A COGNITIVE AND BEHAVIORAL INTERVENTION FOR SMOKING CRAVING: EFFICACY OF HEART RATE VARIABILITY BIOFEEDBACK AND COGNITIVE REAPPRAISAL ON CRAVING, NEGATIVE AFFECT, COGNITIVE PERFORMANCE, AND PSYCHOPHYSIOLOGICAL MEASURES IN WOMEN WHO SMOKE CIGARETTES

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YASMINE OMAR

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ABSTRACT OF THE DISSERTATION

A Cognitive and Behavioral Intervention for Smoking Craving: Efficacy of Heart Rate Variability Biofeedback and Cognitive Reappraisal on Craving, Negative Affect, Cognitive Performance, and Psychophysiological Measures in Women Who Smoke Cigarettes

By YASMINE OMAR

Dissertation Director:

Edward Selby

Stress is a principal factor in promoting smoking lapse and relapse to cigarette smoking in women. Cognitive reappraisal of stress (CR) and heart rate variability biofeedback (HRVb) have both demonstrated positive effects on the stress response and related outcomes, but have never been explored together for their effects on smoking-related outcomes. Sixty-eight adult women, who smoke 10+ cigarettes daily, abstained from nicotine and tobacco for 12 hours prior to a lab visit in which they completed questionnaires and were randomized to practice an intervention of CR and HRVb, or neutral control tasks. All participants then completed three stressful tasks, including a Stroop task to measure cognitive performance. Heart rate variability (HRV) data were collected throughout the study visit, in addition to smoking craving and negative affect assessments at baseline, post-intervention, post-stress, and post-recovery. Results showed a significant difference in craving decrease between experimental conditions, with the Intervention condition showing a significant decrease in smoking craving from baseline after 10 minutes of HRVb. There were no significant differences between conditions in

Stroop task performance or negative affect throughout the study. HRV results revealed group differences in heart rate increase during stress, and decrease after stress, with the Intervention group exhibiting greater volatility in heart rate. Overall, findings of this study indicate that practicing HRVb for 10 minutes results in a significant decrease in smoking craving in female daily smokers with 12 hours of abstinence, and multiple practice sessions of CR and HRVb may be needed to protect against the effect of stress on craving, negative affect, cognitive performance, or HRV. Future research will need to replicate these findings in a larger sample and explore the efficacy of practicing HRVb and CR together over multiple practice sessions in smoking cessation treatment.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Twenty percent of all deaths in the United States, or more than 480,000 deaths each year, are attributable to smoking (USDHHS, 2014). Cigarette smoking remains the leading preventable cause of death, with 16.8% of American adults currently smoking (CDC, 2015) and over 16 million Americans living with a smoking-related disease (USDHHS, 2014). Smoking remains difficult to quit; even with the best available current treatments, quit attempts are successful 35% of the time at most (Garrison & Dugan, 2009). Successful cessation has historically required multiple attempts at quitting (Cohen, et al., 1989), and smoking cessation and relapse to smoking remains by far the most frequent path of cigarette use for smokers (Garcia-Rodriguez et al., 2013; Piasecki, 2006; Rafful et al., 2013).

In order to further reduce smoking rates in the United States, a problem-focused approach would be to re-examine the characteristics of the population of smokers for common trends. Today's adult smokers are distinct in terms of race, socioeconomic status (SES), and comorbidities with psychopathology. *First*, minorities are overrepresented in the population of smokers when compared to the general population, including African Americans (17.5%), American Indians and Alaska Natives (29.2%), and multiple race individuals (27.9%; CDC, 2015). Sexual minorities are also overrepresented, as 23.9% of LGBT adults smoke compared with 16.6% of heterosexual adults (CDC, 2015). *Second*, cigarette smoking is highly comorbid with low socioeconomic status (SES); a recent report released by the CDC found that current smoking rates are lowest in individuals with a graduate degree (5.4%), and highest in individuals with a GED certificate (43%; CDC, 2015). It also reported that smoking rates are higher for those living below the

poverty level (26.3%) than for those living at or above the poverty level (15.2%). *Third*, smoking is comorbid with psychopathology, such that both emotional (Breslau et al., 2004; Kandel et al., 2007; Kendler et al., 1999; Wing et al., 2012) and personality (Grant et al., 2004; Lopez-Quintero et al., 2011) psychopathology have been strongly associated with an increased risk of developing nicotine dependence. Moreover, depressed smokers have a lower likelihood of quitting smoking than non-depressed smokers (Anda et al., 1999). This phenomenon is likely to increase due to emerging evidence that, as smoking rates decline overall, associations between smoking and depression become more prominent (Murphy et al., 2003), suggesting that current smoking cessation treatments are no longer sufficient in addressing obstacles to quitting for specific populations of smokers.

Upon further examination, the distinguishing characteristics of smokers appear to all share one common prominent factor that may shed light on their difficulty with quitting smoking: stress. Minority status, including racial minority status, is associated with greater stress due to racism, discrimination, and marginalization (Meyer, 2003). For example, African American couples disproportionately experience financial strain and racial stress that may compound other relationship stressors (Bryant et al., 2010). Socioeconomic status is also associated with daily stress, which may account for the modulating effect of SES on health outcomes (Baum, Garofalo, & Yali, 1999). Some studies have suggested that smoking prevalence of blue-collar workers is double that of white-collar workers due to psychological stressors associated with low income (Sorensen et al., 2004; Barbeau, Krieger, & Soobader, 2004). Finally, evidence has also shown that stress and depression symptoms are longitudinally related, with a documented

link between exposure to stressful events and depression (Caspi et al., 2003; Hammen, 2005; Kessler, 1997; Turner & Lloyd, 1995) and emotional dysregulation as a mediator of the effect of stress on depression (Abravanel & Sinha, 2015; Compare et al., 2014). Considering its disproportionate permeation of minority status, SES, and emotional psychopathology, it is no wonder that stress has been implicated as a primary mechanism in smoking relapse (McKee et al., 2003; Baer et al., 1989; Cohen & Lichtenstein, 1990).

Laboratory evidence suggests that stress reduces the ability to resist smoking while simultaneously increasing its rewarding effects (McKee et al., 2011). Stress prospectively predicts smoking lapse (Shiffman & Waters, 2004) and lapses triggered by stress progress more quickly to relapse (Shiffman et al., 1996), suggesting deficits in the ability to cope with stress. Early smoking lapse, which is strongly associated with a return to regular smoking (Brown et al., 2009), is often precipitated by increases in negative affect (Shiffman, 2005; Shiffman & Waters, 2004) and difficulties in emotion regulation (i.e., emotional dysregulation, Farris, Zvolensky, & Schmidt, 2015). Such lapse precipitants suggest difficulties with stress responding, as stressful events often trigger significant emotional responses (e.g., negative affect; Lazarus, 1999), and ability to regulate emotion has been proposed as a mediator of stress adjustment (McCarthy, Lambert, & Moller, 2006; Schwartz & Proctor, 2000).

In particular, women's tobacco use patterns show trends that are alarmingly different from men's: reports have shown that women consume more cigarettes than men (Hammond, 2009; Ng et al., 2014) and are less likely to successfully quit smoking than men (Cepeda-Benito et al., 2004; Perkins, 2001; Piper et al., 2010). Depression in women is more frequently comorbid with smoking compared with men (Husky et al., 2008), and

women struggle more than men with smoking cessation, with depressive symptoms cited as a risk factor for relapse (Murphy et al., 2003; Killen et al., 2003). While stress has been implicated as a primary mechanism in smoking relapse for all smokers (McKee et al., 2003; Baer et al., 1989; Cohen & Lichtenstein, 1990), recent evidence indicates that stress is a principal factor in promoting relapse to smoking in females, in part because women appear to be more strongly predisposed to stress responses (Torres & O'Dell, 2016). Moreover, women exhibit a stronger association between measures of psychophysiological reactivity (e.g., heart rate variability) and stress than men (Sloan et al., 1994; Woo & Kim, 2015). It is therefore likely that women are at particular risk for relapse to smoking due to stress. Due to women's increased predisposition to stress responses and the impact of stress on smoking, women who smoke stand to gain the most from improved regulation of their response to stress with adaptive self-regulation strategies, such as cognitive reappraisal and heart rate variability biofeedback.

Cognitive reappraisal has demonstrated positive effects on stress responding and smoking-related outcomes. As a transdiagnostic construct, emotion regulation is highly associated with distress tolerance and other constructs associated with avoidant behavior (Leyro et al., 2010). However, emotion regulation is distinct from similar constructs in its emphasis on efforts or strategies used to regulate one's emotion, like cognitive reappraisal (Gross, 2002). Cognitive reappraisal is the reframing of a situation to influence one's emotional response to it (Gross, 1998). This typically results in a more positive affective response to an otherwise distressing situation or task, as many definitions of emotion regulation highlight the self-regulation of affect (Carver et al., 1996; Breslau, Kilbey, & Andreski, 1991; Gross, 2002; Thompson, 1994). It is possible

that manipulating affective responses then influences both affective and smoking-related outcomes, as a recent meta-analysis found that positive affect manipulations decrease craving to smoke (Heckman et al., 2013). Moreover, practicing emotion regulation strategies like cognitive reappraisal (Lazarus & Folkman, 1984; Gross, 1998) may have beneficial effects on stress responding that further influence smoking-related outcomes, as recent laboratory evidence has indicated that reappraising stress-related arousal improves cognitive performance and physiological reactivity (Jamieson et al., 2012; 2013). Compared with acceptance and suppression, reappraisal as measured by the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) is associated with lower overall craving and negative affect during craving inductions and stress tasks, as well as improved performance on a cognitive stress task (Szasz, Szentagotai, & Hofmann, 2012). Fucito, Juliano, and Toll (2010) found that frequent reappraisal on the ERQ was cross-sectionally associated with smoking fewer cigarettes. Evidence thus far suggests that the use of cognitive reappraisal in particular as a self-regulation strategy may have positive implications for smoking behavior, including cigarette craving.

Heart rate variability biofeedback has also shown promising effects on stress-related outcomes. Utilizing behavioral self-regulation strategies such as heart rate variability biofeedback to increase heart rate variability shows promise in coping with stress. Heart rate variability (HRV) is a measure of fluctuation from the mean heart rate, representing the dynamic interaction between sympathetic and parasympathetic influences on the cardiac system (Siepmann et al., 2008), and serving as a biomarker of autonomic nervous system functioning at both the central and peripheral levels (Zucker et al., 2009; Servant et al., 2008). HRV has been proposed as an important marker of ability

to regulate emotion with implications for emotional psychopathology (Servant et al., 2008). This is supported by the polyvagal theory (Porges, 2003) noting that the vagus nerve serves as a pressed "brake" to reduce heart rate and produce a calm physiological state, which is released during times of threat. Higher HRV is associated with the efficiency of the "vagus brake" to inhibit or disinhibit pacemaker activity according to environmental and social cues (Porges, 2003; Francis et al., 2016). The effects of this dynamic process have been demonstrated in many studies, with evidence showing that high frequency HRV is associated with better emotion recognition in healthy adults (Quintana et al., 2012). Reduced resting HRV, as well as 24-hour HRV, are linked with presence and severity of major depressive disorder (Brunoni et al., 2013; Carney et al., 1995; Imaoka et al., 1985; Agelink et al., 2002; Kemp et al., 2010) and depression treatment outcomes are associated with changes in HRV (Jain et al., 2014). Adverse chronic psychological stress, which is disproportionately experienced by low SES populations, induces low HRV (Delaney & Brodie, 2000; Hjortskov, Rissen, Blangsted, Fallentin, Lundberg, & Sogaard, 2004; Lucini, Di Fede, Parati, & Pagani, 2005; Madden & Savard, 1995) and interferes with cognitive functioning (Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Lupien et al., 1997; Ohman, Nordin, Bergdahl, Slunga, & Stigsdotter, 2007). Notably, smokers have reduced HRV compared with non-smokers and duration of smoking is inversely related with measures of HRV, indicating blunted "vagus brake" activity on the heart in smokers (Barutcu et al., 2005).

HRV biofeedback (HRVb) training aims to improve adaptability to and recovery from fight or flight situations by increasing HRV (Gevirtz, 2013), which is associated with emotional resilience and reduced vulnerability to stress (Appelhans & Luecken,

2006; Ingjaldsson et al., 2003; Thayer, Hansen, & Johnson, 2010). While relaxation and meditation techniques have demonstrated effects on emotional control, HRVb has been argued as a particularly efficient approach due to its combination of feedback, breathing control, relaxation, and meditation (Servant et al., 2008). Past evidence suggests that HRVb training reduces symptoms in disorders characterized by poor emotion regulation (Karavidas et al., 2007; Siepmann et al., 2008; Zucker et al., 2009). A single session of HRVb improves adaptability to stress as measured with improved cognitive performance in a lab-induced stress task (Prinsloo et al., 2010). Several weeks of HRVb practice have shown reductions in substance craving (Penzlin et al., 2015; Eddie et al., 2014) and food craving (Meule et al., 2012), although no studies to date have examined the effect of a single session of HRVb practice on cigarette craving.

THE CURRENT STUDY

Based on promising evidence suggesting that the use of cognitive reappraisal strategies and HRVb may improve adaptation to stress, with implications for smoking outcomes, the current study aimed to assess the efficacy of HRVb and cognitive reappraisal of stress on changes in smoking craving, affect, and HRV, as well as performance on stressful tasks. I recruited adult female daily smokers to be randomized to one of two conditions reflecting the presence or absence of two experimental manipulations: HRVb and cognitive reappraisal of stress (CR). All participants were asked to refrain from nicotine, tobacco, and alcohol use for 12 hours prior to their study visit and to complete a battery of three validated stressful tasks. This study focused on two specific aims:

Aim 1: Examine the effect of using CR and HRVb in a single session on stress task performance. I expected that participants assigned to practice both CR and HRVb would demonstrate greater cognitive performance on mentally stressful tasks during a laboratory visit, as compared to a control group of female smokers who did not practice the two interventions. Specifically, I predicted that, compared with those in the Control condition, participants assigned to practice HRVb and CR would demonstrate shorter response time and higher accuracy on the Stroop task, as the final task in a series of three stressful tasks.

Aim 2: Examine the effect of using CR and HRVb on craving, negative affect, and short-term HRV. I expected that participants assigned to practice an intervention consisting of CR and HRVb would exhibit the greatest HRV adaptations, as measured by changes in HRV at various timepoints of a study visit, as compared to participants randomized to the Control group. I also predicted that participants assigned to practice both HRVb and CR would demonstrate greater reductions in craving and negative affect after the intervention period and by the end of the visit, relative to craving and negative affect at the start of the study, than participants randomized to the Control group.

Examining the relationship between use of CR and HRVb on short term HRV, negative affect, and smoking craving was intended to shed light on the effects of self-regulatory strategies on stress responding and smoking-related variables. As no studies to date had explored the effects of HRVb utility on smoking craving in a single session, this study was the first to address the potential use of a relatively new technology for its short-term effects on variables that increase lapse risk, such as negative affect, craving, and stress responding. This pilot study assessed the efficacy of practicing HRVb using a

device that is available to the general public that had not been previously tested for its short-term effects on craving. As behavioral (e.g., HRVb) and cognitive (e.g., reappraisal) approaches to self-management allow the individual to target both internal and external stimuli when coping with stress (Rokke & Rehm, 2001), both strategies would allow for a more comprehensive approach to managing stress responding, both cognitively and physiologically. This knowledge may assist in bolstering future smoking cessation treatment by reducing vulnerability to stress that frequently precipitates relapse.

RESEARCH METHODOLOGY

Participants

As this was a pilot study intended to inform an ongoing research program, I recruited a sample of 68 female daily smokers ages 18-65 in Central New Jersey. Participants were recruited via waiting room announcements at the Rutgers Robert Wood Johnson Medical School Family Medicine clinic at Monument Square, community flyers and announcements, and Facebook and Craigslist advertisements. To be included in the study, participants had to smoke at least 10 cigarettes per day and agree to complete study procedures, which required a willingness to practice quitting smoking. Exclusionary criteria included being pregnant or trying to become pregnant, color-blindness, current psychosis, current substance dependence, current body mass index (BMI) of over 40, current diagnosis of a cardiac rhythm abnormality (mitrovalve prolapse, frequent premature ventricular contractions, atrial fibrillation, bundle branch block) or a major neurological problem, history of a myocardial infarction, or past-week use of illicit drugs, nicotine replacement therapy, bupropion, varenicline, anti-cholinergic medications, beta blockers, Ritalin, benzodiazepines, tricyclic antidepressants, or antipsychotic medication.

Procedures

Interested participants completed a phone interview or online prescreen for eligibility. Eligible participants were asked to visit the laboratory for 5 minutes in order to provide a breath CO sample of at least 15ppm to confirm eligibility. If eligible, they were scheduled for a 3-hour study visit at least one day later and asked to refrain from alcohol, tobacco, or nicotine use (including e-cigarettes) for 12 hours prior to their appointment time. They were also asked to refrain from caffeine use for 4 hours prior to

their appointment time. On the day of their scheduled study visit, participants provided another breath CO sample of less than 10ppm or a 60% reduction of their eligibility confirmation CO level to confirm tobacco abstinence in the past 12 hours. Evidence shows that daily smokers who have not yet smoked the first cigarette of the day exhibit expired CO levels between 9 and 11ppm (Adan, Prat, & Sanchez-Turet, 2004). The 60% reduction in CO was incorporated to avoid excluding smokers with exceptionally high CO levels. Interested participants who were not CO-eligible were rescheduled for another study visit day. CO confirmation of study eligibility was followed by the informed consent process, and finally the completion of a baseline questionnaire packet (detailed below).

Randomization and experimental conditions

Participants were randomized to one of two conditions, blocked by age and level of motivation to quit smoking in the next 30 days. Participants ages 18-39 were randomized separately from participants ages 40-65. Blocking randomization by age group reduced age confounds within the sample of adult women, as age is correlated with smoking behavior and heart rate variability (Zhang, 2007). Motivation to quit smoking in the next 30 days, on a scale of 1-10, was assessed during the initial CO eligibility visit to block randomization by level of motivation to quit (1-5 versus 6-10). Participants were randomly assigned to one of two study conditions reflecting the presence or absence of the interventions: 1) HRVb and CR instructions, or 2) Sitting quietly while engaging in neutral control tasks and no reappraisal instructions. HRVb involved paced breathing as guided by a moving light on a portable biofeedback device (described below). The three neutral control tasks consisted of two videos depicting nature scenes, in addition to a task

asking participants to press a computer key whenever a shape appears on the computer screen, which occurred approximately every minute. While most controlled studies of HRVb have included control conditions that involved no activities (i.e. no treatment or waitlist control; Wells et al., 2012; Paul & Garg, 2012; Whited, Larkin, & Whited, 2014, Lehrer et al., 2003), I included three minor tasks in order to control for thought processes and potential rumination during the 30-minute sitting period without confounding the CR manipulation with psychoeducation.

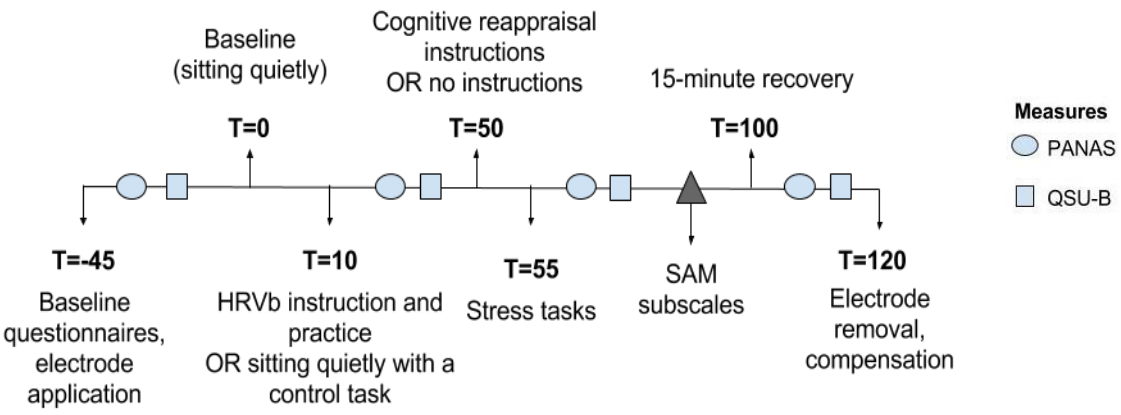
Past studies examining HRVb as an intervention for physiological and mental health conditions have utilized a multi-session protocol, as described in Lehrer et al. (2013). As this was a single-session study, the HRVb protocol was unable to include the resonance frequency determination that would occur between sessions. The current study's HRVb protocol was conducted using the *EmWave2*® device, which trains the user in HRVb and provides readings of HRV, time elapsed, and varying levels of physiological coherence (Edwards, 2014). The term *physiological coherence* is defined by the developers of the *EmWave2*® device as a state characterized by high heart-rhythm coherence (sine-wavelike rhythmic pattern), increased parasympathetic activity, increased entrainment and synchronization between physiological systems, and efficient and harmonious functioning of the cardiovascular, nervous, hormonal, and immune systems (McCraty, 2001). Repeated use of the device has demonstrated clinically significant improvement in performance on tests of executive functioning associated with emotional dysregulation (O'neill & Findlay, 2014) and statistically significant increases on measures of general health and mindfulness (Edwards, 2014). In this study, participants were trained by study staff in how to correctly use the *EmWave2*® device

and interpret the feedback measures it provides on their computer screen, including the real-time graph of HRV as it responds to their breathing and the lights on the device indicating their performance in the breathing task. All participants assigned to the HRVb condition were instructed to practice using the device for 2 minutes while breathing slowly at 5.5 breaths per minute. Study staff then provided them with reinforcement for their performance and constructive feedback for continued improvement before adjusting their breathing rate based on their height (5'4 and below: 6 breaths per minute; 5'5 and above: 5 breaths per minute). After a second 2-minute practice with the new breathing rate, participants were given the opportunity to choose the rate that was most relaxing for them to use for the biofeedback intervention. The biofeedback intervention period was split into three segments (7 minutes, 8 minutes, and 6 minutes) to ensure consistent practice without exhaustion and with regular opportunities for study staff to troubleshoot participant difficulties with the breathing task. The three HRVb segments were of approximately similar length to the three neutral tasks administered in the Control condition.

CR instructions were provided between the second and third HRVb segment, in the form of an interactive Prezi presentation that explained the function of the stress response as an adaptation to effectively overcome challenges, following an example by Jamieson, Nock, and Mendes (2012). They also included practice exercises to apply information in the instructions before the start of the stressful tasks, following the examples by Beadman and colleagues (2015). Participants were provided a worksheet to write down their responses to the exercises in the presentation, and each response was reviewed by research staff using a standard rubric to ensure participants' understanding

of the instructions before proceeding to the next task. As a manipulation check, the "threat," "challenge," and "stressfulness" subscales from the Stress Appraisal Measure (SAM; Peacock & Wong, 1989) were used to assess whether participants instructed to practice CR of stress during the tasks succeeded in reappraising stress. The SAM has demonstrated reliability and good internal consistency of its subscales (Carpenter, 2016). Questions from the SAM were slightly modified to past tense for administration after the tasks. Remaining study visit procedures are detailed in *Figure 1* below.

Figure 1. **Timeline of data collection during a laboratory visit.**



Debriefing and Follow-Up phone call

Following the completion of computer tasks, 15-minute recovery period, and removal of psychophysiological recording equipment, participants were provided with a Debriefing/Resources Sheet to debrief them about the stressful tasks and provide contact information for mental health and tobacco cessation resources. Participants received $100 in cash for completing all study procedures and received instructions to complete a follow-up phone call one week after the visit. Participants who completed the follow-up phone call were entered to win an additional $50. The purpose of the follow-up phone

call was to gather additional quantitative and qualitative information about the aftermath of the study visit and smoking or coping behavior that occurred after the visit in order to inform future studies on HRVb and CR. Questions in the follow-up call assessed changes in smoking craving, smoking behavior, other coping strategies, and perceived stress (using a measure they had completed at baseline) in the aftermath of the study visit.

Measures

Baseline questionnaires. Standard demographics information, in addition to height and self-reported weight for Body Mass Index calculation, was collected. Participants were also asked about hormonal contraceptive use and the date of their last menstrual cycle. Additional baseline measures included the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Garbin, 1988), Perceived Stress Scale (PSS; Cohen, Kamarck & Mermelstein, 1983), Emotion Regulation Questionnaire (ERQ; Gross & John, 2003), Fagerstrom Test for Nicotine Dependence (FTND; Heatherton et al., 1991), and the Timeline Follow-Back (TLFB; Sobell & Sobell, 2000) was used to quantify alcohol use and cigarettes smoked per day over the past 30 days. A complete list of baseline questionnaires is provided in *Table 2*.

Heart Rate Variability Assessment (HRV and HRVb). HRV was examined using electrocardiogram (ECG) measurement with Biopac Acqknowledge software. Three electrodes were positioned subclavicular bilaterally and over the left anterior superior iliac crest after the skin surface was gently cleaned with an alcohol swab. The *EmWave2*® HRVb device (HeartMath®, Boulder Creek, Colorado) was used for HRVb practice and served as an additional measure of HRV for the HRVb

group. The *EmWave2*® device trains the user in HRVb and provides readings of HRV, time elapsed, and varying levels of physiological coherence (Edwards, 2014).

HRV data cleaning followed standard guidelines (Bernston et al., 1990), in which trained research team members screened data for artifacts and corrected when necessary. Each participant's data file was examined by two research team members and cross-validated before the heart period series were analyzed using MindWare analysis software (MindWare, Gahanna, OH). HRV outcome measures included heart rate (HR), as well as frequency-domain measures (respiratory sinus arrhythmia [RSA], Low Frequency HRV) and a time-domain measure (root mean square of successive differences between heartbeats [RMSSD]). RSA refers to the vagus nerve's speeding and slowing of the heart that is driven by respiration (Karemaker, 2009). Teaching participants slow breathing when they experience sympathetic nervous system activation increases RSA (Shaffer & Ginsberg, 2017). Low Frequency HRV (LF HRV) increases with slow breathing that creates a resonant effect, and manipulations that influence LF power do so by affecting modulation of cardiac autonomic outflows by baroreflexes (Goldstein, Bentho, Park, & Sharabi, 2011). LF HRV data was gathered as a manipulation check of the HRVb protocol. RMSSD is a primary time-domain measure used to estimate HRV changes that are mediated by the vagus nerve (Shaffer, McCraty, & Zerr, 2014) and has been shown to be a more reliable estimate of RSA than other measures like the pNN50 (Shaffer & Ginsberg, 2017), although the influence of respiration on RMSSD is unclear (Schipke, Arnold, & Pelzer, 1999; Pentilla et al., 2001). Minute-by-minute measurements of HR, RSA, and RMSSD were calculated using

Mindware software. The average measurement across each segment of the study was then utilized for data analysis.

Blood Pressure. In addition to heart rate variability assessment, blood pressure was measured with an inflating arm cuff and a finger cuff. Changes in blood pressure are detected by blood vessel baroreceptors and further control heart rate through the autonomic nervous system, which may serve as an indication of accurate HRVb practice (Francis, Fisher, Rushby, & McDonald, 2016). Specifically, blood pressure would be expected to decrease as heart rate increases during each inhale during biofeedback practice, and the opposite pattern would be observed during each exhale (Lehrer et al., 2003). Blood pressure variability (BPV) outcome measures included diastolic blood pressure (BP), systolic BP, and mean arterial pressure (MAP), as reported in past research examining biofeedback and emotional stress (Palomba et al., 2011). Continuous non-invasive blood pressure was measured via finger sensors using CNAP®.

Respiration. To control for breathing rate throughout the laboratory visit, non-invasive cardiac output (NICO) electrodes were attached to the participant, with two sensors on each side of the neck and two sensors on each side of the lower ribs, using the Biopac apparatus to measure respiration. The numerical outcome for respiration was calculated by measuring the time interval between successive peaks of the respiration signal.

Affect and Craving. The 20-item Positive and Negative Affect Schedule (PANAS) has demonstrated test-retest reliability, as well as good convergent and divergent validity (Watson, 2000). The 10-item Questionnaire on Smoking Urges-Brief

(QSU-B; Cox, Tiffany, & Christen, 2001) is a measure of smoking urges and cravings, where a higher score indicates a stronger craving. Both measures were administered at four timepoints throughout the study, as shown in *Figure 1*.

Performance on stressful tasks. Past research on gender differences in stress responsivity has yielded inconsistent results, suggesting that gender may interact with the type of stressor or experimental procedure in predicting stress responsivity (Dickerson & Kemeny, 2004). For this reason, three stressful tasks were administered to participants in both experimental conditions in order to elicit different types of stress that may all potentially influence smoking craving in women. The third stressful task was used for data collection on cognitive performance.

Participants ***first*** spent ten minutes completing a Mirror Tracing Task (MTT; Quinn, Brandon, & Copeland, 1996) that has been previously used as a measure of persistence on a stressful task. Performance on the MTT is associated with nicotine dependence treatment outcome (Brandon et al., 2003) and can distinguish cigarette smokers from non-smokers (Quinn et al., 1996). The MTT involves tracing three shapes displayed on a computer screen, using a computer mouse that automatically moves in the opposite direction of one's hand, with a buzzing sound indicating whether the shape is being traced incorrectly. As the primary purpose of this task in the study was to induce stress, participants were instructed to complete the task without the option of termination, and no data was collected for task persistence. The task was terminated by study staff after 10 minutes.

Second, participants completed the validated Montreal Imaging Stress Task (MIST; Dedovic et al., 2005), which involves mental arithmetic with negative

feedback from the investigator (who, in this study, was the research assistant as well as a confederate "supervisor" of the research assistant), to incorporate psychosocial stress. The MIST was specifically designed for eliciting psychosocial stress when participant movement is restricted and has demonstrated effects on biological indices of stress, including cortisol (Dedovic et al., 2005). It has also demonstrated effects on brain areas associated with the limbic system (Dedovic et al., 2009) and HPA axis (Zschucke et al., 2015), which are areas where females have demonstrated greater stress reactivity than males (Oldehinkel & Bouma, 2011; Wang et al., 2007). A nearly identical task to the MIST without the investigator feedback component found that, compared with men, women anticipated that the task would be more difficult and threatening and later rated the task as more difficult (Hughes & Callinan, 2007). The MIST was specifically administered in this study to induce psychosocial stress that may influence performance and cigarette craving.

Third, a computerized Stroop battery (Stroop, 1935) consisting of two tasks was implemented to induce cognitive mental stress and serve as the data collection measure for cognitive performance. The Stroop is a well-known and validated measure of mental stress and cognitive performance that involves reading words and identifying colors on a computer screen. To maximize the diversity of stress task data collection for this study, the Stroop battery consisted of a simple color-word interference test in addition to the Victoria Stroop, which involves responding to many color-word targets on a single screen. Both the Stroop task and the Mirror Tracing Task have demonstrated gender effects with cardiac (heart rate)

reactivity, where women display greater changes in heart rate than men (Allen et al., 1993; Plante, Lantis, & Checa, 1997). A description of tasks is available in *Table 1*.

***Table 1*. Stressful tasks during the laboratory visit.**

Task	Length	Stressor	Data collected
Mirror Tracing Task	10 minutes	Distress	N/A
MIST	10-15 minutes	Psychosocial	N/A
Stroop	15 minutes	Cognitive	Reaction time, response accuracy

DATA ANALYSIS PLAN

Aim 1. To examine the effect of practicing CR and HRVb in a single laboratory session on cognitive performance during stress, reaction time and response accuracy on the Stroop task (as averages for each individual on each of the two Stroop tasks) were examined as outcome measures. Using IBM® SPSS® Statistics Version 25 software, multivariate analyses of variance (MANOVA) and covariance (MANCOVA) examined the effects of the two experimental manipulations (HRVb and CR) and covariates including age, average daily cigarettes smoked, and demographic variables on each of the two Stroop task outcomes (see *Table 2* for a full list of baseline self-report measures that may be included as covariates). Individual analyses of variance (ANOVA) and covariance (ANCOVA) were also utilized to examine each dependent variable separately, which did not significantly alter the results. For this reason, data for Aim 1 are only presented for multivariate analyses.

Aim 2. To examine the effect of practicing CR and HRVb on short-term HRV, craving, and negative affect, changes in craving and negative affect were assessed via changes from baseline craving scores, as well as changes from the post-intervention score (by subtracting the baseline or post-intervention score from later scores).

All HRV data were first cleaned following standard guidelines (Bernston et al., 1990). Group differences in each HRV and BPV outcome were examined as averages for each section of the study (5-minute Baseline, 21-minute Intervention [summing the 7-, 8, and 6-minute sections of the intervention], 10-15 minute MTT, 10-15 minute MIST, 10-minute Stroop, 15-minute Recovery) with change scores for each outcome looking at changes between each study segment's average numerical value. To ensure appropriate

mean values for each section of the study, data segments that were less than 3 minutes long were excluded from analyses.

First, changes in craving and affect from baseline to post-intervention, and from post-intervention to post-stress, were explored using t-tests within the overall sample and within the Control and Intervention groups individually. Change scores for craving, affect, HRV, and BPV were then explored as between-group differences using one-way MANOVAs, which are presented in the results of this paper. One-way ANOVAs were also explored to examine each dependent variable separately and compare results with less statistical power. MANCOVAs were then used to incorporate various covariates into analyses. Respiration rate was included as a covariate for HRV and BPV outcome analyses.

Second, I explored changes over time for the multiple assessments of craving and negative affect by examining within-subject changes across the different tasks of the study. Within-subject data analysis was conducted in Hierarchical Linear Modeling (HLM) Version 7 software (Raudenbush et al., 2011) to account for the nesting structure of the data as well as missingness of data, and allow for analysis with random intercepts. Multilevel models included the covariates mentioned above in addition to the experimental manipulations of CR and HRVb, which were examined as level-2 predictors (fixed effects).

Exploratory analyses. Exploratory analyses were included to further probe details of the analyses for Aims 1 and 2, and to examine changes in PSS score from baseline to the one-week follow-up call. Analyses consisted of one-way ANOVA, MANOVA, or linear regression to elucidate what factors influenced variability in this study's data.

Table 2. Baseline questionnaire measures for use as covariates in analyses.

Measure	Number of items	Justification
Demographic information, height, weight, and smoking history	N/A	General demographic information will be collected, including sex/gender, contraception and menstrual cycle, age, height, weight, sexual orientation, race/ethnicity, education/career history, and mental health history.
Timeline Followback (TLFB)	30	The TLFB will be used to quantify daily smoking behavior over the past 30 days, as a measure of smoking heaviness. It has demonstrated high test-retest reliability for 30-day intervals in cigarette smokers (Robinson et al., 2014).
Fagerstrom Test for Nicotine Dependence (FTND)	6	The FTND is among the most widely used measures of nicotine dependence, with confirmed reliability in different settings and populations that smoke cigarettes (Agrawal et al., 2011; Fagerstrom, 2012).
Reasons for Smoking Questionnaire (RFS)	23	The RFS has been used for over 30 years to assess smokers' motives for smoking, and has demonstrated good convergent validity and internal consistency (Currie, 2004) and adequate test-retest reliability (Tate, Schmitz, & Stanton, 1991).
Beck Depression Inventory-II (BDI-II)	21	The BDI-II has been frequently used to measure depression symptoms and has demonstrated reliability and stability (Beck, Steer, et al., 1988; Beck, Steer, & Brown, 1996).
Emotion Regulation Questionnaire (ERQ)	10	The ERQ is a validated measure of emotion regulation strategies with good test-retest reliability (Gross & John, 2003).
Perceived Stress Scale (PSS)	10	The PSS is the most widely used measure of the perception of stress, and was designed for use in community samples (Cohen, Kamarck & Mermelstein, 1983). Higher PSS scores are associated with failure to quit smoking, greater vulnerability to depression, and more colds (Cohen et al. 1988).
Generalized Anxiety Disorder-7 (GAD-7)	7	The GAD-7 is a brief, validated measure of generalized anxiety with demonstrated reliability that has been recommended for clinical practice and research (Spitzer et al., 2006).
Ruminative Responses Scale (RRS)	22	The RRS will be used to measure the degree to which individuals respond in ruminative ways during depressed moods. It has been demonstrated to have strong inter-item consistency (Treynor, Gonzalez, & Nolen-Hoeksema, 2003).
Emotional Cascade Assessment Measure (ECAM)	9	The ECAM will be used to measure how individuals respond to upsetting situations, and the degree to which they engage in emotional cascades (Selby & Joiner, 2009). This measure is currently being validated in my lab.
Social Desirability Scale	16	The SDS-16 measures social desirability, which can be used to examine potential inconsistencies in objective and subjective data.

RESULTS

Demographic information for my total sample is presented in *Table 3*. This sample was relatively diverse (66.2% European/Caucasian/White), mostly heterosexual (82.4%), and reported great variability in income and relationship status.

Table 3. Sample demographics.

	N	%		N	%
Race			***Relationship Status***		
Hispanic	7	10.3	Single	20	29.4
European/ Caucasian/ White	45	66.2	Dating	6	8.8
African American/ Black/ Caribbean	11	16.2	In a Committed Relationship	27	39.7
Middle Eastern	2	2.9	Married	11	16.2
Asian (Indian subcontinent)	3	4.4	Divorced	2	2.9
More than one race	2	2.9	Other	2	2.9
Rutgers student	6	8.8	***Income***		
Sexual Orientation			Less than 10,000	12	17.6
Straight/Heterosexual	56	82.4	10,000-19,999	10	14.7
Gay/Lesbian/Homosexual	4	5.9	20,000-29,999	10	14.7
Bisexual	5	7.4	30,000-39,999	6	8.8
Other	2	2.9	40,000-49,999	10	14.7
Do not wish to report	1	1.5	50,000-74,999	10	14.7
			75,000-99,999	6	8.8
			100,000 or more	4	5.9
			On birth control	9	13.2

Descriptive information for the total sample, in addition to group differences at baseline, is presented in *Table 4A*. On average, this study's overall sample participant was 38 years old, smoked almost 15 cigarettes per day, and was overweight with a BMI of about 28. Based on scores on the FTND, GAD-7, and BDI-II, the average participant reported moderate nicotine dependence, mild anxiety, and minimal to mild depression. This sample reported an average PSS score of 20.97, which indicates high levels of perceived stress for women (Cohen & Williamson, 1988). High levels of stress in this sample were accompanied by stress-related reasons for smoking on the RFS, as the overall sample endorsed feeling "blue" or wanting to take their mind off of cares and worries, feeling uncomfortable or upset about something, and feeling angry as their strongest reasons for smoking (as demonstrated by an average score of at least 4 out of 5 on each of the items listed). Raw scores on baseline variables examining psychological functioning showed that the Intervention group reported slightly worse functioning overall, with the exception of their depression score on the BDI-II. There were no significant differences between experimental groups on any baseline variable (including baseline measures of craving and affect), with the exception of SDS score. The Intervention group's responses on the SDS indicated a significantly higher level of social desirability than those of the Control group ($p = .004$) with an effect size of Cohen's $d = 0.72$. In addition, 15 participants did not complete the TLFB correctly and therefore had missing information regarding their cigarette and alcohol use over the past 30 days. Analyses using CPD as a covariate were run in two ways – once including only data from participants with CPD information, and once including a separate CPD variable with a

grand mean imputation for the 15 missing participants. Unless otherwise noted, there were no significant differences between the two CPD variables in analyses.

Table 4A. Experimental group differences at baseline.

	Control Group N=34 M (SD)	**Intervention Group N=34 M (SD)**	**Total N=68 M (SD)**
CPD	13.52 (6.05) n=26	15.91 (6.43) n=27	14.74 (6.30) n=53
APD	.35 (.42) n=26	.42 (.76) n=27	.38 (.61) n=53
BMI	27.17 (6.42)	29.62 (6.40)	28.40 (6.48)
Age	37.65 (14.97)	39.12 (13.03)	38.38 (13.95)
SES	2.91 (2.31)	3.03 (2.12)	2.97 (2.21)
Perceived addiction to cigarettes (1-5)	4.50 (.66)	4.32 (.77)	4.41 (.72)
FTND	4.82 (2.10)	4.91 (1.98)	4.87 (2.02)
BDI	15.88 (11.18)	13.62 (10.05)	14.75 (10.61)
ERQ Cognitive Reappraisal	30.12 (6.20)	29.91 (6.66)	30.01 (6.40)
ERQ Expressive Suppression	13.56 (4.95)	14.56 (4.80)	14.06 (4.86)
PSS	20. 74 (7.61)	21.21 (8.06)	20.97 (7.78)
GAD-7	8 (5.18)	8.59 (5.24)	8.29 (5.18)
RRS	21.26 (7.00)	21.24 (6.55)	21.25 (6.73)
SDS**	15.35 (5.04)	18.88 (4.71)	17.12 (5.15)
Baseline QSU-B Factor 1	27.35 (6.89)	29.44 (5.63)	28.40 (6.33)
Baseline QSU-B Factor 2	18.18 (9.00)	21.32 (7.25)	19.75 (8.26)
Baseline QSU-B Total	45.53 (14.59)	50.76 (11.80)	48.15 (13.43)
Baseline PANAS positive	26.94 (8.44)	28.29 (7.82)	27.62 (8.10)
Baseline PANAS negative	16.44 (5.11)	16.53 (4.84)	16.49 (4.95)

Note. **denotes difference between experimental conditions is significant at $p < .01$.

The SAM threat, challenge, and stress subscales were examined to assess the effectiveness of the CR manipulation on participants' appraisal of the stressful tasks. The overall sample reported the level of threat and challenge of the tasks as between slight and moderate, with the degree of stressfulness of the tasks as moderate or considerable. This pattern of reporting is consistent with past research using the SAM, which has found similar values in the threat, challenge, and stressfulness subscales for a variety of stressful tasks (Peacock & Wong, 1990). Of the three subscales, only the SAM threat subscale exhibited a near-significant difference between the experimental groups, $F(1, 65) = 3.69$, $p = .059$, Cohen's $d = 0.47$. This was likely driven by the response to the question, "did this have a negative impact on me?" that contributed to the threat score, where the Intervention condition scored significantly higher than Control, $F(1, 65) = 4.61$, $p = .036$, Cohen's $d = 0.53$. There were no significant differences between groups on any other SAM subscale or specific items. These results suggest that the CR manipulation was not effective at teaching my participants to reappraise stress, and that this study's Intervention condition was more negatively affected by the stressful tasks than the Control condition. Self-reported scores on the SAM subscales, as well as measures of craving and affect after stress, are provided in *Table 4B.*

Table 4B. Experimental group differences post-stress.

	Control Group N=34 M (SD)	**Intervention Group N=33 M (SD)**	**Total N=67 M (SD)**
SAM Threat	2.36 (.68)	2.77 (1.02)	2.56 (.88)
SAM Challenge	2.57 (.87)	2.64 (.95)	2.60 (.90)
SAM Stress	3.11 (.75)	3.34 (.97)	3.22 (.87)
QSU-B Factor 1	30.29 (4.50)	30.24 (5.47)	30.27 (4.96)
QSU-B Factor 2	20.21 (8.24)	23.76 (8.13)	21.96 (8.32)
QSU-B Total	50.50 (11.31)	54.00 (12.77)	52.22 (12.09)
PANAS positive	19.76 (8.26)	21.36 (8.27)	20.55 (8.24)
PANAS negative	20.44 (8.24)	22.91 (8.36)	21.66 (8.33)

***Aim 1*. Stroop Task Performance.**

To examine the effect of practicing CR and HRVb in a single laboratory session on Stroop task performance, reaction time and response accuracy (as averages for each individual) were examined as outcome measures for the two variations of the Stroop task. Three outcome measures (total correct responses, percentage of correct responses out of the total completed, and reaction time) were examined for the Color-Word Stroop task, and two outcome measures (total incorrect responses, and average response time) were examined for the Victoria Stroop. Analyses of covariance examined the effects of the two experimental manipulations (HRVb and CR) on Stroop task outcomes.

Shapiro-Wilks' test of normality found that, with the exception of CWS reaction time for the Intervention group, none of the Stroop outcome variables were normally distributed. Almost all variables were leptokurtotic, ranging from reaction time on the CWS (kurtosis = 2.36, *SE* =. 64) to total incorrect responses on the VS (kurtosis = 13.02,

SE = .64), with only the VS response time variable exhibiting minimal kurtosis (kurtosis = -.09, *SE* = .64). Most variables were within acceptable ranges for skewness (+/- 2.5) with the exception of total incorrect responses on the VS (skewness = 3.15, *SE* = .33). A log10 transformation of all five outcomes did not significantly impact the normality of the variable distributions and did not yield significantly different results in MANOVA analyses. Considering that MANCOVA and ANCOVA analyses are robust to violations of normality, analyses are presented for non-transformed variables.

Primary analyses for Aim 1. A one-way MANOVA was initially performed to examine basic group differences on all five Stroop outcome variables. During this analysis, Levene's statistic was not significant for any variable, suggesting that the homogeneity of variances assumption had been met for all variables. As shown in *Table 5*, there were no significant differences in any Stroop outcome variable between the Control and Intervention conditions. Neither Welch's nor Brown-Forsythe's statistics were significant for any variable. Including age, average cigarettes smoked per day (CPD), FTND, BDI, GAD-7, or SDS score in a MANCOVA (as shown in *Table 5*) or multiple ANCOVAs did not yield significant results. While there was no significant impact of experimental condition on Stroop task performance, means plots revealed that the Intervention group exhibited slightly better performance than the Control group. Therefore, while my hypothesis that experimental condition would have a significant influence on Stroop task performance was not supported, there was a general trend suggesting that the Intervention group performed better than the Control group.

Exploratory analyses. To elucidate other potential driving factors in Stroop task performance for this study, a comprehensive list of covariates (BDI, FTND, ERQ

suppression, ERQ cognitive reappraisal, PSS, RRS, GAD-7, SDS, income, age) were examined as potential predictors of Stroop task performance using linear regression. In all model combinations, age was consistently the only significant predictor of response time on both the CWS (β = .52, *SE* = 2.11, $p < .01$) and the VS (β = .41, *SE* = 6.74, p = .001). Age explained 27% of the variance in response time on the CWS, $F(1, 57) = 20.70$, $p < .01$, and 16.9% of the variance in response time on the VS, $F(1, 62) = 12.37$, $p = .001$.

Outside of reaction time outcomes for the two variations of the Stroop task, nicotine dependence emerged as a significant predictor of accuracy on the CWS. FTND score significantly inversely predicted percent correct responses on the CWS (β = -.26, *SE* = 1.07, $p = .043$) alongside age (β = -.32, *SE* = .18, $p = .019$) and income (β = .29, *SE* = 1.06, $p = .028$) in a model that explained 21.5% of the variance in percent accuracy on the CWS, $F(3, 57) = 4.92$, $p = .004$. FTND score was also a significant inverse predictor of the total correct responses on the CWS, (β = -.39, *SE* = 2.40, $p = .002$) alongside the ERQ cognitive reappraisal score (β = -.29, *SE* = .86, $p = .019$) in a model that explained 23.1% of the variance in total correct CWS responses, $F(2, 57) = 8.27$, $p = .001$.

Accuracy on the VS was not affected by FTND score, however. When all covariates were entered, only RRS score (β = .44, *SE* = .15, $p = .012$) and GAD-7 score (β = -.36, *SE* = .18, $p = .036$) significantly predicted the number of incorrect responses on the VS. This suggests that rumination was a strong predictor of poor performance on the VS, and generalized anxiety score was inversely related to VS performance in a model that predicted 19.7% of the variance in VS incorrect responses, $F(5, 61) = 2.75$, $p = .027$.

Table 5. Group differences in Stroop task performance.

		Color-Word Stroop			Victoria Stroop	
		N=58, 29 per condition			N=63, Control=32, Intervention=31	
		Total correct responses	% correct responses	Average reaction time	Total incorrect responses	Average response time
Kurtosis (*SE*=.64)		5.73	7.90	2.36	13.02	-.09
Skewness (*SE*=.33)		-2.44	-2.69	1.39	3.15	.75
Group differences	F	1.33	.89	.34	.84	.63
	Significance	.255	.367	.562	.364	.431
	Partial eta squared	.03	.02	.01	.02	.01
	Observed Power	.21	.15	.09	.15	.12
Controlling for covariates (age, CPD, FTND, BDI, GAD-7, SDS) N=44 for CWS N=49 for VS	F	1.31	1.50	.14	.06	.02
	Significance	.261	.229	.713	.805	.882
	Partial eta squared	.04	.04	.004	.002	.001
	Observed Power	.20	.22	.07	.06	.05

Note. Results above are presented for MANOVA and MANCOVA analyses. Neither log10 transformations of outcome variables nor using ANOVA and ANCOVA analyses significantly impacted these results.

***Aim 2*. Smoking Craving.**

In order to examine the effect of practicing CR and HRVb on craving using the QSU-B, QSU-B scores were first divided into two factors, as proposed by Cox, Tiffany, and Christen (2001). Factor 1 denotes a "desire and intention to smoke with smoking perceived as rewarding" (p. 11). Factor 2 reflects "an anticipation of relief from negative affect with an urgent desire to smoke" (p. 7). To examine the effect of HRVb, CR, and covariate variables on craving, the QSU-B was examined in terms of each factor and the total score at the four timepoints during which the measure was administered: baseline, post-intervention, post-stress, and after recovery.

Shapiro-Wilk's test of normality was significant for all Factor 1 scores, in addition to the post-stress Factor 2 and Total scores, suggesting a non-normal distribution of variables. With the exception of post-stress Factor 1 (kurtosis = 2.35, *SE* = .58; skewness = -1.33, *SE* = .30), all QSU-B scores were within normal ranges for kurtosis and skewness (+/-1) and revealed no outliers. Considering the intended purpose of the stressful tasks to increase stress levels and smoking craving, it is unsurprising that the post-stress Factor 1 score was leptokurtotic.

Primary analyses for Aim 2. Paired samples t-tests were initially used to examine within-group changes in the overall sample and within each experimental group. Analyses revealed that the average participant reported a significant decrease in Factor 1 ($t(67) = 4.19$, $p < .001$, Cohen's $d = .43$), Factor 2 ($t(67) = 2.95$, $p = .004$, Cohen's $d = .21$), and the total QSU-B score ($t(67) = 4.08$, $p < .001$, Cohen's $d = .34$) from baseline to post-intervention. This overall decrease was driven by changes in the Intervention group,

which demonstrated a significant decrease in Factor 1 ($t(33) = 5.02$, $p < .001$, Cohen's $d = .81$), Factor 2 ($t(33) = 3.17$, $p = .003$, Cohen's $d = .34$), and the total QSU-B score ($t(33) = 4.60$, $p < .000$, Cohen's $d = .60$) from baseline to post-intervention. The Control group, however, did not show a significant decrease in Factor 1 ($t(33) = 1.00$, $p = .325$), Factor 2 ($t(33) = 1.09$, $p = .284$), or the total QSU-B score ($t(33) = 1.23$, $p = .228$). These results suggest that only the Intervention group reported a significant decrease in their smoking craving from baseline to post-intervention, with a large effect size for their reported decrease in QSU-B Factor 1 ($d = .81$).

After the stressful tasks, both the Control and the Intervention groups reported a significant increase in all of the craving subscores from post-intervention to post-stress. Paired samples t-tests revealed that the average participant reported a significant increase in Factor 1 ($t(66) = 6.76$, $p < .001$, Cohen's $d = .79$), Factor 2 ($t(66) = 5.48$, $p < .001$, Cohen's $d = .50$), and the total QSU-B score ($t(66) = 6.60$, $p < .001$, Cohen's $d = .69$).

Experimental group differences in change scores between the various timepoints were then explored for Factor 1 and Factor 2 of the QSU-B as shown in *Table 6* and *Table 7*. A one-way MANOVA with each change score as an outcome found a significant effect of experimental intervention on decrease in Factor 1 score from baseline to post-intervention, $F(1, 64) = 13.23$, $p = .001$, Cohen's $d = .90$. This indicates that participants in the Intervention condition exhibited a greater average decrease in their smoking craving from baseline ($M = -5.36$, $SD = 6.00$) after practicing the study intervention when compared to those in the Control condition ($M = -.52$, $SD = 4.75$), with a large effect size. This was followed by a significant difference in change in smoking craving post-stress $F(1, 64) = 4.99$, $p = .03$, Cohen's $d = -.55$, with the Intervention group reporting a greater

increase in smoking craving between the intervention period and post-stressful tasks (*M* = 6.30, *SD* = 6.31) than the Control group (*M* = 3.24, *SD* = 4.71). Nevertheless, a near-significant difference found that participants practicing the intervention appeared to have a greater overall decrease in their smoking craving from Baseline to post-recovery (the end of the study) compared with the Control group, $F(1, 64) = 3.91$, $p = .052$, Cohen's $d = .49$. Using MANCOVAs to include multivariate outcomes and covariates, these effects remained significant or near-significant after controlling for cigarettes smoked per day (with and without the mean imputation), FTND score, age, BMI, baseline QSU-B Factor 1 score, and SDS score. The group difference in smoking craving change from the beginning to the end of the study visit (baseline to post-recovery) was not significant after controlling for covariates, however.

MANOVA analysis of changes in QSU-B Factor 2 revealed one near-significant difference between experimental groups in the craving change from the intervention period to post-stress, $F(1, 64) = 3.99$, $p = .05$, Cohen's $d = -.49$, with the Intervention group reporting a greater increase (*M* = 5.27, *SD* = 6.59) than the Control group (*M* = 2.45, *SD* = 4.72). A one-way ANOVA examining this difference did not confirm significance of the result, however ($p = .119$). There was no other significant effect of experimental condition on changes in Factor 2 of the QSU-B score throughout the study, with BMI and baseline Factor 2 score emerging as significant covariates as shown in *Table 7*. These results suggest that participants who practiced the study intervention experienced a significant decrease in their desire to smoke and perception that smoking would be rewarding, with little or no significant effects on their anticipation of relief from negative affect by smoking. While this supports my hypothesis that participants

randomized to the Intervention condition would experience a greater decrease in craving from the two interventions, these data do not support my hypothesis that the effects of practicing the intervention would remain throughout the study visit, despite an increase in stress. Trends in changes over time, by experimental group, are visible for QSU-B Factor 1, Factor 2, and Total score in *Figures 2-4*.

Table 6. Group differences in QSU-B Factor 1 changes, with and without covariates.

N=66	Baseline to Intervention	Baseline to stress	Baseline to recovery	Intervention to stress	Intervention to recovery
F	13.23	1.37	3.91	4.99	1.36
Significance	**.001****	.246	**.052***	**.03*[a]**	.248
Partial eta squared	.17	.02	.06	.07	.02
Observed Power	.95	.21	.50	.60	.21
Cohen's *d*	.90		.49	-.55	
Controlling for covariates: Age, CPD, BMI, FTND, baseline F1 craving	Significant covariates: baseline F1 craving	Significant covariates: baseline F1 craving	Significant covariates: baseline F1 craving, CPD	Significant covariates: baseline F1 craving	Significant covariates: baseline F1 craving
F	7.06	.01	1.99	8.04	1.68
Significance	**.011***	.929	.165	**.007****	.202
Cohen's *d*	.90			-.55	
Controlling for SDS					
F	9.70	.90	2.18	3.85	1.54
Significance	**.003****	.346	.144	**.054***	.219
Cohen's *d*	.90			-.55	

Note. **denotes difference between experimental conditions is significant at $p < .01$. *denotes difference between experimental conditions is near-significant at approximately $p < .05$. [a]When using one-way ANOVA, group difference in change from Intervention to stress was near-significant, $p = .097$.

Table 7. Group differences in QSU-B Factor 2 changes, with and without covariates.

N=66	Baseline to Intervention	Baseline to stress	Baseline to recovery	Intervention to stress	Intervention to recovery
F	2.16	.38	.57	3.99	.10
Significance	.146	.539	.452	**.050*[a]**	.754
Partial eta squared	.03	.01	.01	.06	.002
Observed power	.31	.09	.12	.50	.06
Cohen's *d*				-.49	
Controlling for covariates: Age, CPD, BMI, FTND, SDS, baseline F2 craving	Significant covariates: BMI, baseline F2 craving	Significant covariates: baseline F2 craving	Significant covariates: baseline F2 craving	No significant covariates	No significant covariates
F	1.20	3.61	.33	1.97	.04
Significance	.281	.064	.568	.167	.841
Partial eta squared	.03	.08	.01	.04	.001
Observed power	.19	.46	.09	.28	.06

*Note***.** *denotes difference between experimental conditions is near-significant at approximately $p < .05$. [a]When using one-way ANOVA, group difference in change from Intervention to stress was not significant, $p = .119$.

Within-subject analyses for Aim 2. Within-subject changes over time for the four assessments of craving were explored using Hierarchical Linear Modeling (HLM) Version 7 software (Raudenbush et al., 2011) to account for the nesting structure of the data as well as missingness of data, and allow for analysis with random intercepts. The final dataset included a total of 211 timepoint measurements (four per participant) and a total of 54 participants (out of 68 original participants). Due to technical errors in completing the TLFB, 15 participants have missing data for CPD and were excluded from HLM analyses. Time was coded as 0, 1, 2, or 3 in order to interpret baseline as the intercept. Intraclass correlation coefficients were generally appropriate for the three measures of craving (ICC for QSU-B Factor 1: .48; ICC for QSU-B Factor 2: .71; ICC for QSU-B total: .62). In all models, timepoint was included as a level-1 predictor of craving score outcome (Factor 1, Factor 2, and Total QSU-B Score). Level-2 predictors were study condition, cigarettes per day (CPD), BMI, age, and total scores on the FTND, BDI, PSS, GAD-7, SDS, and ERQ expressive suppression and cognitive reappraisal subscores. The greatest decrease in deviance score indicated the best-fitting model for all outcomes.

The best-fitting model, based on deviance score, to predict craving Factor 1 did not include experimental condition as a predictor, which did not support my hypothesis. Exploratory analyses found the best-fitting model to include time as a significant random level-1 predictor, with CPD as a significant level-2 predictor of intercept (β = .277, *SE* = .10, t = 22.71, p = .009). SDS score was a significant level-2 predictor of slope (β = -0.138, *SE* = .05, t = -2.76, p = .009). This suggests that, while experimental condition did not significantly influence within-subject craving Factor 1 throughout the study visit,

participants exhibited within-subject changes in craving across time, which was initially related to their CPD at baseline, but varied in a manner consistent with their level of social desirability.

The best-fitting model, based on deviance score, to predict craving Factor 2 included time as a significant level-1 random predictor. Experimental condition (β = 5.251, *SE* = 1.72, *t* = 3.06, *p* = .004), FTND score (β = 1.05, *SE* = .44, *t* = 2.40, *p* = .02), and ERQ expressive suppression score (β = .841, *SE* = .20, *t* = 4.20, *p* < .001) were all significant level-2 predictors of intercept. ERQ expressive suppression score (β = -0.168, *SE* = .07, *t* = -2.53, *p* = .014) and SDS score (β = -0.163, *SE* = .06, *t* = -2.69, *p* = .01) were both significant predictors of slope of time in the model. This suggests that there were differences across individuals at baseline in craving Factor 2 were associated with differences in total FTND score, experimental condition, and ERQ expressive suppression score. Variability in craving Factor 2 across time was significantly associated with ERQ expressive suppression score and level of social desirability.

For total craving (total QSU-B score including Factor 1 and Factor 2), the best-fitting model, based on deviance score, included time as a significant level-1 random predictor. Experimental condition (β = 12.640, *SE* = 4.16, *t* = 3.04, *p* = .004), CPD (β =.516, *SE* = .23, *t* = 2.24, *p* = .03), and SDS score (β = -0.840, *SE* = .34, *t* = -2.48, *p* = .017) were all significant level-2 predictors of intercept. At baseline, differences in individuals' total craving score were associated with differences in their cigarettes smoked per day, their social desirability score, and experimental condition. Experimental condition (β = -1.942, *SE* = 1.06, *t* = -1.83, *p* = .069) was a near-significant predictor of

slope of time in the model, suggesting that individuals in the Intervention condition were more likely to decrease their overall craving over time.

Figures 2 - 4. Experimental group differences in QSU-B Factor 1, Factor 2, and Total Score.

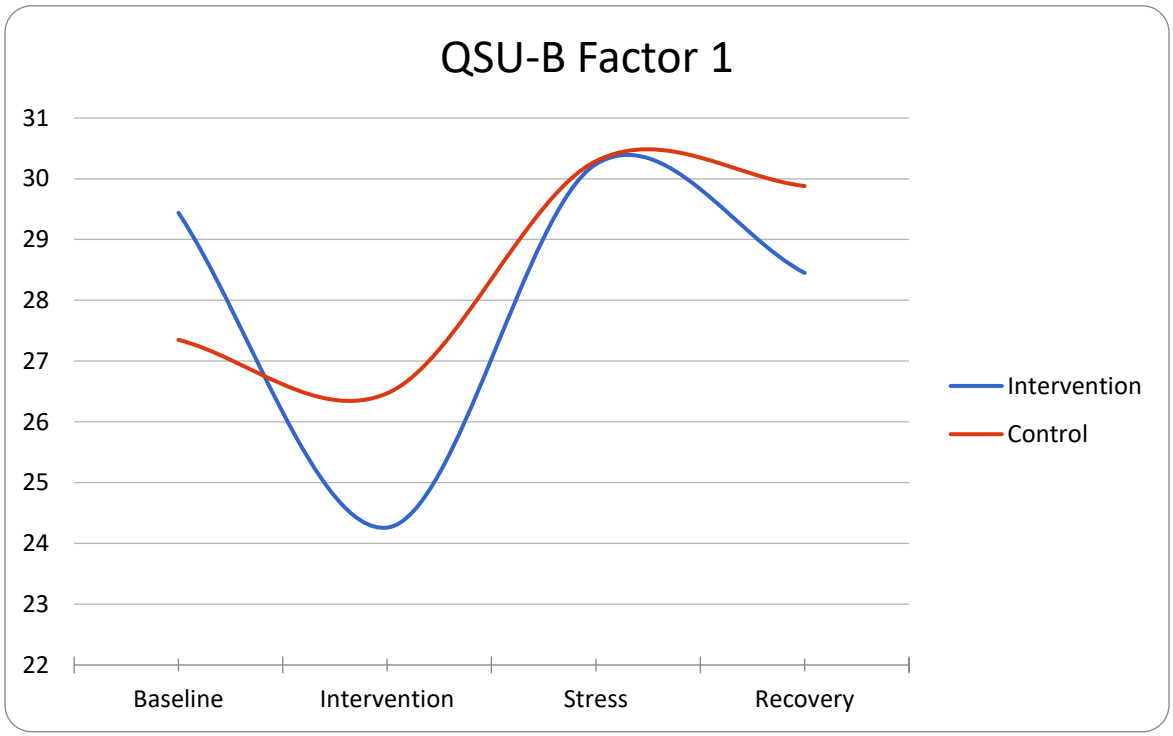


Figure 3.

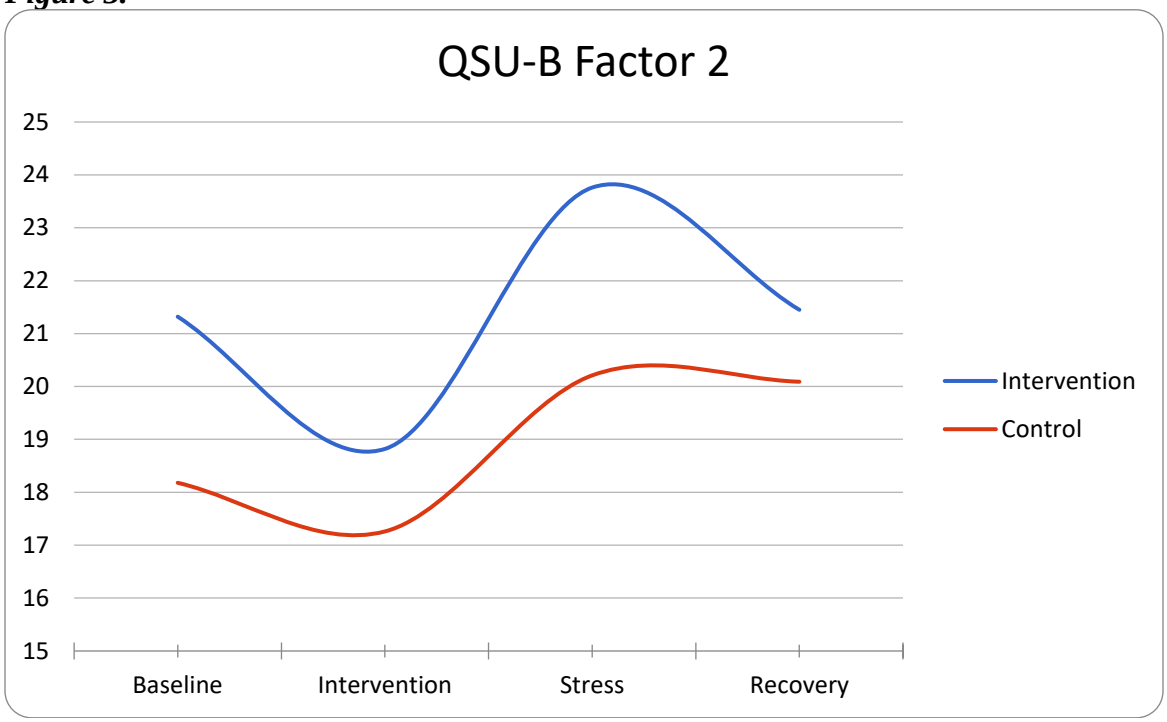
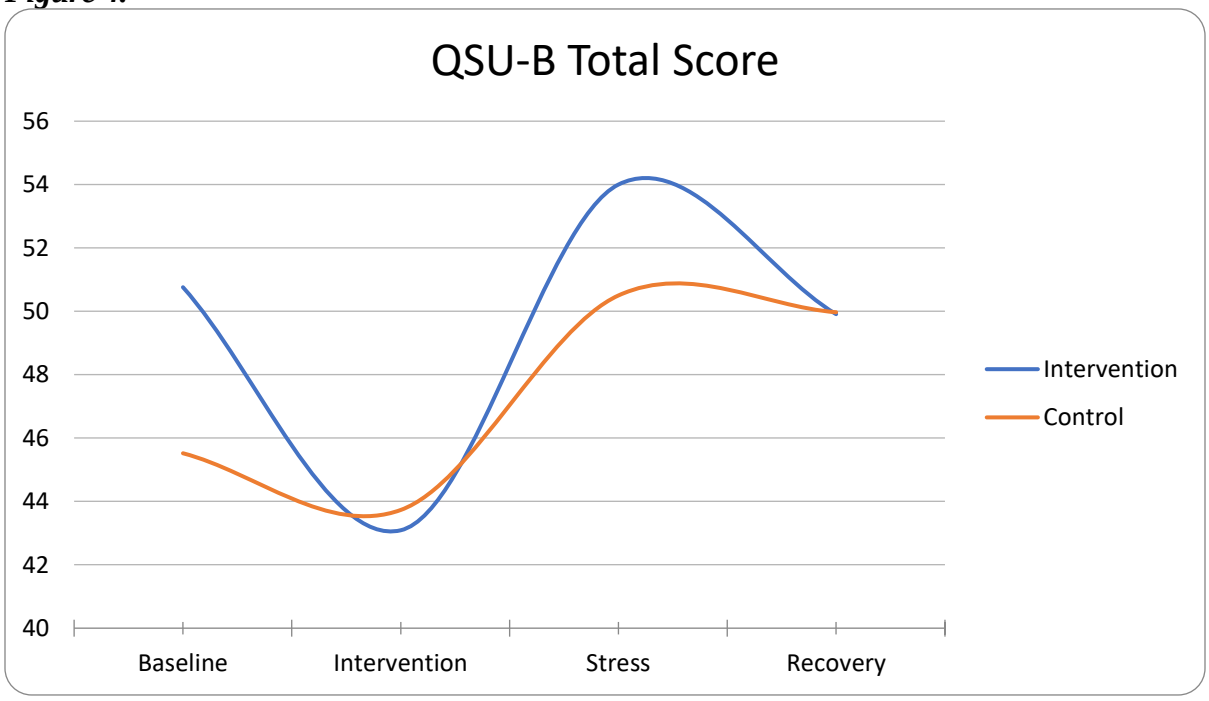


Figure 4.



***Aim 2*. Negative Affect.**

In order to examine the effect of practicing CR and HRVb on negative affect using the PANAS, the positive and negative affect subscores were examined separately, and positive PANAS was examined for exploratory purposes. Shapiro-Wilk's test of normality was significant for all negative PANAS scores, in addition to the majority of the positive PANAS scores with the exception of the baseline positive PANAS score, suggesting a non-normal distribution of variables. With the exception of the post-recovery negative PANAS score (kurtosis = 2.55, *SE* = .58; skewness = -1.45, *SE* = .30), all positive and negative PANAS affect scores were within normal ranges for kurtosis and skewness (+/-1). While there were no outliers in the positive PANAS score distributions, there was one outlier for the negative PANAS score post-intervention, and two outliers for the negative PANAS score post-recovery.

Primary analyses for Aim 2. Independent samples t-tests found no significant differences between the Control or Intervention group in either positive or negative affect at any timepoint in the study. Paired samples t-tests revealed that participants in both the Control and Intervention groups reported similar trends in affect from baseline to post-intervention, with the average participant in the overall sample reporting a significant decrease in both positive affect ($t(67) = 5.66$, $p < .001$, Cohen's $d = .41$), and negative affect ($t(67) = 4.46$, $p < .001$, Cohen's $d = .37$). Both groups showed similar trends in affect after stress, as well, with the average participant reporting a significant decrease in positive affect ($t(66) = 5.86$, $p < .001$, Cohen's $d = .41$), and a significant increase in negative affect ($t(66) = 8.07$, $p < .001$, Cohen's $d = 1.03$) from post-intervention to post-stress. This suggests that, regardless of experimental condition, the overall sample

reported a continuous decrease in positive affect from baseline to post-stress, and a decrease in negative affect from baseline to post-intervention. After stress, however, the overall sample reported a significant increase in negative affect with a large effect size (*d* = 1.03).

Experimental group differences in change scores between the various timepoints were then explored for negative and positive PANAS scores as shown in *Table 8* and *Table 9*. There were no significant differences between the control and Intervention group on changes in negative affect at any timepoint in the laboratory visit, which did not support my hypotheses about the Intervention condition exhibiting less negative affect throughout the study. Incorporating the SDS social desirability score as a covariate did not significantly affect results and yielded nonsignificant models. Potential covariates were then selected based on a Pearson correlation with the outcome variables of above .3, resulting in CPD, BDI, PSS, GAD-7, RRS, and baseline negative PANAS score as covariates.

When covariates were included in analyses for exploratory purposes, the models significantly predicted change scores from baseline to post-stress, baseline to recovery, and post-intervention to post-stress. Experimental condition was not a significant predictor in any model, however. In fact, all significant models were primarily driven by RRS score, which was a significant predictor of changes in scores only when the overall model was significant. Baseline negative PANAS score was also a significant predictor in all models predicting changes from baseline, but unlike the RRS score, baseline negative PANAS score did not significantly predict changes from post-intervention onward. A separate MANOVA examining experimental condition and RRS score as predictors

found RRS to consistently significantly predict change in negative affect from baseline to post-stress, ($F(1, 63) = 12.09$, $p = .001$), baseline to recovery, ($F(1, 63) = 6.00$, $p = .017$), Intervention to post-stress, ($F(1, 63) = 17.79$, $p < .001$), and Intervention to recovery, ($F(1, 63) = 11.44$, $p = .001$). This was particularly evident for the Control group, for which RRS score was a significant predictor of increases in negative affect from baseline to post-stress, ($F(1, 31) = 6.13$, $p = .019$), Intervention to post-stress, ($F(1, 31) = 13.76$, $p = .001$), and Intervention to recovery, ($F(1, 31) = 8.95$, $p = .005$). For the Intervention group, RRS score was also significantly associated with increases in negative affect from baseline to post-stress, ($F(1, 31) = 5.86$, $p = .022$) and Intervention to post-stress, ($F(1, 31) = 5.76$, $p = .023$), but not in changes from Intervention to recovery. Linear regressions examining RRS as a predictor of negative affect during the recovery period of the study found that RRS was a significant predictor for the Control group ($\beta = .63$, $SE = .16$, $t = 4.55$, $p < .001$), predicting 40% of the variance in negative affect during recovery. This relationship was not evident in the Intervention group, ($\beta = .30$, $SE = .16$, $t = 1.75$, $p = .089$), with RRS predicting 9% of the variance in negative affect during recovery. Overall, these exploratory findings suggest that trait rumination was the strongest predictor of changes in negative affect throughout the laboratory visit, regardless of experimental condition, CPD, or scores on the BDI, PSS, or GAD-7. Trait rumination appeared to play a role in negative affect reported during the recovery period by the Control group, and not the Intervention group, despite playing a significant role for both groups at other points in the study.

Table 8. Experimental group differences in negative affect using the PANAS, with and without covariates.

N=66	Baseline to Intervention	Baseline to stress	Baseline to recovery	Intervention to stress	Intervention to recovery
F	2.76	2.00	.22	.54	.17
Significance	.101	.163	.641	.466	.683
Partial eta squared	.04	.03	.003	.01	.003
Observed power	.37	.29	.08	.11	.07
Cohen's *d*	-0.40				
Controlling for covariates: age, CPD, BDI, PSS, GAD-7, RRS, baseline negative affect	Significant covariates: Baseline negative affect	Significant covariates: Baseline negative affect, RRS, GAD-7	Significant covariates: Baseline negative affect, RRS	Significant covariates: RRS	Significant covariates: (RRS p = .10)
F	.66	.06	.01	.02	.28
Significance	.422	.814	.924	.892	.602
Partial eta squared	.02	.001	.000	.000	.000
Observed power	.13	.06	.05	.05	.08
Controlling for SDS					
F	3.01	2.64	.83	.85	.002
Significance	.088	.109	.366	.360	.963
Partial eta squared	.05	.04	.01	.01	.000
Observed power	.40	.36	.15	.15	.05

Note. **denotes difference between experimental conditions is significant at $p < .01$. * denotes difference between experimental conditions is near-significant at approximately $p < .05$.

Exploratory analyses. As with the negative affect change scores, there was no significant effect of experimental condition on changes in positive affect at any timepoint in the laboratory visit. Potential covariates were explored, including Age, CPD, and variables that had a Pearson correlation above .3 with the positive PANAS scores. Adding Age, CPD, BMI, ERQ cognitive reappraisal, and the baseline positive PANAS score as covariates did not significantly alter these results or produce significant models predicting positive affect. Incorporating the SDS social desirability score as a covariate also did not significantly affect results. This suggests that the HRVb and CR intervention did not significantly impact changes in positive affect throughout the laboratory visit.

Table 9. Experimental group differences in positive affect using the PANAS, with and without covariates.

N=66	Baseline to Intervention	Baseline to stress	Baseline to recovery	Intervention to stress	Intervention to recovery
F	.40	.03	.38	.83	1.65
Significance	.528	.859	.543	.365	.204
Partial eta squared	.01	.00	.01	.01	.03
Observed power	.10	.05	.09	.15	.24
Controlling for covariates: age, CPD, BMI, ERQ cognitive reappraisal, baseline positive affect	Significant covariates: ERQ cognitive reappraisal	Significant covariates: Age	Significant covariates: ERQ cognitive reappraisal	Significant covariates: Baseline positive affect	No significant covariates
F	1.12	.04	.10	.77	.37
Significance	.296	.843	.757	.385	.547
Partial eta squared	.02	.001	.002	.02	.01
Observed power	.18	.05	.06	.14	.09
Controlling for SDS					
F	.07	.20	.81	.65	1.55
Significance	.797	.656	.371	.423	.218
Partial eta squared	.001	.003	.01	.01	.02
Observed power	.06	.07	.14	.13	.23

Within-subject exploratory analyses. Due to the largely null findings for positive and negative affect, I did not explore predictors of affect within subjects. Instead, the relationship between affect and craving within subjects was examined across the four assessments in the study using Hierarchical Linear Modeling (HLM) Version 7 software (Raudenbush et al., 2011). Intraclass correlation coefficients were generally appropriate for both positive affect (ICC = .68) and negative affect (ICC = .44). Using total QSU-B score (including Factor 1 and Factor 2) as an outcome, positive PANAS (β = -0.305, *SE* = .13, p = 018) and negative PANAS (β = .993, *SE* = .14, $p < .001$) were both significant predictors of craving that varied across individuals. As positive affect had an inverse relationship with craving and negative affect had a positive relationship with craving, this suggests consistency in participants' reporting of affect and craving that varied at a similar rate throughout the study.

***Aim 2*. HRV.**

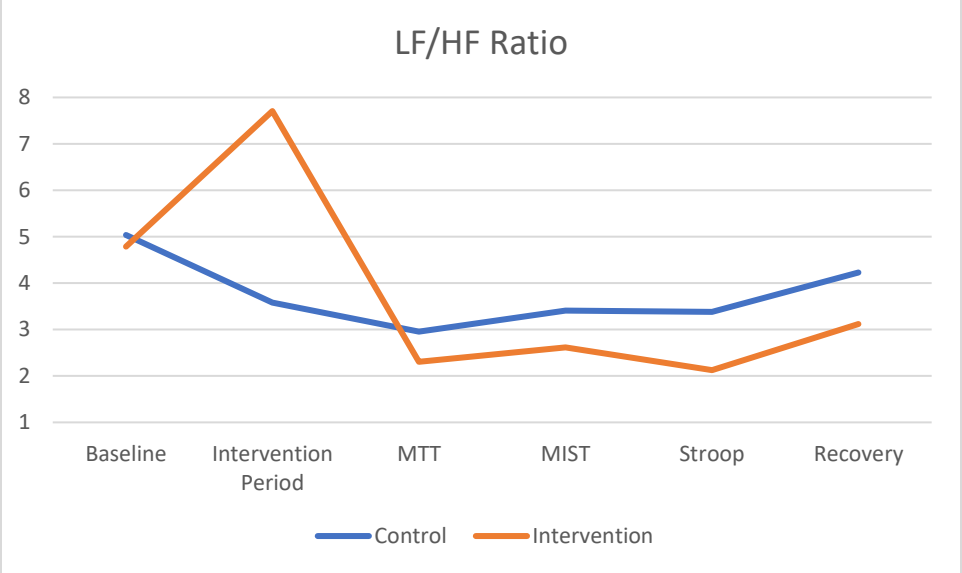
To examine the effect of practicing CR and HRVb on heart rate variability, I first examined changes from baseline to post-intervention in LF HRV to test whether the biofeedback intervention did, in fact, result in slowed breathing in the low frequency range. LF power, or the amplitude in the heart rate power spectrum at the frequency identified as LF Peak Frequency, was natural log transformed to maintain homogeneity of variances. Preliminary LF HRV analyses were followed by the examination of three additional outcome measures of HRV (heart rate, RSA, and RMSSD), which were analyzed separately. *Table 10* and *Table 11* detail experimental group differences in the three measures of HRV throughout the study visit, as well as changes in HRV measures between various segments of the study.

LF HRV. A one-way ANOVA examining the effect of Experimental Condition on natural log transformed LF power at baseline and during the Intervention period found a significant difference between conditions in LF power during Intervention, $F(1, 53) = 19.34$, $p < .001$, with the Intervention group ($M = .80$, $SD = .31$) exhibiting higher LF power than the Control group ($M = .43$, $SD = .32$). There was no significant difference between conditions at baseline, $F(1, 51) = 12.03$, $p = .235$, with the Intervention group ($M = 3.18$, $SD = .56$) exhibiting slightly lower LF power than the Control group ($M = 4.13$, $SD = 4.09$). Although the Intervention group exhibited a greater increase in LF power than the Control group, there was no significant difference in change from baseline to Intervention, $F(1, 51) = 2.23$, $p = .141$. This suggests that, despite an absence of significant differences between the experimental groups at baseline, the Intervention group displayed significantly higher LF power than the Control group during the intervention period, consistent with a slower breathing rate.

LF Peak Power Frequency, the peak frequency in the heart rate power spectrum as measured in the LF frequency band, was also examined for differences between the experimental groups. An independent samples t-test revealed a significant difference between experimental groups in LF Peak Power Frequency change from baseline to the Intervention period of the study, $t(51) = 5.75$, $p < .001$. A paired-samples t-test revealed that the Intervention condition showed a significant increase in LF Peak Power Frequency from baseline to the Intervention period, $t(26) = 4.97$, $p < .001$, with a mean increase of .013 ($SD = .01$). The Control condition, on the other hand, exhibited a significant decrease in LF Peak Power Frequency, $t(25) = 3.34$, $p = .003$, with an average decrease of .011 ($SD = .017$).

A final measure of LF HRV was LF/HF Ratio, or the ratio of low frequency power divided by high frequency power. An independent samples t-test revealed a significant difference between experimental groups in LF/HF Ratio change from baseline to the Intervention period of the study, $t(51) = 3.38$, $p = .001$. Paired-samples t-tests examining changes for both experimental groups revealed that only the Intervention group showed a significant change in LF/HF Ratio from baseline to the Intervention period, $t(26) = 3.08$, $p = .005$, with a mean increase of 2.91 ($SD = 4.91$). As shown in *Figure 5A* below, the significant increase in LF/HF Ratio that was only observable in the Intervention group from baseline to the Intervention period indicates that only the Intervention group stimulated activity of the baroreflex through slowed breathing.

Figure 5A. Group differences in LF/HF Ratio throughout the laboratory visit.



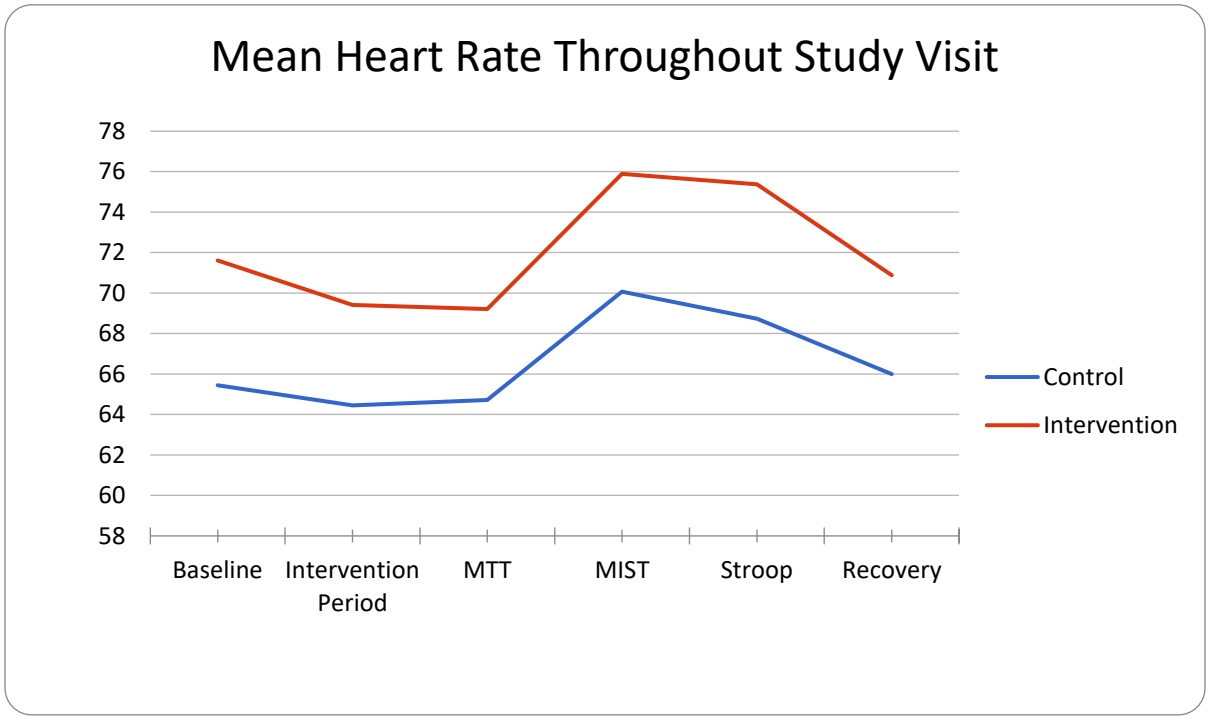
HEART RATE. Paired samples t-tests explored within-group changes in HR for each study condition. While both conditions demonstrated a significant increase in HR from the intervention period to the stressful tasks ($t(54) = 5.73$, $p < .001$, Cohen's d =

.38) and a significant decrease from stress to recovery $t(49) = 3.94$, $p < .001$, Cohen's $d = .24$), only the Intervention group exhibited a significant decrease in HR from baseline to the intervention period, $t(26) = 4.03$, $p < .001$, Cohen's $d = .17$. This suggests that the Intervention group displayed a decrease in HR during the intervention, consistent with physiological relaxation, that was not observed in the Control group.

A two-way ANOVA examining the effect of timepoint in the study and experimental condition on heart rate revealed a significant difference between experimental groups in mean HR, $F(1, 5.092) = 266.93$, $p < .001$, with the average heart rate of the Control group ($M = 66.53$, $SD = 10.15$) being significantly lower than that of the Intervention group ($M = 72.18$, $SD = 9.34$) throughout the study visit, Cohen's $d = 0.58$. Timepoint was also associated with significant differences in heart rate, $F(5, 5) = 43.33$, $p < .001$. There was no significant interaction effect between experimental group and timepoint, however, $F(5, 307) = .10$, $p = .993$. To examine whether these differences were evident at baseline, a one-way ANOVA was conducted and revealed a significant difference in heart rate between experimental groups at baseline, $F(1, 51) = 5.48$, $p = .023$, Cohen's $d = .64$. Changes in heart rate throughout the study visit were therefore explored with baseline heart rate as a covariate. A two-way ANCOVA exploring the effect of experimental group and timepoint in the study, with respiration rate and baseline HR as covariates, found a near-significant effect of experimental group on HR ($F(1, 6.404) = 4.55$, $p = .07$). Respiration rate was not a significant factor in the model ($F(1, 290) = .30$, $p = .584$) and was not included in additional analyses. One-way ANCOVAs exploring the effect of experimental group on changes in heart rate between sections of the study (baseline to HRVb, HRVb to stress tasks, stress tasks to recovery period), with

baseline heart rate as a covariate, revealed a significant difference between experimental groups in HR change from HRVb to stress tasks, and from stress tasks to recovery. After controlling for baseline HRV measures, (HR, RSA and RMSSD), the Intervention group displayed a greater increase in HR from HRVb to the stressful tasks, $F(1, 48) = 4.19$, $p = .046$, Cohen's $d = .22$, and a greater decrease in HR from the stressful tasks to the recovery period, $F(1, 43) = 4.77$, $p = .034$, Cohen's $d = .39$. This suggests that the Intervention group exhibited greater HR reactivity to the stressful tasks, but returned to their "normal" heart rate by the end of the study, as exhibited by no significant differences in HR changes from baseline to recovery between experimental groups, $F(1, 44) = .02$, $p = .886$. Experimental group differences in HR fluctuations throughout the study are visible in *Figure 5B* below.

Figure 5B. Group differences in HR changes throughout the laboratory visit.

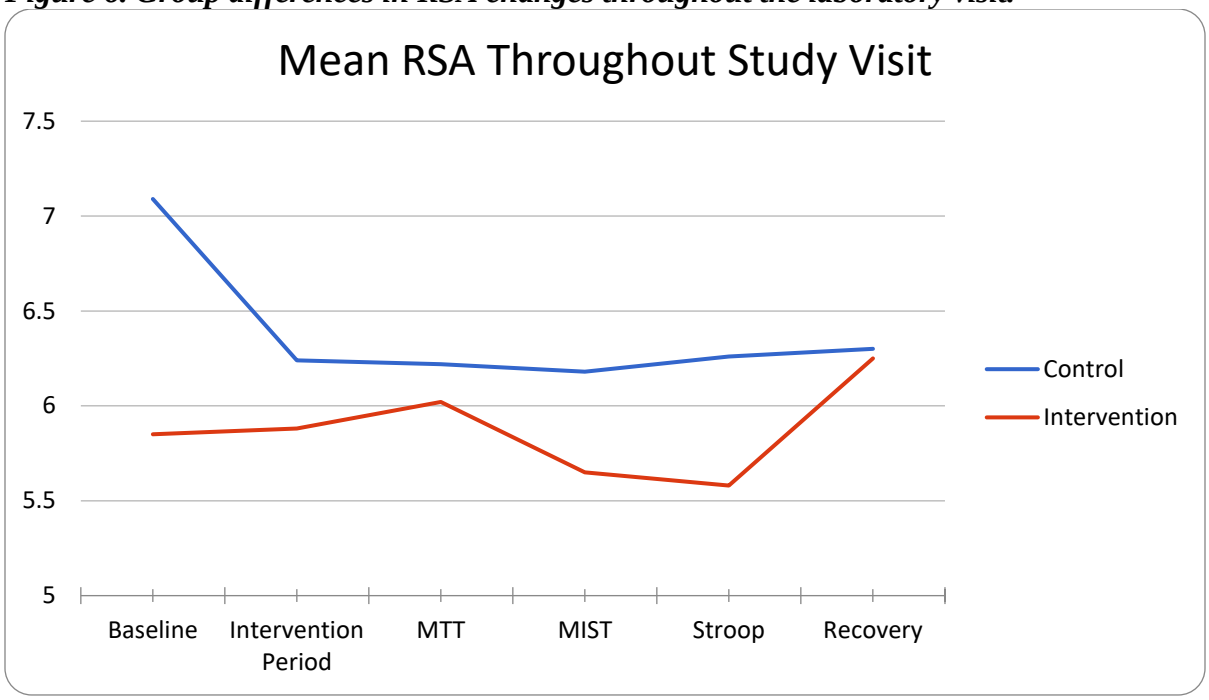


RSA. Paired samples t-tests explored within-group changes in RSA for each study condition. Neither condition demonstrated significant changes in RSA from baseline to the intervention period, ($t(51) = 1.01$, $p = .32$), or from the intervention period to the stressful tasks, ($t(54) = .43$, $p < .67$). The Intervention group did, however, exhibit a near-significant increase in RSA from stress to recovery, $t(25) = 1.78$, $p = .087$, which was driven by the increase in RSA from the final stressful task (Stroop task) to the recovery period, $t(25) = 2.73$, $p = .011$, Cohen's $d = .59$. The Control group did not exhibit a significant change in RSA from stress to recovery.

A two-way ANOVA examining the effect of timepoint in the study and experimental condition on RSA revealed a significant difference between experimental groups on RSA, $F(1, 5.011) = 8.52$, $p = .033$ Cohen's $d = .29$. Unlike the HR findings, timepoint was not associated with significant differences in RSA, $F(5, 5) = 1.20$, $p = .425$. There was also no significant interaction effect between experimental group and timepoint, $F(5, 307) = .78$, $p = .568$. There was no evidence of a significant difference between experimental groups in baseline RSA, $F(1, 51) = 2.13$, $p = .150$. A two-way ANCOVA exploring the effect of experimental group and timepoint in the study, with respiration rate and baseline RSA as covariates, found no significant effect of experimental group on RSA ($F(1, 5.436) = .42$, $p = .545$). Respiration rate was also not a significant factor in the model ($F(1, 290) = 2.12$, $p = .146$) and was not included in additional analyses. After controlling for baseline RSA, there were no significant differences between experimental groups found in RSA change between sections of the study (baseline to HRVb, HRVb to stress tasks, stress tasks to recovery period).

Similarly, there was no significant difference between experimental groups in overall RSA change from baseline to recovery, $F(1, 44) = .001$, $p = .977$.

Figure 6. Group differences in RSA changes throughout the laboratory visit.



RMSSD. Paired samples t-tests explored within-group changes in RMSSD for each study condition. The Control group demonstrated a significant decrease in RMSSD from baseline to the intervention period, $t(24) = 2.15$, $p = .042$, Cohen's $d = .18$. This was not evident in the Intervention group, which instead showed a significant decrease in RMSSD from the intervention period to the stressful tasks, $t(28) = 2.52$, $p = .018$, Cohen's $d = .37$. Neither group exhibited a significant change in RMSSD from stress to recovery, $t(49) = 1.38$, $p = .173$.

A two-way ANOVA examining the effect of timepoint in the study and experimental condition on RMSSD revealed a significant difference between experimental groups in RMSSD, $F(1, 5.013) = 15.82$, $p = .01$, with the Control group

exhibiting higher RMSSD (Cohen's *d* = .36). Similar to the RSA findings, timepoint was not associated with significant differences in RMSSD, $F(5, 5) = 1.27$, $p = .400$. There was also no significant interaction effect between experimental group and timepoint, $F(5, 307) = .66$, $p = .656$. To examine whether these differences were evident at baseline, a one-way ANOVA was conducted and revealed a difference in RMSSD approaching significance between experimental groups at baseline, $F(1, 51) = 3.69$, $p = .06$, with the Control group exhibiting higher RMSSD than the Intervention group, Cohen's *d* = .53. Changes in RMSSD throughout the study visit were therefore explored with baseline RMSSD as a covariate. A two-way ANCOVA exploring the effect of experimental group and timepoint in the study, with respiration rate and baseline RMSSD as covariates, did not find a significant effect of experimental group on RMSSD ($F(1, 5.677) = .06$, $p = .809$). As with the HR and RSA models, respiration rate was not a significant factor in the RMSSD model ($F(1, 290) = .31$, $p = .577$) and was not included in additional analyses. One-way ANCOVAs exploring the effect of experimental group on changes in RMSSD between sections of the study (baseline to HRVb, HRVb to stress tasks, stress tasks to recovery period), with baseline RMSSD as a covariate, revealed a near-significant difference between experimental groups in RMSSD change from HRVb to stress tasks, $F(1, 48) = 3.40$, $p = .072$, with the Intervention group exhibiting a greater decrease in RMSSD than the Control group (Cohen's *d* = .47). This suggests that the Intervention group exhibited greater RMSSD reactivity to the stressful tasks, but were not more reactive than the Control condition overall by the end of the study, as exhibited by no significant differences in RMSSD changes from baseline to recovery between experimental groups, $F(1, 44) = .41$, $p = .526$ and shown in *Figure 7* below. In fact, the

Intervention condition appears to have increased its RMSSD from baseline to recovery throughout the study, whereas the Control condition decreased its RMSSD throughout the study. As observed power for the experimental group differences in RMSSD change throughout the study was .096, it is possible that the Intervention group would increase its overall RMSSD with a larger sample size.

Figure 7. Group differences in RMSSD changes throughout the laboratory visit.

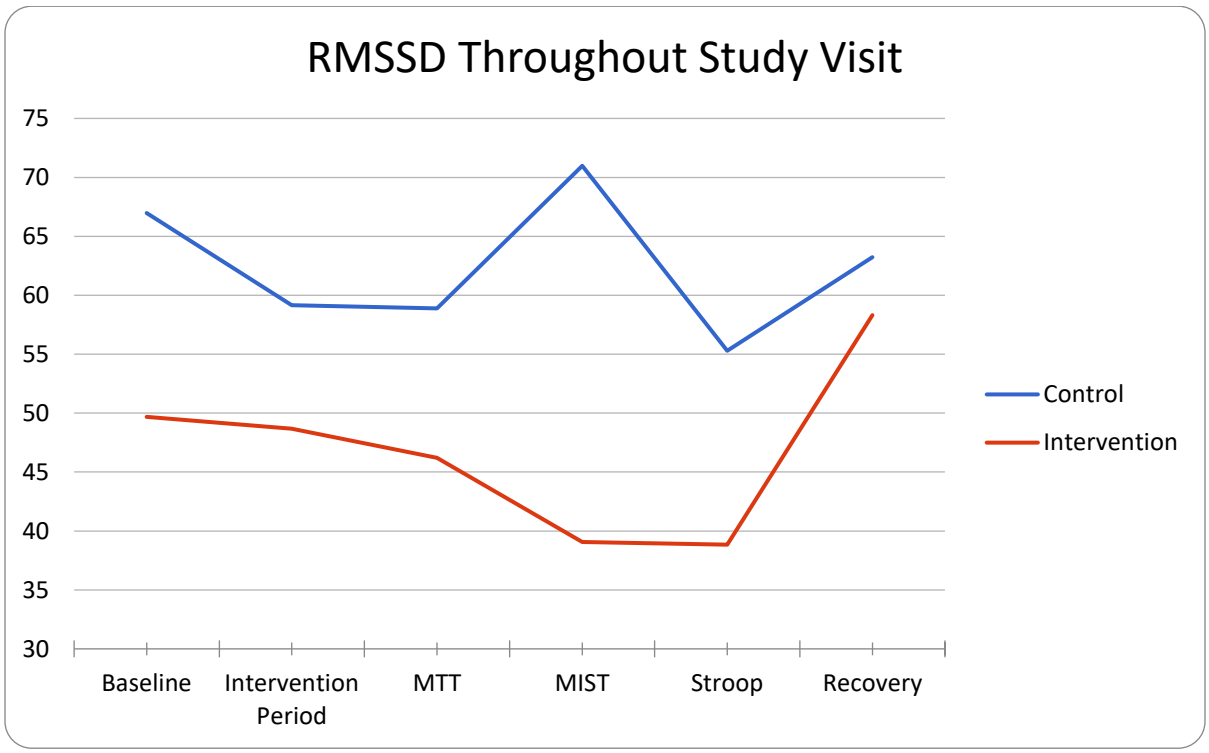


Table 10. Group differences in HRV measures throughout the laboratory visit.

	Heart Rate (F)	Heart Rate (p-value)	RSA (F)	RSA (p-value)	RMSSD (F)	RMSSD (p-value)
Baseline	5.04	**.03***	1.75	.193	2.13	.15
1st HRVb segment	6.34	**.015***	.69	.412	.79	.378
HRVb total	5.06	**.03***	.52	.476	1.11	.30
MTT	4.33	**.043*[a]**	.04	.839	1.46	.234
MIST	7.77	**.008****	1.21	.277	2.88	.10
Stroop	8.89	**.005****	1.52	.224	3.42	.07
Recovery	4.23	**.046*[b]**	.003	.955	.02	.887

Note. **denotes difference between experimental conditions is significant at $p < .01$. *denotes difference between experimental conditions is significant at $p < .05$. [a]When using one-way ANOVA, group difference in HR during MTT was near-significant, $p = .058$. [b]When using one-way ANOVA, group difference in HR during Recovery was not significant, $p = .07$.

Table 11. Group differences in changes in HRV measures at multiple study timepoints, controlling for baseline HRV (HR, RSA, RMSSD).

N=53	**Heart Rate (F)**	**Heart Rate (p-value)**	**RSA (F)**	**RSA (p-value)**	**RMSSD (F)**	**RMSSD (p-value)**
Baseline to 1st HRVb segment	1.79	.148	35.04	.000**	4.70	.003**
Experimental Condition effect	1.38	.247	2.09	.155	.07	.785
Baseline to HRVb total	3.23	.021*	93.26	.000**	4.08	.007**
Experimental Condition effect	.02	.902	.19	.669	.03	.849
HRVb to Stress tasks	8.71	.000**	.32	.86	1.52	.214
Experimental Condition effect	6.99	**.012***	.26	.615	2.16	.149
Stress to Recovery	4.69	.003**	.42	.793	.74	.571
Experimental Condition effect	6.89 Cohen's *d*= .39	**.012***	1.07	.307	1.95	.17

Note. **denotes difference between experimental conditions is significant at $p < .01$. * denotes difference between experimental conditions is significant at approximately $p < .05$.

***Aim 2*. BPV.**

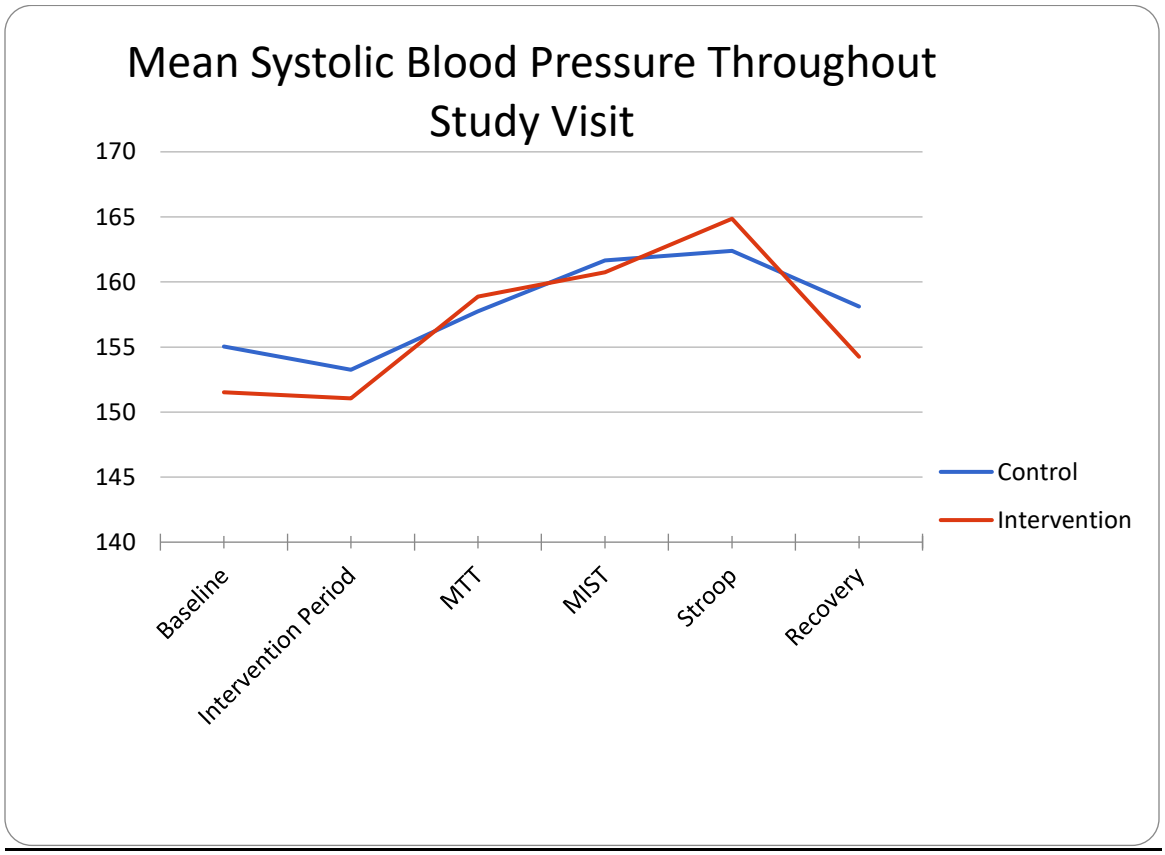
To examine the effect of practicing CR and HRVb on variations in blood pressure, I examined three outcome measures of BPV (systolic BP, diastolic BP, mean MAP) separately. *Table 12, Table 13,* and *Table 14* detail experimental group differences in the three measures of BPV, as well as changes in each measure throughout the study visit with and without covariates included in the analyses.

SYSTOLIC BP. Paired samples t-tests explored within-group changes in systolic BP for each study condition. Neither the Control nor the Intervention group exhibited a significant change in systolic BP from baseline to the intervention period, $t(39) = .43$, $p = .672$. Both experimental groups demonstrated a significant increase in systolic BP from the intervention period to the stressful tasks, $t(41) = 5.40$, $p < .001$, Cohen's $d = .56$. Only the Intervention group displayed a change after stress, showing a significant decrease from stress to recovery, $t(18) = 2.67$, $p = .015$, Cohen's $d = .43$. This suggests that, while both experimental groups experienced an increase in systolic BP during the stressful tasks, only the Intervention condition experienced a significant reduction in blood pressure during recovery.

A two-way ANOVA examining the effect of timepoint in the study and experimental condition on systolic BP revealed no significant difference between experimental groups in systolic BP, $F(1, 5.092) = 1.23$, $p = .317$. Timepoint, however, was associated with significant differences in systolic BP, $F(5, 5) = 12.63$, $p = .007$, Cohen's $d = .06$. There was no significant interaction effect between experimental group and timepoint, however, $F(5, 231) = .23$, $p = .952$. Including respiration rate as a covariate in the model did not significantly alter results, nor was respiration rate a

significant predictor of systolic BP, $F(1, 230) = .34$, $p = .561$. For this reason, respiration rate was not included in additional analyses. Examining systolic BP at baseline also yielded no significant difference between experimental groups, $F(1, 38) = .39$, $p = .539$. One-way ANOVAs explored changes in systolic BP between different segments of the study (baseline to HRVb, HRVb to stress tasks, stress tasks to recovery period) with and without baseline systolic BP as a covariate. In both sets of analyses, there were no significant differences between experimental groups found in systolic BP change between sections of the study. Similarly, there was no significant difference between experimental groups in overall systolic BP change from baseline to recovery, $F(1, 31) = .07$, $p = .801$. These results suggest that, while experimental condition did not exhibit a significant impact on systolic BP throughout the study, the significant effect of timepoint indicates that participants in both study conditions demonstrated significant changes in systolic BP throughout the lab visit, as shown in *Figure 8* below.

Figure 8. Experimental group differences in systolic BP changes throughout the laboratory visit.

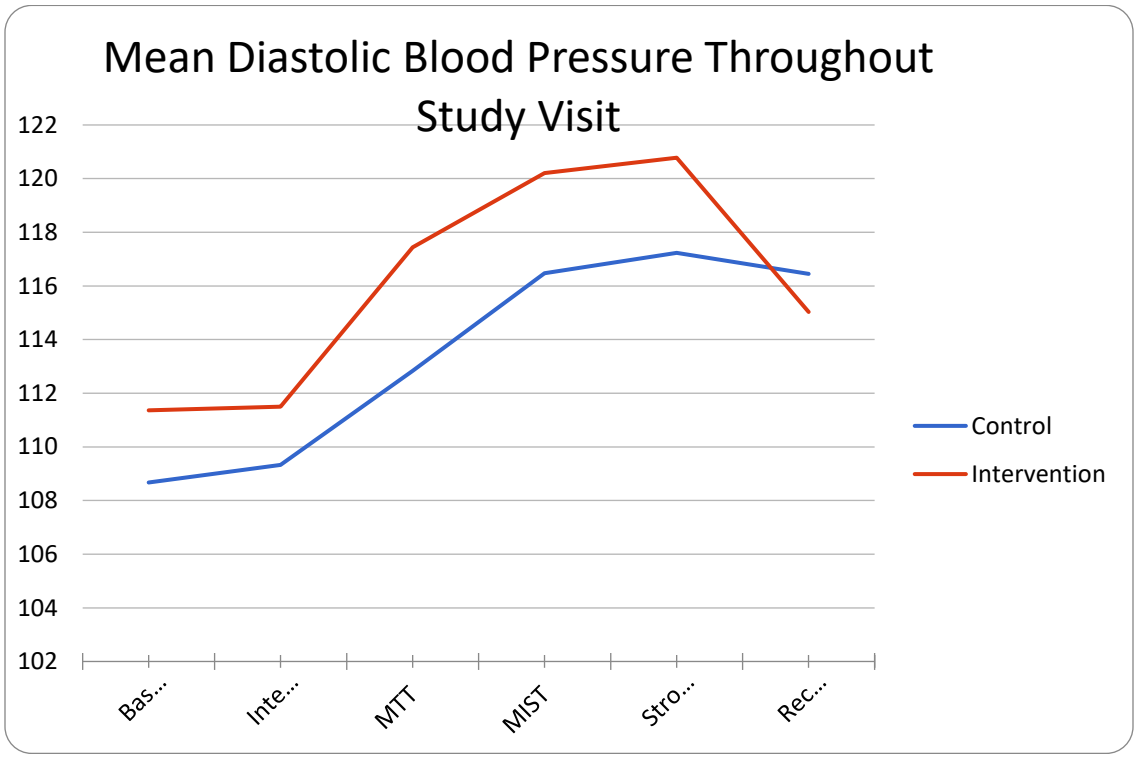


DIASTOLIC BP. Paired samples t-tests explored within-group changes in diastolic BP for each study condition, finding an identical result pattern to the systolic BP t-test analyses. Neither the Control nor the Intervention group exhibited a significant change in diastolic BP from baseline to the intervention period, $t(39) = 1.06$, $p = .295$. Both experimental groups demonstrated a significant increase in diastolic BP from the intervention period to the stressful tasks, $t(41) = 8.79$, $p < .001$, Cohen's $d = .65$. Only the Intervention group displayed a change after stress, showing a significant decrease from stress to recovery, $t(18) = 2.31$, $p = .033$, Cohen's $d = .39$. This suggests that, while both experimental groups experienced an increase in both systolic and diastolic BP during the stressful tasks, only the Intervention condition experienced a significant reduction in blood pressure during recovery.

A two-way ANOVA examining the effect of timepoint in the study and experimental condition on diastolic BP revealed a significant difference between experimental groups in diastolic BP, $F(1, 5.068) = 9.14$, $p = .029$, Cohen's $d = .22$. Timepoint was also associated with significant differences in diastolic BP, $F(5, 5) = 13.87$, $p = .006$. There was no significant interaction effect between experimental group and timepoint, however, $F(5, 231) = .31$, $p = .910$. As with the systolic BP findings, including respiration rate as a covariate in the diastolic BP model did not significantly alter results, nor was respiration rate a significant predictor of diastolic BP, $F(1, 230) = .18$, $p = .675$, and therefore it was not examined in additional analyses. To examine whether these differences were evident at baseline, a one-way ANOVA was conducted and revealed no significant difference in diastolic BP between experimental groups at baseline, $F(1, 38) = .47$, $p = .496$. One-way ANOVAs explored changes in diastolic BP

between different segments of the study (baseline to HRVb, HRVb to stress tasks, stress tasks to recovery period) with and without baseline diastolic BP as a covariate. In both sets of analyses, there were significant or near-significant differences between experimental groups in diastolic BP decrease from stress tasks to recovery with no covariates ($F(1, 30) = 4.17$, $p = .05$, Cohen's $d = .73$) and after controlling for baseline ($F(1, 31) = 2.80$, $p = .104$, Cohen's $d = .60$). Differences were more pronounced when exploring the decrease from the Stroop task (without the other stressful tasks) to recovery, which approached significance even after controlling for baseline diastolic BP, $F(1, 29) = 3.51$, $p = .071$, Cohen's $d = .73$. This suggests that, even when accounting for baseline diastolic BP, the Intervention group exhibited a steeper decline in diastolic BP after stress when compared with the Control group, as shown in *Figure 9*. There was no significant difference between experimental groups in overall diastolic BP change from baseline to recovery, $F(1, 31) = .87$, $p = .359$, suggesting that both experimental groups had similar overall changes from baseline to the recovery period of the study.

Figure 9. Experimental group differences in systolic BP changes throughout the laboratory visit.



MEAN ARTERIAL PRESSURE (MAP). Paired samples t-tests explored within-group changes in MAP for each study condition, finding an identical result pattern to the systolic and diastolic BP t-test analyses. Neither the Control nor the Intervention group exhibited a significant change in MAP from baseline to the intervention period, $t(39) = .52$, $p = .603$. Both experimental groups demonstrated a significant increase in MAP from the intervention period to the stressful tasks, $t(41) = 8.73$, $p < .001$, Cohen's $d = .64$. Only the Intervention group displayed a change after stress, showing a significant decrease from stress to recovery, $t(18) = 2.87$, $p = .01$, Cohen's $d = .43$. This suggests that, while both experimental groups experienced an increase in systolic BP, diastolic BP, and MAP during the stressful tasks, only the Intervention condition experienced a significant reduction in blood pressure during recovery.

Similar to the systolic BP findings, a two-way ANOVA examining the effect of timepoint in the study and experimental condition on MAP revealed no significant difference between experimental groups in MAP, $F(1, 5.079) = 2.41$, $p = .180$. Timepoint, however, was associated with significant differences in MAP, $F(5, 5) = 15.12$, $p = .005$. There was no significant interaction effect between experimental group and timepoint, however, $F(5, 231) = .26$, $p = .934$. As with the systolic and diastolic BP findings, including respiration rate as a covariate in the MAP model did not significantly alter results, nor was respiration rate a significant predictor of MAP, $F(1, 230) = .00$, $p = .998$, and therefore it was not examined in additional analyses. There was no evidence of a significant difference between experimental groups in baseline MAP, $F(1, 38) = .02$, $p = .885$. One-way ANOVAs explored changes in MAP between different segments of the study (baseline to HRVb, HRVb to stress tasks, stress tasks to recovery period) with and without baseline MAP as a covariate. In both sets of analyses, there were near-significant differences between experimental groups in MAP decrease from stress tasks to recovery with no covariates ($F(1, 30) = 2.96$, $p = .096$, Cohen's $d = .61$) and after controlling for baseline ($F(1, 31) = 2.4$, $p = .131$, Cohen's $d = .53$). Differences were more pronounced when exploring the decrease from the Stroop task (without the other stressful tasks) to recovery, which approached significance even after controlling for baseline MAP, $F(1, 29) = 3.28$, $p = .081$, Cohen's $d = .66$. This suggests that, even when accounting for baseline MAP, the Intervention group exhibited a steeper decline in MAP after stress when compared with the Control group, as shown in *Figure 10*. These results follow a similar pattern as diastolic BP results. There was no significant difference between experimental groups in overall MAP change from baseline to recovery, $F(1, 31)$

= .55, p = .462, suggesting that both experimental groups had similar overall changes from baseline to the recovery period of the study.

Figure 10. Experimental group differences in MAP changes throughout the laboratory visit.

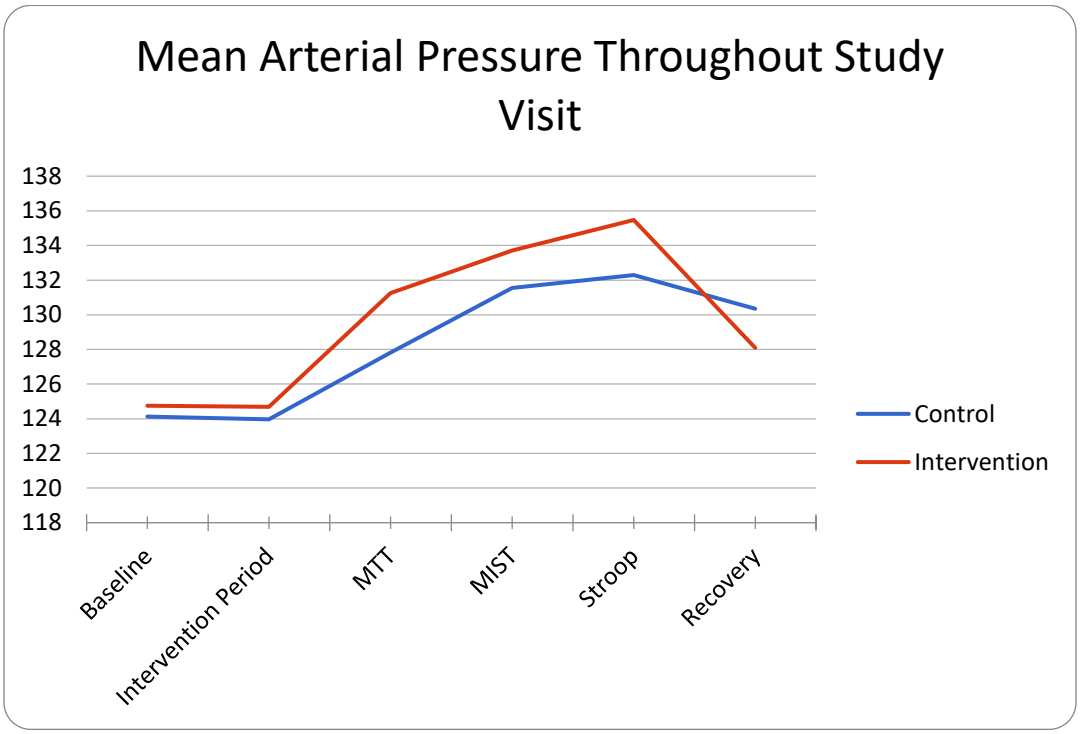


Table 12. Group differences in BPV measures throughout the laboratory visit.

	SysAmp (F)	SysAmp (p-value)	DiasAmp (F)	DiasAmp (p-value)	MAP (F)	MAP (p-value)
Baseline	.39	.539	.47	.496	.02	.885
HRVb total	.21	.648	.46	.50	.04	.841
MTT	.05	.819	1.81	.186	.93	.339
MIST	.03	.87	.81	.374	.24	.631
Stroop	.21	.648	.86	.359	.62	.436
Recovery	.44	.513	.13	.724	.26	.615

Table 13. Group differences in BPV changes between study segments, with baseline of each variable as covariate.

	SysAmp (F)	SysAmp (p-value)	DiasAmp (F)	DiasAmp (p-value)	MeanMAP (F)	MeanMAP (p-value)
Baseline to HRVb total	3.44	.046	3.34	.05	2.69	.085
Experimental Condition effect	.05	.823	.18	.678	.02	.888
HRVb to MTT	.56	.577	2.00	.153	2.23	.126
Experimental Condition effect	.01	.93	1.13	.298	.56	.46
MTT to MIST	.16	.850	.36	.698	.33	.721
Experimental Condition effect	.24	.628	.26	.615	.01	.908
MIST to Stroop	.59	.562	.40	.671	.62	.545
Experimental Condition effect	.46	.505	.44	.512	.79	.381
Stroop to Recovery	.72	.496	3.37	.048*	1.69	.202
Experimental Condition effect	1.35	.254	3.51 Cohen's d= .73	**.071***	3.28 Cohen's d= .66	**.081***
HRVb to Stress (avg of 3 tasks)	1.31	.283	.86	.435	1.29	.291
Experimental Condition effect	.33	.571	.85	.363	.85	.363
Stress to Recovery	.49	.616	2.49	.099	1.42	.258
Experimental Condition effect	.89	.354	2.80 Cohen's d= .60	.104	2.40 Cohen's d= .53	.131

Note. **denotes difference between experimental conditions is significant at $p < .01$. * denotes difference between experimental conditions is nearly significant at approximately $p < .05$.

Table 14. Group differences in BPV changes between study segments, with no covariates.

	SysAmp (F)	SysAmp (p-value)	DiasAmp (F)	DiasAmp (p-value)	MeanMAP (F)	MeanMAP (p-value)
Baseline to HRVb total	.002	.967	.01	.938	.001	.973
HRVb to MTT	.000	.988	.64	.429	.39	.535
MTT to MIST	.27	.609	.18	.679	.01	.937
MIST to Stroop	.54	.467	.56	.461	.75	.393
Stroop to Recovery	1.44	.240	4.18	**.05***	3.43	.074
HRVb to Stress (avg of 3 tasks)	.57	.455	.80	.379	.93	.342
Stress to Recovery	.81	.376	4.17 Cohen's d= .73	**.05***	2.96 Cohen's d= .61	.096

Note. *denotes difference between experimental conditions is significant at approximately $p < .05$.

***Exploratory Analyses*. One-week Follow-Up.**

Exploratory analyses were conducted for my follow-up data in order to examine experimental group differences in perceived stress one week after the laboratory visit. A total of 52 participants were reached for my follow-up survey, with 29 in the Control group and 23 in the Intervention group. Shapiro-Wilk's test of normality was not significant for either the baseline or follow-up PSS score for either experimental group, suggesting a normal distribution of both variables. Both scores were within normal ranges for kurtosis and skewness (+/-1) and revealed no outliers. A one-way ANOVA revealed no significant differences between experimental groups on baseline PSS score ($F(1, 66) = .06$, $p = .805$), follow-up PSS score ($F(1, 50) = 1.00$, $p = .323$), or change from baseline to follow-up PSS score ($F(1, 50) = 2.34$, $p = .132$), although participants in the Intervention group ($M = -6.78$, $SD = 7.70$) did exhibit a seemingly steeper decline in PSS score at follow-up than those in the Control group ($M = -3.52$, $SD = 7.60$) with an effect size of $d = -0.43$.

DISCUSSION

The current study examined the effect of two interventions for coping with stress, HRVb and CR, on multiple outcomes (cognitive performance, cigarette craving, affect, and HRV) in adult women smokers who were nicotine- and tobacco-deprived. All participants were daily moderate-to-heavy smokers recruited from the community in the central New Jersey area, and had not smoked for at least 12 hours prior to the study visit. Primary results suggested that participants who practiced HRVb exhibited a significantly greater decrease in smoking craving when compared with participants who practiced neutral control tasks. Psychophysiological measures during the intervention period indicated that, compared with the Control group, participants practicing HRVb exhibited a significant decrease in heart rate and significantly higher LF power. These measures, combined with the significant decrease in all measures of smoking craving that was reported by the Intervention group after practicing HRVb, suggests that participants were successful in slowing their breathing for the purpose of relaxation, and that this influenced their craving for a cigarette. When three stressful tasks were introduced, however, there appeared to be no difference between experimental groups on any outcome measure. During the post-stress recovery period, differences between the experimental groups emerged once again, with only the Intervention group exhibiting a significant reduction in blood pressure and RSA. The Control group did not exhibit these psychophysiological adaptations, and exploratory analyses found that trait rumination played a role in negative affect reported during the recovery period for this group. Some additional hypotheses were not significantly supported by my data, although general trends in the data were not incongruent with my stated hypotheses. Overall, the evidence

suggests that the Intervention group was successful at practicing HRVb and decreasing their smoking craving, which may have contributed to their psychophysiological recovery after stress.

Baseline Measures, Manipulation Checks, and Follow-up Data

Baseline group differences between my study conditions indicated that my randomization was generally successful. There were no significant differences between my experimental groups in age or any baseline measure, with the exception of social desirability score. My Intervention group reported a significantly higher level of social desirability than my Control group, although including social desirability as a covariate did not significantly impact results in my analyses.

There were also no significant experimental group differences in responses to the stress appraisal measure (SAM) after my stressful tasks. This suggests that my CR intervention was not effective at influencing stress appraisal during this study. The reduced efficacy of this manipulation may have been related to the context of CR practice during this study, including my participants' state of nicotine withdrawal when learning about CR, the long duration of the study overall (3.5-4 hours), and the three stressful tasks immediately following the CR manipulation. Replicating this study with more opportunities for practicing CR over multiple days before stress is introduced may increase its effect during stress.

Follow-up data examining changes in perceived stress one week after the study visit did not suggest that my groups experienced significantly different changes in perceived stress (p = .132), although the Intervention group reported a greater decrease in perceived stress than the Control group.

Cognitive Performance

Contrary to my hypothesis, there were no significant differences between the Control group and the Intervention group on any measure of Stroop task performance. An exploration of covariates as potential predictors of performance revealed that age was a significant predictor of reaction time on both the CWS and VS tasks. This is consistent with past findings on the Stroop task. Previous research using a large sample found that, among healthy volunteers, speed-dependent Stroop scores (i.e., reaction time) were affected by age, with older adults performing at slower speeds than younger adults (Van der Elst, Van Boxtel, Van Breukelen, & Jolles, 2006). Notably, these results are consistent with past Stroop performance results for adult female heavy smokers, which found that age predicted reaction time beyond 2-day abstinence from nicotine (Kos, Hasenfratz, & Battig, 1997). While consistent with past research on the effect of age on reaction time in the Stroop task, these findings suggest that only a robust predictor of Stroop performance was detectable in this small study sample. Means plots comparing the two experimental groups indicated slightly higher performance of the Intervention group above the Control group, an effect in need of further elucidation with a larger study sample.

Baseline nicotine dependence as measured by the FTND also emerged as a significant predictor of accuracy on the CWS, with lower FTND scores predicting better percent correct and total correct responses on the CWS. It is possible that individuals who were more nicotine dependent were more adversely affected by the 12-hour abstinence period and subsequent mental tasks, performing at a lower rate than individuals who were less nicotine-dependent. Past research has indicated that smoking abstinence impairs

performance on the CWS (Pomerleau, Teuscher, Goeters, & Pomerleau, 1994). Results about the influence of nicotine dependence and age on cognitive task performance suggest that additional resources to improve cognitive functioning would need to be included for smoking cessation treatment for older and highly dependent smokers.

Smoking Craving

My results showed a significant effect of the HRVb intervention on decrease in smoking craving reported between the study baseline and immediately after the intervention period, with only the Intervention group reporting a significant decrease in smoking craving. The greatest change was reported in Factor 1 of my craving measure, suggesting that practicing HRVb reduces the desire to smoke with smoking perceived as rewarding within a short period of time, regardless of baseline craving and multiple other covariates. All participants in this study had been asked to refrain from smoking for at least 12 hours and exhibited high smoking craving at the start of the study. The significant decrease in smoking craving that only occurred for the Intervention group suggests a powerful influence of HRVb on smoking craving that future research needs to explore. These results suggest that HRVb may indeed be a useful coping tool for reducing smoking craving that can be incorporated into smoking cessation treatment.

Contrary to my hypothesis, however, HRVb and CR did not protect against the effect of stress on smoking craving. This study's results suggest that, while there was a short-term effect of HRVb on smoking craving, this effect disappeared immediately after stress. This may suggest that a single session of HRVb or CR training may not be sufficient to protect against the effect of stress on smoking-related outcomes and multi-

session treatment may be warranted. Future studies will need to elucidate the effects of multiple sessions of HRVb and CR to determine appropriate intervention dosage.

Negative Affect

Results from this study suggest that HRVb and CR did not have a significant impact on group-level changes in either positive or negative affect when compared with control tasks. This is a surprising difference from my results on changes in cigarette craving throughout the laboratory visit, which found significant differences between my experimental groups in smoking craving, particularly due to past research and theoretical models conceptualizing craving as an affective state (Baker et al., 1987; Zinser, Baker, Sherman, & Cannon, 1992) and a recent meta-analysis identifying that affective manipulations in laboratory settings reduce craving to smoke by reducing negative affect (Heckman et al., 2013). This may suggest that my intervention may target a specific subtype of negative affect (craving), but not negative affect as a whole. The effect of the intervention did not remain during stressful tasks, suggesting that craving to smoke may be more volatile and responsive to brief interventions than negative affect as a whole.

Notably, baseline rumination as measured by the RRS emerged as the strongest predictor of changes in negative affect on the PANAS. As other covariates included scores on measures of depression, perceived stress, generalized anxiety, and past-month cigarettes smoked per day, the relationship between baseline trait rumination and changes in negative affect throughout the lab visit is particularly noteworthy. During the post-stress recovery period, trait rumination appeared to play a role in negative affect reported by the Control group, and not the Intervention group, despite playing a significant role for both groups at other points in the study. Rumination, which involves fixating attention on

negative emotional experiences while repetitively thinking about their presence and meaning in a self-focused manner (Nosen and Woody, 2014; Nolen-Hoeksema, 1991), has been shown to increase negative affect (Just & Alloy, 1997; Nolen-Hoeksema & Morrow, 1993; Nolen-Hoeksema, Morrow, & Fredrickson, 1993). While there is little research on the link between rumination and smoking behavior, Richmond, Spring, Sommerfeld, and McChargue (2001) found that rumination accounts for a significantly larger amount of variance in depression symptoms for smokers (46%) compared with nonsmokers (17%). Future studies will need to further examine this relationship and the influence of interventions that target trait rumination on smoking cessation outcomes.

HRV

An examination of changes from baseline to the intervention period in multiple frequency-domain measures of HRV (LF Power, LF Peak Power Frequency, and LF/HF Ratio) found that, compared with the Control group, the Intervention group exhibited significantly higher LF Power during the intervention period, greater increases in LF Peak Power Frequency and LF/HF Ratio. As LF levels typically increase with slow breathing that creates a resonant effect, these findings indicate that the Intervention group was successfully practicing slow breathing as instructed during the intervention period. The Intervention group exhibited LF Peak Power Frequency of .10 Hz, which is associated with a breathing rate of around 6 breaths per minute and is known to acutely enhance cardiovagal baroreflex sensitivity (Tzeng, Sin, Lucas, & Ainslie, 2009). In addition, the significant increase in LF/HF Ratio that was only observable in the Intervention group from baseline to the intervention period indicates that only the Intervention group stimulated activity of the baroreflex through slowed breathing. Recent

evidence has indicated that, contrary to previous assumptions that LF power is a measure of cardiac sympathetic tone, LF power serves as an index of baroreflex function, and manipulations that influence LF power do so by affecting modulation of cardiac autonomic outflows by baroreflexes (Goldstein, Bentho, Park, & Sharabi, 2011).

There were significant differences between experimental groups in all other measures of HRV examined (HR, RSA, RMSSD), although these were related to baseline differences and not effects of the intervention. There were no significant timepoint x condition interactions for any measure of HRV. When compared with the Control group, the Intervention group exhibited a significantly higher increase in heart rate during stressful tasks, and a significantly greater decrease in heart rate during recovery after the stressful tasks, after controlling for baseline heart rate. This volatility suggests that the intervention did not buffer the influence of stress on heart rate, corroborating past findings for CR that found cognitive reappraisal did not influence the effect of social stress on heart rate (Shermohammed et al., 2017; Denson et al., 2014). Although there were no significant differences in participants' self-reported stress appraisal after the stressful tasks, calling into question whether the CR intervention was successful during the stressful tasks, these past findings make it unclear whether the CR intervention would have impacted experimental group differences in HR had the intervention been successful. Regardless of the CR intervention, this finding may further suggest that practicing the HRVb intervention allowed participants to return to their baseline physiology faster than participants in the Control condition.

Of note, observed power for analyses of RSA and RMSSD changes from baseline to recovery was remarkably low (.05 for RSA and .096 for RMSSD). *Figures 6 and 7*

exhibit a trend of a closing gap between experimental groups in RSA and RMSSD throughout the study, despite larger differences in baseline measures of these outcomes. Replication of these findings with a larger sample size, and possibly with repeated practice sessions of the intervention, may increase both statistical power and effect size in favor of my intervention's hypothesized effect.

At baseline, there was a significant difference in heart rate between conditions, with the Intervention group having a significantly higher heart rate. As shown in *Figures 6 and 7*, the Intervention condition also exhibited lower RSA (p = .15) and RMSSD (p = .06) at baseline. This is interesting because the Intervention group also reported significantly higher craving than the Control group at baseline. This observed consistency in lower HRV and higher self-reported craving suggests that participants with lower parasympathetic resilience also exhibited higher dependence on cigarettes. This is consistent with evidence that links smoking and related outcomes with lower HRV.

BPV

Results of between-group BPV data analyses found timepoint to be consistently associated with significant changes in all three BPV measures (systolic BP, diastolic BP, and MAP), although no time by experimental group interactions were significant. This suggests that participants' blood pressure fluctuated throughout the study, but the HRVb and CR intervention did not have a significant impact on these fluctuations. This was evident throughout multiple timepoints in the study, as well as the overall change from baseline to recovery in all BPV outcome variables. Past research on the effects of biofeedback on blood pressure has been controversial, with some studies indicating that blood pressure may only slightly decrease with HRVb training, and other findings

suggesting that biofeedback lowers systolic BP and MAP reactivity to a challenging task (Palomba et al., 2011).

Within-group BPV results indicated no significant changes across the sample between baseline and the intervention period, followed by a significant increase in systolic BP, diastolic BP, and MAP during the stressful tasks. This suggests that the stressful tasks were successful at inducing psychophysiological stress, regardless of experimental group. Only the Intervention condition experienced a significant reduction in blood pressure during recovery after the stressful tasks, however, which suggests that individuals practicing the intervention displayed resilience by decreasing their blood pressure at a faster rate than those in the Control group.

As with my HRV findings, analyses for BPV were significantly underpowered, with observed power being no higher than .4 (observed in diastolic BP analyses). Diastolic BP and MAP showed similar trends in changes from stress to recovery, but only changes in diastolic BP were statistically significant.

Study limitations

While this study was able to identify interesting relationships between the practice of HRVb and smoking craving in heavy smokers who were nicotine-deprived, several other hypotheses were not supported, possibly due to limitations of the study design. Several analyses were underpowered due to small sample size, although raw data and trends observable in my presented figures suggest that the data would support my hypotheses with adequate statistical power. Future studies would need to include a larger sample size in order to effectively assess whether the data support my hypotheses with adequate power.

Sample characteristics may have also influenced the results of this study and limited the degree to which my findings apply to the general population of adult smokers. My sample consisted exclusively of women; therefore, I cannot assume that the same trends in findings would apply to men practicing HRVb or CR. Moreover, my exclusionary criteria excluded light smokers and individuals with various health conditions (e.g., heart conditions, morbid obesity, etc.), further narrowing the population of smokers to which these findings can generalize. Many exclusionary criteria for this study were required to reduce confounds for psychophysiological data measurement. Future research aimed at examining effectiveness of HRVb and CR within a smoking cessation trial may need to reduce the number of exclusionary criteria to increase external validity of study findings.

Finally, it is important to note that my interventions' limited effects were most likely due to the single-session design of this study. Most studies of HRVb, particularly as an intervention for mental health, have examined it over multiple sessions and found positive effects over time. Similarly, CR appears to have positive effects as a learned skill over time, or as a skill learned preceding a mild stressful task. This study was unique in its inclusion of three stressful tasks that were highly effective at inducing stress. It is unsurprising that a single session of practice for HRVb and CR did not promote resilience against this level of stress, and that more opportunities to practice and learn these skills would be necessary for their benefits to appear during stress. Despite these limitations, HRVb reduced smoking craving within minutes of practice in women who were nicotine-deprived and experiencing withdrawal symptoms, suggesting that it may be a promising intervention for future examination in smokers who are trying to quit. Future research

must explore HRVb and CR over multiple practice sessions in order to account for learning over time, and to increase their chances of success when stress is introduced.

Conclusion and Implications

This study assessed whether combining a cognitive and behavioral approach to improve stress responding in smokers may be efficacious in minimizing the impact of stress on smoking craving and related outcomes. Combining both cognitive and behavioral self-regulation skills for stress adaptation is consistent with cognitive-behavioral theory upon which evidence-based treatments for Axis I pathology were developed, as behavioral (e.g., HRVb) and cognitive (e.g., reappraisal) approaches to self-management allow the individual to target both internal and external stimuli when coping with stress (Rokke & Rehm, 2001). Beyond psychological effects of stress, the combination of such approaches has implications for informing smoking cessation treatments, as it has been previously noted that current treatments for substance addiction "are failing to address important factors that are active in sustaining [such] pathology, because phenomena that lead to relapse... are mediated by physiological as well as cognitive processes" (Eddie, Vaschillo, Vaschillo, & Lehrer, 2015, p. 266). As stress has been implicated in the development of a wide range of psychopathology (Dohrenwend, 2000; Abravanel & Sinha, 2015) and poses an obstacle to successful cessation, the failure of current smoking cessation treatment to improve stress responding indicates a significant gap between science and smoking cessation treatment. Zvolensky et al. (2015) recently noted:

> "*[Evidence suggests] that the use of common strategies that may be successful for the general population of smokers (e.g., behavioral strategies, pharmacotherapy) may be less useful for smokers with emotional disorders who may have tried these methods without success. Given these findings coupled with current evidence that smokers with (vs. without) emotional disorders made more quit attempts and hence appear to be genuinely interested in quitting, efforts should likely be focused on specialized treatment development (rather than solely the application of commonly applied strategies) to identify those treatment strategies to be maximally efficacious for this high-risk subpopulation*" p. 130.

In order to further improve cessation rates, CR instruction and HRVb must be further examined as skills in need of repeated practice in order to be incorporated at low cost into standard smoking cessation treatment, which currently consists of pharmacotherapy and smoking cessation counseling according to the *Clinical Practice Guideline* (Fiore et al., 2008).

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