and neuroticism could be due to the use of different scales. For example, Riese et al. (2007) measured neuroticism four distinct times using the NEO-FFI and the Eysenck Personality Questionnaire, ultimately relating HRV to a latent factor of neuroticism. Regarding hostility, the two studies that found significant relationships between the target variable and HRV used the Cook-Medley Hostility Scale (Demaree& Everhart, 2004; Neumann, Waldstein et al., 2004), whereas I used the Aggression Questionnaire. Although ideally these two scales would measure this factor equally well, it may be that they are measuring slightly different constructs.

Second, the lack of relationship may be due to the HRV recording, particularly at baseline. Virtanen (2003) suggested that they did not find a connection between hostility and tonic levels of HRV due to recording length, as their measurement period was 5 minutes. In the present study, baseline readings were taken for only four minutes, whereas Demaree and Everhart (2004) used a 10-minute baseline period, and others (e.g.,

Sloan et al., 2001) have used 24-hour recordings. A longer recording may be necessary to find trait variation in baseline levels.

Finally, it appears that this was not an anger-inducing task. Although there was evidence of variability in the hostility variable (see Table 1), the mean for post-task anger was only 2.03, which is defined on the rating scale as "slightly." The lack of anger activation may explain why hostility was not related to the reactivity pattern during the task.

Limitations of the Current Study and Directions for Future Research

The findings in this study are limited by our failure to collect several crucial pieces of data that might have been relevant to participants' physiological functioning. Participants in this study were not screened for psychiatric illness, medication usage, history of physical illness, alcohol or drug abuse, smoking or caffeine use. The time of day that participants were involved in the data collection was also not standardized. With more control over these potential sources of error variance, we would have been in a better position to rule out some biomedical variables that could have been responsible for spurious relations between positive reappraisal and HRV.

Analytically, there are several limitations to this study. The large number of variables investigated in the study resulted in an inflation of study-wise alpha, which may have led to a high false-positive rate. Also, as was mentioned above, there were also some variables that lacked elevation and variation (e.g., the post-task affect variables), which makes it more difficult to find a significant relationship with physiological variables. It may be that participants were able to modulate their negative affect through the strategies described here, resulting in the significant relationships using positive

refocusing and positive reappraisal. On the other hand, it appeared that participants did not, on average, become particularly angry during the imagery tasks, so they might not have had much affect to regulate in the first place.

Although I found no relationships between personality and HRV, several other research groups have been successful in this regard (Demaree & Everhart, 2004; Neumann et al., 2004; Riese et al., 2007). Although I examined reactivity in response to a stressor and used a latent growth curve model to separate true change from measurement error, error was also introduced by the lack of controls surrounding data collection. Further work should be done, building upon this model but also including more stringent methods of data collection, to test the potential associations of personality variables with tonic levels of HRV, the magnitude of HRV response, and the HRV response pattern.

If I were to investigate these questions again in a subsequent study, I would alter the design in several ways. First, I would more carefully screen participants for medications, pre-existing conditions, and substance use. Second, I would collect physiological data at a consistent time of day. Third, before to collected physiological data and asked participants to think about a real life negative event, I would prime the relevant emotion regulation strategies, specifically positive reappraisal, suppression, and rumination, using an adaptation of the Sentence Unscrambling Task (Mauss, Cook, & Gross, 2007). Fourth, I would employ both an emotion-priming video, which would be standardized across participants, as well as retrospective life experience. Fifth, I would use alternative self-report scales, specifically the Cook-Medley Hostility scale to measure hostility, the NEO-FFI to measure neuroticism, and Gross's Emotion Regulation Questionnaire (ERQ) for reappraisal, rather than the CERQ. Finally, when measuring

HRV, I would allow participants a longer baseline so they would have more time to become acclimated to the task.

Examining the individual predictors of the other physiological variables measured in this study (i.e., HR, SBP and DBP) was outside of the scope of this inquiry. However, the presence of significant variability in the latent variables of HR, SBP and DBP baseline, reactivity and recovery indicates meaningful individual differences in physiological responses to the laboratory task that are worthy of exploration. Potentially, some of the emotion regulation variables that we considered here as predictors of HF power might also predict differences in task-related changes in heart rate and blood pressure.

It might also be productive to further evaluate linguistic measures of cognitive emotion regulation processes as predictors of HRV (Lepore et al., 2002; Pennebaker et al., 2003). The preliminary relationship between the cognitive mechanisms variable and reactivity that I found in this data set seems like a promising beginning. However, because the linguistic data were not available for all participants, it wasn't possible to completely evaluate cognitive mechanisms in the context of the full model. Furthermore, the relationship between cognitive restructuring and the tendency to use positive reappraisal and other cognitive emotion regulation strategies could be directly tested with more data. Perhaps the tendency for positive reappraisal is indicative of baseline self-regulatory strength, as discussed above, but a language analysis approach would better predict HRV as an indicator of effort during the task.

Given the importance of both self-regulatory strength and effort, it would be useful to target both in interventions. The notion that one could use the feedback

provided by HRV as an indicator of one's self-regulatory reserves was suggested by Segerstrom and Solberg Nes (2007). Given the findings from this study, that individuals who used more positive reappraisal had high levels of HF power HRV, people may benefit most from increasing first their self-regulatory strength (i.e., understanding of and ability to use positive reappraisal) and, consequently, their self-regulatory effort. Determining the directionality in the relationship of positive reappraisal with HRV and self-regulation is crucial: If increasing people's ability to use positive reappraisal in turn impacts HRV and overall self-regulation, this might explain the previously established links between depressive symptoms and HRV (Sharpley, 2002; Udupa et al., 2007; Voss et al., 2006). If a bi-directional relationship exists between these constructs, it would be possible to target either of them and see an increase in self-regulatory ability.

Similarly to Segerstrom and Solberg Nes (2007), Gyurak and Ayduk (2008) found that resting RSA predicted people's ability to effectively regulate their emotions and suggested that it may serve as a protective factor for vulnerable populations (e.g., persons with high rejection sensitivity). Both research groups have suggested targeting HRV using previously validated strategies such as physical exercise (see Sandercock, Bromley &Brodie, 2005, for a review) or biofeedback (Lehrer et al., 1997, 2003; Lehrer, Vaschillo&Vaschillo; 2000). A recent study by Karavidas et al. (2007) provided evidence that HRV biofeedback may impact symptoms of depression. The biofeedback is designed to strengthen baroreflexes, which improves autonomic stability, ultimately resulting in increased emotional stability (Karavidas, 2008). In studies conducted by this research group thus far, the intervention appeared to stimulate the vagus nerve both acutely (i.e., in session) and chronically (i.e., between sessions). Increasing baseline HRV through these

types of interventions may also increase self-regulatory strength. One could also target positive reappraisal as a complementary intervention to increase self-regulatory effort, thus enhancing overall emotion regulation, which is a key component of mental health.

Despite its limitations, the present study provides a more complete model of the process of reactivity and recovery of HRV following an interpersonal stressor and evidence that a specific cognitive regulation process (i.e., positive reappraisal) is related to both tonic individual differences and differences in reactivity to the task. By combining a growth modeling approach to measuring physiological reactivity and recovery with an experimental paradigm that manipulates positive reappraisal, researchers could determine in the future whether these effects are in fact causal, which could have far-reaching implications for self-regulation.

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Tables

Table 1. *Means and Standard Deviations for Self Report Variables*

COVARIATE	**Mean**	**Standard Deviation**
Age	19.65	4.02
Positive Affect (PANAS)	3.66	0.64
Negative Affect (PANAS)	2.25	0.71
Hostility	2.55	0.67
Neuroticism	2.97	0.86
Positive Affect (post-task)	1.08	1.23
Angry Affect (post-task)	2.03	1.37
Negative Affect (post-task)	0.93	0.95
LIWC Variables		
Word Count (Task)*	37.57	20.20
Cognitive Mechanisms (Task)*	13.90	7.25
Positive Emotions (Task)*	1.57	2.51
Negative Emotions (Task)*	4.65	5.56
Subscales from the CERQ		
Self Blame	2.09	0.99
Acceptance	3.69	1.09
Rumination	3.77	0.88
Positive Refocusing	2.71	1.15
Refocus on Planning	3.15	0.95
Positive Reappraisal	3.22	0.95
Putting Into Perspective	30.4	1.16
Catastrophizing	2.82	1.00
Blaming Others	3.11	1.35
Humble Self Appraisals	2.58	0.96
Perspective Taking	3.02	1.15
Benefits to Relationship	2.14	1.20
Benign Causal Attributions	2.83	0.99
Focus of Positive Aspects of Relationship	2.62	1.16
Mental Stimulation of Positive Outcomes	2.95	1.10
Emulation of Moral Exemplars	3.05	1.09
Focus on Positive Aspects of the Transgressor	2.76	1.16

**N* = 89

Table 2. *Means and Standard Deviations for Physiology Variables*

COVARIATE	Mean	Standard Deviation
Ln HF HRV (Minute 1, Baseline)	7.25	1.03
Ln HF HRV (Minute 2, Baseline)	7.15	1.04
Ln HF HRV (Minute 3, Baseline)	7.01	1.05
Ln HF HRV (Minute 4, Baseline)	7.00	1.05
Ln HF HRV (Minute 5, Reactivity)	6.80	1.04
Ln HF HRV (Minute 6, Reactivity)	6.76	1.04
Ln HF HRV (Minute 7, Reactivity)	6.64	1.06
Ln HF HRV (Minute 8, Recovery)	6.96	1.00
Ln HF HRV (Minute 9, Recovery)	6.91	0.97
Ln HF HRV (Minute 10, Recovery)	6.87	1.00
Ln HF HRV (Minute 11, Recovery)	6.95	1.07
Ln HF HRV (Minute 12, Recovery)	6.92	1.02
Ln HF HRV (Minute 13, Recovery)	6.97	1.06
Ln HF HRV (Minute 14, Recovery)	7.02	1.09
Heart Rate (Minute 1, Baseline)	74.08	10.67
Heart Rate (Minute 2, Baseline)	74.45	10.86
Heart Rate (Minute 3, Baseline)	75.46	10.76
Heart Rate (Minute 4, Baseline)	75.30	10.60
Heart Rate (Minute 5, Reactivity)	79.50	11.01
Heart Rate (Minute 6, Reactivity)	77.63	10.90
Heart Rate (Minute 7, Reactivity)	77.41	10.96
Heart Rate (Minute 8, Recovery)	76.38	10.96
Heart Rate (Minute 9, Recovery)	76.60	10.99
Heart Rate (Minute 10, Recovery)	76.50	10.54
Heart Rate (Minute 11, Recovery)	76.04	10.48
Heart Rate (Minute 12, Recovery)	75.90	10.74
Heart Rate (Minute 13, Recovery)	75.97	10.54
Heart Rate (Minute 14, Recovery)	75.76	10.75
Systolic BP (Minute 1, Baseline)	116.29	11.88
Systolic BP (Minute 2, Baseline)	115.87	11.76
Systolic BP (Minute 3, Baseline)	115.43	11.39
Systolic BP (Minute 4, Baseline)	115.17	11.23
Systolic BP (Minute 5, Reactivity)	115.89	11.31
Systolic BP (Minute 6, Reactivity)	116.63	11.57
Systolic BP (Minute 7, Reactivity)	116.46	11.91
Systolic BP (Minute 8, Recovery)	115.50	11.53
Systolic BP (Minute 9, Recovery)	114.99	11.30
Systolic BP (Minute 10, Recovery)	114.50	11.37
Systolic BP (Minute 11, Recovery)	114.12	11.50
Systolic BP (Minute 12, Recovery)	114.25	11.63
Systolic BP (Minute 13, Recovery)	114.08	11.99

COVARIATE	Mean	Standard Deviation
Systolic BP (Minute 14, Recovery)	114.77	12.12
Diastolic BP (Minute 1, Baseline)	65.81	11.11
Diastolic BP (Minute 2, Baseline)	64.40	10.89
Diastolic BP (Minute 3, Baseline)	63.72	10.99
Diastolic BP (Minute 4, Baseline)	62.85	10.88
Diastolic BP (Minute 5, Reactivity)	63.25	11.82
Diastolic BP (Minute 6, Reactivity)	63.51	11.94
Diastolic BP (Minute 7, Reactivity)	63.26	11.86
Diastolic BP (Minute 8, Recovery)	62.49	11.98
Diastolic BP (Minute 9, Recovery)	62.81	12.13
Diastolic BP (Minute 10, Recovery)	63.02	11.83
Diastolic BP (Minute 11, Recovery)	62.09	11.23
Diastolic BP (Minute 12, Recovery)	61.91	11.94
Diastolic BP (Minute 13, Recovery)	62.40	11.70
Diastolic BP (Minute 14, Recovery)	62.61	11.35

Table 3. *Correlations Among Major Study Self-Report Variables*

Measures	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*
1. Self Blame	--	.05	.24**	-.12	.04	.08	-.08	.33**	-.22**	-.04	-.02	.05	-.03	.17*
2. Acceptance		--	.01	.19*	-16	.40**	.25**	-.07	-.05	-.01	.05	-0.01	-0.22**	-0.09
3. Rumination			--	-.06	.22**	.07	-.20*	.40**	.09	.05	.20*	-0.01	0.07	0.16*
4. Positive Refocusing				--	.33**	.25**	.30**	-.21**	.01	.09	-.24**	0.09	-0.24**	-0.21*
5. Refocus on Planning					--	.33**	.18*	.10	.04	.01	-.09	0.13	-0.18*	0.03
6. Positive Reappraisal						--	.39**	-.05	-.14	-.03	-.16	0.14	-0.17*	0.01
7. Putting Into Perspective							--	-.42**	-.04	-.27**	-.20*	0.13	-0.11	-0.08
8. Catastrophizing								--	.01	.11	.17*	-0.04	0.08	0.15
9. Blaming Others									--	.13	.05	-0.05	0.01	-0.13
10. Hostility										--	.38**	-.07	.17*	-.02
11. Neuroticism											--	-.01	.10	.23**
12. Positive Affect												--	-0.29**	0.19*
13. Angry Affect													--	0.41**
14. Negative Affect														--

Table 4. *Fixed and Random Parameter Estimates for Heart Rate in the Quadratic Reactivity Model*

	Estimate (beats/minute)	SE	*t*
Fixed parameters			
Baseline	75.274	0.868	86.746
Task	2.369	0.300	7.909
$Task^2$	-0.540	0.092	-5.884
Random parameters			
Variances			
Baseline	110.577	12.955	8.535
Task	10.327	2.543	4.061
$Task^2$	0.962	0.266	3.615
Covariances			
Baseline/Task	-1.909	3.171	-0.602
Baseline/$Task^2$	0.446	0.970	0.460
Task/$Task^2$	-2.997	0.816	-3.674

Table 5. *Fixed and Random Parameter Estimates for Heart Rate in the Linear and Quadratic Recovery Models*

	Estimate (beats/minute)	SE	*t*
Linear Model			
Fixed parameters			
Task	76.890	0.887	86.705
Recovery	-0.172	0.049	-3.509
Random parameters			
Variances			
Task	114.884	13.576	8.462
Recovery	0.226	0.042	5.364
Covariances			
Task/Recovery	-1.307	0.550	-2.376
Quadratic Model			
Fixed parameters			
Task	77.087	0.895	86.130
Recovery	-0.317	0.122	-2.595
Recovery ^{2}	0.020	0.016	1.198
Random parameters			
Variances			
Task	115.001	13.837	8.311
Recovery	0.549	0.299	1.836
Recovery ^{2}	0.009	0.005	1.747
Covariances			
Task/Recovery	-1.651	1.397	-1.182
Recovery / Recovery ^{2}	0.067	0.182	0.368
Recovery / Recovery ^{2}	-0.056	0.038	-1.466

Table 6. *Fixed and Random Parameter Estimates for Systolic Blood Pressure in the Quadratic Reactivity and Recovery Models*

	Estimate (mmHg)	SE	*t*
Reactivity			
Fixed parameters			
Baseline	115.522	0.908	127.211
Task	0.662	0.392	1.689
Task^2	-0.117	0.120	-0.971
Random parameters			
Variances			
Baseline	122.107	14.374	8.495
Task	12.870	2.782	4.627
Task^2	1.324	0.259	5.110
Covariances			
Baseline/Task	-4.590	4.391	-1.045
Baseline/Task^2	1.520	1.350	1.126
Task/Task^2	-3.782	0.826	-4.580
Recovery			
Fixed parameters			
Task	116.453	0.959	121.490
Recovery	-0.990	0.197	-5.029
Recovery ^{2}	0.104	0.027	3.867
Random parameters			
Variances			
Task	139.526	16.206	8.610
Recovery	3.758	0.841	4.470
Recovery ^{2}	0.052	0.016	3.174
Covariances			
Task/Recovery	-9.253	2.636	-3.510
Recovery / Recovery ^{2}	1.000	0.347	2.882
Recovery / Recovery ^{2}	-0.402	0.110	-3.666

Table 7. *Fixed and Random Parameter Estimates for Diastolic Blood Pressure in the Linear and Quadratic Reactivity and Recovery Models*

	Estimate (mmHg)	SE	*t*
Linear Reactivity Model			
Fixed parameters			
Baseline	63.375	0.875	72.433
Task	-0.050	0.201	-0.249
Random parameters			
Variances			
Baseline	109.419	13.216	8.279
Task	1.902	1.095	1.737
Covariances			
Baseline/Task	-1.284	2.246	-0.572
Quadratic Reactivity			
Fixed parameters			
Baseline	63.432	0.864	73.428
Task	-0.004	0.504	-0.009
$Task^2$	-0.018	0.164	-0.110
Random parameters			
Variances			
Baseline	105.253	12.873	8.176
Task	19.917	4.412	4.514
$Task^2$	2.595	0.468	5.542
Covariances			
Baseline/Task	11.788	5.313	2.219
Baseline/$Task^2$	-4.67	1.790	5.215
Task/$Task^2$	-6.323	1.377	-4.593
Recovery Model			
Fixed parameters			
Task	62.837	0.943	66.616
Recovery	-0.097	0.091	-1.070
Random parameters			
Variances			
Task	126.181	15.663	8.056
Recovery	0.731	0.164	4.462
Covariances			
Task/Recovery	-3.006	1.160	-2.593

Table 8. *Fixed and Random Parameter Estimates for HF power in the Linear Reactivity Model*

	Estimate ($lnms^2$)	SE	*t*
Fixed parameters			
Baseline	7.063	0.080	88.348
Task	-0.140	0.020	-6.940
Random parameters			
Variances			
Baseline	0.897	0.110	8.136
Task	0.055	0.007	7.764
Covariances			
Baseline/Task	-0.044	0.020	-2.179

Table 9. *Fixed and Random Parameter Estimates for HF power in the Quadratic and Linear Recovery Models*

	Estimate ($lnms^2$)	SE	*t*
Quadratic Model			
Fixed parameters			
Task	7.086	0.081	87.586
Recovery	0.064	0.024	2.714
Recovery ^{2}	-0.005	0.003	-1.549
Random parameters			
Variances			
Task	0.784	0.116	6.769
Recovery	0.003	0.012	0.269
Recovery ^{2}	<0.001	<0.001	0.364
Covariances			
Task/ Recovery	0.002	0.207	0.087
Task/ Recovery ^{2}	<0.001	0.004	-0.126
Recovery / Recovery ^{2}	<0.001	0.002	-0.139
Linear Model			
Fixed parameters			
Task	6.796	0.078	87.227
Recovery	0.031	0.008	3.844
Random parameters			
Variances			
Task	0.781	0.105	7.403
Recovery	0.003	0.001	2.348
Covariances			
Task/ Recovery	0.001	0.008	0.070

Table 10. *Fixed and Random Parameter Estimates for HF power in the Combined Reactivity and Recovery Model*

	Estimate ($lnms^2$)	SE	*t*
Fixed parameters			
Baseline	7.069	0.081	87.350
Task	-0.132	0.023	-5.769
Recovery	0.035	0.008	4.476
Random parameters			
Variances			
Baseline	0.883	0.109	8.088
Task	0.032	0.009	3.723
Recovery	0.003	0.001	2.981
Covariances			
Baseline/Task	-0.055	0.022	-2.500
Baseline/Recovery	0.008	0.007	1.075
Task/Recovery	-0.007	0.003	-2.442

Table 11. *Parameter Estimates for Level-2 Predictors Added to the HF power in the Combined Reactivity and Recovery Model*

	Baseline		**Reactivity**		**Recovery**	
COVARIATE	**Est**	**z-score**	**Est**	**z-score**	**Est**	**z-score**
Gender	0.119	0.699	0.021	0.485	0.000	0.011
Age	-0.070	**-3.698**	0.003	0.605	0.001	0.564
Positive Affect (PANAS)	0.293	**2.169**	0.004	0.121	0.004	0.484
Negative Affect (PANAS)	0.048	0.427	-0.006	-0.204	0.002	0.347
Hostility	0.139	1.167	0.039	1.296	0.001	0.181
Neuroticism	-0.046	-0.498	0.027	1.150	0.003	0.455
Positive Affect (post-task)	0.034	0.518	0.009	0.560	0.001	0.313
Angry Affect (post-task)	-0.017	-0.292	-0.013	-0.886	0.002	0.666
Negative Affect (post-task)	-0.013	-0.154	-0.034	-1.582	0.000	0.031
LIWC Variables						
Word Count (Task)			-0.003	-2.007	0.000	0.823
Cognitive Mechanisms (Task)			0.008	**2.260**	0.000	-0.244
Positive Emotions (Task)			0.005	0.412	0.002	0.885
Negative Emotions (Task)			-0.001	-0.161	0.001	0.807
Subscales from the CERQ						
Self Blame	0.075	0.937	0.010	0.484	-0.006	-1.119
Acceptance	0.125	1.730	0.029	1.560	-0.005	-0.981
Rumination	0.134	1.493	-0.021	-0.918	0.005	0.830
Positive Refocusing	0.111	1.626	-0.005	-0.286	-0.099	**-2.077**
Refocus on Planning	0.037	0.441	-0.013	-0.590	-0.004	-0.710
Positive Reappraisal	0.156	**2.255**	0.040	**2.205**	-0.006	-1.217
Putting Into Perspective	-0.003	-0.043	-0.002	-0.101	-0.003	-0.638
Catastrophizing	0.020	0.246	0.021	1.057	0.004	0.775
Blaming Others	0.025	0.422	0.002	0.126	0.004	1.159
Humble Self Appraisals	0.079	0.957	0.029	1.360	-0.004	-0.735
Perspective Taking	0.025	0.202	0.037	1.167	-0.019	**-2.417**
Benefits to Relationship	0.132	**2.021**	0.011	0.638	-0.002	-0.510
Benign Causal Attributions	0.053	0.658	0.013	0.649	-0.003	-0.622
Focus of Positive Aspects of Relationship	0.097	1.418	0.023	1.323	-0.003	-0.595
Mental Stimulation of Positive Outcomes	0.105	1.460	0.017	0.908	-0.003	-0.730
Emulation of Moral Exemplars	0.035	0.476	-0.005	-0.262	0.000	-0.095
Focus on Positive Aspects of the Transgressor	0.095	1.546	0.026	1.638	-0.003	-0.662

Table 12. *Effect Parameters of the Final Model*

Criterion	Predictor	Estimate ($lnms^2$)	SE	*t*
Baseline	Age	-0.068	0.019	-3.679
	Positive Reappraisal	0.153	0.066	2.313
Task Reactivity	Positive Reappraisal	0.036	0.018	2.057
	Baseline	-0.074	0.022	-3.372
Recovery	Positive Refocusing	-0.010	0.004	-2.325
	Task Reactivity	-0.215	0.045	-4.741

Figures

Figure 1. *Heart Rate of 10 randomly selected cases*

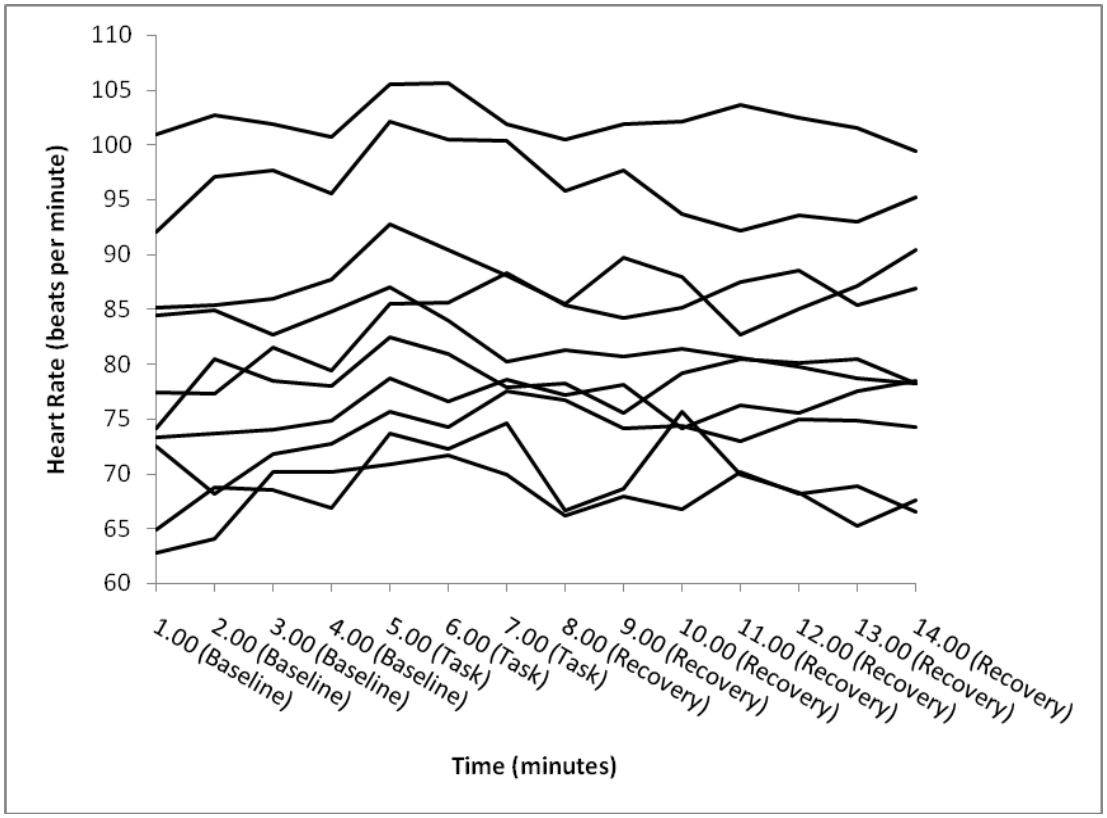


Figure 2. *Natural log HF power of 10 randomly selected cases.*

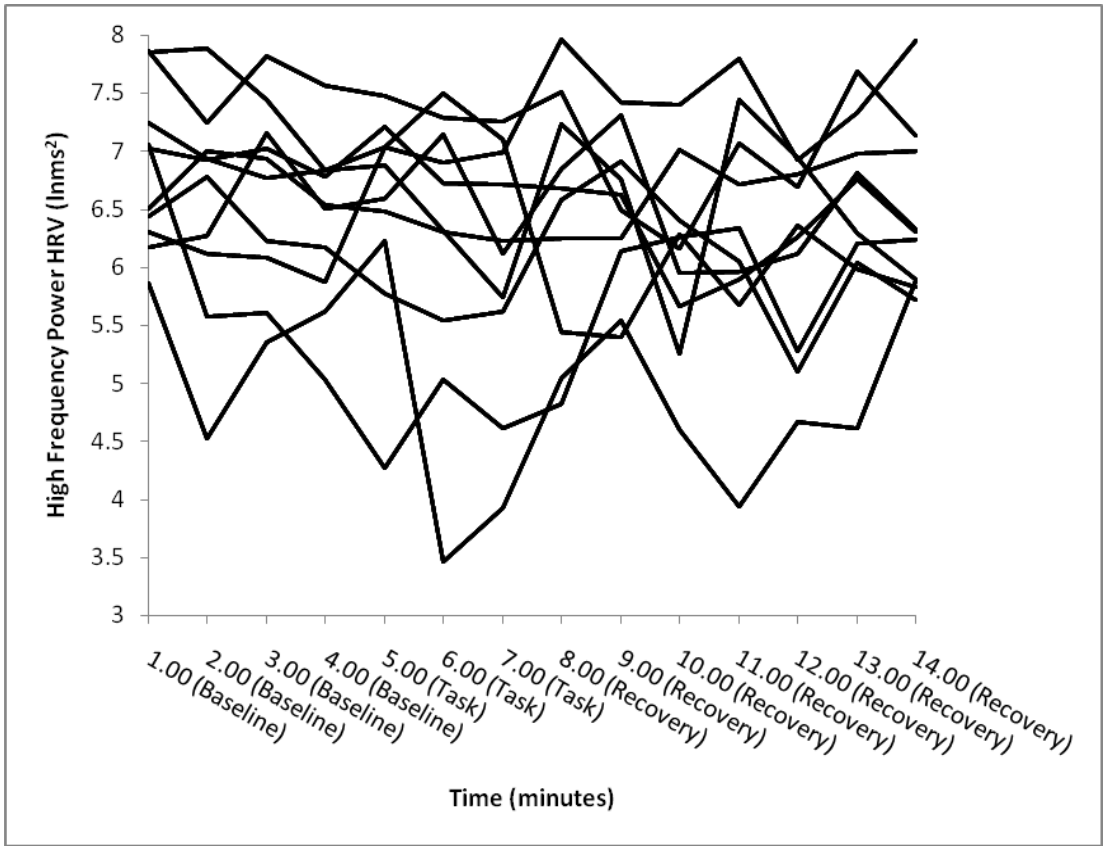


Figure 3. *Latent growth curve model of linear reactivity.*

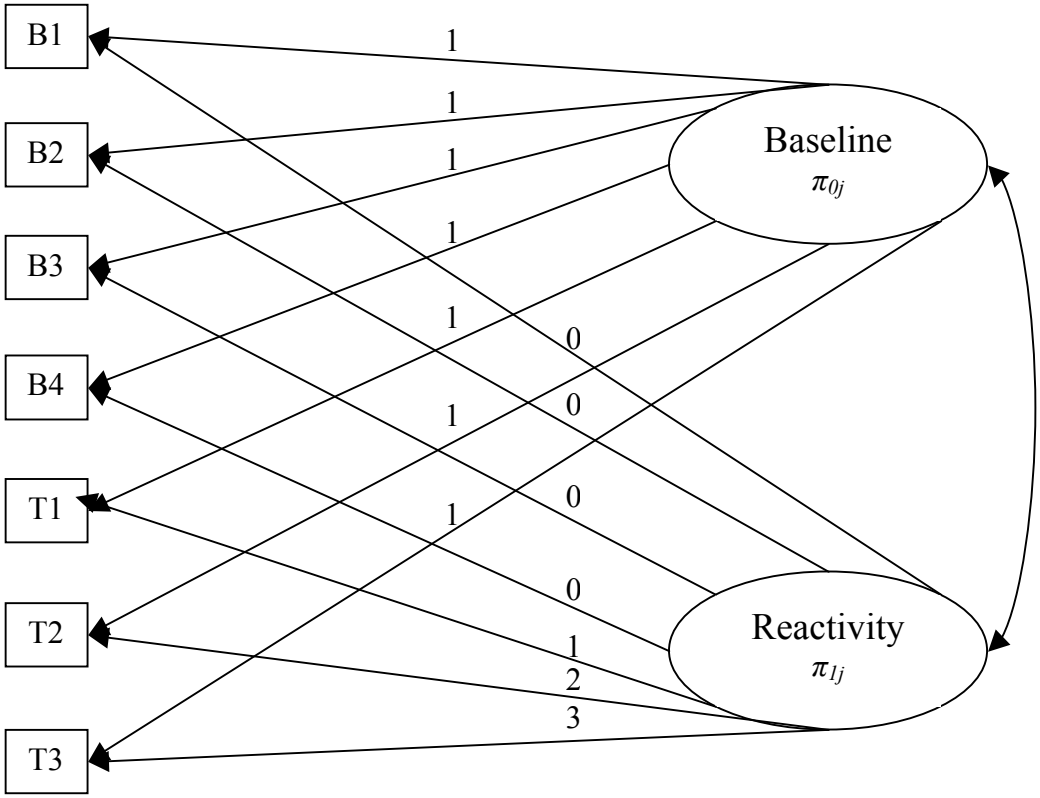


Figure 4. Latent growth curve model of quadratic recovery.

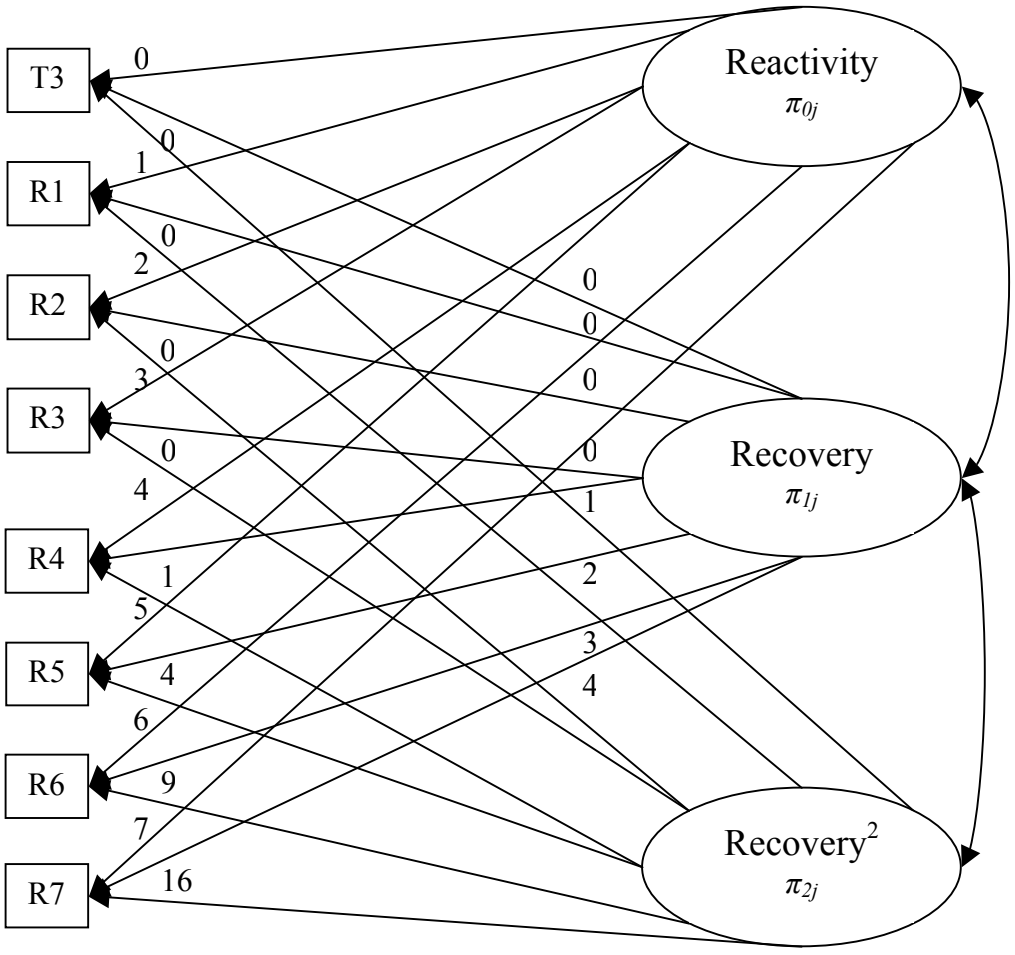


Figure 5.*Piecewise structural model of reactivity and recovery with direct effects.*

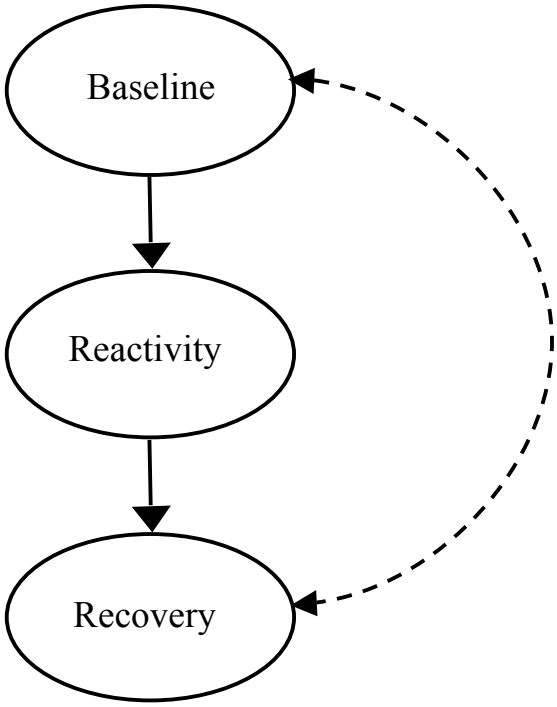


Figure 6. *Piecewise structural model of reactivity and recovery with age as a level-2 predictor.*

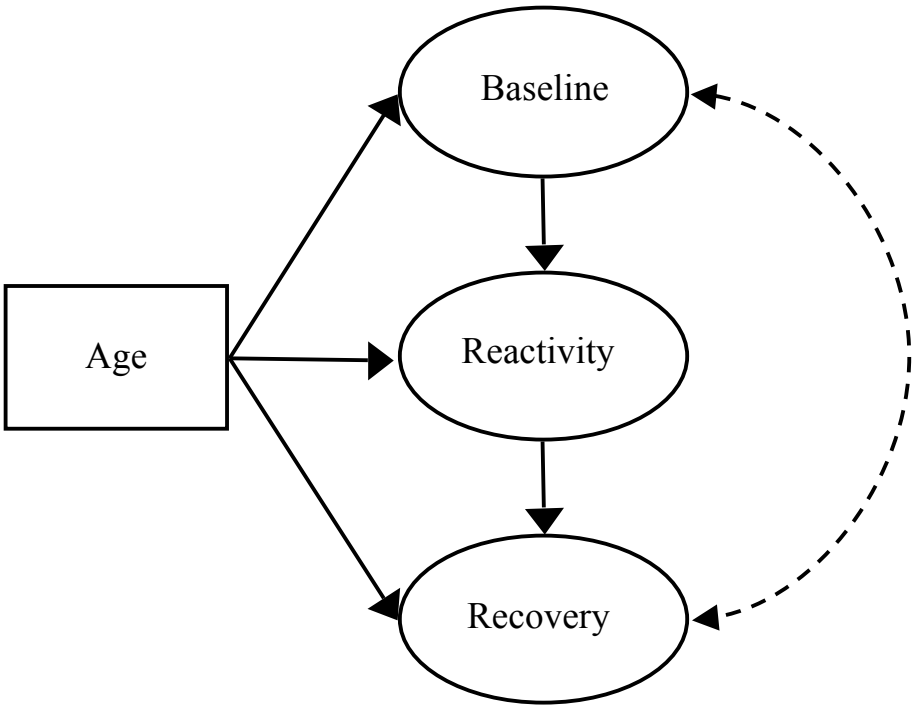
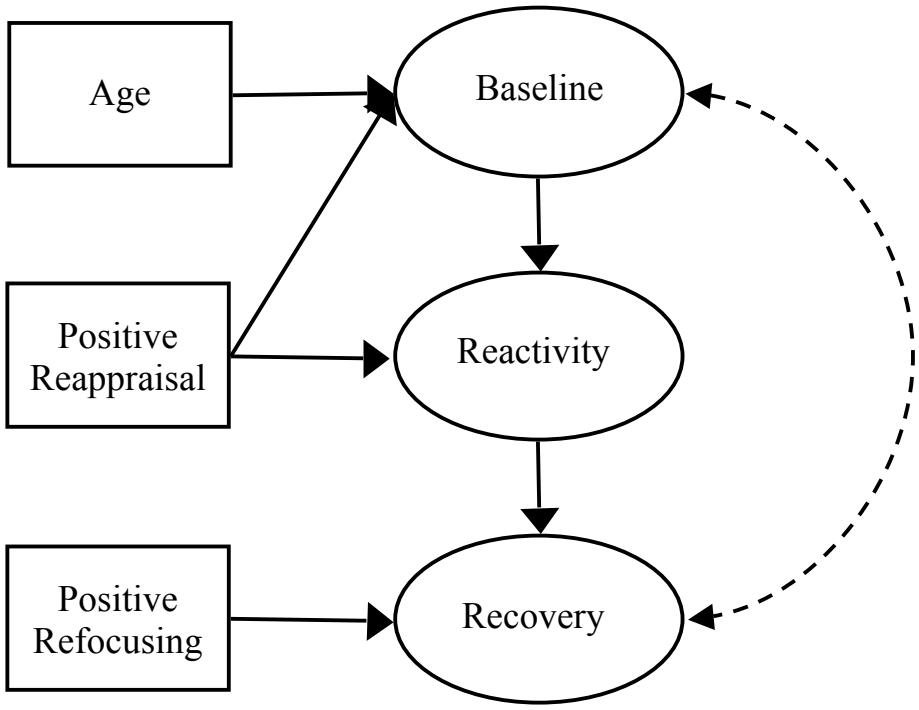


Figure 7.*Final piecewise structural model of reactivity and recovery with level-2 predictors.*



Appendix: Measures

PANAS

This section contains 20 words that describe different feelings and emotions. Please circle the numberthat best indicates**how much you feel each of these emotions in general, that is, on average.**

		1=very slightly or not at all	**2=a little**	**3=moderately**	**4=quite a bit**	**5=extremely**
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

The Aggression Questionnaire

Below are a list of behaviors and feelings that many people experience. Using the scale below, **circle** the number that indicates how much each question does or does not describe you.

1 - - - - - - - - - - 2 - - - - - - - - - - 3 - - - - - - - - - - 4 - - - - - - - - - - 5
Not at all Like Me Not Sure Very much Like Me

1. Once in a while I can't control the urge to strike another person. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	16. I am an even-tempered person. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
2. I am sometimes eaten up with jealousy. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	17. If I have to resort to violence to protect my rights, I will. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
3. I tell my friends openly when I disagree with them. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	18. I know that "friends" talk about me behind my back. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
4. I flare up quickly but get over it quickly. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	19. My friends say that I'm somewhat argumentative. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
5. Given enough provocation, I may hit another person. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	20. Some of my friends think I'm a hothead. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
6. At times I feel I have gotten a raw deal out of life. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	21. There are people who pushed me so far that we came to blows. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
7. I often find myself disagreeing with people. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	22. I am suspicious of overly friendly strangers. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
8. When frustrated, I let my irritation show. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	23. Sometimes I fly off the handle for no good reason. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
9. If somebody hits me, I hit back. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	24. I can think of no good reason for ever hitting a person. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
10. Other people always seem to be getting the breaks. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	25. I sometimes feel that people are laughing at me behind my back. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
11. When people annoy me, I may tell them what I think of them. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	26. I have trouble controlling my temper. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
12. I sometimes feel like a powder keg ready to explode. 1 - - - - - - 2- - - - - - - 3- - - - - - 4- - - - - - - 5	27. I have threatened people I know. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5
13. I get into fights a little more than the average person. 1 - - - - - - 2- - - - - - 3- - - - - - 4- - - - - - - 5	28. When people are especially nice, I wonder what they want.

14. I wonder why sometimes I feel so bitter about things. 1 - - - - - 2- - - - - 3- - - - - 4- - - - - 5 15. I can't help getting into arguments when people disagree with me. 1 - - - - - 2- - - - - 3- - - - - 4- - - - - 5	1 - - - - - 2- - - - - 3- - - - - 4- - - - - 5 29. I have become so mad that I have broken things. 1 - - - - - 2- - - - - 3- - - - - 4- - - - - 5

BFI

Here are a number of characteristics that may or may not apply to you. For example, do you agree that you are someone who *likes to spend time with others?* **Please write a number** next to each statement to indicate the extent to which you agree or disagree with that statement.

1=strongly disagree 2=somewhat disagree 3=neither agree/disagree 4=somewhat agree 5=strongly agree

I See Myself as Someone Who . . . (BFI; John, 1990)

1. ___ is talkative 2. ___ tends to find fault with others 3. ___ does a thorough job 4. ___ is depressed, blue 5. ___ is original, comes up with new ideas 6. ___ is reserved 7. ___ is helpful and unselfish with others 8. ___ can be somewhat careless 9. ___ is relaxed, handles stress well 10. ___ is curious about many different things 11. ___ is full of energy 12. ___ starts quarrels with others 13. ___ is a reliable worker 14. ___ can be tense 15. ___ is ingenious, a deep thinker 16. ___ generates a lot of enthusiasm 17. ___ has a forgiving nature 18. ___ tends to be disorganized 19. ___ worries a lot 20. ___ has an active imagination 21. ___ tends to be quiet 22. ___ is generally trusting	23. ___ tends to be lazy 24. ___ is emotionally stable, not easily upset 25. ___ is inventive 26. ___ has an assertive personality 27. ___ can be cold and aloof 28. ___ perseveres until the task is done 29. ___ can be moody 30. ___ values artistic, aesthetic experiences 31. ___ is sometimes shy, inhibited 32. ___ is considerate and kind to almost everyone 33. ___ does things efficiently 34. ___ remains calm in tense situations 35. ___ prefers work that is routine 36. ___ is outgoing, sociable 37. ___ is sometimes rude to others 38. ___ makes plans and follows through with them 39. ___ gets nervous easily 40. ___ likes to reflect, play with ideas 41. ___ has few artistic interests 42. ___ likes to cooperate with others 43. ___ is easily distracted 44. ___ is sophisticated in art, music, or literature

CERQ

When people remember a negative life event that happened to them, they often have other thoughts in response. We are interested in the thoughts that occurred to you today whenever you thought about the person who hurt you. Please use a number between 1 ("strongly disagree") and 5 ("strongly agree") to indicate whether you had the following thoughts today whenever you thought about the painful event you experienced or the person who hurt you.

1=strongly disagree 2=somewhat disagree3=neither agree/disagree
4=somewhat agree5=strongly agree

TODAY, WHEN I THOUGHT ABOUT WHAT HE/SHE DID TO ME . . .

	1. I felt that I am the one to blame for it
	2. I felt that I am the one who is responsible for what has happened
	3. I thought about the mistakes I have made in this matter
	4. I thought that basically the cause must lie within myself
	5. I thought that I have to accept that this has happened
	6. I thought that I have to accept the situation
	7. I thought that I cannot change anything about it
	8. I thought that I must learn to live with it
	9. I thought about how I feel about what I have experienced
	10. I was preoccupied with what I think and feel about what I have experienced
	11. I wanted to understand why I feel the way I do about what I have experienced
	12. I dwelt upon the feelings the situation has evoked in me
	13. I thought of nicer things than what I have experienced
	14. I thought of pleasant things that nothing to do with it
	15. I thought of something nice instead of what has happened
	16. I thought about pleasant experiences
	17. I thought of what I can do best
	18. I thought about how I can best cope with the situation
	19. It thought about how to change the situation
	20. I thought about a plan of what I can do best
	21. I thought that I can learn something from the situation
	22. I thought that I can become a stronger person as a result of what has happened
	23. I thought that the situation also has its positive sides
	24. I looked for the positive sides to the matter
	25. I tried to see ways that this event can make me a better person
	26. I thought that it all could have been much worse
	27. I thought that other people go through much worse experiences
	28. I thought that it hasn't been too bad compared to other things
	29. I told myself that there are worse things in life
	30. I thought that what I have experienced is much worse than what others have experienced
	31. I kept thinking about how terrible what I have experienced is
	32. I thought that what I have experienced is the worst that can happen to a person
	33. I continually thought about how horrible the situation has been

	34. I felt that others are to blame for it
	35. I felt that others are responsible for what has happened
	36. I thought about the mistakes others have made in this matter
	37. I felt that basically the cause lies with others
	38. I thought about the fact that I could have done the same thing under similar circumstances.
	39. I thought "I could never do something like that to somebody".
	40. I thought about the fact that I have done some things to other people that were not very nice.
	41. It occurred to me that in the right situation, I could be capable of similar behavior.
	42. I thought of the fact that I'm not perfect either.
	43. I thought about how it's sometimes easy to lose sight of how our actions can affect others.
	44. I tried to see things from his/her point of view.
	45. I tried to imagine how things look from his/her perspective.
	46. I try to "put myself in his/her shoes".
	47. I could not imagine how any circumstances under which I would act like that.
	48. I tried to imagine the position/state they were in at the time.
	49. I thought about the ways that this might make our relationship stronger.
	50. I tried to see ways that our relationship could grow from this.
	51. I thought about how this may be an opportunity to communicate about things.
	52. I thought about how we could both really learn from this.
	53. I thought of the ways in which his/her behavior was not completely in his/her control.
	54. I could see some of the pressures that caused him/her to act that way toward me.
	55. I realized that he/she would not normally behave in that way.
	56. I could see how the circumstances in his life are difficult to foresee.
	57. I thought that perhaps the cause of his/her behavior had something to do with his/her upbringing.
	58. I thought that the cause of his/her behavior had something to do with other people in his/her life.
	59. I thought about the things I still like about our relationship.
	60. I realized that there are many good things in our relationship still.
	61. I focused on the positives in our relationship.
	62. I tried to think about the good times we have shared.
	63. I thought about how we have gotten through similarly tough times.
	64. I thought of how nice it would be for us to have a strong relationship again.
	65. I thought about ways to strengthen our relationship.
	66. I thought of how good it would be for us to put this event behind us.
	67. I imagined us having a positive friendship.
	68. I thought about how to confront him/her constructively.
	69. I have thought about ways to communicate constructively.
	70. I imagined ways to keep my cool when I confront him/her.
	71. I imagined how a good person would respond to this situation.
	72. I thought about how certain friends/relatives of mine would respond to the situation.
	73. I tried to use effective/appropriate responses I've seen others use.
	74. I thought about how people in my life who have made me a better person would react.

	75. I thought that difficulties such as the one we are having happen less in other people's relationships.
	76. I thought about how I have worked at this relationship more than other similar relationships in my life.
	77. I thought about his/her strong points.
	78. I focused on the good things about him/her.
	79. I tried to think about all of the things I like about him/her.
	80. I thought about the fact that deep down, we are similar people.
	81. I tried to focus on the nice things he/she has said or done in the past.

Post-Imagery Emotions

Current Feelings toward the Person You Imagined During the Imagery Task. We would like to know how you are feeling AT THIS MOMENT about the person whom you just imagined during the imagery task. That is, we want to know how you are feeling RIGHT NOW regarding the person whom you just imagined yourself interacting with. Using the scale provided, please indicate how much you are currently feeling each of the emotions listed below.

"Regarding the person who hurt me, right now, I feel. . ."	**Not at all**	**Very Slightly**	**Slightly**	**Moderately**	**Considerably**	**To a great extent**	**Extremely**
1. Afraid	0	1	2	3	4	5	6
2. Angry	0	1	2	3	4	5	6
3. Anxious	0	1	2	3	4	5	6
4. Appreciative	0	1	2	3	4	5	6
5. Ashamed	0	1	2	3	4	5	6
6. Compassionate	0	1	2	3	4	5	6
7. Content	0	1	2	3	4	5	6
8. Curious	0	1	2	3	4	5	6
9. Delighted	0	1	2	3	4	5	6
10. Disappointed	0	1	2	3	4	5	6
11. Embarrassed	0	1	2	3	4	5	6
12. Empathic	0	1	2	3	4	5	6

13. Enraged	0	1	2	3	4	5	6
14. Enthusiastic	0	1	2	3	4	5	6
15. Envious	0	1	2	3	4	5	6
16. Fearful	0	1	2	3	4	5	6
17. Grateful	0	1	2	3	4	5	6
18. Guilty	0	1	2	3	4	5	6
19. Happy	0	1	2	3	4	5	6
20. Hostile	0	1	2	3	4	5	6
21. Irritable	0	1	2	3	4	5	6
22. Jealous	0	1	2	3	4	5	6
23. Joyful	0	1	2	3	4	5	6
24. Mad	0	1	2	3	4	5	6
25. Nervous	0	1	2	3	4	5	6
26. Playful	0	1	2	3	4	5	6
27. Scared	0	1	2	3	4	5	6
28. Softhearted	0	1	2	3	4	5	6
29. Sympathetic	0	1	2	3	4	5	6
30. Tender	0	1	2	3	4	5	6
31. Thankful	0	1	2	3	4	5	6
32. Warm	0	1	2	3	4	5	6
33. Worried	0	1	2	3	4	5	6