Emotion Regulation and Smoking:

An Ecological Momentary Assessment Study

by

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Dissertation submitted to the Faculty of the

Medical and Clinical Psychology Graduate Program

Uniformed Services University of the Health Sciences

In partial fulfillment of the requirements for the degree of

Doctor of Philosophy 2018

UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES
SCHOOL OF MEDICINE GRADUATE PROGRAMS
Graduate Education Office (A 1045), 4301 Jones Bridge Road, Bethesda, MD 20814

APPROVAL OF THE DOCTORAL DISSERTATION IN THE DEPARTMENT OF MEDICAL AND CLINICAL PSYCHOLOGY

Title of Dissertation: "Emotion Regulation and Smoking: An Ecological Momentary Assessment Study"

Name of Candidate: Jessica MacIntyre
Doctor of Philosophy Degree
March 12, 2018

DISSERTATION AND ABSTRACT APPROVED:

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ABSTRACT

Emotion Regulation and Smoking: An Ecological Momentary Assessment Study

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Cigarette smoking is the leading cause of preventable death in US. A greater understanding of the psychological processes underlying smoking and relapse is required to develop more effective interventions. Recent research has examined emotion regulation (ER) in addictive behaviors, including tobacco addiction. Previous research has generally conceptualized emotion regulation as a relatively stable construct. However, both theory and data suggest that emotion regulation may also have state-like qualities. The current study builds on the relationship between emotion regulation and smoking by going beyond an examination of overall emotion regulation tendencies and examining fluctuations in emotion regulation abilities. Participants (*N*=29), who were attempting to quit smoking, were given a mobile device for six weeks which prompted them to complete up to four random assessments a day. Participants could also complete participant-initiated assessment if they missed a random assessment. Participants completed assessments of emotion regulation, negative affect, craving, and smoking in the lab and in the field. The primary aims examined the between- and within-subject relationships between emotion regulation, negative affect, craving, and smoking, using

nomothetic and idiographic analyses. The main findings of the study were as follows. First, an association between emotion regulation and negative affect was observed both in the lab and in the field. As emotion dysregulation increased, negative affect increased. Second, there was no evidence for an association between emotion regulation and craving. Similarly, there was little evidence for an association between emotion regulation and smoking during the following week or before the next field assessment. Third, emotion regulation, specifically nonacceptance, may moderate the association between negative affect and craving. Individuals who have generally higher levels of nonacceptance of current emotions report more craving for cigarettes when they experience increases in negative affect, whereas individuals with lower levels of nonacceptance do not. In sum, there is evidence that state emotion regulation is associated with negative affect and that emotion regulation moderates the association between negative affect and craving in smokers attempting to quit.

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INTRODUCTION

Cigarette smoking is the leading cause of preventable death in the US. A greater understanding of the psychological processes underlying smoking and relapse is required to develop more effective interventions. Recent research has examined the role of emotion regulation in addictive behaviors, including tobacco addiction. Previous research has generally conceptualized emotion regulation as a relatively stable construct. However, both theory and data suggest that emotion regulation may also have state-like qualities. State emotion regulation may play a role in when or whether an individual attempting to quit smoking relapses. The over-arching goal of the current study was to examine the role of emotion regulation in smoking behavior. Knowledge gained from this study may improve smoking cessation interventions.

The introduction is organized as follows. First, the literature on the negative health effects of tobacco addiction is reviewed. Second, an overview of theoretical approaches to tobacco addiction is provided, as well as a review of current treatment approaches. Third, the construct of emotion regulation is introduced, and the literature on its role in addiction is described. Last, the methodology used in the study, ecological momentary assessment (EMA), is described.

TOBACCO USE AND HEALTH

In 1898, a German medical student suggested that tobacco dust could be responsible for increased lung tumors observed in tobacco workers (206). In 1912, Isaac Adler attributed the rise in lung cancer occurrence to smoking tobacco, rather than tobacco dust. Despite these hypotheses, the first population study showing a correlation between smoking and lung cancer was not published until 1939, with many more

corroborating the findings published in following years (206). Animal studies published as early as 1931 drew clear connections between tobacco and cancer as mice and rabbits grew tumors where tobacco had been put on their shaved skin. By the 1950's, there was sufficient evidence of numerous carcinogens in cigarette smoke. In 1954, the American Cancer Society, the Public Health Cancer Association, and similar organizations across Europe publicly recognized the evidence of an association between smoking and cancer, and recommended individuals stop smoking to reduce the risk of cancer (206). Lagging behind the scientific evidence, the 1964 Surgeon General's Report was the first government report to officially recognize smoking as a cause of lung cancer and heart disease (259).

Harmful Health Effects of Tobacco Smoking

Scientists have continued to study the negative effects of cigarette smoking on health. Smoking does cause lung cancer (58), as well as increasing the risk of cancer in nearly every area of the body including, but not limited to, the liver (279), stomach (252), larynx (184), esophagus (184), bladder (73), colon (240), pancreas (76), and cervix (275). Smoking also drastically increases the risk of developing various serious respiratory diseases including chronic obstructive pulmonary disease (168) and asthma (130). A large body of research has linked smoking with cardiovascular disease including stroke (187), aneurysmal arterial disease (195), and sudden coronary death (133). Smoking during pregnancy has also been linked to a large number of reproductive health effects such as congenital malformations (165), stillbirth, sudden infant death (276), and later behavioral and cognitive impairments (56). Taken together, these health effects contribute to the fact that cigarette smoking is the leading cause of preventable death in

the United States, accounting for almost one in every five deaths (257). There is a 50% chance that a lifelong smoker will die prematurely from smoking related consequences (59).

Smoking Prevalence

Over 115 years have passed since doctors first guessed at the association between tobacco and tumors, over 75 years have passed since the first evidence of the smoking-cancer link was published, and over 50 years have passed since the historic 1964 Surgeon General's Report was released. However, many individuals continue to smoke. Approximately 40 million (16.8%) American adults currently smoke (257). Smoking is slightly more prevalent in men (18.8%) compared to women (14.8%), with highest rates for individuals between 25 and 44 years of age (20.0%), followed closely by individuals between 45 to 64 years old (18.0%), and individuals 18 to 24 years old (16.7%). Smoking rates have declined in adolescents, but still remain at 9% for high school students and 5% for middle school students (42).

Rates of smoking vary by race and ethnicity, with the highest prevalence of smoking in American Indians/Native Alaskans (29.2%) and multi-race individuals (27.9%), followed by white individuals (18.2%), black individuals (17.5%), Hispanic individuals (11.2%), and Asian individuals (9.5%) (41). Education level is also correlated with smoking rates. Smoking rates decline as education level increases. Almost 23 out of 100 individuals with 12 or fewer years of education smoke cigarettes, a rate that declines to about 20 out of 100 individuals with some college education, all the way down to about 5 out of 100 adults with a graduate degree (41).

Neurobiology of Nicotine Addiction

While there are a myriad of reasons that individuals begin to smoke, it is critical to acknowledge that a primary factor maintaining ongoing smoking behavior is the fact that nicotine, the primary psychoactive (256) ingredient in cigarettes, is addictive (17). A number of factors contribute to nicotine addiction including genetics, environmental factors, and pharmacology (16). When smoke from a cigarette is inhaled, it carries nicotine particles into the lungs, allowing the nicotine to enter the bloodstream and travel to the brain. In the brain, nicotine binds with nicotinic cholinergic receptors triggering the release of neurotransmitters. One of the primary neurotransmitters released is dopamine. The release of dopamine is important in reward and contributes to the reinforcing effects of nicotine.

In addition to dopamine, nicotine increases the release of many primary neurotransmitters including noradrenaline (44), acetylcholine (274), glutamate (177), and GABA (278). The release of these neurotransmitters leads to a wide variety of effects on learning, memory, anxiety, and pain perception (54). Positive psychoactive effects of smoking include improved concentration and attention (105) and reduced levels of anxiety (7).

Nicotine Withdrawal and Cessation

It is difficult to quit smoking. According to data collected by the U.S. Centers for Disease Control (CDC) between 2001 and 2010, 68.8% of current adult smokers wanted to quit, 52.4% had attempted to quit in the last year, and only 6.2% had recently been able to quit (40). In addition to the addictive properties of nicotine and the desirable effects of smoking, a number of adverse withdrawal symptoms help to maintain smoking behavior and thwart quit attempts. Physical symptoms of withdrawal include cough,

constipation, dizziness, poor sleep, bradycardia, and mouth ulcers (120). Additionally, many individuals gain weight after quitting smoking due to increased hunger, a particularly unwanted consequence for individuals with body image concerns or weight-related health concerns (10). Frequently reported cognitive symptoms include impaired memory, confusion, and difficulty concentrating (109; 178). Another feature of nicotine withdrawal is craving. Craving is defined as "a strong, urgent, or abnormal desire for a certain substance or activity" by National Cancer Institute (188).

Most pertinent to the current study, smoking cessation is also associated with significant (acute) increases in negative affect (122; 203). The Diagnostic and Statistical Manual of Mental Disorders (DSM-5) includes irritability, frustration, anger, anxiety, and depressed mood as symptoms associated with nicotine withdrawal (6). Of note, most of these somatic, cognitive, and affective withdrawal symptoms are the opposite of the positive effects associated with smoking.

Withdrawal symptoms begin approximately four to 24 hours following cessation for a chronic smoker. Symptoms are typically most severe three days after the final cigarette and most symptoms return to baseline after about 10 days (231), though increased weight, hunger, and cigarette craving may last up to six months following cessation (121).

Reinforcement Theories of Smoking

Various theories have been developed to explain the psychological underpinnings of nicotine addiction. Three theories will be discussed here due to their connection to the current study.

Positive Reinforcement Theory

Positive reinforcement refers to the pairing of a stimulus with a pleasurable outcome. As discussed above, smoking and the intake of nicotine leads to a number of desirable somatic, cognitive, and psychological effects (7; 54; 105). Individuals begin to strongly associate these pleasurable effects with smoking and smoke in order to achieve these desired outcomes. Research supports this theory as both animals and humans will self-administer nicotine in order to receive the positive benefits (86). It is unlikely that positive reinforcement is a sufficient condition to create and maintain addiction. For example, addicted individuals will continue to administer their drug of choice even after the neurobiological response to the drug is reduced and positive effects are significantly diminished (211). Nonetheless, while not the only factor, the positive reinforcement associated with nicotine is critical to initiating and maintaining smoking (136).

Negative Reinforcement Theory

A complementary theory focuses on the use of drugs in order to avoid or remove unpleasant consequences. Often, due to increased tolerance to a drug, such as nicotine, individuals will feel decreased reactivity to the drug and administer higher and higher quantities of the drug in order to feel the effects. As a consequence of the lowered reactivity of brain reward systems, a return to baseline feels more unpleasant and negative to these individuals, increasing their desire to administer the drug (148). Additionally, as mentioned above, the withdrawal symptoms associated with smoking cessation are aversive. The desire to stop or avoid these symptoms can prevent or thwart attempts to quit smoking (13). As smoking is perceived to reduce negative affect, and nicotine withdrawal is associated with an increase in negative affect, the negative

reinforcement theory is particularly relevant to the current study and will be discussed further below.

Incentive Sensitization Theory

A third explanation for addiction is the Incentive Sensitization Theory (IST; 210). Robinson and Berridge state that addictive drugs sensitize the mesocorticolimbic systems that associate wanting, or "incentive salience," with drug-related stimuli. According to the theory, drug use is maintained by the ability of the conditioned stimuli (i.e., drug cues) to trigger motivation for drugs, similar to the processes of classical conditioning (210). Moreover, drug cues can become so salient that they cause the individual to desire drugs even in the absence of any pleasure from drug use (212). This theory is relevant to the parent study (to be described later) in the current proposal because it suggests that drug cues should grab the attention of drug dependent individuals. As described later, attention to drug cues (or "attentional bias" to drug cues) was the target of the intervention in the parent study although it is not the primary focus of this dissertation.

Treatments for Smoking Cessation

The primary treatments for smoking cessation include nicotine replacement therapy (NRT), prescription medications bupropion and varenicline, and psychological counseling. According to data from the 2010 National Health Interview Surveys (NHIS), 48.3% of adult smokers received advice from a health professional to quit, 5.9% utilized smoking cessation counseling, 30.0% used NRT, bupropion, and/or varenicline, and 31.7% used at least one or more form of counseling or chemical intervention (39).

Pharmacological Treatments

Nicotine Replacement Therapy

The purpose of NRT is to reduce the withdrawal symptoms associated with smoking cessation by replacing the nicotine from cigarettes with nicotine delivered by a different method. NRT includes a variety of administration methods such as the patch, intranasal and oral sprays, gum, lozenges, and sublingual tablets. The transdermal patch is the slowest nicotine delivery method of the various NRT techniques, but all of the methods are slower than smoking (242). Despite the slower speed of delivery, the use of NRT approximately doubles an individual's chance of successfully quitting (191). The odds ratio for smoking abstinence for individuals using NRT compared to controls averages 1.77, with odds ratios ranging from 1.44 to 2.35 across administration methods (235). NRT provides the most flexibility of the first-line cessation therapies as individuals may select the best administration method based on price, awareness, route of administration, adverse effects, and general preference (191).

Bupropion

Originally approved by the Food and Drug Administration (FDA) to treat depression, bupropion was approved in 1997 to treat tobacco dependence. Bupropion was the first non-nicotine substance to show efficacy for smoking cessation (191). When compared to a placebo control group, individuals taking bupropion were significantly more likely to be abstinent at the end of seven weeks of treatment and at a one-year follow-up (124). Eisenberg and colleagues found individuals taking bupropion were about twice as likely to successfully quit (OR 2.07, 95% CI 1.73–2.55) (61). Additionally, individuals taking bupropion to quit showed less weight gain and reported few adverse side effects (124).

It is not clear what specific mechanism of action is responsible for the antismoking effects of bupropion (213). Bupropion inhibits the reuptake of dopamine, noradrenaline, and serotonin and is a non-competitive nicotine antagonist. It is likely that the inhibition of the reduction of dopamine and noradrenaline helps to combat withdrawal symptoms making it easier for individuals to quit and stay abstinent (213).

Varenicline

The FDA approved varenicline, also known as Chantix, in May 2006. Varenicline is a nicotinic receptor partial agonist, meaning that it simulates nicotine receptors ($\alpha_4\beta_2$) but with weaker effects (280). Varenicline's affinity for human nicotinic receptors is about 45% (214). Though the activity is lower than that of nicotine, varenicline's effect on nicotinic receptors helps to reduce craving and withdrawal symptoms (45). Multiple clinical trials examining the efficacy of varenicline have found varenicline to be more effective than placebo and bupropion for smoking cessation (89; 132; 251). Studies have found that varenicline can more than double the odds of cessation when compared to placebo control (OR = 2.41, 95% CrI = 1.91 - 3.12; 61).

Overall, pharmacological treatments offer a very good first line treatment for smoking cessation. Pharmacological treatments have been shown to significantly improve cessation rates. Even so, smoking cessation is very difficult and success rates remain low. Current recommendations suggest combining pharmacological and psychological treatments to address all aspects of smoking addiction (66).

Psychological Treatments

In addition to the pharmacological treatments described above, a number of psychological approaches are available to help smokers quit. Specific approaches include

cognitive behavioral therapy (CBT) and motivational interviewing (MI). Delivery modalities include individual, group, online, and phone-based counseling. In general, research does not reveal significant differences between approach types (154) and success with different theoretical approaches and modalities may depend on which is the best fit with the individual attempting to quit.

Primary targets of counseling usually involve identifying reasons for quitting, increasing motivation to quit, recognizing situations where smoking is likely to occur, planning for such situations, and providing support to the individual throughout the quitting process (154). When compared to control groups with minimal contact, individuals who received face-to-face, individual counseling were significantly more likely to successfully quit (RR = 1.44; CI = 1.25-1.65; 154). Other studies have found proactive telephone counseling to be significantly superior to educational booklets (OR = 2.22; 95% CI = 1.20-4.00; 239). Group counseling, especially in workplace settings, has also been found to increase the odds of successful cessation attempts (OR = 1.77; 95% CI = 1.05-2.80; 35).

In summary, a number of effective smoking cessation treatments are available. Pharmacological treatments target craving and withdrawal symptoms while psychological treatments address motivation, cognitions, and behaviors. Current guidelines from the U.S. Department of Health and Human Services recommend combining pharmacological and psychological approaches as the most effective treatment (258). Despite significant improvements in treatment options over recent decades, cessation rates remain low. One large (N = 20,258), recent tobacco use survey found that of the 38.5% of respondents who had cessation activity over the last year, only 3.1% had succeeded in staying

abstinent for six months or more (255). These numbers illustrate the need to continue to improve cessation treatments. The primary purpose of the current study is to closely examine emotion regulation as a possible target for improving quit attempt outcomes.

EMOTION REGULATION

The concept of emotion regulation has evolved over time. Some of the first work on emotion regulation strategies dates to Sigmund and Anna Freud in the early 20th century in their work on psychological defenses. Anna Freud described these defenses as "the ego's struggle against painful or unendurable ideas or affects (75)." In the more recent past, Susan Folkman and Richard Lazarus have examined "emotion-focused coping," which involves regulating the negative emotions that accompany stressful events (68). Researchers such as Judy Garber and Kenneth Dodge have taken a more developmental perspective, investigating the emergence of emotion regulation skills from early childhood through adulthood (78).

Working Definition

Emotion regulation includes both the awareness and identification of emotions and the set of strategies people use to redirect their emotions and modify their behaviors to accomplish goals (94; 149; 248). Emotions originally evolved to help humans survive. Emotions act as an internal cue to react or respond to a situation. According to the modal model of emotion (Figure 1), emotions are generated when a situation occurs that the individual views as being significant for his or her personal, social, and/or cultural goals. The individual pays attention to the situation due to the perceived importance, gives it a valenced meaning (positive or negative), and reacts with experiential, behavioral, and physiological responses (98). Often, the emotion generated is helpful. Emotions can

enhance our sensory processing (244), help generate behavioral change (217), assist with complex decision-making (182), improve memory (202), and benefit navigation of social interactions (69).

On the other hand, emotions can be harmful when their intensity, duration, frequency, or type is not aligned with the particular situation. Other times, the emotion can bias cognition and/or behavior (126). Frustratingly, emotions can frequently interfere with goals in many of the same areas where emotions are helpful. For example, while emotional judgment can help to navigate social interactions, intense affect can interfere with an individual's ability to correctly read other's emotional states (25). Extremely labile or volatile emotions make it difficult to act in accordance with goals (250). On a clinical level, deficits in emotion regulation are associated with a variety of psychological difficulties including depression (20; 166), posttraumatic stress disorder (PTSD; 24), generalized anxiety disorder (GAD; 219), borderline personality disorder (BPD; 163), eating disorders (82; 107), alcohol-use disorders (226), and substance-abuse (71).

Research examining emotion regulation has increased dramatically in the past two decades, as its importance has become better understood. Unfortunately, this explosion of research also contributed to a proliferation of terms and working definitions that are similar but not always in alignment. This plethora of terms makes literature reviews difficult and the different working definitions mean that similar constructs are measured very differently across studies. The working definition of emotion regulation used for this dissertation was proposed by Kim Gratz and Lizabeth Roemer and presented in their 2004 paper on the development and validation of the Difficulties in Emotion Regulation Scale (DERS; 94). This multidimensional conceptualization is one of the more specific

definitions and has been used across psychological research domains, including substance abuse and addiction (71; 72). The conceptualization specifies four dimensions of emotion regulation:

> (a) awareness, understanding, and acceptance of emotions; (b) ability to engage in goal-directed behaviors and inhibit impulsive behaviors when experiencing negative emotions; (c) flexible use of situationally appropriate strategies to modulate the intensity and/or duration of emotional responses rather than to eliminate emotions entirely; and (d) willingness to experience negative emotions as part of pursuing meaningful activities in life (94).

This definition is used primarily because of the clarity and thoroughness of the conceptualization. It includes both cognitive and behavioral responses to emotion and emphasizes the importance of using these strategies to engage in goal-directed responses. Additionally, this definition corresponds to the DERS, one of the primary measures used to assess emotion regulation both for the current project as well as within the field in general. The terms emotion regulation and emotion dysregulation will be used throughout the paper. Emotion dysregulation refers to times when individuals are experiencing deficits in the areas described above, i.e., low levels of emotion regulation.

Demographic Factors

Emotion regulation patterns do vary some across gender and age. Women have been found to experience more overall stress (176), but also to use more coping strategies of all types including rumination, social support, and suppression (246; 247). Tamres et al (246) argue that it is possible that women use more emotion regulation strategies because they appraise situations as more stressful.

Carstensen et al (38) theorize that as individuals age, they put more emphasis on emotion regulation in order to help create meaning out of life. Some studies have found increased use of effective emotion regulation strategies such as positive reappraisal (22) and older adults believe that they are better at emotion regulation (158). Other studies have found that older adults may engage in more passive regulation strategies such as avoidance, suppression, and withdrawal (23). While the use of these strategies may help older adults protect themselves from unpleasant emotions and maintain energy for other pursuits (49), these strategies have been associated with increased emotional pathology (5).

Emotion Regulation and Affect

There is some disagreement as to whether emotion regulation refers to the control of affect or the control of an individual's response to experiencing intense affect. Frequently, studies will conflate negative affect and emotion dysregulation with the assumption that negative affect is, at the very least, a sign of poor emotion regulation. This conceptualization suggests that intense negative affect is inherently dysregulated and problematic. Research does not support the idea that high emotional reactivity and intensity on their own are accountable for to negative psychological outcomes (155; 156).

Thus, it is important to emphasize that while increased negative affect is often associated with emotion dysregulation, they are two separate constructs with important, distinct clinical implications (28). In other words, while individuals with poor emotion regulation skills may be more likely to have increased or more intense negative affect (101; 174; 218), neither emotion dysregulation nor negative affect is necessary or sufficient for the other to be present. In fact, research suggests that attempts to avoid,

minimize, or suppress negative internal experiences, such as affect, may actually have the opposite effect, increasing the frequency and severity of negative affect (110; 253). Emotion regulation skills allow an individual to respond more effectively to affect in order to act in accordance with his or her goals. Emotion regulation may not necessarily change the presence or initial intensity of an emotion; rather, it changes the individual's reaction to the emotion. For this reason, it is important to examine emotion regulation as a moderator variable as described further below.

Emotion Versus Affect

It is important to note the difference between affect and emotion. While this question is an ongoing debate in the literature and a full discussion is beyond the scope of this dissertation, working definitions for both are briefly provided here. Affect is the individual's current experience measured along two, bi-polar (high/low) dimensions: positive affect and negative affect (268). Unlike some other theories of affect that conceptualize affect as measured along two dimensions, valence (positive to negative) and arousal (high to low; 215), the two-factor model of affect posits that positive and negative affect are independent factors and research supports this supposition, finding that the two factors are not highly correlated and show strong associations with different characteristics and outcomes (267). The measure of affect used for the current study, the Positive and Negative Affect Schedule (PANAS) is based on this two-factor model of affect (267). In contrast to affect, emotion is a more specific, discrete state such as anger, happiness, or fear, where the feeling is tied to a situational cause (207)

The Extended Process Model of Emotion Regulation

James Gross's process model of emotion regulation (97) is the primary model used to conceptualize emotion regulation and is consistent with the working definition described earlier. The model originates from the modal model of emotion (Figure 1), described above. As a brief reminder, the modal model suggests that emotion arises when a situation occurs that an individual views as personally relevant and he or she gives attention to the situation. By nature of attending to the situation, the individual appraises the situation and assesses its relevancy to his or her goals. This appraisal leads to the production of emotion and the subsequent experiential, neurobiological, and behavioral changes (99). These changes then create a feedback loop that can create a new situation, beginning the process over again.

The process model of emotion regulation (Figure 2; 98) expands on the modal model, viewing each step in the model as a possible point to regulate emotion. Moving through time, *situation selection* is the first possible intervention and involves the individual seeking out or avoiding situations based on the expected effect on emotions. If an individual finds him or herself in an uncomfortable or dysregulating situation, he or she may try to change the situation, labeled *situation modification*. When the individual cannot alter the situation, he or she may use *attentional deployment,* focusing on different, less dysregulating aspects of the situation. *Cognitive change* involves altering one's appraisal of the situation to limit or modify the emotional effects. Finally, *response modulation* refers to altering behavioral or physiological responses to the emotion.

The extended process model of emotion regulation adds yet one more level to the model, describing three stages overarching the entire process (100). The first stage is the *identification stage.* In this stage, the triggered emotion is evaluated and the individual

decides whether or not to attempt to regulate the emotion. During this stage, the individual perceives and identifies the emotion, determines whether the affective valence is strong enough to require regulation, and then activates the goal to regulate the emotion. Emotional dysregulation in this stage may stem from poor awareness or acknowledgment of emotion, failure to recognize the need to regulate an emotion, or the decision not to act to regulate the emotion despite the recognition that it would be more effective to do so.

Once the individual decides to regulate the emotion, he or she enters the second stage, the *selection stage*, the goal of which is to decide which emotion regulation strategy to use. In this stage, the individual examines the possible regulation strategies, many of which are described in the following section, and evaluates which strategy is best based on contextual and internal factors such as emotion intensity or cognitive resources available. Breakdowns in emotion regulation at this stage may result from difficulty recognizing the variety of strategies available, preference for less effective strategies, or perceived inability to implement certain strategies.

The third and final stage is the *implementation stage*. At this point, the individual moves from choosing a regulating strategy to actively utilizing the skill within the specific context. Problems at this stage may arise from difficulty recognizing how to implement the chosen strategy in the specific situation or from poor execution of the emotion regulation skill.

In summary, Gross's extended process model of emotion regulation provides a research-based conceptualization of the course of emotion regulation while also attempting to account for why a specific strategy is chosen.

Neurobiology of Emotion Regulation

The regulation of emotion is controlled by a variety of neural networks. Though an extensive review of the neuroscience of emotion regulation is beyond the score of this paper, a brief review follows. All of the following information has been summarized from Silvers et al (237). The amygdala likely plays a large role in the detection of emotionally-salient situations due to its reactivity to both positive and aversive stimuli. The anterior dorsomedial prefrontal cortex helps individuals make judgments about one's own mental state and the mental states of others in order to adapt appropriately. The dorsolateral prefrontal cortex and inferior parietal cortex are primarily associated with concentration, working memory, and focus on goals. The dorsal anterior cingulate and posterior dorsomedial prefrontal cortex are involved in the initiation and maintenance of controlled processing. The ventrolateral prefrontal cortex is implicated in the selection of goal-related responses and information from memory, as well as the production of speech, including internal speech, often necessary to enact the emotion regulation strategies.

Emotion Regulation Strategies

Much of the research conducted in the area of emotion regulation focuses on specific strategies. A brief description of a few of these strategies, as well as related research, follows below. Table 1 provides a review of additional strategies and related research.

Cognitive Strategies

Cognitive strategies are similar to *attentional deployment* and *cognitive change* described in the extended process model and defined as "how individuals direct their attention within a given situation so as to influence their emotions" (102).

Reappraisal

When using reappraisal, an individual thinks about a situation in a way that reduces the emotional impact of the situation (98). For example, an individual feeling hurt and angry that a friend cancelled their plans, may reappraise the situation and focus on the fact that she now has unexpected time to perform a number of tasks that she had been anxious about having time to complete. Research examining self-report of affect, physiological responses, and neural processes supports the effectiveness of using reappraisal to reduce negative affect (87; 103).

Emotion Suppression

Emotion suppression is the inhibition or dampening of an emotion that has already been triggered (99). Some research has found suppression to be effective at reducing negative affect in the short-term, but counterproductive in the long run, leading to increased negative affect after the situation has passed (80). Interestingly, while suppression can reduce negative affect briefly, the neural effects of suppression mirror emotion generation with activation of the amygdala and insular cortex (88).

Behavioral Strategies

Behavioral strategies are outwardly visible strategies described as *situation selection*, *situation modification,* and *response modulation* in the extended process model. These strategies are behaviors that an individual may engage in before, during, or after an emotionally arousing situation in order to modulate his or her emotional response. A primary behavioral strategy is avoidance, the unwillingness to engage in unpleasant feelings, thoughts, memories, and physiological responses in addition to the situations that may cause them (112). While the avoidance of aversive situations may

reduce or evade negative affect in the short-term, research suggests that attempts to avoid negative affect paradoxically increase the intensity and severity of the very feelings that the individual is looking to avoid (271) and may contribute to the development and maintenance of psychopathology (111). Other behavioral strategies such as exercise (175) and progressive muscle relaxation (36) are associated with effective reduction of negative affect.

State vs. Trait

Emotion regulation is usually conceptualized as a stable construct (100), although some theory has suggested more changeable aspects of emotion regulation (157). Overall, it is reasonable to expect that immediate situational factors may influence an individual's ability to regulate emotions at a particular time. Therefore, a person's overall level of emotion regulation ability (i.e., trait emotion regulation) may differ from his or her moment-to-moment emotion regulation ability (i.e., state emotion regulation). For example, an individual may be able to regulate well when the situational intensity is low, but consistently struggle to engage in goal-directed responses when intensity is high. An individual's emotion regulation skills may also vary across situation types. For example, an individual may be very good at regulating emotional responses in a professional context, but terrible at her home with her family. It is also reasonable to expect that aversive experiences (e.g., death of a loved one or an interpersonal argument) and positive experiences (e.g., recent professional or personal success) would influence emotion regulation abilities in the moment. Thus far, this notion has not been explored thoroughly in the literature, though Dr. Kim Gratz's group has developed a state measure of emotion regulation abilities.

Assessment

Assessment of emotion regulation varies across studies, particularly as conceptualizations of emotion regulation differ. Many studies investigate the use of one or more specific emotion regulation strategies and rely on self-report to determine the use of the skill(s) of interest. Experimental laboratory studies may instruct participants to use a particular strategy in order in investigate its effect on an outcome variable of interest such as negative affect (e.g., 97; 127). While informative about the immediate effects of specific strategies, these experiments may not generalize to daily life. Additionally, some research suggests that participants find it difficult to engage in the strategy being targeted (55).

Several self-report questionnaires have been developed to assess more general, dispositional emotion regulation tendencies. James Gross developed the Emotion Regulation Questionnaire (ERQ; 101), a 10-item scale designed to assess expressive suppression and cognitive reappraisal. The Cognitive Emotion Regulation Questionnaire (CERQ; 79) is a 36-item self-report measure designed to assess nine different cognitive regulation strategies. Finally, the Difficulties in Emotion Regulation Scale (DERS; 94), used for the current study, is also a 36-item self-report measure that assesses six areas: 1) nonacceptance of emotional responses; 2) difficulties engaging in goal directed behavior; 3) impulse control difficulties; 4) lack of emotional awareness; 5) limited access to emotion regulation strategies; and 6) lack of emotional clarity. Unlike the CERQ and the ERQ, which only assess cognitive emotion regulation, the DERS measures both behavioral and cognitive emotion regulation.

In addition, a state version of the DERS (S-DERS; 157)) has recently been developed which is ideal for use in an ecological momentary assessment study such as

the current study. The S-DERS has four subscales: 1) nonacceptance of current emotions; 2) limited ability to modulate current emotional and behavioral responses; 3) lack of awareness of current emotions; and 4) lack of clarity about current emotions.

The current study utilized two of these subscales: the Nonacceptance and Modulate scales. The Nonacceptance scale was chosen because of literature supporting the relationship between nonacceptance and recent cigarette smoking (1). Acceptance-based emotion regulation strategies, in contrast to to avoidance-based coping, are associated with lower levels of substance use (70) and improved treatment outcomes (162). A significant amount of research supports the use of substances to minimize or avoid negative affect, described in more detail below ("Emotion Regulation and Addiction"). Building and encouraging the acceptance and tolerance of negative affect may help individuals cope with uncomfortable internal experiences without using substances. There is some evidence supporting the use of acceptance-based treatments such as mindfulness (33) and acceptance and commitment therapy (83) for smoking cessation. The Modulate scale was chosen because of its clear relevance to regulating goal-directed behaviors, such as smoking cessation, in the face of emotional distress. Dr. Gratz explicitly supported the selection of these two subscales for the current study (92).

Thus far, studies have used the S-DERS in laboratory-based studies to measure in the moment emotion regulation skills following tasks expected to impact emotion regulation skills, such as mood induction tasks. For example, Borges and Naugle (27) used the S-DERS to investigate state emotion regulation skills in individuals with personality disorders. Participants completed the S-DERS before and after completing either the Paced Auditory Serial Addition Task (PASAT-C; expected to evoke emotional

distress) or an interpersonally based mood induction essay writing task. Results showed that individuals with a personality disorder reported significantly more state emotion regulation difficulties following the PASAT-C task than the essay task. Unfortunately, the authors did not include an examination of the pre-/post-task S-DERS scores to provide information about the ability of the S-DERS to detect changes in state emotion regulation. Another study (91), utilized the S-DERS to investigate possible differences in state emotion regulation abilities following an emotionally valenced movie clip after participants were instructed to either worry or not worry, and then to be mindful of thoughts and emotions, attempt to suppress certain thoughts, or given no regulation strategy instruction prior to watching the clip. Researchers did not find significant differences on state emotion regulation strategies across conditions. Finally, Arbid (9) used the S-DERS to investigate the relationship between state emotion regulation difficulties and attentional disengagement from negative images. No significant relationship was found.

Emotion Regulation Interventions

Given the well-researched associations between emotion regulation and psychopathology, it is unsurprising that several psychotherapy approaches have been developed that focus specifically on emotion regulation. Dialectical behavior therapy (DBT; 163) was developed to treat individuals with borderline personality disorder (BPD) who are characterized by extreme affective lability (189). Emotion regulation is one of the four sets of skills taught in DBT. DBT emphasizes the effective use of emotion regulation strategies in order to work toward identified goals (163). Studies have found DBT to be effective at improving emotion regulation (11; 143) and some research even

suggests that DBT may alter underlying neural circuitry responsible for emotion generation (90). Other therapeutic approaches that focus on emotion regulation include Emotion Regulation Therapy (ERT), currently being evaluated as a possible treatment for generalized anxiety disorder (181); Acceptance and commitment therapy (ACT; 111) which, as implied by the name, emphasizes the acceptance of negative emotions, rather than the attenuation or removal of the affect, in order to achieve personally relevant and significant goals (21); and emotion-focused therapy (EFT; 96) which concentrates on improving emotion-focused coping skills. Both DBT and ACT have been shown to be effective at treating addiction, including cigarette smoking (53; 57; 159; 164).

Emotion Regulation and Addiction

A large amount of research has found a significant role of emotion regulation in addictive behaviors (129; 146; 208). A growing body of research has revealed that individuals with deficits in emotion regulations are at greater risk of addictive disorders and that individuals with poor emotion regulation find it more difficult to abstain (34). Additionally, some research has found that individuals with addictive disorders may have even greater emotion regulation difficulties during initial periods of abstinence (71). As described by the negative and positive reinforcement theories reviewed above, drugs are often used to reduce unpleasant sensations, such as negative affect, or increase desired sensations, ranging from numbness to euphoria. Thus, it follows that individuals who have difficulty regulating their own emotions may use, and come to rely on, drugs to help regulate unwanted emotions (43). Over time, unwanted emotional states may be learned as cues for substance use (13; 241). In addition to possibly increasing the urge to use the desired substance due to learned associations between negative affect states and

substance use (125), constant, strong, negative emotions can interfere with other objectives, such as desisting from substance use (13).

Emotion Regulation and Tobacco Use

A large body of theory and research suggests that individuals often smoke cigarettes as a means of controlling mood (134; 228; 238), particularly as a way to reduce negative affect (114; 243). Cross-sectional studies have found a strong association between various forms of negative affect, such as depression and anxiety, and smoking initiation (32; 199). Adolescents frequently report that the calming or relaxing effects of cigarettes led to smoking initiation and maintenance (179; 190) and many smokers attempting to quit attribute lapses to stress and negative affect (173; 227). Smokers consistently report a belief that smoking helps to reduce emotional distress (29; 46; 272).

Research often supports this expectancy and smokers tend to report decreases in negative affect and increases in positive affect after smoking (197). A reduction in negative affect following smoking may depend on a variety of factors. For example, research suggests that an individual's expectations about the effects of smoking will influence reported changes in affect (47). Additional research suggests that anxiety may only be reduced by smoking in situations where a benign distraction is present (135; 137). Further, the physiological effects of smoking on blood pressure, heart rate, and cortisol are similar to those produced by stress (200), thus it is surprising that smokers report a relaxing effect. This contrast between the physiological effects and reported psychological effects is known as the "nicotine paradox" (167). This complexity of factors that influence change in negative affect may explain why some studies show mixed findings or no relationship between smoking and negative affect (118; 198).

Some theories attempt to describe and explain this complicated relationship between emotion regulation and smoking. One such theory, the Situation X Trait Adaptive Response (STAR) model (84) attempts to address situation- and personality-dependent cognitive, emotional, and biological effects of drug use. While the STAR model endeavors to account for the array of factors involved in drug use, it has not been widely used or studied, perhaps due to its complexity.

More broadly, the self-medication hypothesis of substance use disorders suggests that individuals with emotion regulation difficulties use substances to help reduce negative affect (144). Many studies have found that smokers have higher levels of negative affect overall when compared to control samples (136). As noted above, there are well-documented associations between depression and smoking (85) and anxiety and smoking (185). The fact that individuals with affect regulation difficulties are significantly more likely to smoke, combined with the well-documented expectancy and perception that smoking reduces negative affect, supports the self-medication hypothesis that individuals may smoke to help cope with emotion regulation difficulties.

A second theory suggests that much of the relationship between emotion regulation and smoking is accounted for by the postponement or easing of withdrawal symptoms. As discussed in the withdrawal section above, negative affect is significantly associated with nicotine withdrawal (123), thus smoking may reduce negative affect primarily due to the removal of the effects of withdrawal. In studies examining the immediate effects of smoking, negative affect directly associated with abstinence is associated with more significant reductions following smoking than negative affect from other sources (201). Other studies show that relative to a nonsmoking control group, the

stress levels of smokers after smoking a cigarette was similar to the baseline stress levels of nonsmokers (197). This finding suggests that smokers may not truly gain a reduction in negative affect, but rather, they smoke to avoid the additional negative effects of withdrawal. All of this research supports the idea that individuals who smoke may have deficits in emotion regulation skills.

Researchers have examined emotion regulation as a predictor variable for craving and smoking. Maladaptive regulation strategies such as suppression have consistently been associated with early smoking initiation, greater smoking urges, and higher rates of cessation relapse (104; 172). On the other hand, research suggests that several emotion regulation strategies are effective at reducing craving. For example, individuals who were instructed to reappraise their emotions about smoking showed less negative affect, reduced craving for cigarettes, and diminished attentional biases to smoking-related cues than individuals who were instructed to suppress or accept their smoking-related emotions (245). Reappraisal has also been found to be associated with weakened smoking expectancies about smoking reducing negative affect (77). The same study found that reappraisal moderated the relationship between induced mood and smoking duration. Overall, these studies support the case that emotion regulation may play a significant role in craving and smoking.

ECOLOGICAL MOMENTARY ASSESSMENT

This study focused on state emotion regulation as well as several other phenomena that often change rapidly over time. Ecological momentary assessment was used in order to more accurately measure changes and patterns of the variables of interest such as emotion regulation, affect, and craving over time. Ecological momentary

assessment (EMA) techniques utilize mobile technology to measure phenomena as they occur in an individual's real-world environment. Assessments may occur randomly, at scheduled times, and/or when a participant initiates an assessment.

EMA techniques have provided an innovative strategy for the assessment of thoughts and behaviors as they occur in the natural environment. Rather than relying upon retrospective reports, data can be collected in real time while individuals are experiencing the phenomenon of interest. This method of data collection is particularly useful because it eliminates recall biases and enhances validity as data are gathered as it occurs in participants' daily lives (232). For example, researchers have found that EMA measurements of affect better predict outcome behaviors when compared to retrospective self-reports of affective lability (8). In addition, data are collected at multiple time points so that changes over time can be considered. The real time, repeated data collection available with EMA is particularly useful in investigating constantly changing psychological states, such as affect.

Ecological Momentary Assessment Studies of Emotion Regulation

Over the past few years, researchers have begun to emphasize the importance of changing contextual factors on emotion regulation and the utilization of real-life, emotion-producing stimuli to examine naturally occurring emotion regulation (3). The field of emotion dynamics has been proposed, consisting of the study of the "trajectories, patterns, and regularities with which emotions, or one or more of their subcomponents (such as experiential, physiological, or behavioral components), fluctuate across time, their underlying processes, and down-stream consequences" (153). A growing body of

research is examining patterns of specific emotions and overall patterns of emotion in specific populations (e.g., 249; 265).

More specific to the current study of emotion regulation, patterns of emotion such as the amount of variability (change from baseline), duration, and co-occurrence assessed by EMA have been suggested as ways to investigate real-time emotion regulation (153) and some studies have begun to use these patterns as measures of emotional instability (128). A couple of studies have used EMA to measure specific emotion regulation strategies and affect in real time. One study used experience-sampling methods to investigate the intensity and lability of emotions as well as the strategies used to regulate them in an adolescent sample (236). The results suggested that participants who responded to negative affect with denial and rumination had higher levels of depression and problem behaviors such as lying, stealing, and fighting.

A second study EMA study examined the spontaneous use of emotion regulation strategies and affect in the daily life of adults. Brans et al. (31) investigated within-person and between-person usage of strategies as well as overall associations between emotion regulation strategy usage and affect. Participants were prompted 10 times a day for seven days to provide current emotions and the extent to which they had used any of six emotion regulation strategies (reflection, reappraisal, rumination, social sharing, expressive suppression, and distraction) since the previous assessment. Researchers found that distraction was the most frequently used strategy, while reappraisal and social sharing were used least. Additionally, participants reported using multiple strategies at once to regulate emotions, a finding which supports prior theoretical work (102). Investigation of patterns over time showed that negative affect at an assessment was

associated with the increased use of emotion regulation strategies at the next assessment. Building on prior research, this study replicated findings (e.g., 269) that rumination and suppression are maladaptive regulation strategies as they were associated with increases in negative affect and decreases in positive affect. This study provides an extension of prior laboratory studies, expanding knowledge of strategy usage in daily life.

Ecological Momentary Assessment Studies of Smoking

The majority of the data described earlier in the introduction was gathered using retrospective data collected in the laboratory. These data, while very useful, are limited by recall errors and bias and low real-world validity. EMA and laboratory studies on smoking suggest that findings from laboratory studies are often not accurate predictions of smoking behaviors in the real-world (230). EMA techniques are particularly suitable for investigating cigarette smoking and its correlates, as it is a discrete behavior that occurs many times throughout the day, often unconsciously (229). EMA allows researchers to not only gather ecologically valid data on smoking, but by collecting data randomly throughout the day, it also allows for an assessment of baseline characteristics in non-smoking situations.

A number of studies have examined precipitants and antecedents to regular and relapse smoking using EMA techniques. Given the well-documented associations between negative affect and smoking, negative affect has been a primary focus of these studies. Shiffman and Waters (233) found that lapses attributed to stress or bad mood were associated with precipitous, rapid changes in negative affect in the hours preceding the lapse. The use of EMA techniques and analyses such as the time-varying lagged effect model allow researchers to build on these findings and show patterns between

negative affect and increases in smoking urges at proximal, later assessments (234). The findings from these studies provide a strong foundation for the current study examining emotion regulation, a factor that is shown to be associated with negative affect, but which has not been examined in the context of EMA and smoking.

Nomothetic and Idiographic Analyses

The use of EMA techniques allows for the application of statistical analyses that investigate time-dependent variation within a single individual in addition to group-level relationships (260). Nomothetic analyses focus on group- or cohort-level phenomena and were used to test null hypotheses in the population, as is typically seen in clinical research. For example, an analysis that examines whether trait emotion regulation is correlated with negative affect would be a nomothetic analysis. Idiographic analyses were conducted on data from individual participants and tested a null hypothesis specific to that individual. For example, an analysis that examines whether there is a correlation between state emotion regulation and negative affect in a particular individual would be an idiographic analysis.

A closely related distinction is between variable centered and person centered analyses. Variable centered analyses encompass many of the traditional approaches of the social sciences, such as correlation and regression. Person centered analyses focus on individuals, and use approaches such as cluster analysis or latent group analyses to cluster individuals together.

Idiographic analyses are appropriate for EMA data (48) because of the number of points collected from each participant. Idiographic models can take time into account, examining patterns of change within individuals across time (260). Idiographic analyses

often involve time series analyses that account for serial dependencies within an individual's data, and time series analyses were used in the current study.

When applicable, idiographic analyses can supplement nomothetic analyses of longitudinal data (222) and can reveal findings that are obscured by nomothetic approaches (e.g., 119). When nomothetic analyses are used, results provided indicate associations across all individuals. As no individual is purely average, these results reveal overall patterns, but may miss important implications for individuals. Individual patterns are particularly important when designing and examining interventions as they can reveal which individuals may respond to an intervention. With a focus on individual patterns over time, idiographic analyses allow researchers to investigate the process of change following an intervention or a change, such as smoking cessation (260).

SUMMARY

Background on cigarette smoking, emotion regulation, and the intersection of the two has been presented. The current study examined these constructs, expanding prior research to investigate state emotion regulation using EMA techniques in order to gain additional knowledge about the relationship between negative affect, craving, and smoking behavior and to provide a possible target for improved interventions. A detailed description of the study follows.

CURRENT STUDY

CAUSAL MODEL

Figure 3 depicts a model of the relationships between emotion regulation, craving, and smoking that was examined in the current study. Those relationships are discussed below. As shown in Figure 3, emotion regulation was investigated both as a predictor variable and as a moderator variable. First, to replicate and extend on the preliminary study (MacIntyre, 2015), this study investigated the association between state and trait emotion regulation and negative affect. Second, this study examined the association between emotion regulation and craving and smoking behavior. Third, this study investigated whether emotion regulation moderates the association between negative affect and craving as well as between craving and smoking behavior.

Overall, the main idea is as follows. It was predicted that individuals with better emotion regulation would report less negative affect and craving in the first place, and that when they did experience negative affect and/or craving, the effect of negative affect and craving on smoking outcomes would be diminished in these individuals.

Emotion Regulation and Negative Affect (Pathway 1)

Overall, poor emotion regulation skills are associated with more negative affect (e.g., 67; 218). This relationship may be due to high usage of ineffective and maladaptive regulation strategies such as suppression which research suggests increases negative affect (31). In addition, negative affect may affect an individual's state emotion regulation skills. Research has found that individuals use different emotion regulation strategies depending on the type and intensity of their emotional experience in the moment (3; 4).

As noted earlier, emotion regulation was assessed as a trait (lab) and a state (field). Trait emotion regulation, as assessed by the DERS, represents the more general, stable emotion regulation abilities of an individual. State emotion regulation, as assessed by the S-DERS, was expected to reflect the impact of contextual factors on the individual's trait emotion regulation (157). Similar to conceptualizations of other variables with state and trait characteristics (e.g., anger or anxiety), it was expected that measures of an individual's state and trait emotion regulation abilities would be correlated, although this was not a primary focus of the dissertation.

Emotion Regulation and Craving/Smoking (Pathway 2)

A significant amount of research supports the finding that negative affect is associated with stronger urges to smoke (30; 113; 160; 209) and is frequently cited as a strong smoking trigger (136; 204). This relationship between negative affect and smoking urges may even become stronger during periods of smoking deprivation such as cessation attempts (161). Therefore, emotion regulation could reduce craving through its impact on negative affect. In addition, some emotion regulation strategies, such as the use of imagery, have been found to reduce craving (245; 266).

As described earlier, there is a well-documented relationship between negative affect and smoking. Negative affect reduction is a primary motivation for smoking (50) thus it follows that an individual's ability to regulate his or her response to negative affect overall and in the moment may affect smoking behavior. The research described above provides support for the associations between specific emotion regulation strategies (e.g., suppression and reappraisal) and craving and smoking. Due to the new focus on state-specific emotion regulation, there is currently no research examining this relationship.

Emotion Regulation as a Moderator Variable (Pathway 3)

A moderator variable is a variable that influences the strength of a relationship between an independent and dependent variable (14). In the current context, a moderator variable would influence the strength of relationship between negative affect and craving, and between craving and smoking. It is important to investigate variables that moderate the association between negative affect and craving, and between craving and smoking, in that such information could be useful for developing treatment plans to help individuals cope with negative affect and craving.

A few studies have investigated the impact of specific emotion regulation strategies on the association between craving and smoking. In one study, cognitive reframing was found to moderate the association between craving and smoking. Specifically, focusing on the long-term health problems associated with smoking is correlated with individuals being less likely to smoke when experiencing craving and smoking for fewer puffs when they do smoke (147). Informal practice of mindfulness has also been found to moderate the relationship between craving and smoking (62).

For cases when emotion regulation is moderating the relationship between emotion regulation and smoking, it is possible that the moderating effect of emotion regulation is due in part to the effect of mood on perceived self-efficacy. Low mood is associated with low perceived self-efficacy, particularly when the behavior is associated with high difficulty and past failures (138; 140). When individuals do not believe they are able to control or resist engaging in a certain behavior such as smoking, they will be less likely to even try to follow through with goal-directed behavior such as cessation (139; 173). Though self-efficacy is not a focus of the current study, it could be an area for future study.

PRELIMINARY DATA

A prior study using laboratory and EMA investigations provided some evidence for state-like qualities of emotion regulation (169). In this study, participants with higher DERS scores (worse emotion regulation) reported more negative affect at laboratory visits. In addition, when a participant reported a higher DERS score than their average, he or she reported higher negative affect. Additionally, the high DERS participants showed more changes in their negative affect, had an overall greater average magnitude of change in negative affect, and had larger "jumps" to their maximum negative affect, all supporting the idea that these individuals have more labile negative affect than the low DERS participants. Overall, these findings suggest the presence of state- and trait-like aspects of emotion regulation.

RATIONALE FOR CURRENT STUDY

Research on emotion regulation is growing, with a particular emphasis on how emotion regulation, often narrowed to a specific emotion regulation technique, is associated with a specific disorder, symptom, or behavior. Despite an expanding body of literature, at this point, while some researchers have suggested the importance of investigating state emotion regulation, no studies have done so at this point. This EMA study was intended to provide some insight into the "movie" associated with emotion regulation, expanding beyond the "snapshot" that laboratory studies provide. Given the number of diagnoses and psychological problems that are significantly associated with emotion regulation difficulties, it is critical to develop a more thorough understanding of emotion regulation.

The well-documented relationship between emotion regulation and smoking, combined with the need for improved smoking cessation treatments creates a promising intersection of research. The current study provided an initial investigation of a more precise understanding of the real-time affective experiences of individuals with varying emotion regulation abilities in the context of smoking.

SPECIFIC STUDY AIMS AND HYPOTHESES

Participants attended seven laboratory visits each one-week apart as well as for a six-week period consecutive with laboratory visits using EMA techniques. The specific aims and corresponding hypotheses based on the literature were as follows:

Specific Aim 1

To examine the association between emotion regulation (trait and state) and negative affect.

Hypothesis 1A

Participants with higher DERS scores would report higher levels of NA in the lab, and on occasions when a participant reported a higher than average DERS rating, he/she would report higher negative affect ratings in the laboratory.

Hypothesis 1B

Participants with higher S-DERS scores would report higher levels of NA in the field and on occasions when a participant reported a higher than average S-DERS rating, he/she would report higher negative affect ratings in the field.

Idiographic Hypothesis 1C

Time series analyses examined associations between S-DERS and NA in individual subjects.

Specific Aim 2

To examine the association between emotion regulation and craving/smoking.

Hypothesis 2A

Participants with higher DERS scores would report higher levels of craving and smoking in the lab, and on occasions when a participant reported a higher than average DERS rating, he/she would report higher craving and smoking in the laboratory.

Hypothesis 2B

Participants with higher S-DERS scores would report higher levels of craving and smoking in the field and on occasions when a participant reported a higher than average S-DERS rating, he/she would report higher craving and smoking in the field.

Idiographic Hypothesis 2C

Time series analyses examined associations between S-DERS and craving and smoking in individual subjects.

Specific Aim 3

To examine emotion regulation, as assessed by the DERS and S-DERS, as a moderator variable.

Hypothesis 3A

Strong trait emotion regulation skills (low DERS scores) were expected to weaken the relationship between 1) negative affect and craving and 2) craving and smoking, assessed in the lab.

Hypothesis 3B

Strong state emotion regulation (low S-DERS scores) were expected to weaken the relationship between 1) negative affect and craving and 2) craving and smoking, assessed during EMA.

Methods

PARTICIPANTS

This study is a secondary analysis of data collected at the Uniformed Services University of the Health Sciences (USUHS) in Bethesda, Maryland. The USUHS Institutional Review Board approved all study procedures.

Participants were adult, community-based smokers in the greater Washington, D.C. metropolitan area recruited using advertisements seeking smokers interested in smoking cessation. Advertisements were displayed on local mass transit, the Express Paper, Craigslist.com, and the use of flyers throughout the community.

INCLUSION AND EXCLUSION CRITERIA

Participants were screened via telephone to determine if they met eligibility criteria for the study. Participants were determined to be eligible if they were a current smoker, aged 18 to 65, who had been smoking at least 10 cigarettes a day for the past two years. Exclusion criteria included current participation in smoking cessation treatment, such as counseling or medication, or the current use of tobacco products other than cigarettes, such as cigars, pipes, and smokeless tobacco. If a participant's expired CO level was less than 10 parts per million (ppm), he or she was excluded, as this would indicate less than regular cigarette use. Participants with a compliance rate of <50% in the first study week were excluded from the study at Week -2, and were referred to local smoking cessation services. Other exclusion criteria included: 1) recent illicit substance use; 2) another household member enrolled in the study; 3) color-blindness; 4) pregnant or breast feeding; 5) Indication of a serious mental illness, as indicated by a history of diagnosis of schizophrenia, schizoaffective disorder, or bipolar disorder, by a score ≥ 25

on the CES-D, or by a score of ≥ 16 on the AUDIT; 6) Inability to follow study procedures; 7) received attentional retraining for smoking cues as part of a prior USUHS study. Finally, participants could be excluded for "any other factor that, in the judgment of the investigators, would likely preclude completion of the protocol."

All participants who were excluded from participation in the study at any point were provided with local and national smoking cessation resources.

STUDY PROCEDURES

The following description of the study procedures is an abbreviated description of the procedures from the original study. Only procedures relevant to the current study will be described in depth.

First Laboratory Visit

Once participants were determined to meet inclusion criteria via the phone screening, they were invited to attend the initial laboratory visit (Week -3 visit). To begin this session, participants were provided with a detailed description of the study and information on risks. Written informed consent was then obtained (Appendix B). Participants were informed of their right to withdraw at any time from the study. Subjects received a copy of the consent form for their records.

After providing consent, participants first completed the breath carbon monoxide (CO) test. Participants were asked to provide a breath CO sample by blowing through a monitor. If the CO monitor indicated that a participant's expired CO level was very low (less than 10 parts per million (ppm)), he or she was excluded from the study. Participants then completed a number of measures assessing a variety of factors including

smoking behavior, physical and mental health history, as well as a demographic questionnaire, and the Positive and Negative Affect Schedule (267). See Table 2 for a complete list of measures used.

Following completion of the questionnaires, eligible participants were trained on the use of the smartphones. Research staff provided a thorough introduction to the basic features of the phone as well as the questionnaires and attention retraining task (not a focus of this study) that participants completed throughout the six-week study period. Participants were informed that completion of the smartphone assessments was monitored daily. If a participant failed to respond with sufficient frequency, e.g., less than two random assessments (RAs) on two consecutive study days, a research assistant contacted the participant to encourage them to maintain compliance.

If individuals were ineligible or declined to participate, they were offered self-help materials and references to local smoking cessation programs. If individuals agreed to participate, they were randomly assigned to either the Attention Retraining or Control training condition. As the intervention is not the focus of the current study, randomization procedures and details on the conditions will not be described here. Finally, the second session (Week -2 visit) was scheduled and a quit date was set three weeks into the study (Week 0 visit).

Additional Laboratory Visits

Participants attended up to eight laboratory sessions. The first seven sessions were weekly during the six weeks of EMA data collection. The eighth session was a follow-up visit approximately 15 weeks after the quit day visit (Week 0 visit).

Ecological Momentary Assessment Procedures

Participants carried a smartphone with them for up to six weeks in the "field" (i.e., as they went about their daily life). Participants were asked to complete at least four field "assessments" (interactions with the smartphone) per day, which included the 3 AR tasks (AR subjects, described in the next section), 3 Control tasks (Control subjects), and 1 Assessment task (all subjects). Participants completed random assessments (RAs) and participant-initiated assessments. For RAs the participants were alerted at random times by the smartphone to complete the tasks. If participants were prompted to complete an assessment at a time when it was difficult to do so, participants could delay the assessment for five minutes, a maximum of four times. If the participant postponed the alert a fourth time, the program cancelled the assessment and participants would no longer be notified to complete an assessment at that time. Participants were instructed that they could complete a participant-initiated assessment ("make-up assessment") if they missed or cancelled an RA. Participants were only compensated for participant-initiated assessments up to four total (random and participant-initiated) assessments per day. Please see Appendix C for the detailed instructions participants were provided for using the smartphones on a daily basis.

Attention Retraining Intervention

Although the attention retraining (AR) intervention of the parent study is not a focus of the current study, a brief description of the intervention follows. As noted above, all participants were required to respond to four RAs per day. Attention retraining (AR) participants were scheduled to complete three AR tasks per day. Control participants were scheduled to complete three control tasks per day. All participants were scheduled to complete one attentional bias task per day on the smartphone.

Attentional bias was assessed on the smartphone using the standard Visual Probe (VP) task. In a VP task, two pictures (one smoking, one neutral) are presented relatively briefly (500 ms) on a computer, one picture on the left and the other on the right. When the two pictures disappear, a probe (a visual stimulus such as a dot) is presented in a location previously occupied by either the neutral or the smoking picture. The task for the participant is to indicate the location of the probe (left or right) as quickly and accurately as possible by pressing one of two response buttons. Typically, smokers are faster to respond to probes that replace a smoking picture than probes that replace a neutral picture because attention has shifted towards the location occupied by the salient stimulus and therefore participants are faster to process and respond to the probe (because attention is already at the location of the salient stimulus).

During the AR (training) tasks, the dot always replaces the neutral picture. There is a perfect correlation between picture type and dot location. The idea behind AR is that participants implicitly learn the rule that the probe always replaces the neutral picture, and therefore attention is automatically shifted to the location of the neutral picture. On the control task, the dot is equally likely to replace the smoking picture and the neutral picture (as in the case for the assessments). There is a zero correlation between picture type and dot location. Therefore, there is no rule to learn. This type of control condition has been used in previous AR studies (e.g., 65). It ensures that: 1) the duration of AR and control training should not differ; 2) AR and control participants receive equal practice on the motoric aspects of the VP tasks; and 3) AR and control participants are exposed to the same smoking and neutral pictures. Consistent with Schoenmakers et al (223), this study used a stimulus presentation durations of 500 ms at each assessment. Based on pilot

data, the mean duration of AR and Control training assessments was expected to be about 7 minutes.

To restate, the treatment variable (AR vs. Control) of the parent study is not under examination in this dissertation. As noted earlier, this dissertation is focused on associations between emotion regulation and negative affect and various smoking outcomes; none of the primary hypotheses relate to treatment condition. It should also be noted that the dissertation study involved assessments on the smartphone (e.g., S-DERS) that only a subset (26.04%) of the parent study subjects completed.

Smoking Cessation Counseling

Every participant, regardless of treatment condition, received face-to-face smoking cessation counseling at each of the eight laboratory visits. Counseling sessions were conducted by graduate students in the laboratory who followed a standardized treatment manual. Treatment consisted primarily of motivational interviewing techniques, problem solving, and psychoeducation. Much of the counseling focused on reasons for quitting described by the individual participant. The counselor explored these reasons for quitting and worked to build and maintain motivation focusing on these reasons. The counselor helped the participant identify triggers of craving and smoking and work to develop strategies to avoid smoking in the face of temptation. Strategies included chewing gum, going for a walk, brushing one's teeth, or drinking water. Participants were encouraged to create a repertoire of tactics to help cope with cravings to smoke. At sessions following the Quit Day visit, counselors helped participants troubleshoot areas where they are struggling to resist temptation or times when they have relapsed. A final major piece of the counseling included educating participants about the process of

quitting. This included handouts and discussions about the effects of smoking, benefits of quitting, and possible side effects of cessation. Appendix F provides the manual pages and handouts for the Week 3 visit, the week prior to Quit Day.

Compensation

Participants received $60 at the completion of the orientation session. If participants were ineligible due to low levels of carbon monoxide or other factors, participants were paid $40 for their time and travel expenses. Participants were paid $40 at each subsequent laboratory visit, except for the 15-week post-quit follow-up visit where they again received $60. Finally, participants were paid $2 for each random assessment they complete on the smartphones. This payment was calculated and made at the following laboratory visit. Any Federal civilians and military personnel enrolled in the study only received compensation for the laboratory sessions and smartphone assessments that occurred during non-duty hours. Participant compensation is outlined in Table 1.

Compliance

The use of the financial compensation as described above is designed to increase compliance with the procedures, particularly completion of RAs. As noted earlier, if a participant failed to respond with sufficient frequency, e.g., less than two RAs on two consecutive study days a research assistant contacted the participant to encourage them to maintain compliance.

MEASURES

A description of laboratory and field measures used for the current study follows below.

Laboratory Measures

The following measures were administered at each of the laboratory visits.

Emotion Regulation

The Difficulties in Emotion Regulation Scale (DERS; 94) was used to measure participants' emotion regulation abilities. The DERS is a 36-item self-report questionnaire on which participants use a five-point Likert scale ranging from 1 ("almost never") to 5 ("almost always") to indicate the extent to which they experience each item. Example items include "When I'm upset, I become out of control" and "I have no idea how I am feeling." Total scores ranged from 36 to 180, with higher scores indicating more difficulties regulating emotion. Internal consistency is excellent (Cronbach's alpha = .93; 94). In order to reduce participant burden, the DERS was administered at Weeks -2, 0, and 2, rather than at every laboratory visit.

Smoking Urges

The Wisconsin Smoking Withdrawal Scale (WSWS) is a 28-item, multifactorial self-report measure that assesses smoking withdrawal (270). For the current study, participants were asked to respond based on how they had felt over the past week, including today. The WSWS has six subscales to assess anger, anxiety, concentration, craving, hunger, sadness, and sleep. The craving subscale of the WSWS was used to assess craving in the laboratory for this study. The craving subscale is comprised of four items including "I have had frequent urges to smoke," "I have been bothered by the

desire to smoke a cigarette," "I have thought about smoking a lot," and "I have trouble getting cigarettes off my mind." Higher scores indicate more severe withdrawal symptoms. Each of the subscales has strong internal consistency and predictive validity. Scores increase with initial nicotine withdrawal and decrease with continued abstinence as expected.

Positive and Negative Affect

The Positive and Negative Affect Schedule (PANAS; 267) was used to measure participants' affect over the past week. The PANAS consists of 20 items, ten negative affect items (PANAS-NA) and ten positive affect items (PANAS-PA). Examples of positive affect items include "excited" and "alert" while negative affect items include "upset" and "hostile." At the laboratory visits, participants were asked to rate how much they felt each affect item "in the past week". Items were rated on a five-point Likert scale ranging from 1 ("very slightly or not at all") to 5 ("extremely"). Total scores could range from 10 to 50, with a higher score indicating more experienced affect. The current study only uses PANAS-NA. For the remainder of the dissertation, PANAS-NA in the lab will referred to simply as PANAS.

Smoking Behaviors

Participants reported the number of cigarettes smoked per day on a pencil and paper smoking diary (days 1-43). Reports of abstinence were validated using salivary cotinine (196) and exhaled CO levels using a CO monitor (Vitalograph, Lexena, KS; 18).

EMA Measures

State Emotion Regulation

Emotion regulation in the field was assessed using the "Nonacceptance" and "Modulate" scales of the S-DERS (157). The entire S-DERS was not used in order to reduce burden on the participants and possible attrition. As mentioned above, the Nonacceptance scale (Nonacceptance of Current Emotions) was chosen because of literature supporting the relationship between nonacceptance and recent cigarette smoking (1). The Modulate scale (Limited Ability to Modulate Current Emotional and Behavioral Responses) was chosen because of its relevance to the engagement in (or cessation of) addictive behaviors. Both scales have exhibited good internal consistency (Nonacceptance (α=0.92) and Modulate (α=0.85; 157).

In the current data (*n*=3901 EMA assessments), internal reliabilities (Cronbach's α) of S-DERS (*n*=3901), S-Nonacceptance (*n*=3901), and S-Modulate (*n*=3668), were .94, .96 and .89 respectively.

Negative Affect

The short form of the Positive and Negative Affect Schedule (PANAS-S; 171) was used to measure affect in the field. The PANAS-S is comparable to the PANAS, but uses a subset of 10 items, five positive and five negative, from the 20 items on the original PANAS. Additionally, participants are asked how much they are feeling each affect item "at this moment" rather than "in the past week." As the current study examined negative affect, only the five negative affect items (scared, nervous, afraid, upset, and distressed) were assessed. In the current dissertation, negative affect (NA) assessed on the PANAS-S will be referred to as PANAS-S.

In the current dataset (*n*=3901 EMA assessments), internal reliability (Cronbach's α) of PANAS-S was .88.

Craving

Participants were asked to use a 7-point Likert scale (1 = no craving, 7 = extreme craving) to respond to the prompt "I am craving a cigarette" at each assessment on the smartphone.

Smoking Behaviors

Participants reported the number of cigarettes smoked since the last phone assessment (five response options: None (coded as "0"); One cigarette (coded as "1"); Two cigarettes (coded as "2"); Three cigarettes (coded as "3"); Four or more cigarettes (coded as "4").

ANALYTIC PLAN

As noted earlier, the analytic plan used both nomothetic and idiographic approaches. For the former, linear mixed models (LMM) was used for the primary analyses of both laboratory and EMA data. SAS PROC MIXED was used for analysis of continuous outcomes assumed to be normally distributed in the population, conditional on model covariates, and SAS PROC GLIMMIX was used for ordinal and non-normal outcomes. LMMs take into account the clustering of data by participants and allow for participants to have different numbers of assessments. For all models using SAS PROC MIXED, a random (subject-specific) intercept and an autoregressive model of order 1 (AR1) for the residuals within subjects was used. AR1 usually provides a good fit in EMA datasets, although alternative specifications (e.g., "modified AR(1)", Schwartz and Stone, 2007) were also tested.

Treatment condition (AR vs. Control) was included as a covariate in all analyses (lab and EMA). For analysis of laboratory data, Visit number (a categorical variable with

3 levels: Week -2, Quitday, Week +2) was included as a covariate. For EMA data, study Day (continuous variable) and Assessment Type (2 levels: RA vs. participant-initiated assessment) was entered as a continuous variable in all models. Regarding Day, for analysis of intensive longitudinal data, such as that collected in the current study, Bolger and Laurenceau (26) recommended adding time (e.g., study day) as a covariate. Day and Assessment Type were not the primary focus of analyses, and their coefficients were treated as fixed (see 225).

All models described conducted using SAS PROC MIXED or SAS PROC GLIMMIX and were confirmed using SPSS MIXED (continuous outcomes) or SPSS GENLINMIXED (ordinal or non-normal outcomes). In all cases in which the model converged, the SPSS analyses replicated the results of the analyses using SAS PROC MIXED or SAS PROC GLIMMIX. To bolster results of primary analyses, the Bootstrapping function in SPSS Version 24 was used to generate robust standard errors using Bias Corrected and Accelerated (BCA) bootstraps for all analyses yielding p values $< .10$. Currently, the use of the Bootstrapping function in SPSS does not permit the use of the repeated statement in SPSS MIXED, and so this statement was removed for these analyses.

The nomothetic analyses described above test null hypotheses related to the population. Idiographic analyses are conducted on data from individual participants (N=1 analyses) and test a null hypothesis specific to each individual. That is, for idiographic analysis the "population" of scores represents all data for an individual, and the data for analysis is conceptualized as a "sample" of that data. As noted earlier, idiographic analyses are appropriate for EMA data (48), can supplement nomothetic analyses of

longitudinal data (221)), and can reveal findings that are obscured by nomothetic approaches (e.g., 119). Idiographic analyses often involve time series analyses that account for serial dependencies within an individual's data, and time series analyses were used in the current study.

A number of procedures are available in SAS (and Version 8 of Mplus) for idiographic analyses. Following Hoeppner et al. (2008), who conducted an idiographic analysis of longitudinal smoking data, this study used SAS PROC ARIMA. (The "AR" of ARIMA refers to "autoregressive" parameters). Hoeppner et al. noted that the identification of the correct ARIMA model for a given dataset is not straightforward (263). They further noted that "..the general transformation approach (264) can be used, which simply uses an ARIMA (5, 0, 0) model (i.e., an autoregressive model of order 5) for all time series." The ARIMA (5, 0, 0) model refers to an autoregressive model with five lags, and no moving average terms included in the model. Hoeppner et al. (119) note that "A simulation study has shown that an ARIMA (5, 0, 0) model adequately approximates most commonly encountered time series in the behavioral sciences (108; 261; 262)…."

For all nomothetic analyses involving the S-DERS, analyses investigated two factor scores, S-Nonacceptance and S-Modulate, as well as S-DERS scores.

Unless otherwise state, alpha was .05, and all tests were 2-tailed.

Specific Aim 1: To examine the association between emotion regulation (trait and state) and negative affect.

Hypothesis 1A: Participants with higher DERS scores would report higher levels of NA in the lab, and on occasions when a participant reported a higher than average DERS rating, he/she would report higher negative affect ratings in the laboratory.

For analyses of laboratory data, the primary independent variables were a *Mean* DERS score and a *Deviation* DERS score assessed at laboratory visits, and the primary dependent variable were PANAS scores assessed in the laboratory. The *Mean* DERS score was computed by aggregating DERS scores over all available visits for each subject (i.e., *Mean* DERS is a level 2 variable). The *Deviation* DERS score (a level 1 variable) was computed as the difference between the DERS score at each visit and the *Mean* DERS score. The *Mean* DERS score and the *Deviation* DERS score was entered together in analyses, along with Visit (as described earlier). A significant coefficient for the *Mean* DERS score would indicate a between-subject association (i.e., that participants who report generally higher DERS scores have higher PANAS scores). A significant coefficient for the *Deviation* DERS score would indicate a within-subject association, i.e., that when a participant reports a higher DERS score than his or her average they report a higher PANAS score (115-117; 169)). In the primary analyses, the coefficient for *Deviation* DERS (a level 1 variable) was treated as random (allowed to vary across individuals). This specification allows the magnitude of the coefficient between *Deviation* DERS and PANAS scores to vary across individuals (see Schwartz and Stone for justification of this approach).

Hypothesis 1B: Participants with higher S-DERS scores would report higher levels of NA in the field and on occasions when a participant reported a higher than average S-DERS score, he/she would report higher negative affect ratings in the field

For the analyses of EMA data, S-DERS was the primary independent variable and the PANAS-S assessed during EMA was the dependent variable. As with analysis of lab data, *Mean* S-DERS and *Deviation* S-DERS scores were entered concurrently to examine between- and within-subject associations (along with other covariates, as described earlier). A significant coefficient for the *Mean* S-DERS score would indicate a between-subject association (i.e., that participants who report generally higher S-DERS scores have higher PANAS-S scores). The analysis between *Deviation* S-DERS and PANAS-S would reveal whether the individuals experience higher PANAS-S when their S-DERS scores are higher than usual. As for hypothesis 1A, the coefficient for *Deviation* S-DERS (a level 1 variable) was treated as random (allowed to vary across individuals). These analyses can therefore reveal <u>when</u> an individual is at risk of high PANAS-S.

Idiographic Analyses: Using an ARIMA (5, 0, 0) model, and including Day as an independent variable, individual models on PANAS-S data for each participant (across all study assessments) were run. The parameter estimate for S-DERS, as well as its standard error and *t* value are reported. Given the large number of analyses, an alpha level of .01 was used for these analyses.

As noted earlier, these analyses are within-subject analyses focused on <u>the individual</u>. The proportion of participants who report a significant (at alpha = .01) within-subject association (positive or negative) between S-DERS and PANAS-S were reported. The expectation was that the individual participants would tend to report a positive association between S-DERS and PANAS-S, such that higher levels of S-DERS were associated with higher levels of negative affect. However, it was possible that individual participants could exhibit a negative association between these two variables.

Specific Aim 2: To examine the association between emotion regulation (trait and state) and craving/smoking.

Hypothesis 2A: Participants with higher DERS scores would report higher levels of craving in the lab and more smoking over the following week, and on occasions when a participant reported a higher than average DERS rating, he/she would report higher craving and smoking in the laboratory, and more smoking over the following week.

For analyses of laboratory data, the primary dependent variables were WSWS craving scores assessed in the laboratory and reported smoking in the week following the laboratory visit, assessed on smoking diaries. The analysis plan followed that used for Hypothesis 1A, except that craving and reported smoking were used as the dependent variables (in separate models). Thus, the *Mean* DERS score and the *Deviation* DERS score were entered together in analyses. The coefficient for *Deviation* DERS (a level 1 variable) was treated as random (allowed to vary across individuals).

Hypothesis 2B: Participants with higher S-DERS scores would report higher levels of craving and smoking in the field, and on occasions when a participant reported a higher than average S-DERS rating, he/she would report higher craving and smoking in the field.

For the analyses of EMA data, S-DERS was the primary independent variable. The dependent variables were craving assessed during EMA (1-7 scale) and reported smoking during EMA before the next assessment (0-4 scale). The analysis plan followed that used for Hypothesis 1B. Thus, *Mean* S-DERS and *Deviation* S-DERS scores were entered concurrently to examine between- and within-subject associations. The analysis between *Deviation* S-DERS and craving/smoking revealed whether the individuals

experienced higher craving or report more smoking before the next assessment when their S-DERS scores were higher than their usual. As for hypothesis 2A, the coefficient for *Deviation* DERS (a level 1 variable) was treated as random (allowed to vary across individuals). We also explored whether the association between S-DERS and craving/smoking was moderated by quit-status by testing the pertinent interaction terms.

Idiographic Analyses: The same approach was taken as used for the idiographic analyses used in Specific Aim 1, i.e., use of an ARIMA (5, 0, 0) model, and including Day as an independent variable. Idiographic analyses revealed the proportion of participants who reported significant within-subject associations (positive or negative) between S-DERS and craving, and between S-DERS and smoking before the next assessment.

Specific Aim 3: To examine emotion regulation (trait and state) as a moderator variable.

Hypothesis 3A: To examine DERS as a moderator variable of the association between 1) negative affect and craving; and 2) craving and smoking. Specifically, strong trait emotion regulation skills (low DERS scores) were expected to weaken the relationship between 1) negative affect and craving and 2) craving and smoking.

Considering first the association between craving and smoking, for analyses of laboratory data, the primary dependent variable was reported smoking in the week following assessment, and the primary independent variable was a DERS x WSWS craving interaction term. A significant parameter estimate for the interaction would reveal that the association between WSWS craving and smoking is moderated by DERS. These analyses involve testing moderation on multilevel data. As described by Preacher et al. (205), testing of moderation hypotheses in multilevel data are more complex than

for non-hierarchical data. The moderator variable, DERS may potentially moderate a between-subject association, a within-subject association, or both. For Hypothesis 3A, both WSWS craving and smoking were assessed at level 1 (i.e., at each visit). This design can be described as a 1 x (1 → 1) design (205). There are three plausible tests of moderation. First, the association between individuals' general level of craving and their general level of smoking may be higher in individuals with high *Mean* DERS (vs. individuals with low *Mean* DERS). This interaction (a significant *Mean* DERS x *Mean* Craving interaction) would represent moderation of a between-subject association. Second, the association between craving and smoking within individuals may be higher in individuals with high *Mean* DERS (vs. individuals with low *Mean* DERS). This interaction (a significant *Mean* DERS x *Deviation* Craving interaction) would represent moderation of a within-subject association. Third, the association between craving and smoking within individuals may be higher when individuals report higher DERS scores (i.e., high *Deviation* DERS) vs. when they report lower DERS scores (low *Deviation DERS*). This interaction (a significant *Deviation* DERS x *Deviation* Craving interaction) also represents moderation of a within-subject association. Following the general approach described by Preacher et al. (2016), these three interaction terms were tested.

When testing a cross-level interaction, Aguinis et al. (2) recommended treating the level 1 coefficients as random. Schwartz and Stone (225) also describe testing a cross-level interaction in EMA data by treating the coefficients of the level 1 variable as random. Consistent with these recommendations the coefficients of *Deviation* scores (level 1 variables) were treated as random (i.e., allowed to vary across subjects). (Note that *Mean* scores are level 2 variables and therefore are treated as fixed.)

The same analyses were used to examine whether DERS moderated the association between negative affect and craving.

Hypothesis 3B: To examine S-DERS, as a moderator variable, of the association between 1) negative affect and craving; and 2) craving and smoking, assessed during EMA. Strong state emotion regulation (low S-DERS scores) were expected to weaken the relationship between 1) negative affect and craving and 2) craving and smoking, assessed during EMA.

Considering first the association between craving and smoking, for the analyses of EMA data, the primary dependent variables was reported smoking in the interval before the next assessment, and the primary independent variable is the S-DERS x Craving interaction term. The same methods as those described above were used to test the three interaction terms in the multilevel data. First, the association between individuals' general level of craving and their general level of smoking during EMA may be higher in individuals with high *Mean* S-DERS (vs. individuals with low *Mean* S-DERS). This interaction (a significant *Mean* S-DERS x *Mean* Craving interaction) would represent moderation of a between-subject association. Second, the association between craving and smoking within individuals may be higher in individuals with high *Mean* S-DERS (vs. individuals with low *Mean* S-DERS). This interaction (a significant *Mean* S-DERS x *Deviation* Craving interaction) would represent moderation of a within-subject association. This also represents a "cross-level interaction" because a level 2 variable (*Mean* S-DERS) is moderating the association between two level 1 variables (WSWS craving and Smoking). Third, the association between craving and smoking within individuals may be higher when individuals report higher DERS scores (i.e., high

Deviation S-DERS) (vs. when they report lower DERS scores (low *Deviation* S-DERS). This (a significant *Deviation* S-DERS x *Deviation* Craving interaction) also represents moderation of a within-subject association.

POWER ANALYSES

Field (64) noted that power analyses for mixed models are complex. To simplify calculations, Field (64) noted a method described in Twisk (254), which uses methods employed for single-level analyses, such as least squares regression, and which involves computing an adjusted sample size (or "effective sample size") that takes into account correlated data. This approach was taken to estimate power in this study.

Power analyses were computed using G*Power 3.1 (63). All analyses assumed alpha = .05 and a 2-tailed test. The power estimates take into account the fact that repeated observations from the same person will be correlated, as captured by the intraclass correlation coefficient (ICC). To compute the adjusted or "effective sample size", the expected total number of assessments, estimated prior to study initiation, was divided by the VIF (Variance Inflation Factor). (VIF = 1+((average number of observations per person) -1)*ICC), where ICC = intraclass correlation coefficient for the dependent variable.

All power analyses assume alpha = .05 and a 2-tailed test and were conducted using G Power version 3.1.3 using data from MacIntyre (169). Given the estimated study completion rate, for Hypothesis 1A, it was anticipated that data would be available from 87 visits from 30 participants; given an estimated intraclass correlation coefficient (estimated ICC = .73), power = .80 to detect a medium-to-large effect size of *rho* = .44 for the between-subject association. The power to detect a within-subject association can

be conceptualized as the power to reject the null hypothesis that the mean within-subject correlation in the population = 0. With *N*=30, the study has power = .80 to reject the null hypothesis if the mean within-subject correlation, rho, = .12, assuming that the SD of the within-subject correlations is 0.25. For Hypothesis 1B (within-subject association), the power was greater, due to the larger number of assessments. For the between-subject association (Hypothesis 1B) with an estimated ICC = .5, given that the mean number of assessments completed by each participant will be ~134 (assuming 80% compliance), the study has power = .80 to detect a medium-to-large effect size of *rho* = .35 (between-subject association).

For Hypotheses 2A and 2B, power depends on the ICCs for craving and smoking, but power is expected to be greater than that for Hypotheses 1A and 1B because the ICCs are expected to smaller (~.30).

Specific Aim 3 requires testing interactions in multilevel data, and power calculations become even more complex (64). Some guidance has been provided by Kreft and de Leeuw (151). They stated that when testing cross-level interactions (i.e., an interaction between a level 2 and a level 1 variable) one should have more than twenty level 2 units (corresponding to subjects in the current study), and that subject sizes "should not be too small". For EMA data, the current study was expected to have ~30 level 2 units (subjects), each with an expected average of ~134 assessments. Thus, for analysis of EMA data, the current study appears to meet Kreft and de Leeuw's informal criterion. For analysis of laboratory data, each subject was expected to have 3 data points (corresponding to the 3 visits), and so power will be lower for these analyses.

COVARIATES

Age, gender, education level, and income were considered as level 2 covariates. For each covariate, we examined whether the covariate was associated with DERS and S-DERS. *Mean* DERS scores were not correlated with age (r = -.08, p = .67), gender (r = .01, p = .97), years of education (r = .13, p = .52), and income (r = -.03, p = .69). *Mean* S-DERS scores were correlated with age (r = .43, p = .04), but not with gender (r = -.10, p = .64), years of education (r = .20, p = .38), and income (r = -.16, p = .48).

Parameter estimates from LMMs may be biased if the missing data are not missing at random (denoted be "NMAR"). Parameter estimates are unbiased if missingness is completed "explained" by the independent variables (denoted as "missing at random", "MAR"). We examined whether age, gender, education level, and income were associated with two indices of missingness, the number of RAs completed, and total number of assessments completed. None of the variables that significantly predicted these variables (all ps > .07), although age was marginally correlated with number of RAs completed (r = .38, p = .07).

Given the above, age was included as a covariate in secondary analyses involving S-DERS. None of the main findings changed when including age as a covariate. Therefore, only results unadjusted for age are presented in the dissertation.

EXPLORATORY ANALYSES

Primary analyses used all available data from both phases of the study. Exploratory analyses were also conducted to determine whether results differed between the pre-quit phase and post-quit phase.

Treatment condition was included as a covariate in all analyses. Exploratory analyses also examined whether patterns of associations differed testing by Treatment condition by testing the pertinent Treatment by (S-)DERS interaction term.

The primary analyses involving negative affect and craving were cross-sectional in that the independent and dependent variables were assessed at the same time point. (Within-subject analyses involving smoking were prospective in that the dependent variable was amount smoked before the next assessment). For negative affect, to get further information on temporal relationships between S-DERS and negative affect, analyses involving lagged values of independent variable were conducted. This is described in more detail in the results section.

Finally, exploratory analyses were conducted using different specifications of the R matrix (within-subject correlation of residuals), following the guidance of Schwartz and Stone (225).

Results

Overall, the 29 participants all completed at least one DERS assessment, and 25 participants contributed data on the S-DERS (Table 3, Figure 6). As noted in Table 3, participants were predominantly African American, with low socioeconomic status. Participants reported smoking approximately 13 cigarettes per day.

DESCRIPTIVE STATISTICS

Of the 29 participants, 28 remained in the study until quit-day, and 27 (93.10%) completed treatment and attended the Week +3 visit. Of these 27 participants, smoking data from EMA assessments were available for 24 participants. Participants who reported no smoking during EMA during the final week of treatment, and who had a CO level ≤ 8 ppm at Week +3, were designated as "abstinent". Of the 3 participants without EMA data, 2 participants had a CO level ≥ 9 ppm at Week +3 and were designated as "non-abstinent"; 1 participant reported no smoking on the smoking log for that week and had a CO level ≤ 8 ppm at Week +3, and was designated as "abstinent". (Reported smoking on the smoking log during the final week was strongly correlated with reported smoking during EMA during the final week, Spearman $r = .87$, $p < .001$). Using these criteria, 6 of the 27 participants (22.22%) were designated as abstinent at Week +3.

Overall, the 25 participants with S-DERS data initiated 4045 EMA assessments (1248 RAs; 2797 participant-initiated assessments) and completed 3901 EMA assessments (94.46% of participant-initiated assessments). Participants completed an average of 48.60 RAs ($SD = 41.89$) and an average of 107.44 participant-initiated assessments ($SD = 54.65$), an average of 156.04 total assessments ($SD = 51.48$). Participants completed EMA assessments for an average of 41.64 days ($SD = 2.91$).

Participants completed 93.80% (SD = 2.90%) of the expected number of assessments (4 assessments per day for the duration of the study). As noted above, the majority of assessments were participant-initiated (68.85%).

Aggregated over all EMA assessments, average S-DERS scores (1-5 scale) were 1.20 (SD=0.35), 1.22 (SD=0.44), and 1.15 (SD = 0.31) for S-DERS, S-Nonacceptance, and S-Modulate respectively. The minimum possible score (i.e., 1.00) was reported on 64.82%, 72.36%, and 75.73% of assessments for S-DERS, S-Nonacceptance, and S-Modulate respectively.

To evaluate within-subject variability, the standard deviation of S-DERS data was computed for each participant separately. The average within-participant standard deviation was 0.17, 0.25, and 0.15 for S-DERS, S-Nonacceptance, and S-Modulate respectively. For S-DERS and S-Nonacceptance, all participants had non-zero standard deviations. For S-Modulate, 5 out of 23 participants (21.74% of participants) had no variability in S-Modulate across the study.

Descriptive statistics for laboratory and field data are reported in Tables 4 and 5 respectively.

Correlations between Lab and Field Data

Using Spearman's correlation, average DERS scores from the lab, aggregated across all visits, correlated with average S-DERS (n=25, r=.45, p=.02), S-Nonacceptance (n=25, r=.50, p=.01), and S-Modulate scores (n=23, r=.47, p=.02), aggregated over all EMA assessments. However, these correlations were not significant using Pearson's r (rs = .34, .20, and .39 respectively).

SPECIFIC AIM 1

Hypothesis 1A tested whether participants with higher DERS scores reported higher levels of NA in the lab, and whether on occasions when a participant reported a higher than average DERS rating, he/she reported higher negative affect ratings in the laboratory. Table 6 reveals that there was a significant positive association between the *Mean* DERS score and PANAS (a between-subject association; Figure 7), and a significant positive association between the *Deviation* DERS score and PANAS (a within-subject association), supporting hypothesis 1A (Figure 8).

Hypothesis 1B tested whether participants with higher S-DERS scores reported higher levels of NA in the field, and whether on occasions when a participant reported a higher than average S-DERS rating, he/she reported higher negative affect ratings in the field. Table 7 reveals that there was a significant positive association between the *Mean* S-DERS score and PANAS-S (a between-subject association; Figure 9), and a significant positive association between the *Deviation* S-DERS score and PANAS-S (a within-subject association), supporting hypothesis 1B (Figure 10). Regarding the two subscales, the between-subject association was significant for both scales, and the within-subject association was significant for S-Modulate but not S-Nonacceptance.

For hypothesis 1C, time series analyses examined associations between S-DERS and NA in individual subjects. Table 14 reports results of ARIMA analyses conducted on 25 individual subjects with S-DERS and Day as predictor variables and PANAS-S as the dependent variable. Using an alpha of .01, 9 subjects (36% of subjects) exhibited significant positive associations between S-DERS and PANAS-S. For example, subject 1043 exhibited a parameter estimate (PE) of 0.67, with a significant t value. This indicates that the null hypothesis that the regression coefficient (PE) in the "population"

for subject 1043 is equal to zero (when controlling for Day and autoregressive parameters) can be rejected. The PE of 0.67 can be interpreted in the usual way, i.e., for every 1 unit increase in S-DERS the predicted negative affect increases by 0.67 units, when controlling for Day and autoregressive parameters. No subjects (0% of subjects) exhibited a significant negative association between S-DERS and PANAS-S. Consistent with the results of within-subject nomothetic analyses above, the mean *t* value was significantly different from 0, $t(24) = 2.74$, $p = .005$.

As noted earlier, there was limited within-subject variability of S-DERS, which may reduce the ability to detect significant associations. Consistent with this, there was a significant correlation between *SD* of S-DERS and the magnitude of the *t* value reported in Table 14 ($r = .57$, $p = .003$). That is, participants who exhibited more variability in S-DERS exhibited stronger associations between S-DERS and PANAS-S.

SPECIFIC AIM 2

Hypothesis 2A tested whether participants with higher DERS scores reported higher levels of craving in the lab and smoking over the following week, and whether on occasions when a participant reported a higher than average DERS rating, he/she reported higher craving and smoking. There was no evidence for a significant positive association between the *Mean* DERS score and WSWS craving (a between-subject association; Table 8), or a significant positive association between the *Deviation* DERS score and WSWS craving (a within-subject association; Table 9). Similarly, there was no evidence for a significant positive association between the *Mean* DERS score and smoking during the following week (a between-subject association), or a significant positive association

between the *Deviation* DERS score and smoking during the following week (a within-subject association). In sum, there was no evidence to support hypothesis 2A.

Hypothesis 2B tested whether participants with higher S-DERS scores reported higher levels of craving and smoking in the field, and whether on occasions when a participant reported a higher than average S-DERS rating, he/she reported higher craving and smoking in the field. There was no evidence for a significant positive association between the *Mean* S-DERS score and craving (a between-subject association; Table 10), or a significant positive association between the *Deviation* S-DERS score and craving (a within-subject association; Table 11). Similarly, there was no evidence for a significant positive association between the *Mean* S-DERS score and smoking (a between-subject association), or a significant positive association between the lagged *Deviation* S-DERS score and Smoking before the next assessment (a within-subject association). The same was true for S-Nonacceptance and S-Modulate scores. In sum, there was no evidence to support hypothesis 2B.

For hypothesis 2C, time series analyses examined associations between S-DERS and Craving in individual subjects. Table 14 reports results of ARIMA analyses conducted on 25 individual subjects with S-DERS and Day as predictor variables and Craving as the dependent variable. Using an alpha value of .01, 1 subject (4% of subjects) exhibited significant positive associations between S-DERS and Craving. One subject (4% of subjects) exhibited a significant negative association between S-DERS and Craving. Consistent with the results of within-subject nomothetic analyses above, the mean t value ($M = 0.28$, $SD = 1.87$) was not significantly different from 0, $t(24) = 0.75$, $p = .46$.

Time series analyses also examined associations between S-DERS and Smoking before the next assessment in individual subjects. Table 14 reports results of ARIMA analyses conducted on 25 individual subjects with S-DERS and Day as predictor variables and Smoking as the dependent variable. Using an alpha value of .01, 2 subjects (8% of subjects) exhibited significant positive associations between S-DERS and Smoking before the next assessment. No subjects (0% of subjects) exhibited a significant negative association between S-DERS and Smoking before the next assessment. Consistent with the results of within-subject nomothetic analyses above, the mean *t* value ($M = 0.22$, $SD = 1.20$) was not significantly different from 0, $t(24) = 0.91$, $p = .37$.

As noted earlier, there was limited within-subject variability of S-DERS, which may reduce the ability to detect significant associations. Contrary to the results for Specific Aim 1, there was no significant correlation between *SD* of S-DERS and the magnitude of the *t* value reported in Table 14 for Craving ($r = .25$, $p = .22$) or Smoking ($r = .01$, $p = .97$).

SPECIFIC AIM 3

Hypothesis 3A tested whether strong trait emotion regulation skills (low DERS scores) weakened the relationship between 1) negative affect and craving and 2) craving and smoking, assessed in the lab. As noted in Table 12, there was no evidence for a significant interaction between DERS and PANAS in the prediction of craving in the lab. There was, however, evidence for an interaction between *Mean* DERS and *Deviation* Craving in the prediction of Smoking (Figure 11), and between *Deviation* DERS and *Deviation* Craving in the prediction of Smoking (Figure 12). However, as noted in the Table, these results were obtained when the coefficients for *Deviation* Craving were

treated as fixed, rather than random. The model failed to converge in either SPSS or SAS when the coefficients for *Deviation* Craving were treated as random. Treating the coefficients of fixed increases power of the tests but may also increase the type I error rate if there truly are differences in associations between craving and smoking in the population. Therefore, the reported *p* values should be treated with caution.

Hypothesis 3B tested whether strong state emotion regulation (low S-DERS scores) weakened the relationship between 1) negative affect and craving and 2) craving and smoking, assessed during EMA. As noted in Table 12, there was evidence for a significant interaction between *Mean* S-Nonacceptance and *Deviation* NA in the prediction of craving. This interaction is illustrated in Figure 13. As implied by the Figure, the association *Deviation* NA and Craving tends to be greater in individuals with higher levels of S-Nonacceptance than in individuals with lower levels of S-Nonacceptance. For example, there was a significant association between *Deviation* NA and Craving for individuals with higher than median *Mean* S-Nonacceptance scores, $PE = 0.45$, $SE = 0.10$, $F\ (1, 1841) = 21.84$, $p < .001$, but not for individuals with lower than median *Mean* S-Nonacceptance scores, $PE = 0.13$, $SE = 0.09$, $F\ (1, 1672) = 2.20$, $p = .13$. This interaction is in the predicted direction, and provides some support for hypothesis 3B. Confidence in this interaction (*Mean* S-Nonacceptance x *Deviation* NA interaction) is bolstered by the fact that it was observed when treating craving as an ordered categorical variable (using SAS PROC GLIMMIX), $PE = 0.49$, $SE = 0.18$, $F(1, 3823) = 7.14$, $p = .008$.

There was no evidence that strong state emotion regulation weakened the relationship between craving and smoking (Table 13)

EXPLORATORY ANALYSES

Effect of Phase

Exploratory analyses examined whether the significant associations reported earlier differed by Phase. For these analyses, Phase (Pre-quit vs. Post-quit) was included as a covariate.

For hypothesis 1A, there was no evidence that the association between *Mean* DERS and PANAS was higher at the Pre-quit visit (*PE* = 0.016) than at the Post-quit visits (*PE* = 0.023) (*p* value for Phase x *Mean* DERS interaction = .48). Likewise, there was no evidence that the association between *Deviation* DERS and PANAS was higher at the Pre-quit visits (*PE* = 0.006) than at the Post-quit visits (*PE* = 0.038) (*p* value for Phase x *Deviation* DERS = .45)

For hypothesis 1B, there was evidence that the association between *Mean* S-DERS and PANAS-S was higher during the Pre-quit phase (*PE* = 0.76) than during the Post-quit phase (*PE* = 0.64) (*p* value for Phase x *Mean* S-DERS interaction = .005), although the association was robust for both phases. There was no evidence that the association between *Deviation* S-DERS and PANAS-S was higher during the Pre-quit phase (*PE* = 0.31) than Post-quit (*PE* = 0.25) (*p* value for Phase x *Deviation* S-DERS interaction = .54).

For hypothesis 3A, there was no evidence that the interaction between *Mean* DERS and *Deviation* Craving, and the interaction between *Deviation* DERS and *Deviation* Craving, were moderated by Phase (*p* values for Phase x *Mean* DERS x *Deviation* Craving and Phase x *Deviation* DERS x *Deviation* Craving interaction terms = .43 and .46 respectively).

For hypothesis 3B, there are no evidence that the *Mean* S-Nonacceptance x *Deviation* NA interaction in the Pre-quit phase (*PE* = 0.63) was different from the *Mean* S-Nonacceptance x *Deviation* NA interaction in the Post-quit phase (*PE* = 0.76) (*p* value for Phase x *Mean* S-Nonacceptance x *Deviation* NA interaction term = .66)

Moderation by Treatment

Exploratory analyses examined whether significant associations observed differed by Treatment condition. For hypothesis 1A, there was no evidence that the association between *Mean* DERS and *Deviation* DERS was moderated by Treatment (*p* values for Treatment x *Mean* DERS and Treatment x *Deviation* DERS interaction terms = .84 and .66 respectively). For hypothesis 1B, there was likewise no evidence that the association between *Mean* S-DERS and PANAS-S, and *Deviation* S-DERS and PANAS-S, were moderated by Treatment (*p* values for Treatment x *Mean* S-DERS and Treatment x *Deviation* S-DERS interaction terms = .23 and .83 respectively).

For hypothesis 3A, there was no evidence that the interaction between *Mean* DERS and *Deviation* Craving, and the interaction between *Deviation* DERS and *Deviation* Craving, were moderated by Treatment (*p* values for Treatment x *Mean* DERS x *Deviation* Craving and Treatment x *Deviation* DERS x *Deviation* Craving interaction terms = .35 and .06 respectively).

For hypothesis 3B, there was no evidence that the *Mean* S-Nonacceptance x *Deviation* NA interaction was moderated by Treatment (*p* value for Treatment x *Mean* S-Nonacceptance x *Deviation* NA interaction term = .86).

Temporal Relationships

As noted earlier, there was a significant association between *Deviation* S-DERS and PANAS-S, meaning that when a participant reported higher levels of S-DERS than his or her average, he or she also reported higher levels of negative affect. This analysis is cross-sectional, in that *Deviation* S-DERS and PANAS-S are assessed at the same time. To gain additional information on temporal relationships, two additional analyses were conducted: 1) *Deviation* S-DERS at time t_{-1} served as the (lagged) predictor variable and PANAS-S at time *t* as the dependent variable; and 2) *Deviation* PANAS-S (NA) at time t_{-1} served as the (lagged) predictor variable and S-DERS at time *t* as the dependent variable.

Lagged *Deviation* S-DERS predicted PANAS-S, $PE = 0.10$, $SE = 0.03$, $p = .02$, when controlling for lagged *Deviation* PANAS-S (and other covariates included in earlier models). That is, when a participant reported higher levels of S-DERS than his or her average, he or she also reported higher levels of negative affect at the next assessment. However, the reverse was not true: Lagged *Deviation* PANAS-S did not predict S-DERS, $PE = 0.00$, $SE = 0.02$, $p = .98$, when controlling for lagged *Deviation* DERS (and other covariates included in earlier models). In sum, for the within-subject association there was more evidence for a causal relationship from S-DERS to PANAS-S than vice versa.

R-Sided Covariance Structure

The reported analyses using SAS and SPSS for continuous outcomes used "type=AR(1)" for the R-sided covariance structure (R matrix). It has been argued that this specification assumes equal intervals (spacing) between assessments, which is true for daily diary data (as used by Hoeppner et al. (119)) but not quite true for assessment-level EMA data (225). Schwartz and Stone (216) suggested alternative code that applies to

unequal intervals (Schwartz and Stone p. 88-89 provide a detailed description of the method). They termed this specification “modified first order autoregressive” and argued that it may provide an optimal fit for mixed models with unequal intervals. In the abundance of caution, all key analyses were computed using the Schwartz and Stone (216) specification for the R-matrix; there was no change in the key findings. It should be noted that the same issue also applies to the idiographic analyses in that ARIMA models assume equal spacing (equal intervals between assessments). For this reason, the *p* values from ARIMA models should be treated with caution pending replication using a robust method.

Discussion

The main findings of the study were as follows. First, regarding Specific Aim 1, an association between emotion regulation and negative affect was observed both in the lab and in the field. As emotion dysregulation increased, negative affect increased. Second, for Specific Aim 2, there was no evidence for an association between emotion regulation and craving. Similarly, there was no evidence for an association between emotion regulation and smoking during the week following each, or before the next EMA assessment. Third, for Specific Aim 3, there is evidence that emotion regulation, specifically the nonacceptance of emotion, may moderate the association between negative affect and craving in field data. There is no evidence that emotion regulation moderates the association between craving and smoking in field, and some tentative evidence in lab data. In general, the pattern of data did not differ from pre-quit to post-quit. These findings are discussed in more detail below.

Association Between Emotion Regulation and Negative Affect

Emotion regulation was significantly associated with negative affect assessed in the laboratory and field, such that more difficulty with emotion regulation is associated with increased levels of negative affect. Moreover, for both settings, there was evidence for both between- and within-subject associations between emotion regulation and negative affect. These results are described further below.

In the laboratory, *Mean* DERS score across visits was significantly associated with negative affect across visits, such that individuals with poor overall emotion regulation abilities had higher intensity of negative affect assessed in the lab. These findings support the majority of emotion regulation conceptualizations that view emotion

regulation as a trait measure (e.g., 94; 131). The significant association between the *Deviation* DERS score and negative affect at each visit indicates that visit-to-visit changes in emotion regulation abilities are themselves related to negative affect. The results support the notion that better or worse emotion regulation over a certain time period has a significant relationship with the negative affect the individual experiences during that time period and support the state-like aspects of emotion regulation as well.

Similar to the results seen in the lab, *Mean* S-DERS score across all field assessments was significantly associated with negative affect in the field. Individuals who reported higher average S-DERS scores in the field reported higher negative affect. Additionally, the significant association between the *Deviation* S-DERS score and negative affect indicates that the relationship between fluctuations in emotion regulation and changes in negative remains significant when examined outside of the laboratory setting and over a significantly larger number of assessments. That is, when an individual is experiencing more emotion dysregulation, he or she is more likely to be experiencing higher levels of negative affect. When examining the two subscale scores, the association remains significant for S-Modulate, and becomes non-significant for S-Nonacceptance.

The idiographic analyses examining associations between S-DERS and negative affect supported the results from the within-subject analyses from mixed models, with 9 participants (36% of 25 participants, 95% CIs, 20.25%, 55.48%) showing a significant positive association and 0 participants showing a negative association. One might wonder why the association is not observed for all participants, or a large majority of participants. As noted earlier, the limited variability in S-DERS scores within participants may have made it difficult to detect associations between these variables in some individuals.

While the relationships were significant for both the between- and within-subject associations, the between-subject associations appear to be stronger. As noted above, it is possible that the limited variability of S-DERS response within participants weakened our ability to find within-subject associations. Notably, as reported earlier, at about two thirds of all assessments participants reported the minimum possible S-DERS score (1.00).

To determine whether scores in the current study were lower than that observed in previous studies, the scores obtained in the current study were compared to scores obtained in the original study validating the S-DERS measure (157). S-Modulate scores in the current study (M = 1.15, SD = 0.31) were lower than those in the Lavender et al. study (M = 1.44, SD = 0.64), and this difference was significant, t = 2.09, p = .04. However, it is important to note that the Lavender et al. scores were obtained following a mood induction task which was expected to elevate S-DERS scores. S-Nonacceptance scores for the current study (M = 1.22, SD = 0.43) were also lower than those in Lavender et al. (M = 1.46, SD = 0.76), although this difference was not significant, t = 1.43, p = .15. In sum, the scores obtained in the current study were not only low in an absolute sense, i.e., close to 1.00, but were also lower than scores obtained in the validation study.

This is the first study to utilize the S-DERS in an EMA study. It is possible that while the S-DERS does detect significant changes in emotion regulation, such as before and after an intervention designed to elicit a strong emotional response (27; 91), the moment-to-moment changes in emotion regulation throughout daily life being measured in the current study may simply not have been strong enough to produce a large change in response on the S-DERS.

Overall, the results on negative affect replicate and extend the findings of MacIntyre and colleagues (170). It replicates the finding that emotion regulation is significantly associated with negative affect assessed in the laboratory, when examined both between- and within-subjects. It extends these findings by finding the same pattern of results when using EMA and a measure specifically designed to assess state emotion regulation. As noted earlier, several studies have used the S-DERS in laboratory studies to investigate changes in emotion regulation following tasks expected to impact emotion regulation (9; 12; 27; 91). To our knowledge, the current study is the first study to utilize the S-DERS to examine state changes in emotion regulation abilities in daily life using EMA methodology.

The current study focuses on cross-sectional analyses between emotional dysregulation and negative affect, for both between and within-subject associations. To restate, analyses address the question as to whether individuals who are report generally higher levels of trait and state emotion dysregulation report more generally higher levels of negative affect (between-subject association), and whether individuals report higher levels of negative affect when they report more trait/state emotion dysregulation than usual (within-subject association). Given the expected temporal relationships between emotion dysregulation and negative affect, any relationships that exist between these two variables would be most likely detected in cross-sectional analyses. Future research, preferably using larger sample sizes, could also benefit from prospective analyses, for both between- and within-subject associations. For the latter, exploratory analyses revealed that there was more evidence for a prospective association from *Deviation* S-DERS to negative affect at the subsequent assessment, than from *Deviation* PANAS-S to

S-DERS at the subsequent assessment. Stated another way, there was more evidence for a causal relationship between emotion regulation and negative affect, than vice versa. (This is consistent with the causal model of the study). This finding supports some previous literature that found emotion regulation to be more predictive of emotional adjustment than the other way around (19). For prospective between-subject associations, one could examine whether *Mean* S-DERS in the pre-quit phase predicts negative affect during post-quit.

ASSOCIATIONS BETWEEN EMOTION REGULATION AND CRAVING/SMOKING

Contrary to hypothesis, there was no evidence for a significant association between emotion regulation and craving. This was true for both laboratory data and field data. Moreover, it was true both when examining between-subject association and within-subject associations. For the field data, the conclusion remained true when examining S-DERS scores as well as the two S-DERS subscale scores. The idiographic analyses examining associations between S-DERS and craving supported the results from the within-subject analyses from nomothetic analyses, with one participant showing a significant positive association and one participant showing a significant negative association.

Also, contrary to hypothesis, there was also little evidence for a significant association between emotion regulation and smoking. Again, this was true for both laboratory data and field data, and when examining between-subject association and within-subject associations examining S-DERS scores as well as the two subscale scores. The idiographic analyses revealed that 2 participants (8% of 25 participants, 95% CI = 2.22%. 24.97%) exhibited a significant association between S-DERS and smoking before

the next assessment. Therefore, it is possible that there exists a small proportion of participants who do exhibit a robust within-subject association. Future research using larger samples will be required to examine this further. However, the current data suggest that, even if this were true, only a small minority of participants would likely exhibit this association.

Based on previous literature, it was expected that results would support a relationship between emotion regulation and both craving and smoking. As described in the section titled "Emotion Regulation and Tobacco Use," previous studies have documented the relationship between negative affect and smoking and the perceived use of smoking as an emotion regulation strategy to reduce or avoid negative affect (113; 136; 243). Additionally, poor emotion regulation has been associated with early smoking initiation, greater smoking urges, and higher rates of cessation relapse (104; 172). Despite this, there are some possibilities for why a significant association was not found.

One possibility is that there is lower power to detect an association between emotion regulation and craving than between emotion regulation and negative affect. Theoretically, one would expect an effect of emotion regulation on smoking (or craving) to be (at least partly) mediated by its effect on negative affect. Despite the hypothesis of an association, there are two primary reasons a relationship between emotion regulation and craving/smoking does not emerge. First, there is lower power for testing the association with craving/smoking than with negative affect, (see Kenny and Judd (142) for detailed account). Second, theory suggests that the association between emotion regulation and negative affect is likely to be a larger effect than the association between emotion regulation and craving/smoking, because affect/affective lability are more

proximal to emotion regulation than craving/smoking. If it exists, this relationship would be more difficult to find, particularly with a relatively small sample size.

Indeed, while some studies have found significant relationships between specific emotion regulation strategies and smoking/craving (e.g., 77; 104; 141; 172; 245) there is a clear dearth of findings relating general emotion regulation skills to craving or smoking behavior. On the other hand, there is a large amount of research connecting emotion regulation and affect (e.g., 101; 174; 218) a relationship further supported by the significant results of Aim 1 in the current study. It is possible that while there is a strong theoretical case for a relationship between emotion regulation and craving or smoking, the relationship is too distant to be detected using the methods used in the current study. It is also possible that while specific emotion regulation strategies are related to craving and smoking, the relationship does not hold for the more general emotion regulation skills assessed by the DERS or the two subscales used for the S-DERS.

In addition, regarding the field data, as discussed in the previous section the scores on the S-DERS showed minimal elevation and variability, and this may have made it difficult to detect associations with craving/smoking. It is possible that the S-DERS is not well-suited to measuring the slight changes in emotion regulation skills that occur throughout the day with minimal provocation. It is also possible that while there are clear fluctuations in emotion regulation skills, individuals remain relatively stable in the absence of major disruptions. As mentioned above, some studies have found changes in emotion regulation skills following laboratory interventions (e.g., 27) and following remission of psychological symptoms (52), but few have examined naturally occuring changes throughout the day. Given the random timing of the assessments in the current

study, it is very possible that assessments did not occur close in time to situations in which a participant may have been experiencing strong fluctuations in emotion regulation and therefore missed the time to detect variability. While the relationship between emotion regulation and negative affect discussed above may have been strong enough to overcome this limited variability, the more distant relationship between emotion regulation and craving/smoking would have been more difficult to detect if the factors discussed impacted the detection of emotion regulation fluctuations.

Previous literature supports a relationship between nonacceptance and general smoking behavior (37) and nonacceptance and recent smoking behavior (1). While the Adams et al. study did not specifically examine the association between nonacceptance and craving, it is unclear why the current study did not replicate the relationship between nonacceptance and smoking. One possible explanation could be due to the fact that craving was assessed at the same time point as the S-DERS in the current study. Given that the participants in this study were smokers who were hoping to quit, it is possible that individuals were reporting elevated levels of nonacceptance of current emotions at times of elevated craving because they were feeling angry or weak for craving a cigarette.

Additionally, it was expected that a relationship between the Modulate subscale (i.e., difficulties modulating emotional and behavioral responses in the moment) and smoking would be found. The Modulate subscale includes items such as "I feel out of control" and "I am having difficulty controlling my behaviors (157)." Endorsement of such statements would conceivably occur at times when an individual was having trouble resisting his or her craving and gave in to the urge to smoke. It is possible that the relationship was not significant due to the relatively low endorsement and variability

found on the S-DERS (discussed in more detail in the previous sections). It is also possible that while individuals in the study did report a desire to quit smoking, they may not have felt "out of control" when they smoked, particularly given the common nature of smoking for participants in the current study. Participants may have refrained from endorsing these items unless they were about to engage in a behavior that truly felt "out of control" or out of the ordinary. Some studies suggest that while smokers may feel out of control of their craving, they do not lose control of their behavior (15).

EMOTION REGULATION AS A MODERATOR

Overall, there was evidence that nonacceptance of emotions may moderate the association between negative affect and craving. The findings suggest that individuals who have generally higher levels of nonacceptance of current emotions report more craving for cigarettes when they experience increases in negative affect. The moderation of the association between negative affect and craving by nonacceptance makes strong conceptual sense. The Nonacceptance subscale includes items such as "I am angry with myself for feeling this way" and "I feel like I am a weak person for feeling this way (157)." Individuals who more frequently endorse these items may be more likely to look for ways to help themselves stop feeling those distressing emotions. When considered in the context of smokers' belief that smoking helps to reduce emotional distress (29; 46; 272), it would make sense to find a relationship between individuals who tend to feel more upset about their emotions and craving a perceived "cure" when they feel those upsetting emotions. This result supports the focus on acceptance-based treatments for smoking cessation to help individuals cope with and accept negative affect without relapsing (37; 83).

The moderating effect was only found when mean levels of nonacceptance and deviation scores for negative affect were examined. It is possible that a moderation relationship when using deviation nonacceptance scores was not found because of the low variability of scores discussed above. A moderating relationship between mean nonacceptance and mean negative affect may not have been found because the power to detect such a relationship is lower.

We did not find any moderating effect of S-DERS on the relationship between craving and smoking. While such a relationship was expected to be found, it is possible that if such an effect exists, it is too small to have been found in the current study. It was expected that emotion regulation would have a stronger effect on the relationship between negative affect and craving because these are purely internal responses. In contrast, the transition from craving to smoking could be impacted by a number of factors beyond internal regulation such as current access to cigarettes or ability to smoke in present location due to restrictions. If a participant experienced emotion dysregulation that may have altered her decision to smoke in a moment of craving but was at work where smoking is prohibited, our data would not detect any relationship between craving and smoking because the smoking did not occur for reasons not measured by the current study. Examining such factors will be important in future studies.

In contrast to the field data, there was evidence that DERS scores moderated the association between craving assessed in the lab and subsequent smoking. However, these results are treated with caution for the following reasons. First, due to participant non-compliance on the smoking diaries, the analyses were restricted to 26 individuals with data. Second, the results were obtained when treating coefficients for craving as fixed, as

the model did not converge when treated as random. This specification may inflate the risk of a type I error if the coefficients are truly different between subjects in the population. Third, the data were not consistent in that regression coefficients for the (significant) interaction terms had different signs for between- and within- subject associations. It is difficult to think of a theoretical basis for why the regression coefficients (for the interaction terms) should have different signs in the two analyses. In sum, these results are treated with caution, pending replication.

STUDY STRENGTHS

The study had several strengths. First, this study is a preliminary examination of state, versus trait, emotion regulation, an investigation that has been called for by a variety of researchers in the field (150; 153). Assessment of state (as well as trait) emotion regulation may reveal information as to when an individual is at risk of negative outcomes, and therefore provide guidance on when treatments might be most effective.

Second, the relatively long duration of the study permitted the use of both nomothetic and idiographic analyses, which could have revealed patterns among individuals that could help tailor treatment in the future. Last, the current study also examined emotion regulation as both a predictor variable and as a moderator variable. This focus is consistent with theory but has been rarely examined in the same study.

STUDY LIMITATIONS

There are several limitations to the current study that should be noted. First, this study was incorporated into a parent study that included an intervention. Although the intervention was not expected to alter emotion regulation, and although Treatment was

controlled for in all analyses, the presence of the intervention may limit the generalizability of the current findings.

Second, the data are correlational. Relatedly, as noted earlier, analyses focused primarily on cross-sectional analyses. It is not possible to state that emotion regulation causes the patterns of negative affect observed in the lab or field data.

Third, in order to reduce participant burden and increase adherence, only two subscales of the S-DERS were administered in the field. Therefore, the assessment of state emotion regulation was not comprehensive. Furthermore, two items from the S-Modulate subscale administered in the field were not included. One item, "I believe that I am going to end up feeling very depressed," was not included due to safety monitoring concerns. A second item, "I am having difficulty doing the things I need to do right now," was inadvertently excluded when programming the smartphones.

Fourth, emotion regulation measured in the laboratory used the total scale while the field data is drawn from data from two selected subscales, complicating direct comparisons between lab and field data This is also true for measures of craving; the WSWS is used in the laboratory while a single item was used to assess craving in the field. Finally, while the WSWS uses four items to assess craving, it is not a dedicated craving assessment like the Questionnaire for Smoking Urges (QSU; 51).

Fifth, in order to increase the number of trainings/assessments completed during this demanding 6-week protocol, participants were permitted to enter participant-initiated assessments when they missed a random assessment. This had the desired effect of increasing the overall number of assessments (and therefore trainings) completed, with participants completing 93.70% of expected assessments. However, this procedure

clearly reduced the proportion of random assessments that were completed, meaning that the data from random assessments may be less "random" than other EMA studies. Moreover, because participants could select the time for entering participant-initiated assessments, these assessments are also likely to occur at non-random times. Stated another way, emotion dysregulation may be reduced in this study if participants tend to enter assessments when feeling emotionally regulated.

Last, the exclusion criteria may have excluded participants with the highest levels of emotional dysregulation, potentially limiting the range of DERS and S-DERS observed. That is, participants who were most likely to reveal large fluctuations in emotional dysregulation were excluded from the study. When taken together with the previous limitation, this consideration provides further context for evaluating the limited range of emotion regulation scores in the study.

IMPLICATIONS

Theoretical

This study expands on theory surrounding emotion regulation, particularly on the relationship between emotion regulation and negative affect. Some studies have focused on specific emotion regulation skills, as a means to control or reduce internal emotional responses. For example, several studies have found that instructions to use reappraisal have reduced the amount of negative affect participants experience when compared to control conditions (97; 101). Other researchers push back against the idea of conflating negative emotions with emotion dysregulation (95). These theorists emphasize that the presence of negative emotions is not inherently disruptive or problematic. Studies examining suppression and avoidance support the idea that attempting to prevent or

minimize uncomfortable negative internal experiences, such as negative affect, can actually increase these responses (110; 220). Indeed, studies have found that an inability to tolerate distress is closely related to relapse, suggesting that it is the reaction to negative affect, rather than the presence of negative affect that is associated with replapse (34). This line of theory emphasizes the control of behavior in the presence of negative emotion, rather than the control of the emotion itself (180).

The results of the current study provide some evidence for each theoretical approach. The significant association between emotion regulation and negative affect found in both the field and laboratory strongly suggests that individuals with more emotion regulation difficulties also experience more negative affect. Additionally, the lagged analyses suggest that when an individual is particularly struggling with emotion regulation (as indicated by a high *Deviation* DERS score) he or she is more likely to experience higher negative affect at the subsequent assessment. This finding does suggest that while increased negative affect does not necessarily indicate emotion dysregulation, poor emotion regulation may lead to increased negative affect. While a significant relationship between emotion regulation and smoking would have provided evidence for the theory that good emotion regulation skills help individuals control their behavior, the moderating effect of emotion regulation on the relationship between negative affect and craving does lend support to the idea that individuals with strong emotion regulation skills are better able to control behavioral urges in the face of negative affect. Further analyses could examine whether emotion regulation measures correlate with quit status even in the absence of shorter term associations examined in the current study.

Clinical

The current study suggests that individuals with more emotion regulation difficulties are more likely to experience higher negative affect during smoking cessation attempts. Even if this increase in negative affect does not appear to be significantly related to elevations in craving or smoking, it is not beneficial for individuals' mental health. Additionally, results suggest that individuals who struggle with the acceptance of emotions may be particularly vulnerable to affect-induced craving for cigarettes. It may be beneficial for clinicians to assess for higher levels of nonacceptance to help identify individuals who may struggle more with craving during periods of negative affect. Given that previous studies have also emphasized the role of nonacceptance in smoking behavior and depression (1), nonacceptance seems to be a promising target to improve smoking cessation attempts. Future research should focus on acceptance-based treatments such as ACT for smoking cessation (83) which encourage individuals to learn how to tolerate negative affect without using cigarettes to regulate.

FUTURE DIRECTIONS

There are a number of directions for future research. First, as noted earlier, further analyses can be conducted on the current dataset to address additional questions. For example, analyses using lagged predictor variables can examine prospective between- and within-subject associations between S-DERS and negative affect/craving. For example, for between-subject prospective associations, one can examine whether *Mean* S-DERS in the pre-quit phase predicts PANAS-S scores in the post-quit phase (and vice versa). Another approach is to use measures of variability of S-DERS scores, rather than, or in addition to, mean scores, as the predictor variable. For example, in the risk taking literature, the coefficient of variability (CV) on the BART task exhibits different patterns

of associations with drinking variables (compared to mean scores) (DeMartini et al., 2014). For smoking outcomes, one can examine whether pre-quit assessments of DERS/S-DERS are associated with abstinence at end of treatment, although this analysis will have limited power given the small sample size and the small number of individuals who were able to maintain abstinence. However, there are many different outcome measures for smoking cessation studies, and some may be more related to emotion regulation than others. For example, one might expect the influence of emotion regulation to occur quite quickly, meaning that time-to-lapse, or time-to-relapse outcomes may be appropriate.

Second, future studies can make methodological changes to the study protocol to address some of the unanswered questions from the current study. For example, future research could take steps to investigate and possibly address the very low variability of S-DERS scores seen in the current study. One hypothesis for this low variability is that the typical moment to moment changes in emotion regulation abilities are not large enough to be detected by the S-DERS, or else are occurring at times when participants are not completing assessments. Alternatively, participants may not experience large changes in emotion regulation. In addition to including random assessments like the current study, a future study could ask participants to initiate an assessment at times when they are feeling upset or dysregulated. For example, Preston and colleagues (personal communication to A. Waters) have used participant-initiated "stress assessments" to gain a better understanding of the role of stressful events in drug use and relapse. The use of participant-initiated "stress" or "dysregulation" assessments would presumably capture more occasions when S-DERS scores are elevated (if indeed, participants do experience

such elevations in S-DERS) which would presumably increase variability in emotion regulation.

As another methodological change, it may be beneficial to reduce the amount of time between assessments, particularly if participants initiate an assessment at a time of distress or dysregulation. It is likely that the effects of emotion regulation, negative affect, craving, and smoking on each other may occur over short amounts of time, rather than over the timespan of hours. A review of literature did not find any studies investigating the time course of emotion regulation ability changes at this point, but other studies that have utilized the S-DERS have investigated changes after a 10-minute mood induction task (27) suggesting that emotion regulation changes are likely to occur over a span of minutes, rather than hours. A study of controlled attention and emotional responding found that reactions to unpleasant pictures were reliably detected after 160 ms and differences in passive versus directed viewing were detected after 620 ms (106). A second study, designed to examine the impact of emotion regulation strategies on emotional responding, researchers showed that emotional responses occur within seconds of the presentation of emotionally salient stimuli (224). Furthermore, the authors found that the impact of emotion regulation strategies, specifically distraction and reappraisal, impacted the emotional response in this time period of seconds, suggesting that these processes both occur very rapidly.

Third, to build on this need to hone in on moments of dysregulation, future studies could also blend naturalistic, EMA studies with laboratory interventions designed to manipulate negative affect and/or emotion regulation. Such laboratory investigations could be paired with measures of craving pre- and post-manipulation and smoking

behavior. While the laboratory portion would still create an artificial environment to study these relationships, results in the laboratory could be compared to data collected in the field to possibly support the applicability of laboratory findings to real world situations. The addition of a laboratory-based intervention mentioned above could provide an opportunity to investigate the time course of emotion regulation changes in a more molecular "minute-by-minute" manner.

Fourth, as noted earlier, one limit with the approach used in the current study is that the data are correlational. However, experimental manipulation of emotion regulation in the field could help to determine the causal relation between variables. EMA could be used to induce the use of regulation strategies in real-life situations and investigate the effect on smoking and craving in the moment. Additionally, smartphones could provide an excellent opportunity for clinicians to teach individuals how to use emotion regulation strategies in their day-to-day life. Such research and clinical implementation may eventually converge to create trainings that are delivered via smartphones as stand-alone treatment or additional support for other smoking cessation treatments (273).

Last, and more broadly, future research could also examine the course of emotion regulation as identified in the process model of emotion regulation (Figure 2). Multidimensional assessments could ask participants to report the type of situation they encountered, their affective response, the behavioral and cognitive regulation strategies they attempted, and outcomes such as change in affect or problematic behaviors (e.g. smoking or substance use). Such an investigation could provide insight into the patterns of strategy use that are associated with successful and failed cessation attempts or even momentary attempts to resist craving. In addition to strategies that have already shown

associations with smoking behavior such as suppression and reappraisal (104; 172; 245), specific behavioral strategies that are often suggested to individuals attempting to quit (e.g., eating, drinking, chewing gum, and exercising) could be investigated. Such an investigation could help to validate the real-world effectiveness of specific strategies and provide treatment targets.

Table 1. Review of Emotion Regulation Strategies and Related Research

Strategy	Definition	Effectiveness	Research
Reappraisal	Individual thinks about a situation in a way that reduces the emotional impact of the situation (98)	Effective	Reappraisal associated with reduced negative affect (87) and general emotional reactivity (103).
Acceptance	"Adoption of an intentionally open, receptive, flexible, and nonjudgmental posture with respect to moment-to-moment experience" (111)	Effective	Acceptance helps to reduce negative affect (277) and behaviors associated with poor emotion regulation such as self-harm (93)
Distraction	Focusing on or thinking about something unrelated to the emotionally salient aspect of the situation (Gross, 1998)	Effective	Effective at modulating negative emotion including depression (186; 193) and anger (81; 216)
Emotion Suppression	Attempted inhibition or dampening of an emotion that has already been triggered (99)	Possibly effective in the short term; ineffective in the long run	Effective at reducing negative affect in the short-term, but counterproductive in the long run, leading to increased negative affect after the situation has past (80).

Avoidance	Avoidance of unpleasant private experiences (emotions, thoughts, memories, etc.) and the contexts that may cause them to occur (Hayes, et al., 1996).	Possibly effective in the short term; ineffective in the long run	Avoidance of negative emotions, feelings, or situations will reduce negative affect in the short-term, but is believed to maintain problematic patterns of behavior and thinking that contribute to overall higher negative affect, particularly anxiety (5; 112).
Rumination	"A mode of responding to distress that involves repetitively and passively focusing on symptoms of distress and on the possible causes and consequences of these symptoms" (194)	Ineffective - may actually increase NA	Particularly ineffective at reducing negative affect (145; 183). High levels of rumination are associated with increased depressive symptoms (192), PTSD symptoms (60), anxiety symptoms (74), and disordered eating (152)

Table 2: Study Design and Lab Measures for Parent and Proposed Study

Week→	Scr.	-3	-2	-1	0	1	2	3	15
Day of Study		1	8	15	22	29	36	43	127
Setting	T/P	Lab	Lab	Lab	Lab	Lab	Lab	Lab	Lab
Inclusion/Exclusion	X	X							
Smoking Cessation Counseling		X	X	X	X	X	X	X	X
AR or Control Intervention		X	X	X	X	X	X	X	
Phase (Pre-/Post- Quit, Follow Up)		Pre-	Pre-	Pre-	Post-Quit	Post-	Post-	Post-	FU
LAB MEASURES									
Visual Probe task		X			X			X	X
Smoking Status		X	X	X	X	X	X	X	X
WSWS		**X**	**X**	**X**	**X**	**X**	**X**	**X**	**X**
SABQ		X	X	X	X	X	X	X	X
CES-D		X	X	X	X	X	X	X	X
Demographics	X	X							
Health History & AUDIT		X							
Tobacco History & NDSS	X	X							
REALM-SF		X							
CO & Cotinine		X	X	X	X	X	X	X	X
PANAS			**X**		**X**		**X**		
DERS			**X**		**X**		**X**		
COMPENSATION									
Study Visits		$60	$40	$40	$40	$40	$40	$40	$60
Per RA		$2	$2	$2	$2	$2	$2	$2	
Visit Duration (Min.)	20	100	60	60	70	60	60	60	70

Table Note: Key: Scr. = Screening; T/P = telephone; Week 0 = Quit Day; FU = Follow-Up; WSWS = Wisconsin Smoking Withdrawal Scale (Welsch et al., 1999); SABQ = Self-report Attentional Bias Questionnaire; CES-D = The CES-D Scale: A Self-Report Depression Scale for Research in the General Population; REALM-SF = Rapid Assessment of Adult Literacy in Medicine Short Form (Arozullah, 2007); AUDIT = Alcohol Use Disorder Identification Test; NDSS = Nicotine Dependence Syndrome Scale (Shiffman et al., 2004); PANAS=Positive and Negative Affect Scale; DERS = Difficulties in Emotion Regulation Scale; items from the S-DERS (State version) were added to EMA; CO = carbon monoxide. Primary variables in the current study are bolded. At the orientation session, if a participant was ineligible because the breath test indicates low levels of carbon monoxide, the participant was given $40 for time and travel expenses. If the participant was ineligible for another reason, he or she received $40 after completing the questionnaires.

Table 3. Demographics

		DERS	S-DERS
		N_2=29	N_2=25
Age (years)		47.41 (11.27)	48.20 (10.01)
Gender (%)			
	Male	55.2	60.0
	Female	44.8	40.0
Race (%)			
	White	17.2	12.0
	AA	62.1	72.0
	No Response	20.7	16.0
Education (years)		12.89 (1.81)	12.65 (1.70)
Income (0-10 scale)		1.96 (2.57)	1.86 (2.71)
Smoking Rate		13.14 (4.02)	12.64 (3.48)
Cotinine (ng/ml)		465.87 (254.25)	430.57 (203.88)

Table Note: Date are Mean (*SD*) (continuous variables) or % (Categorical variables). Ns as stated except for education and income where data is missing for one participant due to an error in data collection. Average income translates to between $0 to $19,999 per year. Income levels and percentage reported as follows: 0= <$10,000/year (34.5% of participants); 1=$10,000 to $19,999/year (20.7%); 2=$20,000 to $29,999/year (10.3%); 3=$30,000 to $39,999/year (6.9%); 4=$40,000 to $49,999/year (0.0%); 5=$50,000 to $59,999/year (6.9%); 6=$60,000 to $69,999/year (0.0%); 7=$70,000 to $79,999/year (6.9%); 8=$80,000 to $89,999/year (0.0%); 9=$90,000 to $99,999/year (3.4%); 10=$100,000 or more/year; 10.3% missing data.

Table 4. Summary Statistics for Lab Measures

Visit →		**Week -2**	**Week 0**	**Week +2**	**All**
Variable ↓	Scale	n_2=28	n_2=27	n_2=27	n_1=82
DERS	36-180	62.22 (17.14)	59.76 (12.37)	58.30 (11.28)	60.10 (13.79)
PANAS	1-5	1.21 (0.35)	1.22 (0.37)	1.23 (0.55)	1.22 (0.43)
WSWS-Craving	0-4	2.37 (0.72)	2.28 (1.01)	2.19 (0.98)	2.28 (.90)
Cigs Following Week	0-	10.26 (5.41)	4.46 (4.77)	3.18 (4.19)	5.96 (5.65)

Table Note: Data are Mean (*SD*). n_2=number of subjects. *n*s for variables for Visit -2, Visit 0, Visit +2 respectively are as follows: DERS, *n*s = 27, 25, 27; PANAS, *n*s = 27, 25, 27; WSWS-Craving, *n*s = 28, 27, 27; Cigs Following Week, *n*s = 21, 23, 20.

Table 5. Summary Statistics of EMA Measures

Time →		**Pre-Quit**	**Post-Quit**	**All**
		n_1=1875	n_1=2026	n_1=3901
Variable ↓		n_2=25	n_2=25	n_2=25
	Scale			
S-DERS	1-5	1.27 (0.53)	1.25 (0.44)	1.26 (0.48)
S-Nonacceptance	1-5	1.32 (0.70)	1.25 (0.48)	1.28 (0.60)
S-Modulate	1-5	1.18 (0.44)	1.22 (0.47)	1.20 (0.45)
PANAS-S	1-5	1.25 (0.54)	1.29 (0.54)	1.27 (0.54)
Craving	1-7	2.57 (1.81)	2.48 (1.69)	2.52 (1.75)
Cigarettes since last assessment	0-4	2.39 (1.46)	1.12 (1.32)	1.73 (1.53)

Table Note: Data are Mean (SD). n_1 = no. assessments; n_2 = number of subjects. Data exclude 2039 who only completed 2 EMA assessments. Only complete assessments are included.

Table 6. Results of LMMs for Hypothesis 1A

DV →					**PANAS**				
IV ↓	H	n_1	n_2	*df*	*PE*	*SE*	*F*	*p*	*95% CI*
Mean DERS	1A	79	29	13.05	0.014	0.002	37.72	<.001	0.011, 0.025
Deviation DERS	1A	79	29	20.02	0.026	0.001	7.06	.021	0.006, 0.030

Table Note: n_1 = number of visits; n_2 = number of subjects. H = Hypothesis; *PE* = parameter estimate; *SE* = standard error; *F* = *F* value from LMM. Covariates are Treatment (AR vs. Control) and Visit (categorical variable with 3 levels) (parameter estimates for covariates not shown)
Note: For 1A data show results from SAS PROC MIXED when treating coefficients for *Deviation* DERS as random. The output was accompanied by a notification that convergence criteria were met, and the "Estimated G matrix is not positive definite". When coefficients for *Deviation* DERS were treated as fixed, the results for *Deviation* DERS were as follows: F (1, 31.7) = 8.02, p = .008. 95% CI were derived using bootstrapping when treating coefficients for *Deviation* DERS as fixed.

Table 7. Results of LMMs for Hypothesis 1B

DV →				**PANAS-S**					
IV ↓	H	n_1	n_2	*df*	*PE*	*SE*	*F*	*p*	*95% CI*
Mean S-DERS	1B	3901	25	22.12	0.70	0.14	25.99	<.001	0.42, 0.99
Deviation S-DERS	1B	3901	25	12.41	0.28	0.10	7.94	.015	0.10, 0.56
Mean S-Nonacceptance	1B	3901	25	22.09	0.42	0.14	9.40	.006	0.38, 0.44
Deviation S- Nonacceptance	1B	3901	25	15.92	0.10	0.06	2.73	.118	
Mean S-Modulate	1B	3668	23	19.35	0.89	0.12	59.53	<.001	0.85, 0.93
Deviation S- Modulate	1B	3668	23	8.60	0.44	0.19	5.46	.046	0.23, 1.33

Table Note: n_1 = no. assessments; n_2 = number of subjects. H = Hypothesis; *PE* = parameter estimate; *SE* = standard error; *F* = F value from LMM. Covariates are Treatment (AR vs. Control), Assessment Type (level 1 variable with 2 levels), Day of Study (continuous) (parameter estimates for covariates not shown)
Note: Two subjects did not complete S-Modulate.

Table 8. Results of LMMs for Hypothesis 2A Craving

DV →		**WSWS_Craving**						
IV ↓	H	n_1	n_2	*df*	*PE*	*SE*	*F*	*p*
Mean DERS	2A	79	29	24.39	0.007	0.001	0.45	.509
Deviation DERS	2A	79	29	10.64	0.008	0.02	0.20	.660

Table Note: n_1 = number of visits; n_2 = number of subjects. H = Hypothesis; *PE* = parameter estimate; *SE* = standard error; *F* = *F* value from LMM. Covariates are Treatment (AR vs. Control) and Visit (categorical variable with 3 levels) (parameter estimates for covariates not shown).

Table 9. Results of LMMs for Hypothesis 2A Smoking

DV →		**Smoking**						
IV ↓	H	n_1	n_2	*df*	*PE*	*SE*	*F*	*p*
Mean DERS	2A	61	26	23.72	0.07	0.04	2.74	.132
Deviation DERS	2A	61	26	73.78	0.11	0.09	1.61	.237

Table Note: n_1 = number of visits; n_2 = number of subjects. H = Hypothesis; *PE* = parameter estimate; *SE* = standard error; *F* = F value from LMM. Covariates are Visit (categorical variable with 3 levels) and Treatment (AR vs. Control) (parameter estimates for covariates not shown).
Note: For 2A, when smoking is the DV, the table shows results from SAS PROC MIXED when treating coefficients for *Deviation* DERS as random. The output was accompanied by a notification that convergence criteria were met, and the "Estimated G matrix is not positive definite". When coefficients for *Deviation* DERS were treated as fixed, the results were as follows: *Mean* DERS: F (1, 18.9) = 0.79, p = .39; *Deviation* DERS: F (1, 31.7) = 4.53, p = .04.

Table 10. Results of LMMs for Hypothesis 2B Craving

DV →				**Craving**					
IV ↓	H	n_1	n_2	*df*	*PE*	*SE*	*F*	*p*	*95% CI*
Mean S-DERS	2B	3901	25	18.82	0.27	0.59	0.21	.656	
Deviation S-DERS	2B	3901	25	14.06	0.44	0.28	2.51	.135	
Mean S-Nonacceptance	2B	3901	25	20.39	-0.08	0.49	0.02	.878	
Deviation S-Nonacceptance	2B	3901	25	11.62	0.21	0.20	1.17	.302	
Mean S-Modulate	2B	3668	23	14.84	0.39	0.72	0.29	.595	
Deviation S-Modulate	2B	3668	23	10.55	0.59	0.31	3.53	.088	-0.77, 0.85

Table Note: n_1 = no. assessments; n_2 = number of subjects. H = Hypothesis; *PE* = parameter estimate; *SE* = standard error; *F* = F value from LMM. Covariates are Treatment (AR vs. Control), Assessment Type (level 1 variable with 2 levels), and Day in Study (continuous) (parameter estimates for covariates not shown)

Table 11. Results of LMMs for Hypothesis 2B Smoking

DV →				**Smoking**				
IV ↓	H	n_1	n_2	*df*	*PE*	*SE*	*F*	*p*
Mean S-DERS	2B	3901	25	15.71	0.47	0.35	1.79	.200
Deviation S-DERS	2B	3901	25	13.04	0.03	0.09	0.11	.742
Mean S-Nonacceptance	2B	3901	25	12.11	0.45	0.26	2.86	.116
Deviation S- Nonacceptance	2B	3901	25	10.64	0.02	0.08	0.07	.797
Mean S-Modulate	2B	3668	23	12.91	-0.005	0.42	0.00	.991
Deviation S- Modulate	2B	3668	23	11.44	0.01	0.09	0.02	.887

Table Note: n_1 = no. assessments; n_2 = number of subjects. H = Hypothesis; *PE* = parameter estimate; *SE* = standard error; *F* = F value from LMM. Covariates are Treatment (AR vs. Control), Assessment Type (level 1 variable with 2 levels), and Day in Study (continuous) (parameter estimates for covariates not shown)

Table 12. LMMs for Specific Aim 3 Negative Affect to Craving

				NA→Craving					
IV ↓	H	n_1	n_2	*df*	*PE*	*SE*	*F*	*P*	*95% CI*
DERS as Moderator									
Mean DERS x *Mean* NA	3A	82	29	25.18	-0.05	0.04	1.71	.203	
Mean DERS x *Deviation* NA	3A	79	29	40.83	-0.03	0.03	0.81	.374	
Deviation DERS x *Deviation* NA	3A	79	29	65.91	0.12	0.08	2.23	.140	
Mean S-DERS x *Mean* NA	3B	3901	25	12.58	0.30	1.70	0.03	.864	
Mean S-DERS x *Deviation* NA	3B	3901	25	8.42	0.48	0.26	3.36	.102	
Deviation S-DERS x *Deviation* NA	3B	3901	25	1126.27	-0.14	0.13	1.26	.261	
Mean S-Nonacceptance x *Mean* NA	3B	3901	25	9.96	0.18	1.85	0.01	.925	
Mean S-Nonacceptance x *Deviation* NA	3B	3901	25	8.86	0.64	0.27	5.51	.044	0.52, 1.13
Deviation S-Nonacceptance x *Deviation* NA	3B	3901	25	2233.56	-0.18	0.11	2.54	.111	
Mean S-Modulate x *Mean* NA	3B	3668	23	8.98	1.22	1.84	0.44	.525	
Mean S-Modulate x *Deviation* NA	3B	3668	23	8.04	0.40	0.30	1.76	.221	
Deviation S-Modulate x *Deviation* NA	3B	3668	23	1433.68	-0.17	0.14	1.47	.226	

Table Note: n_1 = no. of visit (3A) or assessments (3B); n_2 = number of subjects. H = Hypothesis; *PE* = parameter estimate; *SE* = standard error; *F* = F value from LMM. Covariates are Treatment, Assessment Type (Participant-initiated vs. RA) (3B), Visit (3A), and Day in Study (3B).

For 3A, none of the models converged when treating coefficients for *Deviation* NA as random. Data shown are output when coefficients for *Deviation* NA is treated as fixed. Given that no *p* values are significant, the effects would be not significant if coefficients for *Deviation* NA could be treated as random.

95% CIs derived when coefficients for *Deviation* S-Nonacceptance treated as fixed

Table 13. LMMs for Specific Aim 3 Craving to Smoking

					Craving→Smoking			
IV ↓	H	n_1	n_2	*df*	*PE*	*SE*	*F*	*p*
DERS as Moderator								
Mean DERS x *Mean* Craving	3A	61	26	16.13	-0.13	0.08	2.45	.137
Mean DERS x *Deviation* Craving	3A	61	26	27.10	-0.29	0.08	11.62	.002
Deviation DERS x *Deviation* Craving	3A	61	26	47.86	0.38	0.16	5.62	.022
Mean S-DERS x *Mean* Craving	3B	3901	25	16.07	-0.13	0.40	0.10	.760
Mean S-DERS x *Deviation* Craving	3B	3819	25	18.13	0.10	0.09	1.22	.283
Deviation S-DERS x *Deviation* Craving	3B	3819	25	2800.75	0.04	0.04	1.16	.281
Mean S-Nonacceptance x *Mean* Craving	3B	3901	25	15.59	0.06	0.31	0.04	.839
Mean S-Nonacceptance x *Deviation* Craving	3B	3819	25	19.09	0.06	0.08	0.71	.410
Deviation S-Nonacceptance x *Deviation* Craving	3B	3819	25	1632.19	0.04	0.03	1.26	.261
Mean S-Modulate x *Mean* Craving	3B	3668	23	17.21	0.40	0.73	0.30	.591
Mean S-Modulate x *Deviation* Craving	3B	3592	23	3490.78	0.06	0.05	1.46	.227
Deviation S-Modulate x *Deviation* Craving	3B	3592	23	3331.98	0.04	0.04	0.76	.382

Table Note: n_1 = no. of visit (3A) or assessments (3B); n_2 = number of subjects. H = Hypothesis; *PE* = parameter estimate; *SE* = standard error; *F* = F value from LMM. Covariates are Treatment, Assessment Type (Participant-initiated vs. RA) (3B), Visit (3A), and Day in Study (3B).

For 3A, none of the models converged when treating coefficients for *Deviation* Craving as random. Data shown are output when coefficients for *Deviation* Craving is treated as fixed.

For 3B, none of the models converged when treating coefficients for *Deviation* Craving and *Deviation* S-Modulate as random. Given that no *p* values are significant, the effects would be not significant if coefficients for *Deviation* Craving and *Deviation* S-Modulate could be treated as random.

Table 14: Results of ARIMA Analyses

Subject		**PANAS-S**			**CRAVING**			**SMOKING**		
	n	***PE***	***SE***	***t***	***PE***	***SE***	***t***	***PE***	***SE***	***t***
1043	152	0.67	0.04	**17.88***	1.32	0.21	**6.44***	0.05	0.17	0.29
1049	125	1.19	0.10	**12.00***	1.07	0.57	1.88	-0.14	0.21	-0.65
2027	75	0.17	0.02	**7.25***	1.54	0.60	2.58	-0.10	0.55	-0.19
1054	137	0.72	0.11	**6.68***	0.89	0.57	1.57	-0.05	0.29	-0.19
1053	143	0.63	0.12	**5.38***	-0.53	0.25	-2.15	-0.01	0.16	-0.03
2028	158	0.27	0.08	**3.40***	-0.01	0.19	-0.04	0.12	0.30	0.40
2037	89	0.41	0.13	**3.13***	-0.12	0.59	-0.20	-0.05	0.43	-0.13
1056	320	0.28	0.10	**2.83***	-0.07	0.16	-0.46	0.36	0.25	1.46
1055	155	0.32	0.12	**2.71***	-1.95	1.24	-1.58	-0.09	0.29	-0.31
1051	154	0.60	0.23	2.54	0.84	1.13	0.75	-0.27	0.47	-0.59
2036	179	1.71	0.83	2.06	2.00	1.77	1.13	1.28	0.46	**2.80***
1050	165	2.14	1.12	1.92	0.94	3.12	0.30	1.46	2.47	0.59
2032	179	1.38	0.88	1.57	-5.71	12.54	-0.46	-4.98	7.52	-0.66
2038	195	0.07	0.05	1.44	0.00	0.17	0.01	-0.20	0.15	-1.34
2035	187	0.05	0.12	0.42	3.08	2.92	1.05	1.80	2.29	0.78
2031	103	0.01	0.06	0.11	-0.59	0.41	-1.44	-0.43	0.19	-2.27
2034	179	0.01	0.82	0.01	1.18	1.11	1.06	0.97	2.01	0.48
1048	117	-0.01	0.09	-0.08	1.69	1.80	0.94	0.88	1.24	0.71
2040	199	0.00	0.00	-0.17	-0.35	0.10	**-3.56***	0.26	0.14	1.92
1045	182	-0.21	1.07	-0.20	-1.61	9.54	-0.17	0.03	0.18	0.19
1052	181	-0.08	0.31	-0.27	-0.35	1.47	-0.24	0.29	0.42	0.69
1046	105	-0.31	0.80	-0.38	1.10	1.75	0.63	1.13	0.42	**2.70***
1047	193	-0.24	0.44	-0.54	-4.86	3.49	-1.39	-0.56	0.46	-1.23
1042	64	-0.26	0.48	-0.54	2.25	1.94	1.16	2.03	1.84	1.10
1044	165	-0.36	0.35	-1.04	-2.49	2.89	-0.86	-1.56	1.40	-1.11

Table Note: Data are results of ARIMA analyses with PANAS-S (left side of table), Craving (middle of table) and Smoking before the next assessment (right side of table) as the dependent variables and S-DERS as the independent variable. Day was also included in all models (parameter estimate for Day not shown). *n* = number of assessments for each subject. Subjects; *PE* = Parameter Estimate for model; *SE* = Standard Error; *t* = *t* value for S-DERS; entries are ordered by the *t* value for the association between S-DERS and PANAS-S. *p < .01

Figure 1. Modal model of emotion.

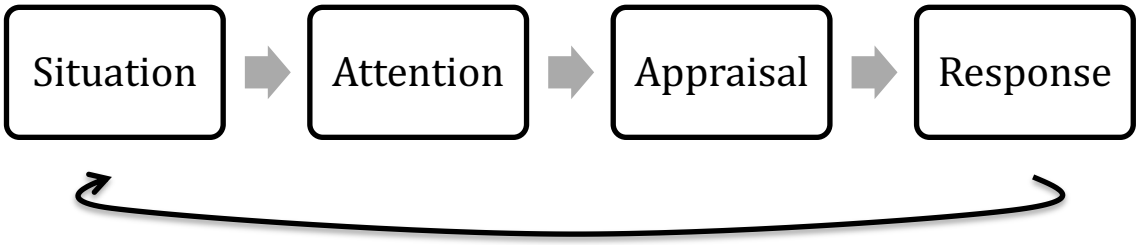


Figure 1 Note: According to the modal model of emotion, emotions are generated when a situation occurs that the individual views as being significant for his or her personal, social, and/or cultural goals. The individual pays attention to the situation due to the perceived importance, gives it a valenced meaning (positive or negative), and reacts with experiential, behavioral, and physiological responses (97).

Figure 2. The process model of emotion regulation.

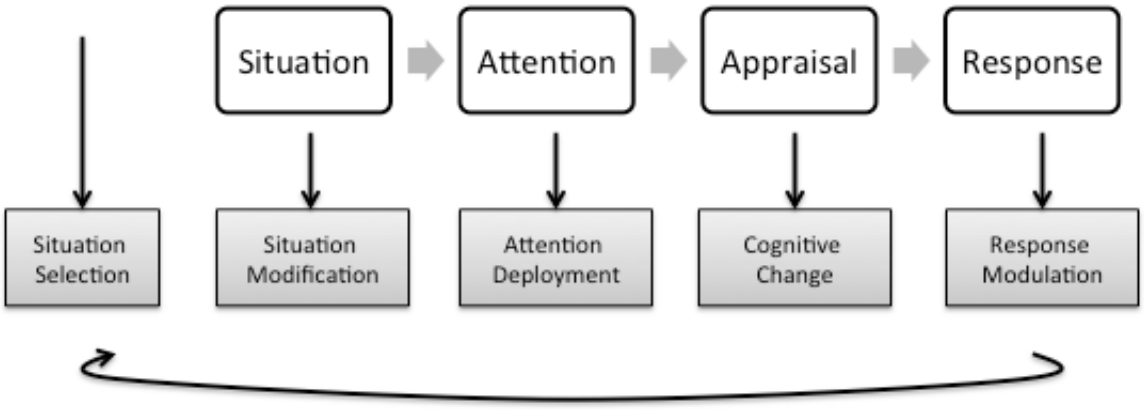


Figure 2 Note: The process model of emotion regulation (98) expands on the modal model, viewing each step in the model as a possible point to regulate emotion. *Situation selection* is the first possible intervention and involves the individual seeking out or avoiding situations based on the expected effect on emotions. If an individual finds him or herself in an uncomfortable or dysregulating situation, he or she may try to change the situation, labeled *situation modification*. When the individual cannot alter the situation, he or she may use *attentional deployment,* focusing on different, less dysregulating aspects of the situation. *Cognitive change* involves altering one's appraisal of the situation to limit or modify the emotional effects. Finally, *response modulation* refers to altering behavioral or physiological responses to the emotion.

Figure 3. Model of the Proposed Study.

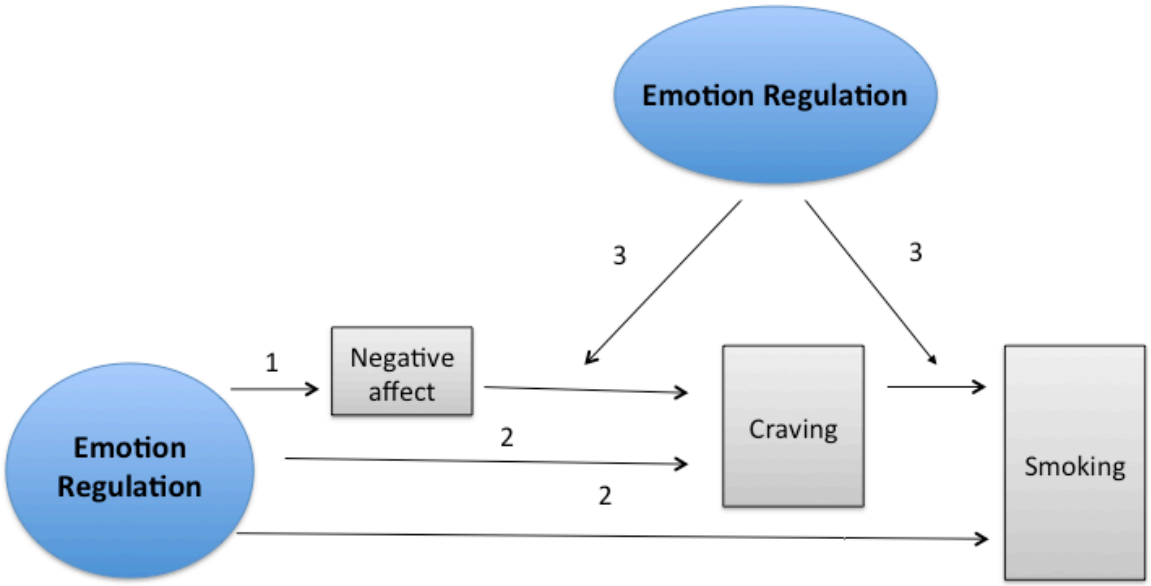


Figure 3 Note: Pathway 1 examined the association between emotion regulation (trait and state) and negative affect. Pathway 2 examined the association between emotion regulation and craving/smoking. Finally, pathway 3 examined emotion regulation, as assessed by the DERS and S-DERS, as a moderator variable.

Figure 4. Timeline of the Study.

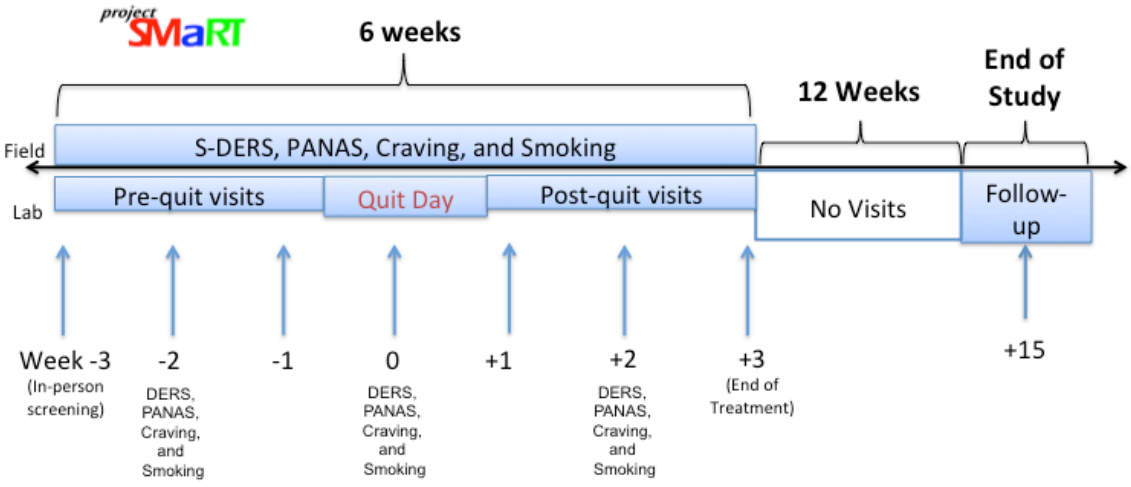


Figure 5. CONSORT Chart for Parent Study

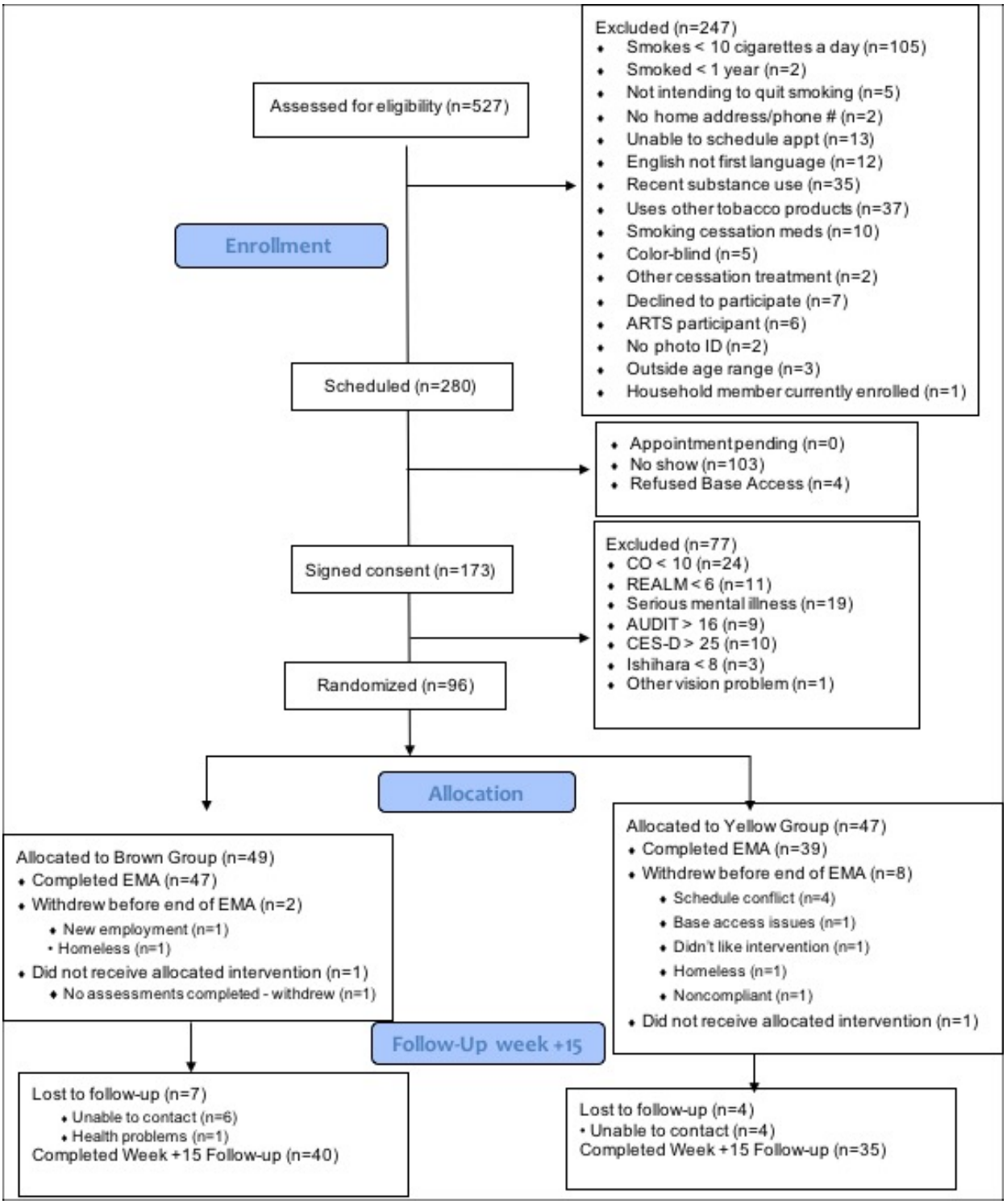
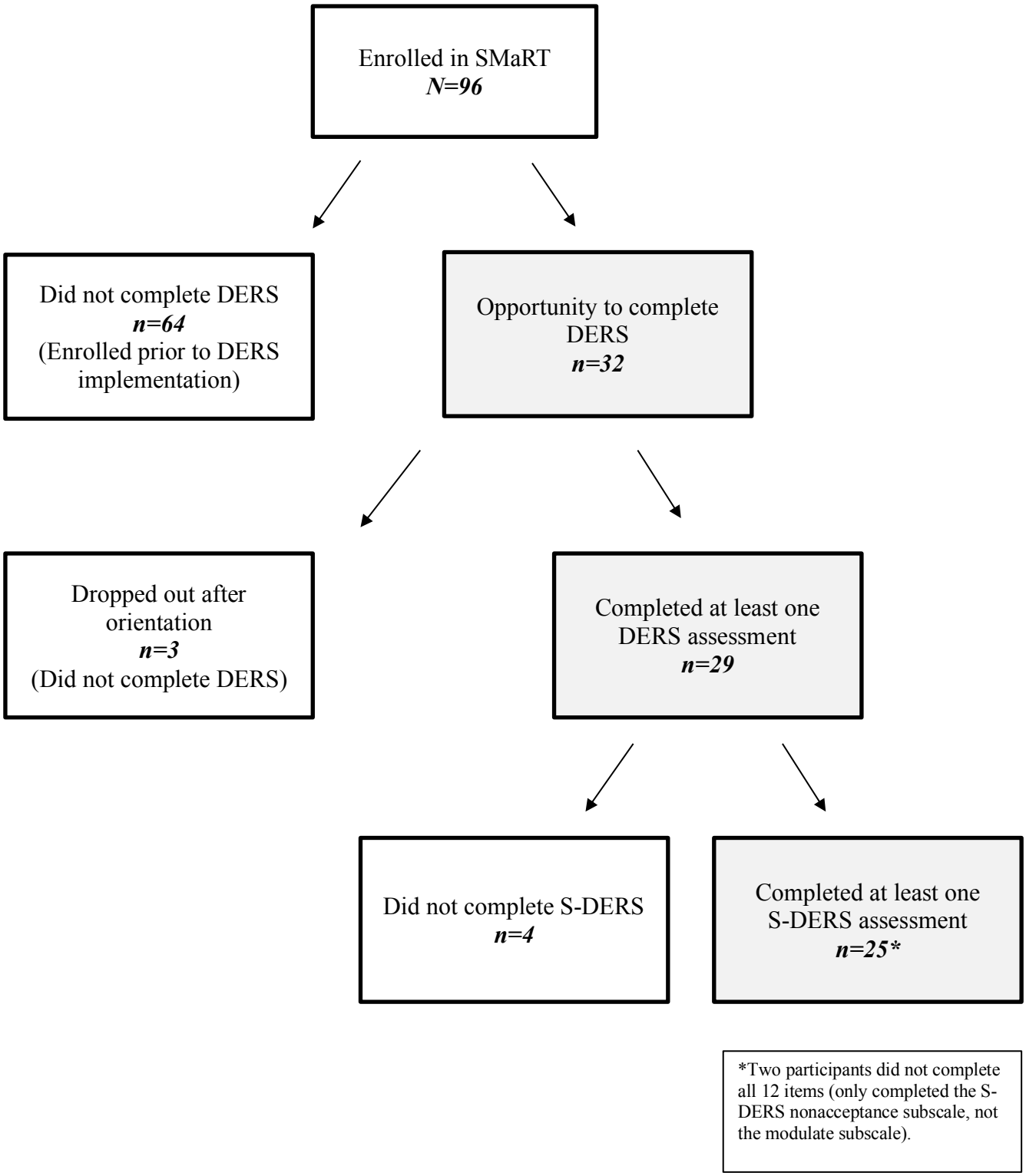


Figure 6. CONSORT Chart for Dissertation Study



Variable↓	**Visit -2[a]**	**Visit 0[b]**	**Visit +2[c]**
	***n*2**	***n*2**	***n*2**
DERS	27	25	27
PANAS	27	25	27
WSWS	28	27	27
Cigarettes Following Week	21[#]	23[#]	20[#]
	Phase 0		**Phase 1**
	***n*2**		***n*2**
S-DERS	25		25
S-Nonacceptance	25		25
S-Modulate	23*		23*

Figure Note. n_2 = Number of Participants. Of the 29 participants who completed at least 1 DERS assessment, 25 participants had the opportunity to complete the S-DERS.
[a]1 participant did not attend the Week -2 visit, and therefore did not complete the DERS, PANAS, and WSWS. Due to researcher error, 1 participant was run on the incorrect version of QDS, and did not have DERS and PANAS data at Week -2.
[b]1 participant dropped out before Week 0, and data from 1 participant on Week 0 were lost. Due to researcher error, 2 participants were run on the incorrect version of QDS, and did not have DERS and PANAS data at Week 0.
[c]2 participants dropped out before Week +2.
[#]Data for Cigarettes Following Week were missing due to participant attrition or to participant non-compliance in completing the smoking log.
*2 participants completed EMA before the S-Modulate was added to the program.

Figure 7. Results for Hypothesis 1A Mean DERS

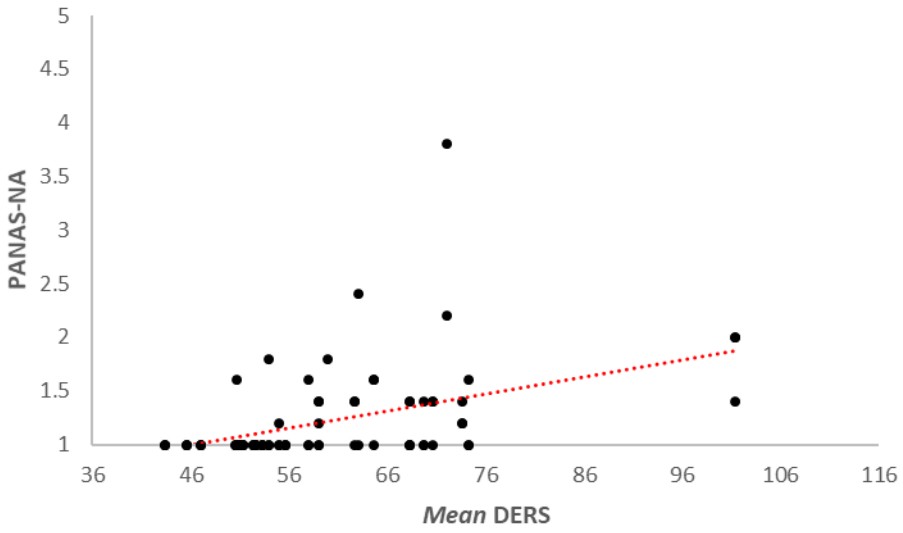


Figure 7 Note: Association between DERS and PANAS-S (1-5 scale) in the lab (Hypothesis 1A). Dots represent visits. Each participant had up to three visits (79 visits). Figure illustrates the correlation between *Mean* DERS and PANAS-S (between-subject association). For illustrative purposes, the dotted lines represent the best fit line for data presented on the graphs (rather than predicted scores implied by LMMs).

Figure 8. Results for Hypothesis 1A Deviation DERS

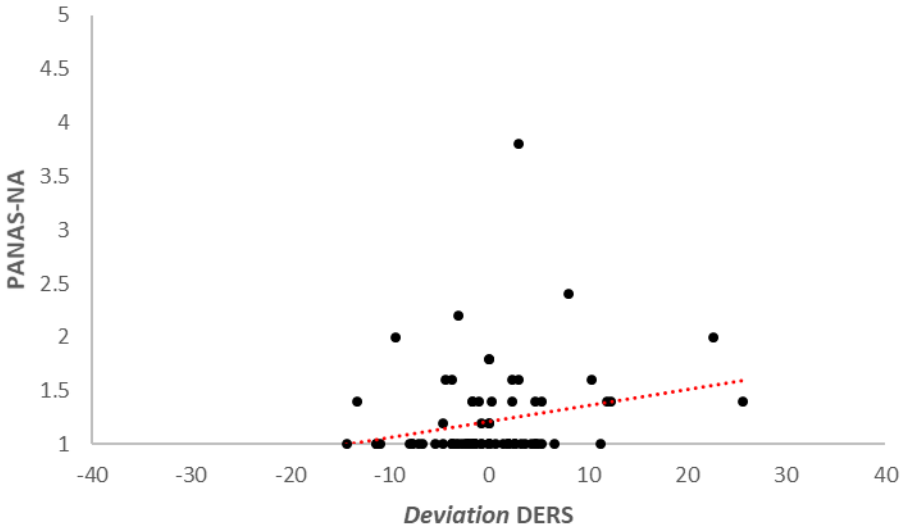


Figure 8 Note: Association between DERS and PANAS-S (1-5 scale) in the lab (Hypothesis 1A). Dots represent visits. Each participant had up to three visits (79 visits). Figure illustrates the correlation between *Deviation* DERS and PANAS-S (within-subject association). For illustrative purposes, the dotted lines represent the best fit line for data presented on the graphs (rather than predicted scores implied by LMMs).

Figure 9. Results for Hypothesis 1B Mean S-DERS

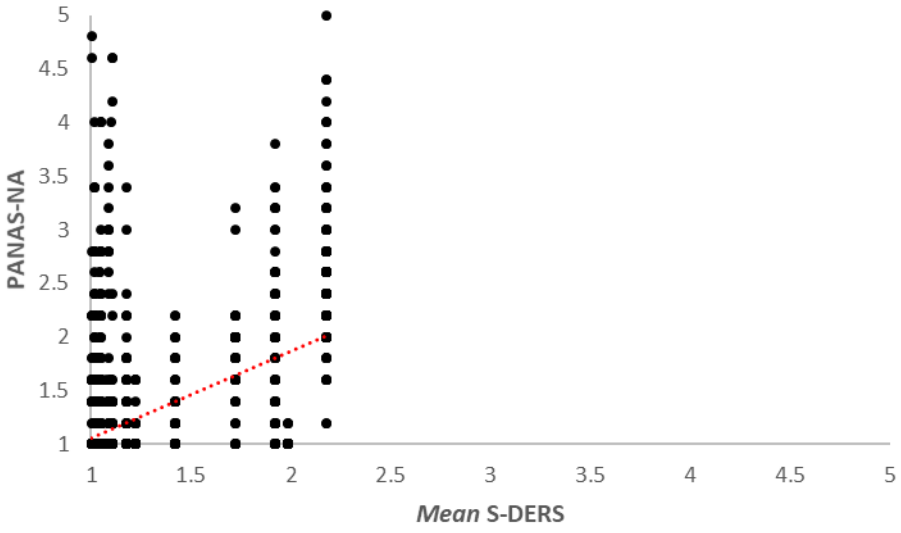


Figure 9 Note: Association between S-DERS and PANAS-S (1-5 scale) in the field (Hypothesis 1B). Dots represent EMA assessments (3901 assessments). The figure illustrates the correlation between *Mean* S-DERS and PANAS-S (between-subject association). For illustrative purposes, the dotted lines represent the best fit line for data presented on the graphs (rather than predicted scores implied by LMMs).

Figure 10. Results for Hypothesis 1B Deviation S-DERS

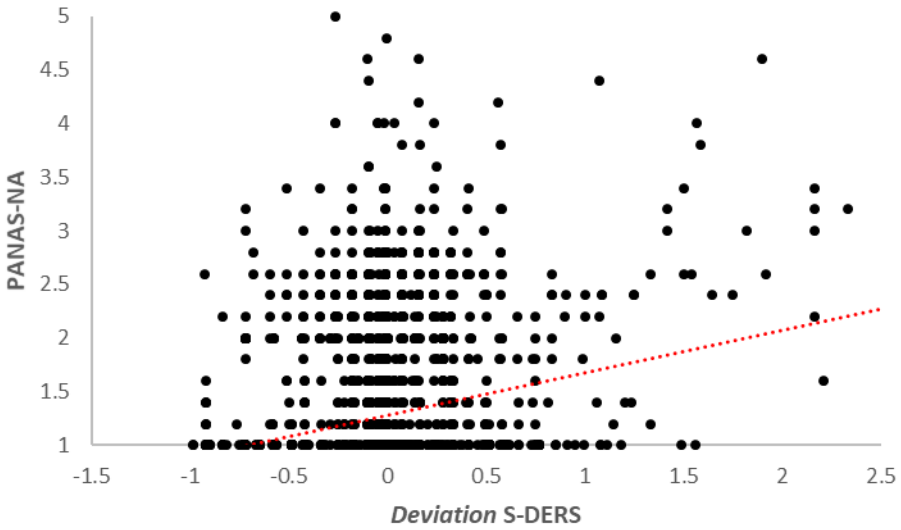


Figure 10 Note: Association between S-DERS and PANAS-S (1-5 scale) in the field (Hypothesis 1B). Dots represent EMA assessments (3901 assessments). This figure illustrates the correlation between *Deviation* S-DERS and PANAS-S (within-subject association). For illustrative purposes, the dotted lines represent the best fit line for data presented on the graphs (rather than predicted scores implied by LMMs).

Figure 11. Results for Hypothesis 3A Interaction Between Mean DERS and Deviation Craving in Prediction of Smoking

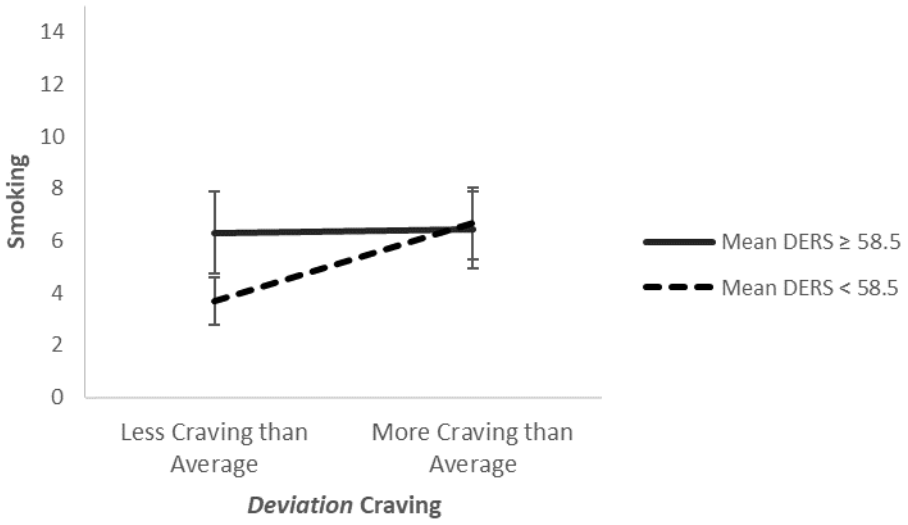


Figure 11 Note: Interaction between *Mean* DERS and *Deviation* Craving in prediction of Smoking in the lab (Hypothesis 3A). For clarity, *Mean* DERS is dichotomized as < 58.5 (less than the median *Mean* DERS) and ≥ 58.5 (greater than or equal to median *Mean* DERS). *Deviation* Craving is dichotomized as < 0 (less than subject-specific average) and ≥ 0 (greater than, or equal to, subject-specific average). Data are Mean (± 1 Standard Error) aggregated over all pertinent visits.

Figure 12. Results for Hypothesis 3A Interaction Between Deviation DERS and Deviation Craving in Prediction of Smoking

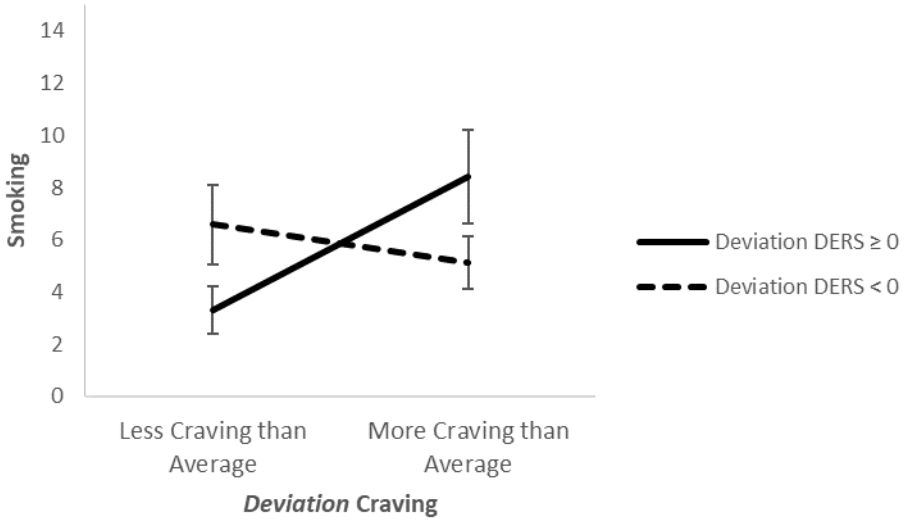


Figure 12 Note: Interaction between *Deviation* DERS and *Deviation* Craving in prediction of Smoking in the lab (Hypothesis 3A). *Deviation* DERS and *Deviation* Craving are dichotomized as < 0 (less than subject-specific average) and ≥ 0 (greater than, or equal to, subject-specific average). Data are Mean (± 1 Standard Error) aggregated over all pertinent visits.

Figure 13. Results for Hypothesis 3B Interaction Between Mean S-Nonacceptance and Deviation PANAS-NA in Prediction of Craving

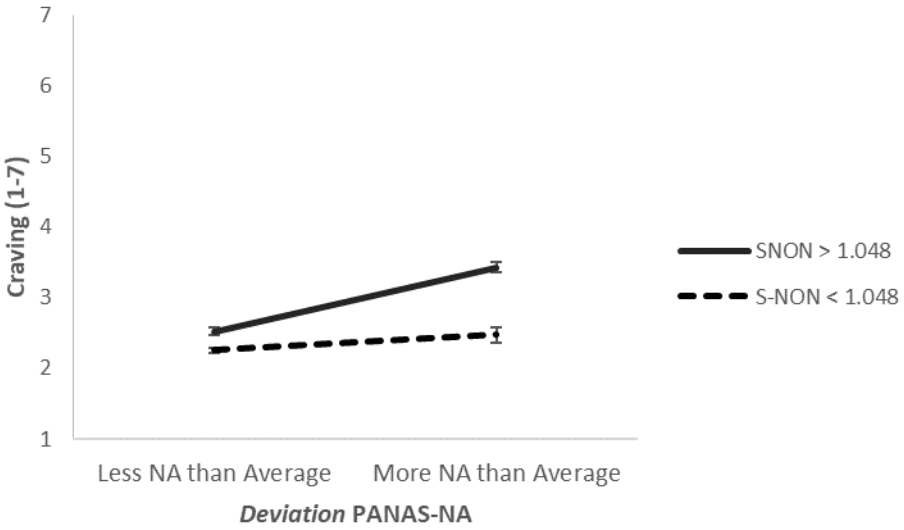


Figure 13 Note: Interaction between *Mean* S-Nonacceptance and *Deviation* PANAS-S in prediction of Craving (1-7) in the field (Hypothesis 3B). For clarity, *Mean* S-Nonacceptance is dichotomized as <1.048 (less than or equal to median *Mean* S-Nonacceptance) and >1.048 (greater than median *Mean* S-Nonacceptance). *Deviation* PANAS-S is dichotomized as < 0 (less than subject-specific average) and ≥ 0 (greater than, or equal to, subject-specific average). Data are Mean (± 1 Standard Error) aggregated over all pertinent EMA assessments.

Appendix A: Recruitment Advertisements

The above ad was placed on Washington, D.C. area metro trains and buses.

HEADING: Thinking About Quitting Smoking Cigarettes in the New Year?

Are you thinking about quitting smoking cigarettes in the New Year?
We may be able to help! Project SMaRT is now enrolling participants for a research study on smoking cessation with appointments beginning in January 2015.
You will receive smoking cessation guidance at no cost. Compensation may be provided for time and travel expenses. You must be between 18 and 65 to take part in this study.
This study takes place at the Uniformed Services University in Bethesda, MD.

If you are interested, call 301-295-1535, to speak with a researcher, or email projectsmartparticipants@usuhs.edu .
Keywords: Pay, paid, compensation, reimbursement, smoking, cigarette, Bethesda

The above wording was used for advertisements placed on local Craigslist websites.

Appendix B. Consent Form for Current Study

UNIFORMED SERVICES UNIVERSITY OF THE HEALTH SCIENCES
F. Edward Hébert School of Medicine
4301 JONES BRIDGE ROAD
BETHESDA, MARYLAND 20814-4799

This consent form is valid only if it contains the IRB stamped date. Do not sign this form if the IRB stamped date is not present or has expired.

Consent for Voluntary Participation in a Non-Clinical Research Study

1. INTRODUCTION OF THE STUDY

You are being asked to be in a research study entitled, "Attentional Retraining for Tobacco Dependence", at the Uniformed Services University (USU), Bethesda, Maryland. You have been asked to take part in this study because you are a smoker, and you want to quit smoking. Your participation is voluntary. Refusal to participate will not result in any punishment or loss of benefits to which you are otherwise permitted. Please read the information below, and ask questions about anything you do not understand, before deciding whether to take part in the study.

2. PURPOSE OF THE STUDY

The purpose of this behavioral research study is to evaluate a new method of influencing smokers' attention, cravings and smoking. Results from this study may help researchers create more effective cessation (quitting) programs in the future. If you agree to be part of the study, you will be randomly assigned to one of two training conditions. You will not know which condition you are in. This is the normal procedure in this type of study. In previous research by other investigators, the attention training has been delivered on a desktop computer in a laboratory setting. This research has shown that these two conditions can influence smokers' attention, cravings, and smoking differently. In this study, we want to see if we can deliver the training on a Smart Phone. We also want to see if the attention training can help you reduce your smoking or help you quit smoking.

3. PROCEDURES TO BE FOLLOWED

If you agree to be in this study, you will be asked to attend a total of 8 sessions in Building 28 at USUHS. At the first session, you will complete a breath test that allows the investigators to know how much you smoke. You will also complete a number of questionnaires. The questionnaires will ask about you and your health, your smoking habits, your drinking habits, and your emotional state. There will also be a brief reading test and a color vision check. You will then complete a series of computerized reaction-time tests.If you are found to be eligible and you wish to take part in this study, you will given a Smart Phone and asked to carry it around with you for 6 weeks. You will be trained to use the Smart Phone at the first visit.

The Smart Phone will beep you at random times during the day (about 4 times each day). After the Smart Phone beeps you, you will be asked to respond to a series of questions which ask you how you are feeling at that time. You will also be asked to do a brief reaction-time test. Each assessment on

the Smart Phone will last in total around 10-12 minutes.

You will be asked to attend 6 further weekly laboratory sessions at USUHS, and then a final visit 18 weeks after the first visit. At each laboratory session, you will complete a series of questionnaires and computerized evaluations. You will also be asked to complete a breath test and to provide a saliva sample. The breath test and the saliva sample will help the researchers find out how much you have smoked. At each visit, including the first, you will also be given a smoking diary and asked to record the number of cigarettes you smoke each day between visits. In total, the first laboratory visit will last around 2-2 ½ hours and each laboratory session (other than the first) will last around 60 to 70 minutes.

At each laboratory session during the study, a staff member will meet with you for 15 to 30 minutes and help you to try and quit. You will set a quit day after 3 weeks in the study. Every participant will receive the same help.

Participation in this study will be over after your final visit to USUHS, which will be 15 weeks after your quit day.

The research staff will monitor your mood during the study. If you experience significant distress or anxiety, you will be provided with mental health resources.

4. NUMBER OF PEOPLE THAT WILL TAKE PART IN THIS STUDY

Up to 250 subjects are expected to take part in this study at USUHS.

5. AMOUNT OF TIME FOR YOU TO COMPLETE THE STUDY

If you are eligible, you will be part of this study for about 18 weeks. The study will require approximately 36-40 hours of your time.

ELIGIBILITY AND PAYMENT FOR BEING IN THIS STUDY

Civilians and military personnel are eligible to take part in this study.

Non-federal civilians, federal civilians, and military personnel may be paid for participation in this study. Payments will be made after each visit.

Civilians (non-federal). You will receive $60 for completing the first session. You will also receive $40 for completing each laboratory session, except the last for which you will receive $60. You will also receive $2 for each random assessment that you complete on the Smart Phone. If you attend all laboratory sessions and complete 75% of the random assessments on the Smart Phone you will receive a total of $360 (for laboratory visit) + $252 (Smart Phone assessments) = $612. You will

receive compensation in cash at each lab visit.

Civilians (federal). You will only receive compensation for laboratory sessions and Smart Phone assessments if those sessions occur during non-duty hours. In addition, if you wish to be compensated for participation during non-duty hours, you must file a request for outside activity. If the request is approved and the sessions and Smart Phone assessments occur during non-duty hours, the payment will be same as for the non-federal civilians (as written above).

Uniformed Personnel. You will only receive compensation for laboratory sessions and Smart Phone assessments if those sessions occur during non-duty hours. In addition, if you wish to be compensated for participation during non-duty hours, you must file a request for outside activity. If the request is approved and the sessions and Smart Phone assessments occur during non-duty hours, the payment will be same as for the non-federal civilians (as written above).

Federal Civilians and Uniformed Personnel should inform their supervisors about the study for which they are volunteering whether or not they will receive compensation.

At the orientation session, if you are ineligible for the study because the breath test indicates that you have low levels of carbon monoxide in your breath, you will receive $40 for your time and travel expenses. If you are ineligible for another reason, the session will last for a longer duration and you will receive $40 for your time and travel expenses. Payments to ineligible participants follow the same rules as those stated above for the eligible participants.

7. POSSIBLE RISKS OR DISCOMFORTS FROM BEING IN THIS STUDY

The expected risks or discomforts from being in this study are expected to be minimal. There are no known risks associated with the assessments in the laboratory on the Smart Phone. When you try to quit, you may experience symptoms of nicotine withdrawal, which include restlessness, difficulty concentrating, and/or mood changes.

You may refuse to answer any question that makes you feel uncomfortable. If you have concerns after completing the questionnaires, you are encouraged to contact your doctor or the study chair.

If something in this research makes you uncomfortable or upset, you may choose to stop taking part in this research at any time without loss of benefits; you may contact the investigator for referral. If the investigators note any distress or anxiety associated with the research, you will receive referrals, if appropriate.

DO NOT USE THE SMARTPHONE WHILE DRIVING OR OPERATING A MOTOR VEHICLE. This is dangerous and may be illegal. If you ignore this safety warning and use the Smart Phone while driving, we will not be responsible for any accidents or fines resulting from this hazardous behavior.

POSSIBLE BENEFITS FROM BEING IN THIS STUDY

You may benefit from this study because if you are able to quit, this may be very beneficial to your health. Future smokers may benefit from what is learned. The information we learn may help us learn to develop better smoking cessation programs.

However, no benefit can be guaranteed.

9. CONFIDENTIALITY/PRIVACY AND HOW YOUR IDENTITY AND YOUR RESEARCH RECORDS WILL BE MAINTAINED

All information you provide as part of this study will be confidential and will be protected to the fullest extent provided by law. If you are a military member please be advised that under Federal Law a military member's confidentiality cannot be strictly guaranteed. Your responses to our laboratory and Smart Phone assessments will be maintained in a locked filing cabinet or on a password-protected computer in lab offices in the Department of Medical and Clinical Psychology. All records directly related to this study will be accessible only to those persons directly involved in conducting this study and members of the USUHS Institutional Review Board (IRB), which provide oversight for protection of human research volunteers. In addition, the IRB at USUHS and other federal agencies that help protect people who are involved in research studies may need to see the information you give us. Other than those groups, records from this study will be kept private to the fullest extent of the law. Scientific reports that come out of this study will not use your name or identify you in any way.

The breath sample you provide will allow us to measure carbon monoxide (CO) levels in your breath. This will allow us to measure how much you have smoked. We will use a standard CO monitor (Vitalograph, Lexena, KS) according to the manufacturer's instructions. Data on your CO levels will be stored on a password-protected computer in Room 113 of Building 28. The password is only known to the research staff.

The saliva samples you provide us will help us find out how much you have smoked. The saliva samples will be stored in a freezer (-80F) in Building 28 for up to three months. Batches of saliva samples will be sent to Salimetrics, Inc, LLC. (www.salimetrics.com). Salimetrics will perform an assay (a test) on each sample to determine the level of cotinine in the saliva. Cotinine is a breakdown product of nicotine and tells us how much you smoked during the past few days. No other tests will be performed on the saliva samples. Salimetrics will not be provided with your name or any other health information.

Only the study researchers will have access to the saliva samples prior to shipment. The samples are labeled with the participant study number and visit number; only the USUHS research staff will know the linkages between study numbers and participants and this information will be stored in a

locked file cabinet or on password-protected computers. Thus, confidentiality will be maintained during storage and distribution. The shipping procedures follow the U.S. Centers for Disease Control guidelines for transport of biological specimens. Once the cotinine assay is performed, Salimetrics will destroy the samples. Because you are free to drop out of the study at any time, you can request that your saliva samples be destroyed. Saliva samples will only be stored at USUHS.

10. CONDITIONS WHICH YOUR PARTICIPATION IN THIS STUDY MAY BE STOPPED WITHOUT YOUR CONSENT

The investigator may stop you from taking part in this study if being in the study is unsafe or dangerous to you. The investigator may also stop you from participating if you experience difficulty in following the procedures.

11. IF YOU DECIDE TO STOP TAKING PART IN THIS STUDY AND THE INSTRUCTIONS FOR STOPPING EARLY

You have the right to withdraw from this study at any time. If you decide to stop taking part in this study, you should tell the principal investigator as soon as possible. If you decide to withdraw from the study, you will receive compensation for all study activities completed prior to withdrawing from the study.

12. RECOURSE IN THE EVENT OF INJURY

This study should not entail any physical or mental risk beyond those described above. We do not expect complications to occur, but if, for any reason, you feel that continuing this study would constitute a hardship for you, we will end your participation in the study. If at any time you believe you have suffered an injury or illness as a result of participating in this research project, you should contact the Director of Human Research Protections Program at the Uniformed Services University of the Health Sciences, Bethesda, Maryland 20814-4799 at (301) 295-9534. This office can review the matter with you, can provide information about your rights as a subject, and may be able to identify resources available to you. If you believe the government or one of the government's employees (such as a military doctor) has injured you, a claim for damages (money) against the federal government (including the military) may be filed under the Federal Torts Claims Act. Information about judicial avenues of compensation is available from the University's General Counsel at (301) 295-3028.

13. CONTACT FOR QUESTIONS OR PROBLEMS

If you have questions about this research, you should contact Andrew J. Waters, Ph.D. the person in charge of the study. His phone number at USUHS is 301 295-9675, or you may call the study office at (301) 295-1535. Even in the evening or on weekends, you can leave a message at either number. If you have questions about your rights as a research subject, you should call the

Director of Human Research Protections Program, at USUHS at (301) 295-9534. She is your representative and has no connection to the researcher conducting this study.

SIGNATURE OF RESEARCH PARTICIPANT OR LEGAL RESPRESENTATIVE

You have read (or someone has read to you) the information in this consent form. You have been given a chance to ask questions and all of your questions have been answered to your satisfaction.

BY SIGNING THIS CONSENT FORM, YOU FREELY AGREE TO TAKE PART IN THE RESEARCH IT DESCRIBES.

_____ _____

Participant's Signature Date

_____

Participant's Printed Name

SIGNATURE OF INVESTIGATOR/RESEARCH TEAM MEMBER

You have explained the research to the participant, or his/her legal representative, and answered all of his/her questions. You believe that the volunteer subject understands the information described in this document and freely consents to participate.

_____

Investigator's/Research Team Member's Signature Date (must be the same as the participant's)

_____

Investigator's/Research Team Member's Printed Name

SIGNATURE OF WITNESS

Your signature as witness is intended to attest that the information in the consent document and any other information was explained to and apparently understood by the participant, or the participant's legal representative, that questions and concerns were addressed and that informed consent was freely given.

_____

Witness' Signature Date (must be the same as the participant's)

_____

Witness' Printed Name

Appendix C. Smartphone Instructions

project SMaRT

Smartphone Instructions

Uniformed Services University of the Health Sciences
Department of Medical and Clinical Psychology
Laboratory of Cognitive Interventions

8901 Rockville Pike
Building 28, Room 113
Bethesda, MD 20889

301-295-1535

1

Using the touchscreen

- This phone has a resistive touch screen which is different from most other touch screens.
- You need to apply pressure to get the phone to respond. This takes a little practice to get the hang of it.
- If you have trouble getting the screen to respond, you may prefer to use the stylus.

2

Stylus is stored at the back of the phone

3

When you are doing the reaction time test on the phone, you must use the hard keys. The keys on the screen will not work.

4

This is the home screen. The phone should return to this screen between assessments.

5

You can return to the home screen from any program screen by tapping Return.

6

Setting the Wake up and Bed times

You can specify the earliest and latest times that random assessments will occur.

Tap the Wake up time or Bed time button from the home screen.

7

Use the drop down arrow to change the time, then tap OK.

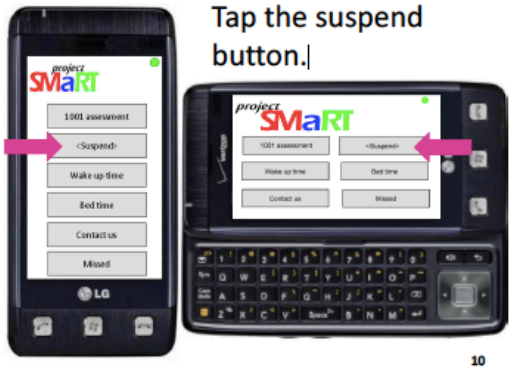
8

Using the suspend function

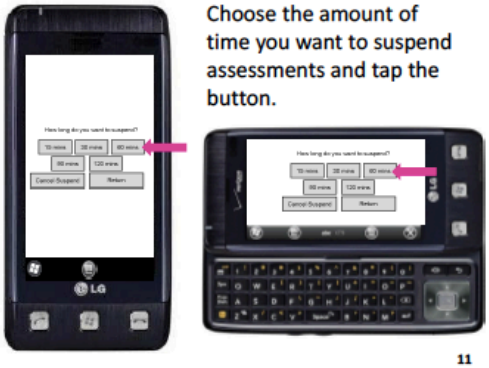
- You can suspend the phone so that it does not present assessments for a specific time period.
- Use this function if you are driving, in a meeting, or somewhere the alarm will be a disturbance.
- Please note that assessments that would have been presented during suspended function will not be rescheduled, and you will not be paid for missed assessments.
- Use the suspend function only when necessary.

9

Tap the suspend button.

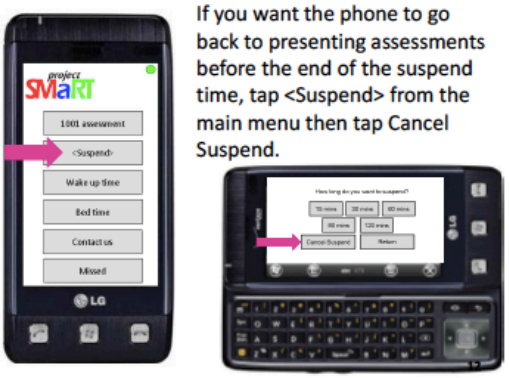


10



Choose the amount of time you want to suspend assessments and tap the button.

11



If you want the phone to go back to presenting assessments before the end of the suspend time, tap <Suspend> from the main menu then tap Cancel Suspend.

Using the delay function

- When the phone beeps you for a random assessment, you will have the choice of completing the assessment at that time, or of delaying the assessment for 5 minutes.
- You can hit the delay button up to 4 times, but after the 4th time, the assessment will be cancelled.

13

This is the screen you will see when it is time for a random assessment.

- Tap the Delay 5 minutes button to delay the assessment.
- The alert will sound again in 5 minutes.

14

Do not use the Smart phone while driving or operating any type of motor vehicle. This is dangerous and may be illegal.

It is best to put the phone in suspend mode or simply ignore the assessment alert and do a make-up assessment when you are done driving.

15

Make-up assessments

If you miss an assessment for any reason, you may complete a makeup assessment. Tap the Assessment button on the top line to Start.

16

Contact us

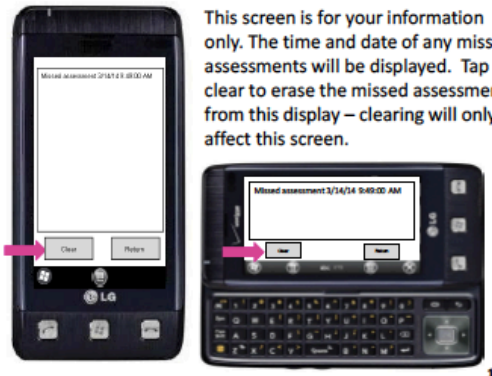
- To find our contact information, tap Contact us. Our phone number and address will be displayed.

17

Missed Assessments

- If you want to see if you have missed any assessments, Tap Missed.

18



This screen is for your information only. The time and date of any missed assessments will be displayed. Tap clear to erase the missed assessments from this display – clearing will only affect this screen.

19

Adjusting the volume

You can change the volume of the Random Assessment alert. We recommend keeping the volume as loud as possible, because the alert can be hard to hear, even at full volume

20

Charging the phone

- The phone has a very short battery life. You can expect 8-10 hours from a full charge.
- **You will need to charge the phone every day.**
- An alarm will sounds if the battery gets too low.
- If the battery gets too low, the phone will stop presenting random assessments.
- If the battery is not lasting at least 8 hours, please let us know so we can replace the battery.

21

Left edge

- There is a panel on the left edge of the phone that covers the charger.
- Gently lift and turn the panel to expose the charging port.
- Insert the charger with the mini-USB symbol or letter B facing upwards.

22

Resetting the phone

- If your phone freezes or you are not getting 4 assessments per day, you can reset the phone.
- If resetting the phone does not fix the problem, please call us at 301-295-1535 so we can help you fix the problem.

23

Left edge

- The reset button is located on the left edge of the phone, under the panel that covers the power/mini-USB port. It is the small inset button just above the mini-USB port.
- To reset the phone, press and release the reset button with the stylus. The phone will restart and it will automatically return to the program home screen.
- This process may take a few minutes.

24

Appendix D: Laboratory Self-Report Measures

DEMOGRAPHICS QUESTIONNAIRE

Q1. What is your date of birth? __ __ / __ __ / __ __ __ __
mm / dd / yyyy

Refuse to Answer

Q2. What is your gender?
1 Male
2 Female

Q3. What is your present marital status? (Choose one)

1 Single
2 Married
3 Divorced
4 Widowed
5 Living with significant other
6 Separated
8 Refuse to Answer

Q4. How many years of education have you completed? (Choose one)

01 1 (Elementary School)
02 2 (Elementary School)
03 3 (Elementary School)

04 4 (Elementary School)
05 5 (Elementary School)
06 6 (Middle School)
07 7 (Middle School)
08 8 (Middle School)
09 9 (High School)
10 10 (High School)
11 11 (High School)
12 12 (High School)
13 13 (Some College)
14 14 (Vocational or Community College Degree)
16 16 (Four Year College Degree)
17 17 (Some Postgraduate Work)
18 18 (Postgraduate Degree; Master Degree)
20 20 (Postgraduate Degree; M.D., Ph.D., DDS, Dr.P.H., etc.)
98 Refuse to Answer

Q5. Are you of Hispanic/Latino origin?
1 Yes
0 No
8 Refuse to Answer

Q6. What category best describes your race? (Choose one)

1	Anglo American/Euro American/White
2	African American/Black
3	Asian American
4	Native of Hawaii or other Pacific Islander
5	Native American or Alaska Native
6	Mixed Race
7	Other
8	Refuse to Answer

If Q6 is equal to 8 or Q6 is less than 7, then skip to Q8.

Q7. Please specify your race_ __ __ __ __ __ __

Q8. Do you receive Medicare, Medicaid, or Medical Assistance currently?

1	Yes
0	No
7	Don't Know
8	Refuse to Answer

Q9. Do you have private insurance or group insurance?

1	Yes
0	No
7	Don't Know
8	Refuse to Answer

Q10. What is your total family income per year, before taxes? (Choose one)

01	Less than $10,000 per year or less than about $833 per month
02	$10,000 to $19,999 per year or less than about $1250 per month
03	$20,000 to $29,999 per year or less than about $2083 per month
04	$30,000 to $39,999 per year or less than about $2916 per month
05	$40,000 to $49,999 per year or less than about $3750 per month
06	$50,000 to $59,999 per year or less than about $4583 per month
07	$60,000 to $69,999 per year or less than about $5416 per month
08	$70,000 to $79,999 per year or less than about $6250 per month
09	$80,000 to $89,999 per year or less than about $7083 per month
10	$90,000 to $99,999 per year or less than about $7916 per month
11	$100,000 or more per year or more than $8333 per month
98	Refuse to Answer

Q11. Generations in the U.S. Please choose the best response: (Choose one)

1 I'm an immigrant of the US
2 I was born in the US
3 One of my parents and I were born in the US (the other parent immigrated)
4 My parents and I were born in the US
5 My grandparents, my parents, and I were born in the US
6 My great-grandparents and ancestors were born in the US
8 Refuse to Answer

If Q11 is greater than 1, then skip to Q13.

Q12. What year did you immigrate to the US? __ __ __ __

Refuse to Answer

Q13. Employment Status. Please choose the best response: (Choose one)

01 Regular full-time (30 or more hours per week)
02 Regular part-time (less than 30 hours per week)
03 Unemployed, currently *looking* for work
04 Unemployed, currently *NOT looking* for work
05 Homemaker
06 Student
07 Retired
08 Unable to work or disabled
09 Other
98 Refuse to Answer

If Q13 is less than 9, then skip to Q15.

Q14. Please specify your employment status.

Q15. In the past 30 days, what was the primary source of your income? (Choose one)

1 A job
2 Unemployment Benefits
3 VA/Disability/Social Security Income
4 Welfare/Food Stamps/Aid to Family with Dependent Children
5 Alimony or Child Support
6 Spouse/partner is main source of income
8 Refuse to Answer

POSITIVE AND NEGATIVE AFFECT SCHEDULE (PANAS)

PANAS Questionnaire

This scale consists of a number of words that describe different feelings and emotions. Read each item and then list the number from the scale below next to each word. **Indicate to what extent you feel this way right now, that is, at the present moment *OR* indicate the extent you have felt this way over the past week (circle the instructions you followed when taking this measure)**

1	2	3	4	5
Very Slightly or Not at All	A Little	Moderately	Quite a Bit	Extremely

_____ 1. Interested	_____ 11. Irritable
_____ 2. Distressed	_____ 12. Alert
_____ 3. Excited	_____ 13. Ashamed
_____ 4. Upset	_____ 14. Inspired
_____ 5. Strong	_____ 15. Nervous
_____ 6. Guilty	_____ 16. Determined
_____ 7. Scared	_____ 17. Attentive
_____ 8. Hostile	_____ 18. Jittery
_____ 9. Enthusiastic	_____ 19. Active
_____ 10. Proud	_____ 20. Afraid

Scoring Instructions:

Positive Affect Score: Add the scores on items 1, 3, 5, 9, 10, 12, 14, 16, 17, and 19. Scores can range from 10 – 50, with higher scores representing higher levels of positive affect. Mean Scores: Momentary = 29.7 (*SD* = 7.9); Weekly = 33.3 (*SD* = 7.2)

Negative Affect Score: Add the scores on items 2, 4, 6, 7, 8, 11, 13, 15, 18, and 20. Scores can range from 10 – 50, with lower scores representing lower levels of negative affect. Mean Score: Momentary = 14.8 (*SD* = 5.4); Weekly = 17.4 (*SD* = 6.2)

Note: For the laboratory visits, participants were asked to report how they had felt over the last week.

DIFFICULTIES IN EMOTION REGULATION SCALE (DERS)

Response categories:

. 1 Almost never (0-10%)
. 2 Sometimes (11-35%)
. 3 About half the time (36-65%)
. 4 Most of the time (66 – 90%)
. 5 Almost always (91-100%)

1. I am clear about my feelings.
2. I pay attention to how I feel.
3. I experience my emotions as overwhelming and out of control.
4. I have no idea how I am feeling.
5. I have difficulty making sense out of my feelings.
6. I am attentive to my feelings.
7. I know exactly how I am feeling.
8. I care about what I am feeling.
9. I am confused about how I feel.
10. When I'm upset, I acknowledge my emotions.
11. When I'm upset, I become angry with myself for feeling that way.
12. When I'm upset, I become embarrassed for feeling that way.
13. When I'm upset, I have difficulty getting work done.
14. When I'm upset, I become out of control.
15. When I'm upset, I believe that I will remain that way for a long time.
16. When I'm upset, I believe that I'll end up feeling very depressed.
17. When I'm upset, I believe that my feelings are valid and important.
18. When I'm upset, I have difficulty focusing on other things.
19. When I'm upset, I feel out of control.
20. When I'm upset, I can still get things done.
21. When I'm upset, I feel ashamed with myself for feeling that way.
22. When I'm upset, I know that I can find a way to eventually feel better.
23. When I'm upset, I feel like I am weak.
24. When I'm upset, I feel like I can remain in control of my behaviors.
25. When I'm upset, I feel guilty for feeling that way.
26. When I'm upset, I have difficulty concentrating.
27. When I'm upset, I have difficulty controlling my behaviors.
28. When I'm upset, I believe there is nothing I can do to make myself feel better.
29. When I'm upset, I become irritated with myself for feeling that way.
30. When I'm upset, I start to feel very bad about myself.
31. When I'm upset, I believe that wallowing in it is all I can do.
32. When I'm upset, I lose control over my behaviors.
33. When I'm upset, I have difficulty thinking about anything else.
34. When I'm upset, I take time to figure out what I'm really feeling.
35. When I'm upset, it takes me a long time to feel better.
36. When I'm upset, my emotions feel overwhelming.

WISCONSIN SMOKING WITHDRAWAL SCALE (WSWS)

Please answer the following questions based on how you have felt or what you have noticed during the past week, including today.

0	1	2	3	4
Strongly Disagree	*Disagree*	*Feel Neutral*	*Agree*	*Strongly Agree*

1. Food is not particularly appealing to me.
   0 1 2 3 4
2. I am getting restful sleep.
   0 1 2 3 4
3. I have been tense or anxious.
   0 1 2 3 4
4. My level of concentration is excellent.
   0 1 2 3 4
5. I awaken from sleep frequently during the night.
   0 1 2 3 4
6. I have felt impatient.
   0 1 2 3 4
7. I have felt upbeat and optimistic.
   0 1 2 3 4
8. I have found myself worrying about my problems.
   0 1 2 3 4
9. I have had frequent urges to smoke.
   0 1 2 3 4
10. I have felt calm lately.
    0 1 2 3 4
11. I have been bothered by the desire to smoke a cigarette.
    0 1 2 3 4
12. I have felt sad or depressed.
    0 1 2 3 4
13. I have been irritable, easily angered.
    0 1 2 3 4
14. I want to nibble on snacks or sweets.
    0 1 2 3 4
15. I have been bothered by negative moods such as anger, frustration, and irritability.
    0 1 2 3 4
16. I have been eating a lot.
    0 1 2 3 4
17. I am satisfied with my sleep.
    0 1 2 3 4
18. I have felt frustrated.
    0 1 2 3 4
19. I have felt hopeless or discouraged.
    0 1 2 3 4
20. I have thought about smoking a lot.
    0 1 2 3 4
21. I have felt hungry.
    0 1 2 3 4
22. I feel that I am getting enough sleep.
    0 1 2 3 4
23. It is hard to pay attention to things.
    0 1 2 3 4
24. I have felt happy and content.
    0 1 2 3 4
25. My sleep has been troubled.
    0 1 2 3 4
26. I have trouble getting cigarettes off my mind.
    0 1 2 3 4
27. It has been difficult to think clearly.
    0 1 2 3 4
28. I think about food a lot.
    0 1 2 3 4

WSWS note: Items 9, 11, 20, and 26 constitute the Craving subscale and were used for the current study.

SMOKING DIARY

project SMaRT **Smoking Log**

Instructions:

- At the end of each day, indicate how many cigarettes you have smoked that day.
- Complete this record each day. Don't try to remember several days back.
- Be honest… Accurate information is important to success!

Study ID number: _____

Study week	Appointment date	Appointment time
2 weeks to quit day		
1 week to quit day		
QUIT DAY!!!		
Week 1		
Week 2		
Week 3		
Follow-up visit		

	Sun	Mon	Tues	Wed	Thurs	Fri	Sat
Week –3 Orientation							
Week –2							
Week –1							
QUIT WEEK							

	Sun	Mon	Tues	Wed	Thurs	Fri	Sat
Week +1							
Week +2							
Week +3							
Week +4							

Appendix E: EMA Measures

STATE DIFFICULTIES IN EMOTION REGULATION (S-DERS)

S-DERS

Please read each statement and indicate how much it applies to **YOUR EMOTIONS RIGHT NOW**.

1	2	3	4	5
Not at all	Somewhat	Moderately	Very much	Completely

1) I feel guilty for feeling this way.

2) I am embarrassed for feeling this way.

3) I am feeling very bad about myself.

4) I feel ashamed with myself for feeling this way.

5) I am angry with myself for feeling this way.

6) I am irritated with myself for feeling this way.

7) I feel like I'm a weak person for feeling this way.

8) I am having difficulty controlling my behaviors.

9) My emotions feel out of control.

10) I believe that I will continue feeling this way for a long time.

11) I feel out of control.

12) My emotions feel overwhelming.

POSITIVE AND NEGATIVE AFFECT SCHEDULE – SHORT FORM (PANAS-S)

Indicate to what extent you feel this way at this moment. Use the following scale to record your answers:

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

1) Scared
2) Nervous
3) Afraid
4) Upset
5) Distressed

VISUAL PROBE TASK

Note: In the visual probe (VP) task used to deliver the attention retraining intervention, participants are asked to focus on the fixation cross in the center of the smartphone screen. The cross is then replaced by a series of picture pairs (one smoking and the other neutral) that are presented relatively briefly (500 ms) with one picture on the left and the other on the right. When the picture pair disappears, a probe (the dot) is presented in a position formerly occupied by one of the pictures. Participants are asked to hit the C key if the probe appears on the left side of the screen and the M key if the probe appears on the right side of the screen.

Appendix F. Smoking Cessation Counseling Workbook (Excerpt)

Session 3 (Week -1): Tools for quitting

Date: **Study ID:**

Session goals: (1) Review motivation, (2) Develop cessation plan – triggers and craving

1. Reminder: Motivation for Quitting

2. Working together to create a plan to help you quit
    1. Quit date: _____
    2. List your usual triggers (can be physical, emotional, or situational):

Example Triggers
- Being around smokers
- Starting the day.
- Feeling stressed.
- Being in a car
- Drinking coffee or tea.
- Enjoying a meal.
- Drinking alcohol.
- Feeling bored.

    a. Prepare for triggers (work through Managing Triggers Worksheet)
    b. Reward yourself
3. What do you think quit day will be like?

4. How to handle unavoidable triggers (circle which ones will work for you and add others)

    c. Relaxation techniques (progressive muscle relaxation)
    d. Deep breathing
    e. Reciting mantra – to remind yourself of primary motivation for quitting
    f. Staying busy – (e.g., taking walks or meeting with friends in non-smoking places)
    g. Have healthy snacks on hand to replace the desire for the oral satisfaction of smoking.
    h. _____
    i. _____
    j. _____
    k. _____

5. Urges and cravings
    a. Immediately after quitting: urges to smoke may seem random, frequent, and strong
    b. The longer you stay quit: urges become less frequent and less intense; occur mainly in the presence of your triggers
    c. How have you dealt today (or in the past) with urges to smoke in situations when smoking was not allowed?

    _____

    _____

    _____

    d. Could those methods work for you as you try quitting?

6. Dealing with cravings
    a. What does craving feel like to you?

    _____

    _____

    _____

7. Circle the techniques below that will help you when you have cravings:

    h. Delay until the urge passes - usually within 3 to 5 minutes.

i. Distract yourself. Call a friend or go for a walk.
j. Drink water to fight off cravings.
k. Deep Breaths - Relax! Close your eyes and take 10 slow, deep breaths.
l. Discuss your feelings with someone close to you.
m. Add your own ideas to help with cravings:

_____

_____

_____

8. Review Handling Temptations to Smoke During Stressful Times worksheet and go over Relaxation Techniques if applicable.

9. Develop social support plan using Social Support for Nonsmoking worksheet.

10. Handouts

_____ Managing Triggers
_____ Handling Temptations /Relaxation Technique
_____ Social Support for Nonsmoking
_____ Progressive Muscle Relaxation (if applicable)
_____ Deep Breathing (if applicable)

Handout 6: Managing Triggers

MANAGING TRIGGERS FOR SMOKING WORKSHEET

How did you manage your triggers for smoking this week?

Keep a record of your triggers for smoking and the strategies you used to deal with them.	
Trigger	**Strategy**
Example: Driving to work	Chew gum, listen to relaxing music

From Abrams, Niaura, Brown, Emmons, Goldstein, & Monti (2003) The Guilford Press.

Handout 7: Handling Temptation

Handling Temptations to Smoke During Stressful Times

- Separate the cigarette from the situation. Think back to a recent stressful situation that you went through. Ask yourself what a cigarette could have done to make the situation any better.
- Step back, take a deep breath, and say to yourself, "I am in control" or "I can handle this." Then deal with the problem.
- If you become angry or upset with someone, tell yourself, "If I smoke I am only hurting myself, not that person." Remind yourself that smoking is not a good way to get back at anyone.
- Leave the room or setting if necessary.
- Distract yourself. Always have something to read or do with you. (Do a crossword puzzle, write your next shopping list, read a magazine, knit, etc.)

Relaxation Technique

Taking some time to relax each day will reduce the stress in your life. It will also give you the energy to deal with problems when they do occur. Practice this simple routine for at least fifteen minutes each day. You will be surprised at how calm and refreshed you will feel!

FIND
a quiet place where no one will bother you.

SIT
in a comfortable chair or lie on the floor.

THINK
of a sound or a short word that you can say over and over to yourself.

CLOSE
your eyes.

LET
yourself relax.

BEGIN
to breathe deeply and slowly, taking the air in through your nose and letting it out through your mouth. As you do this, say your special word over and over to yourself.

DO
this for fifteen minutes each day.

DON'T WORRY
if you have trouble clearing your mind the first few times you do this. Just like everything else, it will become easier as you practice.

Handout 8: Social Support

SOCIAL SUPPORT FOR NONSMOKING WORKSHEET

Getting support and encouragement from others while you quit and work at being a non-smoker can be very helpful. Complete this handout to help you determine what other people do that is helpful or not helpful to you and what you can do the ask them to be more helpful.

Supportive Behaviors for Nonsmoking

List behaviors from other that you consider to be helpful or supportive to your nonsmoking efforts:

1.
2.
3.
4.

Non-supportive Behaviors for Nonsmoking

List behaviors from other that you consider to be not helpful or that interfere with your efforts to quit smoking:

1.
2.
3.
4.

Requesting Behavior Changes from Others

What can you ask or request of others to have them engage in more actions or behaviors that you find supportive of nonsmoking?

1.
2.
3.
4.

What can you ask or request of others to have them engage in fewer (or eliminate completely) behaviors that you find not helpful or that interfere with your effort to quit smoking?

1.
2.
3.
4.

From Abrams, Niaura, Brown, Emmons, Goldstein, & Monti (2003) The Guilford Press.

Handout 9: Progressive Muscle Relaxation

progressive muscle relaxation

One of the body's reactions to fear and anxiety is muscle tension. This can result in feeling "tense", or can lead to muscle aches and pains, as well as leaving some people feeling exhausted. Think about how you respond to anxiety. Do you "tense up" when you're feeling anxious? Muscle relaxation can be particularly helpful in cases where anxiety is especially associated to muscle tension. This information sheet will guide you through a common form of relaxation designed to reduce muscle tension.

Muscle tension

Muscle tension is commonly associated with stress, anxiety and fear as part of a process that helps our bodies prepare for potentially dangerous situations. Even though some of those situations may not actually be dangerous, our bodies respond in the same way. Sometimes we don't even notice how our muscles become tense, but perhaps you clench your teeth slightly so your jaw feels tight, or maybe your shoulders become. Muscle tension can also be associated with backaches and tension headaches.

Progressive Muscle Relaxation

One method of reducing muscle tension that people have found helpful is through a technique called Progressive Muscle Relaxation (PMR). In progressive muscle relaxation exercises, you tense up particular muscles and then relax them, and then you practise this technique consistently.

preparing for relaxation

When you are beginning to practice progressive muscle relaxation exercises keep in mind the folloing points.

- **Physical injuries.** If you have any injuries, or a history of physical problems that may cause muscle pain, always consult your doctor before you start.
- **Select your surroundings.** Minimise the distraction to your five senses. Such as turning off the TV and radio, and using soft lighting.
- **Make yourself comfortable.** Use a chair that comfortably seats your body, including your head. Wear loose clothing, and take off your shoes.
- **Internal mechanics.** Avoid practicing after big, heavy meals, and do not practice after consuming any intoxicants, such as alcohol.

general procedure

1 Once you've set aside the time and place for relaxation, slow down your breathing and give yourself permission to relax.
2 When you are ready to begin, tense the muscle group described. Make sure you can feel the tension, but not so much that you feel a great deal of pain. Keep the muscle tensed for approximately 5 seconds.
3 Relax the muscles and keep it relaxed for approximately 10 seconds. It may be helpful to say something like "Relax" as you relax the muscle.
4 When you have finished the relaxation procedure, remain seated for a few moments allowing yourself to become alert.

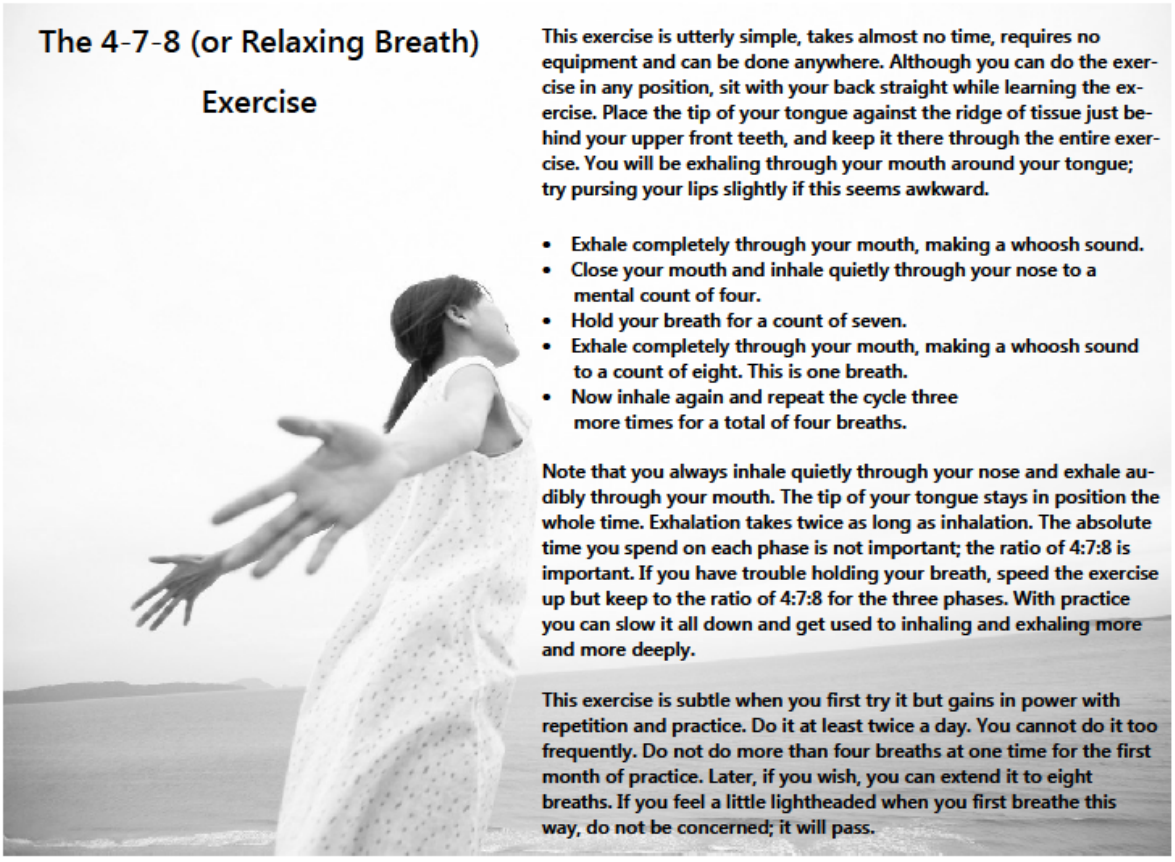
Relaxation sequence

1. **Right hand and forearm.** Make a fist with your right hand.
2. **Right upper arm.** Bring your right forearm up to your shoulder to "make a muscle".
3. **Left hand and forearm.**
4. **Left upper arm.**
5. **Forehead.** Raise your eyebrows as high as they will go, as though you were surprised by something.
6. **Eyes and cheeks.** Squeeze your eyes tight shut.
7. **Mouth and jaw.** Open your mouth as wide as you can, as you might when you're yawning.
8. **Neck.** !!! Be careful as you tense these muscles. Face forward and then pull your head back slowly, as though you are looking up to the ceiling.
9. **Shoulders.** Tense the muscles in your shoulders as you bring your shoulders up towards your ears.
10. **Shoulder blades/Back.** Push your shoulder blades back, trying to almost touch them together, so that your chest is pushed forward.
11. **Chest and stomach.** Breathe in deeply, filling up your lungs and chest with air.
12. **Hips and buttocks.** Squeeze your buttock muscles
13. **Right upper leg.** Tighten your right thigh.
14. **Right lower leg.** !!! Do this slowly and carefully to avoid cramps. Pull your toes towards you to stretch the calf muscle.
15. **Right foot.** Curl your toes downwards.
16. **Left upper leg.** Repeat as for right upper leg.
17. **Left lower leg.** Repeat as for right lower leg.
18. **Left foot.** Repeat as for right foot.

Practice means progress. Only through practice can you become more aware of your muscles, how they respond with tension, and how you can relax them. Training your body to respond differently to stress is like any training – practising consistently is the key.

Centre for Clinical Interventions

Handout 10: Deep Breathing Exercise



Appendix G. Participant Contract

project SMaRT

Participant Requirements

I, _____, agree to participate in the study "Attentional Retraining for Tobacco Dependence" (Project SMaRT). I am aware of the following conditions of my participation:

- I understand that I will not receive any medication to help me quit smoking in this study. I will not use nicotine replacement products or any other medication to help me quit smoking.
- I understand that I need to smoke normally until my quit day.
- I will attend all study visits at the USUHS campus; building 28.
- If it is necessary for me to miss any of the study visits, I will reschedule the missed appointment within a 3-5 day window.
- I understand that if I miss any appointments, a member of the staff will contact me by phone and/or mail to reschedule to visit.
- I understand that in order to receive compensation, I must be present at the study visit. **No compensation will be mailed.**
- I agree to fill out the tobacco record log at the end of each day, as a part of study requirements.
- I understand that my data is very valuable to the study, even if I am not able to quit smoking, and I will make an effort to complete the entire study. Compensation does not depend on how much or how little I smoke.
- I understand that I will be asked to provide breath and saliva samples at each study visits.
- I understand that it is important to complete the random assessments daily on the Smart Phone, and that if I miss two or more assessments on two consecutive days; I will receive a reminder call from the researchers.
- I understand that the Smart Phone needs to be charged every evening so that it will be ready to use the next day.
- **I understand that the Smart Phone is the property of the United States Government and must be returned to the researchers, even if I drop out of the study.**
- I understand that if I consistently miss more than half the assessments, I might be excluded from further participation in the study.

_____ _____

Signature **Date**

Appendix note: This participant contract was signed once final eligibility was determined during the initial laboratory visit and the individual expressed interest in participating in the study. This contract was designed to make explicit and emphasize the study commitments and expectations up front in order to maximize adherence and compliance from participants. Each participant was provided with a copy of his or her signed contract.

REFERENCES

1. Adams CE, Tull MT, Gratz KL. 2012. The role of emotional nonacceptance in the relation between depression and recent cigarette smoking. *The American Journal on Addictions* 21:293-301

2. Aguinis H, Gottfredson RK, Culpepper SA. 2013. Best-practice recommendations for estimating cross-level interaction effects using multilevel modeling. *Journal of Management* 39:1490-528

3. Aldao A. 2013. The Future of Emotion Regulation Research: Capturing Context. *Perspectives on psychological science : a journal of the Association for Psychological Science* 8:155-72

4. Aldao A, Nolen-Hoeksema S. 2012. The influence of context on the implementation of adaptive emotion regulation strategies. *Behaviour research and therapy* 50:493-501

5. Aldao A, Nolen-Hoeksema S, Schweizer S. 2010. Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical psychology review* 30:217-37

6. American Psychiatric Association. 2013. *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub

7. Anderson SM, Brunzell DH. 2012. Low dose nicotine and antagonism of beta2 subunit containing nicotinic acetylcholine receptors have similar effects on affective behavior in mice. *PloS one* 7:e48665

8. Anestis MD, Selby EA, Crosby RD, Wonderlich SA, Engel SG, Joiner TE. 2010. A comparison of retrospective self-report versus ecological momentary assessment measures of affective lability in the examination of its relationship with bulimic symptomatology. *Behaviour research and therapy* 48:607-13

9. Arbid NX. 2017. *Attentional and Emotional Consequences of Emotional Acceptance and Suppression in an Elevated Anxiety Sample*. University of Massachusetts Boston

10. Audrain-McGovern J, Benowitz NL. 2011. Cigarette Smoking, Nicotine, and Body Weight. *Clinical pharmacology and therapeutics* 90:164-8

11. Axelrod SR, Perepletchikova F, Holtzman K, Sinha R. 2011. Emotion Regulation and Substance Use Frequency in Women with Substance Dependence and Borderline Personality Disorder Receiving Dialectical Behavior Therapy. *The American journal of drug and alcohol abuse* 37:37-42

12. Baker AW, Keshaviah A, Goetter EM, Bui E, Swee M, et al. 2017. Examining the Role of Anxiety Sensitivity in Sleep Dysfunction Across Anxiety Disorders. *Behavioral Sleep Medicine* 15:216-27

13. Baker TB, Piper ME, McCarthy DE, Majeskie MR, Fiore MC. 2004. Addiction Motivation Reformulated: An Affective Processing Model of Negative Reinforcement. *Psychological review* 111:33-51

14. Baron RM, Kenny DA. 1986. The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of personality and social psychology* 51:1173

15. Baumeister RF. 2017. Addiction, cigarette smoking, and voluntary control of action: Do cigarette smokers lose their free will? *Addictive Behaviors Reports* 5:67-84 %@ 2352-8532

16. Benowitz NL. 2008. Neurobiology of nicotine addiction: implications for smoking cessation treatment. *The American journal of medicine* 121:S3-10

17. Benowitz NL. 2009. Pharmacology of Nicotine: Addiction, Smoking-Induced Disease, and Therapeutics. *Annual review of pharmacology and toxicology* 49:57-71

18. Benowitz NL, Jacob III P, Ahijevych K, Jarvis MJ, Hall S, et al. 2002. Biochemical verification of tobacco use and cessation. *Nicotine & Tobacco Research* 4

19. Berking M, Orth U, Wupperman P, Meier LL, Caspar F. 2008. Prospective effects of emotion-regulation skills on emotional adjustment. *Journal of Counseling Psychology* 55:485 %@ 1939-2168

20. Berking M, Wirtz CM, Svaldi J, Hofmann SG. 2014. Emotion regulation predicts symptoms of depression over five years. *Behaviour research and therapy* 57:13-20

21. Blackledge JT, Hayes SC. 2001. Emotion regulation in acceptance and commitment therapy. *Journal of clinical psychology* 57:243-55

22. Blanchard-Fields F, Mienaltowski A, Seay RB. 2007. Age differences in everyday problem-solving effectiveness: Older adults select more effective strategies for interpersonal problems. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 62:P61-P4

23. Blanchard-Fields F, Stein R, Watson TL. 2004. Age differences in emotion-regulation strategies in handling everyday problems. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 59:P261-P9

24. Boden M, Matthew Tyler B, Stefan W, Kateri M, Janice K. 2013. Emotion Regulation and Posttraumatic Stress Disorder: A Prospective Investigation. *Journal of social and clinical psychology* 32:296

25. Bodenhausen GV, Sheppard LA, Kramer GP. 1994. Negative affect and social judgment: The differential impact of anger and sadness. *European Journal of social psychology* 24:45-62

26. Bolger N, Laurenceau J. 2013. Intensive longitudinal methods: An introduction to diary and experience. New York, NY: Guilford

27. Borges L, Naugle A. 2015. An Experimental Examination of the Interaction between Mood Induction Task and Personality Psychopathology on State Emotion Dysregulation %M doi:10.3390/bs5010070 %U http://www.mdpi.com/2076-328X/5/1/70. *Behavioral Sciences %@ 2076-328X* 5:70

28. Bradley B, DeFife JA, Guarnaccia C, Phifer J, Fani N, et al. 2011. Emotion Dysregulation and Negative Affect: Association With Psychiatric Symptoms [CME]. *The Journal of clinical psychiatry* 72:1,478-691

29. Brandon TH, Baker TB. 1991. The Smoking Consequences Questionnaire: The subjective expected utility of smoking in college students. *Psychological Assessment: A Journal of Consulting and Clinical Psychology* 3:484

30. Brandon TH, Wetter DW, Baker TB. 1996. Affect, expectancies, urges, and smoking: Do they conform to models of drug motivation and relapse? *Experimental and Clinical Psychopharmacology* 4:29

31. Brans K, Koval P, Verduyn P, Lim YL. 2013. The Regulation of Negative and Positive Affect in Daily Life. *Emotion (Washington, D.C.)* 13:926-39

32. Breslau N. 1995. Psychiatric comorbidity of smoking and nicotine dependence. *Behavior genetics* 25:95-101

33. Brewer JA, Mallik S, Babuscio TA, Nich C, Johnson HE, et al. 2011. Mindfulness training for smoking cessation: results from a randomized controlled trial. *Drug and alcohol dependence* 119:72-80

34. Brown RA, Lejuez CW, Kahler CW, Strong DR, Zvolensky MJ. 2005. Distress tolerance and early smoking lapse. *Clinical psychology review* 25:713-33

35. Cahill K, Moher M, Lancaster T. 2008. Workplace interventions for smoking cessation. *Cochrane database of systematic reviews*:CD003440

36. Carlson CR, Hoyle RH. 1993. Efficacy of abbreviated progressive muscle relaxation training: a quantitative review of behavioral medicine research. *Journal of consulting and clinical psychology* 61:1059

37. Carmody TP, Vieten C, Astin JA. 2007. Negative affect, emotional acceptance, and smoking cessation. *Journal of Psychoactive Drugs* 39:499-508 %@ 0279-1072

38. Carstensen LL, Fung HH, Charles ST. 2003. Socioemotional selectivity theory and the regulation of emotion in the second half of life. *Motivation and emotion* 27:103-23

39. Centers for Disease Control and Prevention. 2011. Quitting Smoking Among Adults - United States, 2001-2010, Centers for Disease Control and Prevention

40. Centers for Disease Control and Prevention. 2011. Quitting smoking among adults--United States, 2001-2010. *MMWR. Morbidity and mortality weekly report* 60:1513-9

41. Centers for Disease Control and Prevention. 2015. *Current Cigarette Smoking Among Adults in the United States*. http://www.cdc.gov/tobacco/data_statistics/fact_sheets/adult_data/cig_smoking/

42. Centers for Disease Control and Prevention. 2016. Youth and Tobacco Use. ed. NCfCDPaHP Office on Smoking and Health

43. Cheetham A, Allen NB, Yücel M, Lubman DI. 2010. The role of affective dysregulation in drug addiction. *Clinical psychology review* 30:621-34

44. Clarke PB, Reuben M. 1996. Release of [3H]-noradrenaline from rat hippocampal synaptosomes by nicotine: mediation by different nicotinic receptor subtypes from striatal [3H]-dopamine release. *British journal of pharmacology* 117:595-606

45. Coe JW, Brooks PR, Vetelino MG, Wirtz MC, Arnold EP, et al. 2005. Varenicline: an alpha4beta2 nicotinic receptor partial agonist for smoking cessation. *Journal of medicinal chemistry* 48:3474-7

46. Cohen LM, McCarthy DM, Brown SA, Myers MG. 2002. Negative affect combines with smoking outcome expectancies to predict smoking behavior over time. *Psychology of addictive behaviors* 16:91

47. Cohen S, Tyrrell DA, Smith AP. 1993. Negative life events, perceived stress, negative affect, and susceptibility to the common cold. *Journal of personality and social psychology* 64:131

48. Conner TS, Tennen H, Fleeson W, Barrett LF. 2009. Experience sampling methods: A modern idiographic approach to personality research. *Social and personality psychology compass* 3:292-313

49. Consedine NS, Magai C, Bonanno GA. 2002. Moderators of the emotion inhibition-health relationship: A review and research agenda. *Review of General Psychology* 6:204

50. Copeland AL, Brandon TH, Quinn EP. 1995. The Smoking Consequences Questionnaire-Adult: Measurement of smoking outcome expectancies of experienced smokers. *Psychological Assessment* 7:484

51. Cox LS, Tiffany ST, Christen AG. 2001. Evaluation of the brief questionnaire of smoking urges (QSU-brief) in laboratory and clinical settings. *Nicotine & tobacco research : official journal of the Society for Research on Nicotine and Tobacco* 3:7-16

52. D'Avanzato C, Joormann J, Siemer M, Gotlib IH. 2013. Emotion regulation in depression and anxiety: examining diagnostic specificity and stability of strategy use. *Cognitive Therapy and Research* 37:968-80 %@ 0147-5916

53. De Groot F, Morrens M, Dom G. 2014. [Acceptance and commitment therapy (ACT) and addiction: a literature review]. *Tijdschrift voor psychiatrie* 56:577-85

54. Decker MW, Brioni JD, Bannon AW, Arneric SP. 1995. Diversity of neuronal nicotinic acetylcholine receptors: Lessons from behavior and implications for cns therapeutics. *Life Sciences* 56:545-70

55. Demaree HA, Robinson JL, Pu J, Allen JJ. 2006. Strategies actually employed during response-focused emotion regulation research: Affective and physiological consequences. *Cognition and Emotion* 20:1248-60

56. DiFranza JR, Aligne CA, Weitzman M. 2004. Prenatal and postnatal environmental tobacco smoke exposure and children's health. *Pediatrics* 113:1007-15

57. Dimeff LA, Linehan MM. 2008. Dialectical behavior therapy for substance abusers. *Addict Sci Clin Pract* 4:39-47

58. Doll R, Hill AB. 1956. Lung cancer and other causes of death in relation to smoking. *British medical journal* 2:1071

59. Doll R, Peto R, Boreham J, Sutherland I. 2004. Mortality in relation to smoking: 50 years' observations on male British doctors. *BMJ (Clinical research ed.)* 328:1519

60. Ehlers T, Ehring T, Ehlers A. 2014. Does rumination mediate the relationship between emotion regulation ability and posttraumatic stress disorder? *European journal of psychotraumatology* 5:508

61. Eisenberg MJ, Filion KB, Yavin D, Bélisle P, Mottillo S, et al. 2008. Pharmacotherapies for smoking cessation: a meta-analysis of randomized controlled trials. *Canadian Medical Association Journal* 179:135-44

62. Elwafi HM, Witkiewitz K, Mallik S, Thornhill IV TA, Brewer JA. 2013. Mindfulness training for smoking cessation: Moderation of the relationship between craving and cigarette use. *Drug and alcohol dependence* 130:222-9

63. Faul F, Erdfelder E, Lang AG, Buchner A. 2007. G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behav Res Methods* 39:175-91

64. Field A. 2013. *Discovering Statistics Using IBM SPSS Statistics*. SAGE Publications

65. Field M, Duka T, Eastwood B, Child R, Santarcangelo M, Gayton M. 2007. Experimental manipulation of attentional biases in heavy drinkers: do the effects generalise? *Psychopharmacology* 192:593-608

66. Fiore MC, Jaen CR, Baker TB, Bailey WC, Benowitz NL, et al. 2008. Treating Tobacco Use and Dependence: 2008 Update. ed. USDoHaH Services. Rockville, MD: Public Health Service

67. Flett GL, Blankstein KR, Obertynski M. 1996. Affect intensity, coping styles, mood regulation expectancies, and depressive symptoms. *Personality and individual differences* 20:221-8

68. Folkman S, Lazarus RS. 1985. If it changes it must be a process: study of emotion and coping during three stages of a college examination. *J Pers Soc Psychol* 48:150-70

69. Forgas JP. 1991. Affect and social judgments: An introductory review. In *Emotion and social judgments*, ed. JP Forgas:3-30: Psychology Press. Number of 3-30 pp.

70. Forys K, McKellar J, Moos R. 2007. Participation in specific treatment components predicts alcohol-specific and general coping skills. *Addictive behaviors* 32:1669-80

71. Fox HC, Axelrod SR, Paliwal P, Sleeper J, Sinha R. 2007. Difficulties in emotion regulation and impulse control during cocaine abstinence. *Drug and alcohol dependence* 89:298-301

72. Fox HC, Hong KA, Sinha R. 2008. Difficulties in emotion regulation and impulse control in recently abstinent alcoholics compared with social drinkers. *Addictive behaviors* 33:388-94

73. Freedman ND, Silverman DT, Hollenbeck AR, Schatzkin A, Abnet CC. 2011. Association between smoking and risk of bladder cancer among men and women. *Jama* 306:737-45

74. Fresco DM, Frankel AN, Mennin DS, Turk CL, Heimberg RG. 2002. Distinct and Overlapping Features of Rumination and Worry: The Relationship of Cognitive Production to Negative Affective States. *Cognitive Therapy and Research* 26:179-88

75. Freud A. 1937. *The Ego and the Mechanisms of Defencs*. pp 42. London: Hogarth

76. Fuchs CS, Colditz GA, Stampfer MJ, Giovannucci EL, Hunter DJ, et al. 1996. A prospective study of cigarette smoking and the risk of pancreatic cancer. *Archives of Internal Medicine* 156:2255-60

77. Fucito LM, Juliano LM, Toll BA. 2010. Cognitive reappraisal and expressive suppression emotion regulation strategies in cigarette smokers. *Nicotine & Tobacco Research*

78. Garber J, Dodge KA, eds. 1991. *The development of emotion regulation and dysregulation*. New York: Cambridge University Press.

79. Garnefski N, Kraaij V. 2007. The cognitive emotion regulation questionnaire. *European Journal of Psychological Assessment* 23:141-9

80. Geraerts E, Merckelbach H, Jelicic M, Smeets E. 2006. Long term consequences of suppression of intrusive anxious thoughts and repressive coping. *Behaviour research and therapy* 44:1451-60

81. Gerin W, Davidson KW, Christenfeld NJ, Goyal T, Schwartz JE. 2006. The role of angry rumination and distraction in blood pressure recovery from emotional arousal. *Psychosomatic medicine* 68:64-72

82. Gianini LM, White MA, Masheb RM. 2013. Eating pathology, emotion regulation, and emotional overeating in obese adults with binge eating disorder. *Eating behaviors : an international journal* 14:309-13

83. Gifford EV, Kohlenberg BS, Hayes SC, Antonuccio DO, Piasecki MM, et al. 2004. Acceptance-based treatment for smoking cessation. *Behavior therapy* 35:689-705

84. Gilbert DG. 1997. The situation x trait adaptive response (STAR) model of drug use, effects, and craving. *Human Psychopharmacology-Clinical and Experimental* 12:S89

85. Glassman AH, Helzer JE, Covey LS, Cottler LB, Stetner F, et al. 1990. Smoking, smoking cessation, and major depression. *Jama* 264:1546-9

86. Glautier S. 2004. Measures and models of nicotine dependence: positive reinforcement. *Addiction (Abingdon, England)* 99:30-50

87. Goldin PR, McRae K, Ramel W, Gross JJ. 2008. The Neural Bases of Emotion Regulation: Reappraisal and Suppression of Negative Emotion. *Biological Psychiatry* 63:577-86

88. Goldin PR, McRae K, Ramel W, Gross JJ. 2008. The neural bases of emotion regulation: reappraisal and suppression of negative emotion. *Biol Psychiatry* 63:577-86

89. Gonzales D, Rennard SI, Nides M, Oncken C, Azoulay S, et al. 2006. Varenicline, an $\alpha 4 \beta 2$ nicotinic acetylcholine receptor partial agonist, vs

sustained-release bupropion and placebo for smoking cessation: a randomized controlled trial. *Jama* 296:47-55

90. Goodman M, Carpenter D, Tang CY, Goldstein KE, Avedon J, et al. 2014. Dialectical behavior therapy alters emotion regulation and amygdala activity in patients with borderline personality disorder. *Journal of psychiatric research* 57:108-16

91. Goodnight JRM. 2016. Returning to presence: the effects of mindfulness on emotion regulation following worry among individuals with analogue generalized anxiety disorder.

92. Gratz KL. 2015. E-mail communication. ed. JM MacIntyre

93. Gratz KL, Gunderson JG. 2006. Preliminary Data on an Acceptance-Based Emotion Regulation Group Intervention for Deliberate Self-Harm Among Women With Borderline Personality Disorder. *Behavior Therapy* 37:25-35

94. Gratz KL, Roemer L. 2004. Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment* 26:41-54

95. Gratz KL, Tull MT. 2010. Emotion regulation as a mechanism of change in acceptance- and mindfulness-based treatments. In *Assessing mindfulness and acceptance processes in clients: Illuminating the theory and practice of change.*:107-33. Oakland, CA, US: Context Press/New Harbinger Publications. Number of 107-33 pp.

96. Greenberg LS. 2004. Emotion–focused therapy. *Clinical Psychology & Psychotherapy* 11:3-16

97. Gross JJ. 1998. Antecedent-and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *Journal of personality and social psychology* 74:224

98. Gross JJ. 1998. The emerging field of emotion regulation: an integrative review. *Review of general psychology* 2:271

99. Gross JJ. 2013. *Handbook of emotion regulation*. Guilford publications

100. Gross JJ. 2015. Emotion Regulation: Current Status and Future Prospects. *Psychological inquiry* 26:1-26

101. Gross JJ, John OP. 2003. Individual differences in two emotion regulation processes: implications for affect, relationships, and well-being. *J Pers Soc Psychol* 85:348-62

102. Gross JJ, Thompson RA. 2007. Emotion regulation: Conceptual foundations. In *Handbook of emotion regulation*, ed. JJ Gross:3-24. New York, NY: Guilford Press. Number of 3-24 pp.

103. Gruber J, Hay AC, Gross JJ. 2014. Rethinking Emotion: Cognitive Reappraisal is an Effective Positive and Negative Emotion Regulation Strategy in Bipolar Disorder. *Emotion (Washington, D.C.)* 14:388-96

104. Haaga DA, Allison ML. 1994. Thought suppression and smoking relapse: A secondary analysis of Haaga (1989). *British Journal of Clinical Psychology* 33:327-31

105. Hahn B. 2015. Nicotinic receptors and attention. *Current topics in behavioral neurosciences* 23:103-35

106. Hajcak G, Dunning JP, Foti D. 2009. Motivated and controlled attention to emotion: Time-course of the late positive potential. *Clinical Neurophysiology* 120:505-10

107. Harrison A, Sullivan S, Tchanturia K, Treasure J. 2010. Emotional functioning in eating disorders: attentional bias, emotion recognition and emotion regulation. *Psychological medicine* 40:1887-97

108. Harrop JW, Velicer WF. 1985. A comparison of alternative approaches to the analysis of interrupted time-series. *Multivariate behavioral research* 20:27-44

109. Hatsukami DK, Hughes JR, Pickens RW, Svikis D. 1984. Tobacco withdrawal symptoms: an experimental analysis. *Psychopharmacology* 84:231-6

110. Hayes SC, Luoma JB, Bond FW, Masuda A, Lillis J. 2006. Acceptance and commitment therapy: Model, processes and outcomes. *Behaviour research and therapy* 44:1-25

111. Hayes SC, Strosahl KD, Wilson KG. 1999. *Acceptance and commitment therapy: An experiential approach to behavior change*. Guilford Press

112. Hayes SC, Wilson KG, Gifford EV, Follette VM, Strosahl K. 1996. Experiential avoidance and behavioral disorders: A functional dimensional approach to diagnosis and treatment. *Journal of consulting and clinical psychology* 64:1152

113. Heckman BW, Carpenter MJ, Correa JB, Wray JM, Saladin ME, et al. 2015. Effects of experimental negative affect manipulations on ad libitum smoking: a meta‐analysis. *Addiction (Abingdon, England)* 110:751-60

114. Heckman BW, Kovacs MA, Marquinez NS, Meltzer LR, Tsambarlis ME, et al. 2013. Influence of affective manipulations on cigarette craving: a meta-analysis. *Addiction (Abingdon, England)* 108:2068-78

115. Hedeker D, Demirtas H, Mermelstein RJ. 2009. A mixed ordinal location scale model for analysis of Ecological Momentary Assessment (EMA) data. *Statistics and its interface* 2:391

116. Hedeker D, Gibbons RD. 2006. *Longitudinal data analysis*. John Wiley & Sons

117. Hedeker D, Mermelstein RJ, Berbaum ML, Campbell RT. 2009. Modeling mood variation associated with smoking: an application of a heterogeneous mixed-effects model for analysis of ecological momentary assessment (EMA) data. *Addiction (Abingdon, England)* 104:297-307

118. Herbert M, Foulds J, Fife‐Schaw C. 2001. No effect of cigarette smoking on attention or mood in non‐deprived smokers. *Addiction (Abingdon, England)* 96:1349-56

119. Hoeppner BB, Goodwin MS, Velicer WF, Mooney ME, Hatsukami DK. 2008. Detecting longitudinal patterns of daily smoking following drastic cigarette reduction. *Addictive behaviors* 33:623-39

120. Hughes JR. 2007. Effects of abstinence from tobacco: valid symptoms and time course. *Nicotine & Tobacco Research* 9:315-27

121. Hughes JR, Gust SW, Skoog K, Keenan RM, Fenwick JW. 1991. Symptoms of tobacco withdrawal. A replication and extension. *Archives of general psychiatry* 48:52-9

122. Hughes JR, Hatsukami D. 1986. Signs and symptoms of tobacco withdrawal. *Archives of general psychiatry* 43:289-94

123. Hughes JR, Higgins ST, Hatsukami D. 1990. Effects of abstinence from tobacco. In *Research advances in alcohol and drug problems*:317-98 %@ 1489916717: Springer. Number of 317-98 %@ 1489916717 pp.

124. Hurt RD, Sachs DPL, Glover ED, Offord KP, Johnston JA, et al. 1997. A Comparison of Sustained-Release Bupropion and Placebo for Smoking Cessation. *New England Journal of Medicine* 337:1195-202

125. Hyman SE. 2005. Addiction: a disease of learning and memory. *American Journal of Psychiatry* 162:1414-22

126. Isen AM. 1987. Positive affect, cognitive processes, and social behavior. *Advances in experimental social psychology* 20:203-53

127. Jackson DC, Malmstadt JR, Larson CL, Davidson RJ. 2000. Suppression and enhancement of emotional responses to unpleasant pictures. *Psychophysiology* 37:515-22

128. Jahng S, Wood PK, Trull TJ. 2008. Analysis of affective instability in ecological momentary assessment: Indices using successive difference and group comparison via multilevel modeling. *Psychological methods* 13:354-75

129. Janfaza M. 2015. The predictive role of difficulties in emotion regulation and self-control with susceptibility to addiction in drug-dependent individuals. *Research on Addiction Quarterly Journal of Drug Abuse* 9:45-54

130. Jindal S, Gupta D. 2004. The relationship between tobacco smoke & bronchial asthma. *Indian Journal of Medical Research* 120:443

131. John OP, Gross JJ. 2004. Healthy and unhealthy emotion regulation: Personality processes, individual differences, and life span development. *Journal of personality* 72:1301-34 %@ 467-6494

132. Jorenby DE, Hays JT, Rigotti NA, Azoulay S, Watsky EJ, et al. 2006. Efficacy of varenicline, an $\alpha 4 \beta 2$ nicotinic acetylcholine receptor partial agonist, vs placebo or sustained-release bupropion for smoking cessation: a randomized controlled trial. *Jama* 296:56-63

133. Kannel WB, Doyle JT, McNamara PM, Quickenton P, Gordon T. 1975. Precursors of sudden coronary death. Factors related to the incidence of sudden death. *Circulation* 51:606-13

134. Kassel JD, Evatt DP, Greenstein JE, Wardle MC, Yates MC, Veilleux JC. 2007. The acute effects of nicotine on positive and negative affect in adolescent smokers. *Journal of abnormal psychology* 116:543-53

135. Kassel JD, Shiffman S. 1997. Attentional mediation of cigarette smoking's effect on anxiety. *Health Psychology* 16:359

136. Kassel JD, Stroud LR, Paronis CA. 2003. Smoking, stress, and negative affect: correlation, causation, and context across stages of smoking. *Psychological bulletin* 129:270

137. Kassel JD, Unrod M. 2000. Smoking, anxiety, and attention: support for the role of nicotine in attentionally mediated anxiolysis. *Journal of abnormal psychology* 109:161-6

138. Kavanagh DJ. 1992. Self-efficacy and depression. In *Self-efficacy: Thought Control of Action*, ed. R Schwarzer:177-93. New York, NY: Routledge. Number of 177-93 pp.

139. Kavanagh DJ, Andrade J, May J. 2005. Imaginary relish and exquisite torture: the elaborated intrusion theory of desire. *Psychological review* 112:446

140. Kavanagh DJ, Bower GH. 1985. Mood and self-efficacy: Impact of joy and sadness on perceived capabilities. *Cognitive Therapy and Research* 9:507-25

141. Keenan NKX. 2013. *Emotion regulation and health behavior: effects of negative affect and emotion regulation strategies on eating and smoking*. University of Minnesota

142. Kenny DA, Judd CM. 2014. Power Anomalies in Testing Mediation. *Psychological Science* 25:334-9

143. Keuthen NJ, Rothbaum BO, Welch SS, Taylor C, Falkenstein M, et al. 2010. Pilot trial of dialectical behavior therapy-enhanced habit reversal for trichotillomania. *Depression and anxiety* 27:953-9

144. Khantzian EJ. 1985. The self-medication hypothesis of addictive disorders: focus on heroin and cocaine dependence. *American journal of Psychiatry* 142:1259-64

145. Kirkegaard Thomsen D. 2006. The association between rumination and negative affect: A review. *Cognition and Emotion* 20:1216-35

146. Kober H. 2013. Emotion Regulation in Substance Use Disorders. In *Handbook of Emotion Regulation*, ed. JJ Gross. New York, NY: The Guilford Press. Number of.

147. Kober H, Kross EF, Mischel W, Hart CL, Ochsner KN. 2010. Regulation of craving by cognitive strategies in cigarette smokers. *Drug and alcohol dependence* 106:52-5

148. Koob GF, Le Moal M. 2001. Drug addiction, dysregulation of reward, and allostasis. *Neuropsychopharmacology* 24:97-129

149. Koole SL. 2009. The psychology of emotion regulation: An integrative review. *Cognition and Emotion* 23:4-41

150. Koval P, Kuppens P. 2012. Changing emotion dynamics: individual differences in the effect of anticipatory social stress on emotional inertia. *Emotion (Washington, D.C.)* 12:256

151. Kreft I, de Leeuw J. 1998. *Introducing multilevel modeling*. Sage

152. Kubiak T, Vogele C, Siering M, Schiel R, Weber H. 2008. Daily hassles and emotional eating in obese adolescents under restricted dietary conditions--the role of ruminative thinking. *Appetite* 51:206-9

153. Kuppens P, Verduyn P. 2011. Looking at Emotion Regulation Through the Window of Emotion Dynamics. *Psychological inquiry* 26:72-9

154. Lancaster T, Stead LF. 2005. Individual behavioural counselling for smoking cessation. *Cochrane database of systematic reviews*

155. Larsen RJ, Diener E. 1987. Affect intensity as an individual difference characteristic: A review. *Journal of Research in personality* 21:1-39

156. Larsen RJ, Diener E, Emmons RA. 1986. Affect intensity and reactions to daily life events. *Journal of personality and social psychology* 51:803

157. Lavender JM, Tull MT, DiLillo D, Messman-Moore T, Gratz KL. 2015. Development and Validation of a State-Based Measure of Emotion Dysregulation: The State Difficulties in Emotion Regulation Scale (S-DERS). *Assessment*

158. Lawton MP. 2001. Emotion in later life. *Current directions in psychological science* 10:120-3

159. Lee EB, An W, Levin ME, Twohig MP. 2015. An initial meta-analysis of Acceptance and Commitment Therapy for treating substance use disorders. *Drug and alcohol dependence* 155:1-7

160. Leventhal AM. 2010. Do Individual Differences in Reinforcement Smoking Moderate the Relationship Between Affect and Urge to Smoke? *Behavioral medicine (Washington, D.C.)* 36:1-6

161. Leventhal AM, Greenberg JB, Trujillo MA, Ameringer KJ, Lisha NE, et al. 2013. Positive and negative affect as predictors of urge to smoke: temporal factors and mediational pathways. *Psychology of addictive behaviors : journal of the Society of Psychologists in Addictive Behaviors* 27:262-7

162. Levin C, Ilgen M, Moos R. 2007. Avoidance coping strategies moderate the relationship between self-efficacy and 5-year alcohol treatment outcomes. *Psychology of addictive behaviors : journal of the Society of Psychologists in Addictive Behaviors* 21:108-13

163. Linehan M. 1993. *Cognitive-behavioral treatment of borderline personality disorder*. Guilford press

164. Linehan MM, Schmidt H, Dimeff LA, Craft JC, Kanter J, Comtois KA. 1999. Dialectical behavior therapy for patients with borderline personality disorder and drug‐dependence. *The American journal on addictions* 8:279-92

165. Little J, Cardy A, Munger RG. 2004. Tobacco smoking and oral clefts: a meta-analysis. *Bulletin of the World Health Organization* 82:213-8

166. Liverant GI, Brown TA, Barlow DH, Roemer L. 2008. Emotion regulation in unipolar depression: The effects of acceptance and suppression of subjective emotional experience on the intensity and duration of sadness and negative affect. *Behaviour research and therapy* 46:1201-9

167. Lombardo TW, Epstein LH. 1986. The nicotine paradox: effect of smoking on autonomic discrimination. *Addictive behaviors* 11:341-4

168. Lundbäck B, Lindberg A, Lindström M, Rönmark E, Jonsson A-C, et al. 2003. Not 15 but 50% of smokers develop COPD?—report from the obstructive lung disease in Northern Sweden studies. *Respiratory medicine* 97:115-22

169. MacIntyre JM. 2015. *Emotion Regulation and Negative Affect: Laboratory and Field Investigations*. Uniformed Services Univeristy of the Health Sciences

170. MacIntyre JM, Ruscio A, Brede E, Waters AJ. 2018. Emotion dysregulation and negative affect: Laboratory and EMA investigations in smokers. *Addictive Behaviors Reports*

171. Mackinnon A, Jorm AF, Christensen H, Korten AE, Jacomb PA, Rodgers B. 1999. A short form of the Positive and Negative Affect Schedule: evaluation of factorial validity and invariance across demographic variables in a community sample. *Personality and Individual Differences* 27:405-16

172. Magar EC, Phillips LH, Hosie JA. 2008. Self-regulation and risk-taking. *Personality and individual differences* 45:153-9

173. Marlatt GA, Gordon JR. 1980. Determinants of relapse. Implications for the maintenance of behavior change. In *Behavioral medicine: Changing health lifestyles*, ed. PO Davidson, SM Davidson. New York, NY: Brunner/Mazel. Number of.

174. Martin RC, Dahlen ER. 2005. Cognitive emotion regulation in the prediction of depression, anxiety, stress, and anger. *Personality and Individual Differences* 39:1249-60

175. Mata J, Hogan CL, Joormann J, Waugh CE, Gotlib IH. 2013. Acute exercise attenuates negative affect following repeated sad mood inductions in persons who have recovered from depression. *Journal of abnormal psychology* 122:45

176. Matud MP. 2004. Gender differences in stress and coping styles. *Personality and individual differences* 37:1401-15

177. McGehee DS, Heath MJ, Gelber S, Devay P, Role LW. 1995. Nicotine enhancement of fast excitatory synaptic transmission in CNS by presynaptic receptors. *Science (New York, N.Y.)* 269:1692-6

178. McLaughlin I, Dani JA, De Biasi M. 2015. Nicotine withdrawal. *Current topics in behavioral neurosciences* 24:99

179. McNeill AD, Jarvis M, West R. 1987. Subjective effects of cigarette smoking in adolescents. *Psychopharmacology* 92:115-7

180. Melnick SM, Hinshaw SP. 2000. Emotion regulation and parenting in AD/HD and comparison boys: linkages with social behaviors and peer preference. *Journal of abnormal child psychology* 28:73-86

181. Mennin DS. 2004. Emotion regulation therapy for generalized anxiety disorder. *Clinical Psychology & Psychotherapy* 11:17-29

182. Mikels JA, Maglio SJ, Reed AE, Kaplowitz LJ. 2011. Should I go with my gut? Investigating the benefits of emotion-focused decision making. *Emotion (Washington, D.C.)* 11:743

183. Moberly NJ, Watkins ER. 2008. Ruminative self-focus and negative affect: an experience sampling study. *Journal of abnormal psychology* 117:314

184. Moore C. 1965. Smoking and cancer of the mouth, pharynx, and larynx. *Jama* 191:283-6

185. Morissette SB, Tull MT, Gulliver SB, Kamholz BW, Zimering RT. 2007. Anxiety, anxiety disorders, tobacco use, and nicotine: a critical review of interrelationships. *Psychological bulletin* 133:245

186. Morrow J, Nolen-Hoeksema S. 1990. Effects of responses to depression on the remediation of depressive affect. *J Pers Soc Psychol* 58:519-27

187. Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, et al. 2015. Heart Disease and Stroke Statistics—2016 Update A Report From the American Heart Association. *Circulation*:CIR. 0000000000000350

188. National Cancer Institute. 2016. *NCI Dictionary of Cancer Terms*. https://www.cancer.gov/publications/dictionaries/cancer-terms

189. Nica EI, Links PS. 2009. Affective instability in borderline personality disorder: experience sampling findings. *Current psychiatry reports* 11:74-81

190. Nichter M, Vuckovic N, Quintero G, Ritenbaugh C. 1997. Smoking experimentation and initiation among adolescent girls: qualitative and quantitative findings. *Tobacco control* 6:285-95

191. Nides M. 2008. Update on Pharmacologic Options for Smoking Cessation Treatment. *The American journal of medicine* 121:S20-S31

192. Nolen-Hoeksema S. 2000. The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of abnormal psychology* 109:504-11

193. Nolen-Hoeksema S, Morrow J, Fredrickson BL. 1993. Response styles and the duration of episodes of depressed mood. *Journal of abnormal psychology* 102:20-8

194. Nolen-Hoeksema S, Wisco BE, Lyubomirsky S. 2008. Rethinking Rumination. *Perspectives on psychological science : a journal of the Association for Psychological Science* 3:400-24

195. Norman PE, Curci JA. 2013. Understanding the effects of tobacco smoke on the pathogenesis of aortic aneurysm. *Arteriosclerosis, thrombosis, and vascular biology* 33:1473-7

196. Ossip-Klein DJ, Bigelow G, Parker SR, Curry S, Hall S, Kirkland S. 1986. Task Force 1: Classification and assessment of smoking behavior. *Health Psychology*

197. Parrott AC. 1995. Stress modulation over the day in cigarette smokers. *Addiction (Abingdon, England)* 90:233-44

198. Parrott AC, Kaye FJ. 1999. Daily uplifts, hassles, stresses and cognitive failures: in cigarette smokers, abstaining smokers, and non-smokers. *Behavioural Pharmacology* 10:639-46

199. Patton GC, Hibbert M, Rosier MJ, Carlin JB, Caust J, Bowes G. 1996. Is smoking associated with depression and anxiety in teenagers? *American Journal of Public Health* 86:225-30

200. Perkins KA, Grobe JE, Fonte C, Breus M. 1992. "Paradoxical" effects of smoking on subjective stress versus cardiovascular arousal in males and females. *Pharmacology, biochemistry, and behavior* 42:301-11

201. Perkins KA, Karelitz JL, Conklin CA, Sayette MA, Giedgowd GE. 2010. Acute negative affect relief from smoking depends on the affect situation and measure but not on nicotine. *Biological psychiatry* 67:707-14

202. Phelps EA. 2004. Human emotion and memory: interactions of the amygdala and hippocampal complex. *Current opinion in neurobiology* 14:198-202

203. Piasecki TM, Kenford SL, Smith SS, Fiore MC, Baker TB. 1997. Listening to nicotine: Negative affect and the smoking withdrawal conundrum. *Psychological Science* 8:184-9

204. Piper ME, Piasecki TM, Federman EB, Bolt DM, Smith SS, et al. 2004. A multiple motives approach to tobacco dependence: the Wisconsin Inventory of Smoking Dependence Motives (WISDM-68). *Journal of consulting and clinical psychology* 72:139-54

205. Preacher KJ, Zhang Z, Zyphur MJ. 2016. Multilevel structural equation models for assessing moderation within and across levels of analysis. *Psychological methods* 21:189

206. Proctor RN. 2012. The history of the discovery of the cigarette-lung cancer link: evidentiary traditions, corporate denial, global toll. *Tobacco control* 21:87-91

207. Quigley K, Lindquist KA, Barrett LF. 2014. Inducing and measuring emotion and affect: Tips, tricks, and secrets. *Handbook of research methods in social and personality psychology*:220-52

208. Quirk SW. 2001. Emotion concepts in models of substance abuse. *Drug and Alcohol Review* 20:95-104

209. Robinson JD, Lam CY, Carter BL, Minnix JA, Cui Y, et al. 2011. A Multimodal Approach to Assessing the Impact of Nicotine Dependence, Nicotine Abstinence, and Craving on Negative Affect in Smokers. *Experimental and clinical psychopharmacology* 19:40-52

210. Robinson TE, Berridge KC. 1993. The neural basis of drug craving: an incentive-sensitization theory of addiction. *Brain research reviews* 18:247-91

211. Robinson TE, Berridge KC. 1993. The neural basis of drug craving: an incentive-sensitization theory of addiction. *Brain research. Brain research reviews* 18:247-91

212. Robinson TE, Berridge KC. 2008. The incentive sensitization theory of addiction: some current issues. *Philosophical Transactions of the Royal Society of London B: Biological Sciences* 363:3137-46

213. Roddy E. 2004. Bupropion and other non-nicotine pharmacotherapies. *BMJ (Clinical research ed.)* 328:509-11

214. Rollema H, Chambers LK, Coe JW, Glowa J, Hurst RS, et al. 2007. Pharmacological profile of the $\alpha 4 \beta 2$ nicotinic acetylcholine receptor partial agonist varenicline, an effective smoking cessation aid. *Neuropharmacology* 52:985-94

215. Russell JA. 2003. Core affect and the psychological construction of emotion. *Psychological review* 110:145

216. Rusting CL, Nolen-Hoeksema S. 1998. Regulating responses to anger: effects of rumination and distraction on angry mood. *J Pers Soc Psychol* 74:790-803

217. Saffrey C, Summerville A, Roese NJ. 2008. Praise for regret: People value regret above other negative emotions. *Motivation and Emotion* 32:46-54

218. Salters-Pedneault K, Roemer L, Tull MT, Rucker L, Mennin DS. 2006. Evidence of broad deficits in emotion regulation associated with chronic worry and generalized anxiety disorder. *Cognitive Therapy and Research* 30:469-80

219. Salters-Pedneault K, Roemer L, Tull TM, Rucker L, Mennin SD. 2006. Evidence of Broad Deficits in Emotion Regulation Associated with Chronic Worry and Generalized Anxiety Disorder. *Cognitive Therapy and Research* 30:469-80

220. Salters-Pedneault K, Tull MT, Roemer L. 2004. The role of avoidance of emotional material in the anxiety disorders. *Applied and Preventive Psychology* 11:95-114

221. Schmidt M, Perels F, Schmitz B. 2010. How to perform idiographic and a combination of idiographic and nomothetic approaches. *Zeitschrift für Psychologie/Journal of Psychology* 218:166-74

222. Schmidt M, Perels F, Schmitz B. 2010. How to perform idiographic and nomothetic approaches: A comparison of time series analyses and hierarchical modeling. *Zeitschrift für Psychologie/Journal of Psychology* 218:166-74

223. Schoenmakers TM, de Bruin M, Lux IF, Goertz AG, Van Kerkhof DH, Wiers RW. 2010. Clinical effectiveness of attentional bias modification training in abstinent alcoholic patients. *Drug and alcohol dependence* 109:30-6

224. Schönfelder S, Kanske P, Heissler J, Wessa M. 2014. Time course of emotion-related responding during distraction and reappraisal. *Social cognitive and affective neuroscience* 9:1310-9

225. Schwartz JE, Stone AA. 2007. The analysis of real-time momentary data: A practical guide. In *The Science of Real-Time Data Capture: Self-Reports in Health Research*, ed. A Stone, S Shiffman, A Atienza, L Nebeling:76-113: Oxford University Press. Number of 76-113 pp.

226. Sher KJ, Grekin ER. 2007. Alcohol and Affect Regulation. In *Handbook of emotion regulation*, ed. JJ Gross:560-80. New York, NY: Guildford Press. Number of 560-80 pp.

227. Shiffman S. 1982. Relapse following smoking cessation: a situational analysis. *Journal of consulting and clinical psychology* 50:71

228. Shiffman S. 1993. Assessing smoking patterns and motives. *Journal of consulting and clinical psychology* 61:732-42

229. Shiffman S. 1993. Assessing smoking patterns and motives. *Journal of consulting and clinical psychology* 61:732

230. Shiffman S, Li X, Dunbar MS, Tindle HA, Scholl SM, Ferguson SG. 2015. Does laboratory cue reactivity correlate with real-world craving and smoking responses to cues? *Drug and alcohol dependence* 155:163-9

231. Shiffman S, Patten C, Gwaltney C, Paty J, Gnys M, et al. 2006. Natural history of nicotine withdrawal. *Addiction (Abingdon, England)* 101:1822-32

232. Shiffman S, Stone AA, Hufford MR. 2008. Ecological momentary assessment. *Annu. Rev. Clin. Psychol.* 4:1-32

233. Shiffman S, Waters AJ. 2004. Negative affect and smoking lapses: a prospective analysis. *Journal of consulting and clinical psychology* 72:192-201

234. Shiyko M, Naab P, Shiffman S, Li R. 2014. Modeling complexity of EMA data: time-varying lagged effects of negative affect on smoking urges for subgroups of nicotine addiction. *Nicotine & tobacco research : official journal of the Society for Research on Nicotine and Tobacco* 16 Suppl 2:S144-50

235. Silagy C, Lancaster T, Stead L, Mant D, Fowler G. 2004. Nicotine replacement therapy for smoking cessation. *The Cochrane Library*

236. Silk J, Laurence S, Morris AS. 2003. Adolescents' Emotion Regulation in Daily Life: Links to Depressive Symptoms and Problem Behavior. *Child development* 74:1869-80

237. Silvers JA, Buhle JT, Ochsner KN, Silvers J. 2013. The neuroscience of emotion regulation: Basic mechanisms and their role in development, aging and psychopathology. *The handbook of cognitive neuroscience* 1:52-78

238. Sjöberg L, Johnson T. 1978. Trying to give up smoking: A study of volitional breakdowns. *Addictive behaviors* 3:149-64

239. Skov-Ettrup LS, Lise SS-E, Peter D, Mickael B, Janne ST. 2016. The effectiveness of telephone counselling and internet- and text-message-based support for smoking cessation: results from a randomized controlled trial. *Addiction (Abingdon, England)* 111:1257-66

240. Slattery ML, Potter JD, Friedman GD, Ma KN, Edwards S. 1997. Tobacco use and colon cancer. *International journal of cancer* 70:259-64

241. Stasiewicz PR, Maisto SA. 1993. Two-factor avoidance theory: The role of negative affect in the maintenance of substance use and substance use disorder. *Behavior Therapy* 24:337-56

242. Stead LF, Perera R, Bullen C, Mant D. 2012. Nicotine replacement therapy for smoking cessation. *Cochrane database of systematic reviews* 11:CD000146

243. Stevens SL, Colwell B, Smith DW, Robinson J, McMillan C. 2005. An exploration of self-reported negative affect by adolescents as a reason for smoking: implications for tobacco prevention and intervention programs. *Preventive medicine* 41:589-96

244. Susskind JM, Lee DH, Cusi A, Feiman R, Grabski W, Anderson AK. 2008. Expressing fear enhances sensory acquisition. *Nature neuroscience* 11:843-50

245. Szasz PL, Szentagotai A, Hofmann SG. 2012. Effects of emotion regulation strategies on smoking craving, attentional bias, and task persistence. *Behaviour research and therapy* 50:333-40

246. Tamres LK, Janicki D, Helgeson VS. 2002. Sex differences in coping behavior: A meta-analytic review and an examination of relative coping. *Personality and social psychology review* 6:2-30

247. Thoits PA. 1991. Gender differences in coping with emotional distress. In *The social context of coping*, ed. J Eckenrode:107-38: Springer. Number of 107-38 pp.

248. Thompson RA. 1994. Emotion regulation: A theme in search of definition. *Monographs of the society for research in child development* 59:25-52

249. Thompson RJ, Mata J, Jaeggi SM, Buschkuehl M, Jonides J, Gotlib IH. 2012. The everyday emotional experience of adults with major depressive disorder: Examining emotional instability, inertia, and reactivity. *Journal of abnormal psychology* 121:819-29

250. Tice DM, Bratslavsky E. 2000. Giving in to feel good: The place of emotion regulation in the context of general self-control. *Psychological Inquiry* 11:149-59

251. Tonstad S, Tønnesen P, Hajek P, Williams KE, Billing CB, et al. 2006. Effect of maintenance therapy with varenicline on smoking cessation: a randomized controlled trial. *Jama* 296:64-71

252. Trédaniel J, Boffetta P, Buiatti E, Saracci R, Hirsch A. 1997. Tobacco smoking and gastric cancer: Review and meta‐analysis. *International Journal of cancer* 72:565-73

253. Tull MT, Gratz KL, Salters K, Roemer L. 2004. The role of experiential avoidance in posttraumatic stress symptoms and symptoms of depression, anxiety, and somatization. *The Journal of nervous and mental disease* 192:754-61

254. Twisk JWR. 2006. *Applied Multilevel Analysis: A Practical Guide for Medical Researchers*. Cambridge University Press

255. U.S. Department of Commerce. 2012. National Cancer Institute-sponsored Tobacco Use Supplement to the Current Population Survey (2010-2011). ed. C Bureau

256. U.S. Department of Health and Human Services. 1988. The Health Consequences of Smoking: Nicotine Addiction. A Report of the Surgeon General. ed. USDoHaH Services. Atlanta, GA

257. U.S. Department of Health and Human Services. 2014. The Health Consequences of Smoking - 50 Years of Progress: A Report of the Surgeon General, U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, Atlanta

258. U.S. Department of Health and Human Services. 2016. *Explore Quit Methods*. https://smokefree.gov/tools-tips/explore-quit-methods

259. U.S. Department of Health E, and Welfare,. 1964. Smoking and Health. ed. E U.S. Department of Health, and Welfare. Washington, D.C.: United States Public Health Service Office of the Surgeon General

260. Velicer WF, Babbin SF, Palumbo R. 2013. Idiographic Applications: Issues of Ergodicity and Generalizability. In *Handbook of developmental systems theory and methodology*, ed. PC Molenaar, RM Lerner, KM Newell: Guilford Publications. Number of.

261. Velicer WF, Colby SM. 2005. A Comparison of Missing-Data Procedures for Arima Time-Series Analysis. *Educational and Psychological Measurement* 65:596-615

262. Velicer WF, Colby SM. 2005. Missing Data and the General Transformation Approach to Time Series Analysis. In *Contemporary psychometrics: A festschrift*

for Roderick P. McDonald.:509-35. Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers. Number of 509-35 pp.

263. Velicer WF, Harrop J. 1983. The reliability and accuracy of time series model identification. *Evaluation Review* 7:551-60

264. Velicer WF, McDonald RP. 1984. Times Series Analysis Without Model Identification. *Multivariate behavioral research* 19:33-47

265. Verduyn P, Delvaux E, Van Coillie H, Tuerlinckx F, Van Mechelen I. 2009. Predicting the duration of emotional experience: two experience sampling studies. *Emotion (Washington, D.C.)* 9:83

266. Versland A, Rosenberg H. 2007. Effect of brief imagery interventions on craving in college student smokers. *Addiction Research & Theory* 15:177-87

267. Watson D, Clark LA, Tellegen A. 1988. Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of personality and social psychology* 54:1063

268. Watson D, Tellegen A. 1985. Toward a consensual structure of mood. *Psychological bulletin* 98:219

269. Webb TL, Miles E, Sheeran P. 2012. Dealing with feeling: a meta-analysis of the effectiveness of strategies derived from the process model of emotion regulation. *Psychol Bull* 138:775-808

270. Welsch SK, Smith SS, Wetter DW, Jorenby DE, Fiore MC, Baker TB. 1999. Development and validation of the Wisconsin Smoking Withdrawal Scale. *Experimental and Clinical Psychopharmacology* 7:354-61

271. Wenzlaff RM, Wegner DM. 2000. Thought suppression. *Annual review of psychology* 51:59-91

272. Wetter DW, Smith SS, Kenford SL, Jorenby DE, Fiore MC, et al. 1994. Smoking outcome expectancies: factor structure, predictive validity, and discriminant validity. *Journal of abnormal psychology* 103:801

273. Whittaker R, Borland R, Bullen C, Lin RB, McRobbie H, Rodgers A. 2009. Mobile phone-based interventions for smoking cessation. *Cochrane database syst Rev* 4

274. Wilkie GI, Hutson PH, Stephens MW, Whiting P, Wonnacott S. 1993. Hippocampal nicotinic autoreceptors modulate acetylcholine release. *Biochemical Society transactions* 21:429-31

275. Winkelstein Jr W. 1990. Smoking and cervical cancer--current status: a review. *American Journal of Epidemiology* 131:945-57

276. Wisborg K, Kesmodel U, Henriksen TB, Olsen SF, Secher NJ. 2001. Exposure to tobacco smoke in utero and the risk of stillbirth and death in the first year of life. *American journal of epidemiology* 154:322-7

277. Wolgast M, Lundh LG, Viborg G. 2011. Cognitive reappraisal and acceptance: An experimental comparison of two emotion regulation strategies. *Behaviour research and therapy* 49:858-66

278. Xiao C, Yang KC, Zhou CY, Jin GZ, Wu J, Ye JH. 2009. Nicotine modulates GABAergic transmission to dopaminergic neurons in substantia nigra pars compacta. *Acta pharmacologica Sinica* 30:851-8

279. Zhu K, Moriarty C, Caplan LS, Levine RS. 2007. Cigarette smoking and primary liver cancer: a population-based case–control study in US men. *Cancer Causes & Control* 18:315-21

280. Zierler-Brown SL, Kyle JA. 2007. Oral varenicline for smoking cessation. *Annals of Pharmacotherapy* 41:95-9