

## ABSTRACT

Title of Document: A COMPREHENSIVE ASSESSMENT OF  
DISTRESS TOLERANCE AS A PREDICTOR  
OF EARLY SMOKING LAPSE

Brooke A. Stipelman, PhD, 2008

Directed By: Carl W. Lejuez, PhD, Assistant Professor,  
Department of Psychology

The CDC estimates that approximately 20.9% of U.S. adults currently smoke. Moreover, cigarette smoking continues to be the leading preventable cause of death and disability in the United States making it is a significant public health problem. Although 70% of smokers express a desire to quit, relapse is quite common, with rates as high as 60-90% depending on the method of quitting used. Moreover, many smokers who attempt to quit, lapse within a few days, and many of these individuals ultimately resume smoking and are not able to recover to achieve abstinence. The initial experience of smoking cessation is stressful and is associated with a number of unpleasant withdrawal symptoms. Therefore, one particular hypothesis suggests that how an individual reacts to and tolerates these uncomfortable feelings may be a key contributing factor of relapse. This threshold for tolerating physical and psychological stress is known as distress tolerance. While early evidence has suggested that distress tolerance is associated with duration of quit attempts, to date, no study has examined the effects of distress tolerance across physical, psychological and biological domains on a number of other relapse predictors (e.g. negative affect,

anxiety sensitivity and withdrawal symptoms) in determining smoking outcome. Therefore, the following study looked at the role of these variables in predicting smoking outcome in a group of 58 adult smokers who entered a smoking cessation treatment study. As hypothesized, both measures of physical distress tolerance and one measure of psychological distress tolerance significantly predicted time to smoking lapse above and beyond other smoking related variables. There was no relationship between smoking abstinence and self-report and biological measures of distress tolerance. There were also no significant findings with respect to any affect related smoking variables. Implications and future directions are addressed.

A COMPREHENSIVE ASSESSMENT OF DISTRESS TOLERANCE AS A  
PREDICTOR OF EARLY SMOKING LAPSE

By

Brooke A. Stipelman, M.A.

Dissertation submitted to the Faculty of the Graduate School of the  
University of Maryland, College Park, in partial fulfillment  
of the requirements for the degree of  
Doctor of Philosophy  
2008

Advisory Committee:  
Professor Carl W. Lejuez, Chair  
Professor Jack Blanchard  
Professor Harold Sigal  
Professor Matthew Tull  
Professor Eric Wish

© Copyright by  
Brooke A. Stipelman  
2008

## Table of Contents

Table of Contents.....	ii
List of Tables.....	iv
List of Figures.....	v
Chapter 1: Theoretical Rational.....	1
Public Health Significance of Cigarette Smoking.....	1
Smoking Cessation Treatment.....	2
Pharmacotherapy.....	3
Nicotine Replacement Therapy.....	3
Bupropion.....	5
Standard Behavioral Treatment.....	7
Overview of Smoking Relapse.....	11
Theory of Smoking Relapse.....	11
Importance of Early Smoking Relapse.....	13
Predictors of Early Smoking Lapse/Relapse.....	16
Withdrawal Symptoms.....	16
Negative Affect-specific Withdrawal Symptoms.....	19
Depression.....	21
Anxiety Sensitivity.....	24
HPA Reactivity/Cortisol.....	27
Distress Tolerance.....	30
Statement of the Problem.....	34
Hypotheses.....	37
Chapter 2: Method and Procedure.....	38
Participants.....	38
Screening Measures.....	38
Procedure.....	41
Laboratory Tasks .....	42
Paced Auditory Addition Task.....	42
Computerized Mirror-Tracing Persistence Task.....	43
Physical Challenge: Breath Holding Task.....	44
Pain Challenge: Cold Pressor Task.....	45
Self Report Measures.....	45
Smoking History, Nicotine Dependence .....	45
Distress Tolerance.....	46
Affective States.....	47
Nicotine Withdrawal Symptoms.....	47
Outcome Measures of Distress Tolerance.....	48
Task Persistence.....	48
Dysphoria.....	48
Biological Measures.....	48
Outcome Measures of Smoking Status.....	48
Chapter 3: Results.....	50
Relationships among Distress Tolerance Measures.....	50

Psychological Distress Tolerance.....	50
Physical Distress Tolerance.....	51
Physiological Distress Tolerance.....	51
Distress Tolerance Scale Self-Report.....	51
Relationship among Affective Self-Reports and DT Measures.....	52
Smoking Status.....	53
Mean Differences between Early and Delayed Lapsers.....	53
Demographic and Smoking History.....	53
Affective Self-report Measures.....	54
Distress Tolerance Measures.....	54
Predictors of Smoking Abstinence.....	54
Chapter 4: Discussion.....	65
Distress Tolerance.....	65
Affect-Related Variables.....	69
Convergent Validity.....	72
Limitations and Future Directions.....	75
Implications.....	79
Bibliography.....	81

## List of Tables

1. Means, standard deviations and intercorrelations among distress tolerance measures and affect self-reports.....56
2. Frequency distribution table for number of days to first smoking lapse.....57
3. Means and standard deviations of demographics and smoking history variables among the entire sample as well as group differences between early and delayed lapsers.....58
4. Means, standard deviations, and group differences on self-report and distress tolerance measures between early and delayed lapsers.....59
4. Logistic regression analysis of immediate and delayed lapsers with covariates gender and average number of cigarettes smoked per day, and relevant measures of distress tolerance.....64

## List of Figures

1. Mean task persistence (seconds) between early and delayed lapsers on the PASAT and MTPT-C tasks.....60
2. Mean difference score (seconds) between early and delayed lapsers on the BH and CP tasks.....61
3. Mean scores between early and delayed lapsers on the DTS (total score and subscales).....62
4. Mean salivary cortisol difference scores between early and Delayed lapsers.....63



## Chapter 1: Theoretical Rationale

### Public Health Significance of Smoking

Cigarette smoking is highly prevalent both in the United States and throughout the world. In the United States, approximately 44.5 million adults (20.9%) currently smoke, with over 81% endorsing daily use and 19% reporting less consistent use (CDC, 2005b). In general, it is estimated that about 25% of individuals living in developed nations are habitual smokers, with daily consumption ranging from around 14 cigarettes per day in low income countries to 22 cigarettes per day in high income countries (Zaher et al., 2004). The DSM-IV defines nicotine dependence as a maladaptive pattern of substance use resulting in clinically significant impairment or distress as evidenced by three or more criterion symptoms of dependence. These symptoms include: tolerance, withdrawal, nicotine being consumed in larger amounts than intended, persistent desire to cut down or control nicotine use, a great deal of time spent in activities to obtain, use, or recover from the effects of nicotine, reduction in social, occupational, or recreational activities due to nicotine use, and continued use of nicotine despite having knowledge of specific psychological or physical harm caused by or exacerbated by the use nicotine (American Psychiatric Association, 1994). It is assumed that within a few years of daily smoking, most smokers will begin to develop dependence and notice withdrawal symptoms upon smoking cessation (UDHHS, 1994). For example, approximately 50% of current smokers in their twenties meet criteria for nicotine dependence (Breslau, Kilbey, & Andreski, 1994). The National Comorbidity Survey Replication (NCS-R) estimated the 12-month prevalence of DSM-IV nicotine dependence at 11%, with about

10.5% of females and 11.6% of males meeting criteria (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Although this rate is substantially lower than that published in the original NCS (24%; Breslau, Johnson, Hiripi, & Kessler, 2001) and the Epidemiological Catchment Area Study (ECA; 36.6%; Robins, Helzer, Przybeck, 1986), overall prevalence rates of smoking have declined over the past two decades due to increased public health efforts (CDC, 2005b). In addition, these surveys used older versions of the DSM which were thought to be over inclusive (Hughes, Gust, & Pechacek, 1987), and employed less stringent criteria (i.e. three or more lifetime criteria versus two or more in the 12-month period).

Cigarette smoking is the leading preventable cause of death in the United States (USDHHS, 1990). It has been estimated that smoking causes an average of 440,000 deaths, which accounts for about one out of five deaths per year (CDC, 2003). In fact, more deaths are caused each year from smoking than from human immunodeficiency virus (HIV), illegal drug use, alcohol use, motor vehicle injuries, suicides, and murders combined (CDC, 2002; McGinnis & Foege, 1993). Smoking is associated with many diseases such as lung, oral, and a host of other cancers, cardiovascular disease, chronic obstructive pulmonary disease, peptic ulcers, gastrointestinal disorders, maternal/fetal complications and a variety of other serious health complications (Bartecchi, MacKenzie, & Schrier, 1994; Peto, Lopez, Boreham, Thun, & Heath, 1992). It is estimated that about 45% of all smokers will die of a tobacco-related disorder and smokers tend to die about 14 years earlier than nonsmokers (Peto, Lopez, Boreham, Thun, & Heath, 1992; CDC, 2002). In addition to the direct impact of smoking, secondhand smoke causes about 35,000 deaths and associated morbidity in relatives and children annually (CDC, 2002a),

and almost 1,000 infant deaths can be attributed to smoking during pregnancy (CDC, 2005a). In addition to the human cost, it is estimated that \$92 billion (1997-2001) in productivity is lost due to deaths from smoking, with the economic costs of smoking totaling more than \$167 billion, including an additional \$75.5 billion in smoking-related medical expenditures (CDC, 2005a). These sobering statistics offer a clear picture of huge public health impact that cigarette smoking has on our society.

### *Smoking Cessation Treatments*

Given the issues related to the morbidity and mortality of chronic cigarette smoking, there has been a huge public health campaign aimed at reducing the prevalence of smoking in the United States. While the ultimate public health goal may be to develop primary prevention strategies aimed at curbing the initiation of smoking behavior, smoking cessation treatments can aid those individuals who already smoke and are motivated to quit. Although approximately 70% of current smokers maintain that they would like to quit smoking, relapse rates are high, with the one-year quit ratio in the general smoking population at less than 1% (Fiore et al., 1989). The majority of individuals who attempt to quit smoking (about 90%), do so on their own without the aid of any formalized treatment (USDHHS, 1990). Within this group of self-quitters, 33% remain abstinent after two days and only about 5% are still smoke free after one year (CDC, 2002b). In particular, one study followed a group of 235 self-quitters and found that over 1/3 of smokers relapsed within 24 hours and another 1/3 within 48 hours (Garvey, Bliss, Hitchcock, & Heingold, 1992). In fact, by six months only 3% of the sample had remained abstinent. However, it is important to note that most smokers make a number of quit attempts so that eventually 50% of smokers end up quitting (USDHHS,

1990). Those individuals who seek out formal treatments fare slightly better, however relapse rates are still as high as 70-85% (Fiore, Bailey, & Cohen, 2000). However, compared to relapse rates following a self-quit attempt, one can conclude that smoking cessation treatments do contribute to improved smoking outcomes for many individuals. While there are a number of different treatment modalities currently available, the two with the most scientific evidence are pharmacotherapy and behavioral treatments.

### *Pharmacotherapy*

Guidelines for the treatment of tobacco use and dependence offered by the Department of Health and Human Services cite the effectiveness of pharmacotherapy in increasing smoking abstinence, with the recommendation that these treatments be used in all individuals who are trying to quit smoking and for whom it is not contraindicated for physical reasons (Fiore et al., 2001). In particular, the guidelines highlight four nicotine replacement therapies (NRT; gum, inhaler, nasal spray, and patch) and one antidepressant (Bupropion SR) as being safe and effective methods for quitting smoking.

*Nicotine Replacement Therapy.* Nicotine replacement therapies work by providing the smoker with an alternative safer form of nicotine that directly relieves some of the symptoms of withdrawal and craving that commonly occur during abstinence from smoking (Jaffe, 1985). Depending upon the type of product used, these treatment can provide a continuous, stable dose of nicotine over an extended period of time (i.e. patch) or a more rapid onset and shorter duration to help curb symptoms and cravings as they occur (i.e. spray, gum, inhaler). All forms of NRTs appear to be equally as effective (Hajek, West, Foulds, Nilsson, Burrows, & Meadow, 1999), though compliance was found to be highest for the patch and lowest for the nasal spray and inhaler. Data from a

host of clinical trials support the efficacy of NRTs in helping increase rates of smoking cessation. For example, a meta-analysis by Fiore, Smith, Jorenby & Baker (1994) examined the efficacy of the nicotine patch in increasing smoking abstinence. They reviewed 17 double-blind placebo-controlled studies ( $n=5098$ ) and found that six-month abstinence rates for smokers on the patch was 22% compared to 9% for placebo. Similarly, Silagy, Mant, Fowler, and Lodge (1994) conducted a meta-analysis of the effects of NRTs broadly on the smoking abstinence rates across 53 randomized controlled trials ( $n = 17,703$ ). They found that the use of an NRT increased the odds of remaining abstinent by almost two-fold ( $OR = 1.71$ ) compared to control interventions (range of 1.61 for gum to 3.05 for inhaler). More recently, Silagy, Lancaster, Stead, Mant, & Fowler (2002) conducted a Cochrane Library Systems Review to examine the effectiveness of five different types of NRTs (i.e. gum, patch, nasal spray, inhaler, and tablets) on smoking abstinence. They identified one hundred randomized clinical trials (48 gum, 30 patch, four nasal spray, four inhaler, and two tablet) and similar to their earlier study found that the use of NRTs overall increases the odds of quitting smoking by almost two fold ( $OR = 1.71$ ). In addition, NRTs have also been shown to be effective in light smokers (i.e. 15 or fewer cigarettes per day; Shiffman, 2005), very heavy smokers (i.e. 40 or more cigarettes per day; Shiffman, Di Marino, & Pillitteri, 2005), and those who have failed previous quit attempts using other pharmacological treatments such as different NRTs and Zyban (Shiffman, Dresler, & Rohay, 2004). Finally, because most data on the efficacy of NRTs have been conducted using clinical trials, Alberg and colleagues (2005) looked at both the prevalence and effectiveness of NRT use in the general population. Using data collected from a cohort in Maryland in 1989, they found

that 36% of smokers had endorsed using NRTs. However, interestingly, 30% of those individuals who used NRTs had quit smoking compared to 39% of nonusers. The authors hypothesize that users of NRTs may be more severe cases and only turn to this method after several unsuccessful quit attempts using other means.

*Bupropion*, Bupropion (Zyban) is a selective re-uptake inhibitor of dopamine and noradrenalin. While the exact mechanisms through which Bupropion works to promote smoking cessation is unclear, it is believed to be related to the reduced re-uptake of dopamine in the mesolimbic system (Ascher et al., 1995), and of noradrenalin in the locus coeruleus (Ferry, 1999). These areas are believed to be stimulated by nicotine and associated with symptoms of cravings and withdrawal. Consistent with this hypothesis, Bupropion has been shown to reduce the severity of craving during smoking cessation compared to other types of treatments (Hurt et al., 1997; Jorenby et al., 1999; Tashkin et al., 2001). Results from a number of clinical trials support the efficacy of Bupropion for smoking cessation. For example, in the guidelines for smoking treatment provided by DHHS, Fiore et al. (2000) reported that at the end of six months approximately 30% of smokers who used Bupropion were no longer smoking compared to 18-30% of those using nicotine replacement therapy. In addition, a Cochrane Library Systematic Review pooled results from 19 clinical trials of Bupropion found that use of the drug more than doubled the odds of remaining abstinent from smoking ( $OR = 2.06$ ; Hughes, Stead, & Lancaster, 2003). Compared to other pharmacotherapies, Bupropion has been shown to be more effective. One study examining the efficacy of Bupropion versus the nicotine patch found significantly higher rates of smoking abstinence at both six and 12-month follow-up in the Bupropion and Bupropion plus patch condition compared to use of the

patch alone or placebo (Jorenby et al., 1999). In addition, a recent six-week, randomized open label trial comparing Bupropion to Gabapentin found Bupropion to be superior in reducing withdrawal symptoms and promoting smoking cessation (White, Crockford, & Patten, 2005). Finally, two studies looked at the efficacy of retreatment using Bupropion in smokers who previously failed to respond. They found 6-to-12 month continuous abstinence rates of 12% and 9%, respectively for Bupropion compared to 2.0% for placebo (Gonzales, Nides, Ferry, 2001), and a 12-month point prevalence abstinence of 19% for Bupropion and 9% for placebo (Gonzales, Nides, Ferry, 2002).

The overall findings of pharmacological treatments for smoking are promising in helping to reduce relapse and promote smoking abstinence, however relapse rates are still high and there is a need for improvement. While NRTs help smokers alleviate some of the unpleasant physical and psychological symptoms associated with nicotine withdrawal, behavior therapy can address the conditioned cues, reinforcers and social context associated with smoking and can help the individual make lifestyle changes to effectively live smoke free. In fact, studies have shown that pharmacological treatments are most successful when implemented in a context that includes behavioral interventions (Fagerstrom, 1988; Fiore, Bailey, Cohen, Dorfman, Goldstein, & Gritz, 2000).

#### *Standard Behavioral Treatment*

Behavioral techniques for smoking cessation emerged in the 1960s and continue to be an effective tool for smoking cessation (Fiore et al., 2000). In particular, cognitive social learning theory (Bandura, 1997) provides a good behavioral framework for understanding these intervention strategies. Specifically, this model conceptualizes smoking as a learned behavior that is acquired through a number of behavioral principles

such as classical and operant conditioning, modeling, behavioral self-control, self-efficacy, and outcome expectancies (Brown & Emmons, 1991). While the exact components of behavioral smoking treatments vary, they typically address three distinct stages of treatment: preparation for quitting, quitting, and maintenance or relapse prevention. In line with this, Fiore and colleagues (2000) cite practical skills counseling (i.e. problem solving/skills training/stress management/relapse prevention), social support both in and out of the treatment setting, and aversive smoking interventions to be techniques associated with improved smoking outcomes.

In terms of preparing the individual to begin the process of quitting, motivational interviewing (MI) is a popular behavioral technique used for the treatment of alcohol use and other addictive disorders (Hettema, Steele, & Miller, 2005) that has also been applied to smoking cessation (Rollnick, Butler, & Stott, 1997). Motivational interviewing is a direct yet client centered technique which works to enhance intrinsic motivation to change through exploring and resolving the individual's ambivalence (Miller & Rollnick, 2002). In particular, Miller and Rollnick (2002) outline four basic principles of MI which include; expressing empathy, developing discrepancy, rolling with resistance, and supporting self-efficacy. The main advantage of MI is that it works to motivate individuals who may not be entirely ready to give up smoking to begin making changes in regards to their thoughts and behaviors that will eventually lead them to quit. There is some evidence to suggest that MI is efficacious in smoking cessation (Butler et al., 1999; Colby et al., 1998). For example, Soria and colleagues (2006) found that a combination of motivational interviewing and bupropion was over 5 times more effective in getting



smokers to quit compared to bupropion and anti-smoking advice (abstinence rates of 18.4 versus 3.4%, respectively).

According to the guidelines constructed by Fiore et al (2000), practical counseling is an important and effective component of any behavioral smoking treatment. They outline a number of examples of practical counseling that are utilized to some degree in most behavioral treatments. The first of these includes skills related to identifying high risk situations that may contribute to relapse. This can include any situational variable (e.g. being around other smokers, exposure to alcohol), internal state (e.g. stress, negative affect, cravings), or activity (e.g. being in a rush) that can precipitate an early relapse. In addition, practical counseling can incorporate the development and mastery of coping skills used to help deal with these potential high risk situations. This can include elements such as learning to anticipate and avoid high-risk situations, developing strategies and making changes to deal with stress, negative affect or any other unpleasant mood state that could contribute to relapse, and learning new activities and habits that help suppress cravings or urges. Finally, it is important to engage in some form of relapse prevention. Specifically, this includes basic information about what to expect during the smoking withdrawal process, the addictive nature of smoking, and the danger of a smoking lapse progressing into relapse (for reviews on practical counseling techniques see Brown 2003; Curry & McBride, 1994).

Social support has been identified as a key factor in promoting smoking abstinence; therefore it recommended that behavioral treatment incorporate some type of social support element (Fiore et al. 2001). The focus of social support can vary significantly across treatment interventions and can include elements such as listening

and encouragement by the therapist and group members (if applicable) and acquiring skills to help the individual seek social support outside of treatment. One example of this would be behavioral contracting where the patient enlists in the aid of a friend or loved for help in abstaining from cigarettes and maintaining a smoke free lifestyle. Studies have found that social support when used in conjunction with other behavioral techniques significantly improves the chances of quitting smoking (Etringer, Gregory, & Lando, 1984; Hamilton & Bornstein, 1979; Lichtenstein, E., Glasgow, R. E., & Abrams, D. B., 1986). For example, West, Edwards, and Hajek (1998) found that when smokers attending a smoking clinic were paired with a “buddy” (another smoker attempting to quit), they reported significantly higher four week abstinence rates compared to those smokers in the solo condition (27% vs. 12%, respectively). In addition, a longitudinal study examined three types of social support: support from a partner directly related to quitting, perceptions of the availability of general support resources, and the presence of smokers in the subject’s social network (Mermelstein, Cohen, Lichtenstein, Baer, & Kamarck, 1986). They found that all three elements of social support were important, but at different points in the cessation process. Specifically, high levels of partner support and perceived availability of support were associated with smoking cessation and short-term abstinence (i.e. up to 3 months) while smokers in the subject’s social network was associated with maintenance and long-term abstinence

Finally, aversive smoking techniques pair smoking with negative sensations to reduce the pleasure associated with smoking and extinguish the behavior. Rapid smoking is the most commonly employed aversive technique and typically involves having the smoker take a puff from their cigarette every few seconds until they feel unable to

continue. While rapid smoking, the individual is directed to focus on the unpleasant sensations produced. Other aversive techniques include rapid puffing, smoke holding, excessive smoking, self-paced smoking, covert sensitization and use of electric shocks or bitter pills (Hajek & Stead, 2000). Results from a Cochrane Database System included twenty-five trials using either rapid smoking or some other type of aversion method and found an almost two-fold ( $OR = 1.98$ ) increase in smoking cessation compared to control groups (Hajek & Stead, 2000). However, the authors note a number of methodological limitations across these studies that may have resulted in spurious results, so more work needs to be done to determine the effectiveness of this technique. Therefore, they are recommended in situations where more desirable treatments may have failed (Fiore et al. 2000).

Overall, behavioral theories offer a number of different techniques aimed at quitting smoking and maintaining a smoke free lifestyle. Although pharmacological agents work to help alleviate some of the unpleasant withdrawal symptoms associated with smoking, behavioral techniques offer the coping skills, support and psychoeducation that are necessary in reducing relapse rates.

### Overview of Smoking Relapse

#### *Theory of Smoking Relapse*

The most well-known and utilized theory of relapse prevention is the cognitive-behavioral model proposed by Marlatt and Gordon (1980, 1985). According to this model, relapse (i.e. a setback in an individual's attempt to change or modify a particular behavior) occurs as the result of exposure to a high-risk situation. Specifically, if an individual is confronted with a high risk situation and has either not learned an effective

coping response to deal with the situation, or chooses not to implement it due to factors such as motivation or anxiety, then there is an increased risk of a smoking lapse (i.e. a single slip or mistake). In addition, this increased probability of a lapse is mediated by other factors such as the individual's positive expectancies regarding the outcome of smoking and decreased self-efficacy regarding their perceived lack of ability to cope with the situation. However, according to this model, a smoking lapse does not necessarily have to result in a full-blown smoking relapse. In cases where the individual possess effective skills to cope with the situation, they can prevent a lapse from progressing into relapse and can proceed with smoking abstinence. However, following a lapse, many individuals may experience what is known as the abstinence violation effect (AVE), a phenomenon which basically refers to the loss of perceived control that is experienced following a lapse in smoking (Curry, Marlatt, & Gordon, 1987). The AVE operates on three distinct levels to increase the likelihood of full blown relapse. First, the AVE causes the smoker to feel negative emotions such as shame, guilt and blame which can lead the individual to want to smoke to alleviate some of these feelings. Secondly, the AVE can lead to cognitive dissonance due to the inconsistency between the individual's desire to be a nonsmoker and their reality of just having smoked a cigarette. Finally, the AVE can lead the individual to make stable, internal, and global attributions about their failure to remain abstinent which can contribute to feelings of low self-efficacy regarding their ability to remain abstinent. All of these consequences associated with the AVE can operate independently or in conjunction with one another to increase the probability that once these individuals smoke one cigarette they will abandon their quit attempt and resume smoking. Finally, the process of smoking is likely to provide the individual with

both positive and negative reinforcement which further increases the probability of continuing to smoke.

There have been a number of studies conducted evaluating this relapse prevention model which focus on either cognitive behavioral treatments designed to address relapse prevention strategies or the AVE (e.g. Baer & Lichtenstein, 1988; Brandon et al., 2000; Shiffman, Hitchcox 196 and 1997). For example, Bliss and colleagues (1989) conducted a prospective study that observed self-quitters over the course of one-month following smoking cessation to determine what variables contributed to a relapse crisis. In this particular study a relapse crisis was defined as either a smoking lapse or a situation in which the individual experienced the strongest temptation to smoke. They found that variables such as the presence of other smokers, withdrawal symptoms and negative affect were common precipitants of the relapse crisis. However, consistent with Marlatt and Gordon (1985), smoking abstinence was strongly related to the number of cognitive-behavioral coping strategies used during the situation. In addition, Curry, Marlatt, and Gordon (1987) looked at the validity of the AVE as defined by internal, stable and global causal attributions for cigarette smoking following a period of abstinence. Following completion of a smoking cessation treatment, those individuals who relapsed were asked to provide retrospective causal attributions for initial smoking lapses. They found that those smokers who relapsed following a slip reported significantly more AVEs than those who regained abstinence following their lapse. In addition, AVE was the most robust predictor of subsequent smoking following an initial smoking lapse. However, it is important to note that not all studies have found support for Marlatt and Gordon's relapse model (Donovan 1996b; Hall et al. 1997; Hall et al., 1990; Ockene 2000). In fact, a

review by Irvin and colleagues (1999) looked at 26 different studies that employed some type of relapse prevention treatment consistent with the model and found that while relapse prevention was reliable and effective, it was more successful in treating alcohol and illicit substance use than smoking. However, despite these inconsistencies in the literature, Marlatt and Gordon's model (1980) continues to be the most commonly ascribed theory of relapse.

#### *Importance of Early Smoking Lapse and Relapse*

Although Marlatt and Gordon's (1985) model of relapse highlights the importance of situational factors leading to relapse during the initial three to six months following cessation, there is evidence suggesting that the majority of individuals who lapse do so within the first couple of weeks of quitting. For example, Hughes, Keely, and Naud (2004) conducted a review of seven studies including either self-quitters or a no-treatment control group and found that the majority of smokers relapsed within 8 days of quitting. In addition, Gulliver, Hughes, Solomon, and Dey (1995) followed a group of 630 smokers attempting to self-quit and found that over 50% of the sample had endorsed smoking more than one cigarette by day 2 with an additional 21% lapsing within two weeks. This risk of early smoking lapse is evident in both self-quitters and those who receive formal smoking cessation treatment (Cook, Gerkovich, O'Connell, & Potocky, 1995; Doherty, Kinnunen, Militello, & Gravey, 1995; Shiffman et al., 2000; Shiffman, Hickcox, Paty, Gnys, Richards, & Kassel, 1997; Spanier, Shiffman, Maurer, Renyolds, & Quick, 1996; Westman, Behm, Simel, & Rose, 1997) For example, Zhu and colleagues (1996) conducted a large scale study where smokers were assigned to one of three telephone counseling sessions designed to aid in smoking cessation. They found that

59% of subjects in self-help, 54% in single and 49% in multiple contact groups smoked within the first week.

Although pharmacological treatments are designed to minimize withdrawal symptoms thus theoretically reducing early relapse, the data still evidence high rates of early relapse. For example, Shiffman and colleagues (2006) looked at the effects of a high-dose nicotine patch versus placebo in a group of adult smokers. They found that the patch was effective in providing initial abstinence (i.e. 24 hours without smoking), but amongst those smokers who were able to achieve initial abstinence, 67.2% lapsed. In fact, the median duration of abstinence achieved in the patch condition was six days compared to two days for the placebo. These findings are consistent with other studies that have found even with the NRT, most lapses occurred within the first week (Doherty, Kinnunen, Militello, & Garvey, 1995; Shiffman et al., 2000 and 1997). Although another set of studies fared slightly better, 43-50% of participants receiving the nicotine spray or inhaler endorsed smoking within the first week, which is still considerably high (Schneider et al., 1995; Schneider, Olmstead, Nilsson, Mody, Franzon, & Doan, 1996). Therefore, the overall conclusion gleaned from these results is that the initial first weeks of smoking cessation tend to be the most vulnerable to smoking lapses.

There is ample evidence to suggest that the majority of individuals who lapse within the first couple weeks of smoking cessation will ultimately progress to full relapse. It has been estimated that between 80% to almost 100% of individuals who experience a smoking lapse return to some form of regular smoking (Brandon et al., 1990; Cook, Gerkovich, O'Connell, & Potocky, 1995; Brown, Herman, Ramsey, & Stout, 1998; Doherty, Kinnunen, Militello, & Garvey, 1995; Garvey, Bliss, Hitchcock, Heinold, &

Rosner, 1992; Kenford et al., 1994; Shiffman, Hickcox, Paty, Gnys, Richards, & Kassel, 1997; Norregaard, Tonnesen, & Petersen, 1993; Smith, Jorenby, Fiore, Anderson, Mielke, & Beach, 2001). Chornock, Stitzer, Gross, & Leischow (1992) looked prospectively at the effects of smoking exposure following a brief period of abstinence. Sixty-seven smokers who had completed three days of smoking abstinence were randomly assigned to either smoke five of their own cigarettes or remain abstinent during a discrete period of time on the fourth day. Afterwards, all subjects were informed that they were free to resume smoking. They found that subjects from the programmed lapse condition were quicker to return to smoking compared to those in the abstinence condition suggesting that exposure to cigarettes following smoking cessation increases the probability of subsequent smoking. A similar result was found in a more recent study employing similar methodology but controlling for nicotine exposure (Juliano, Donny, Houtsmuller, & Stitzer, 2006). Therefore, these studies suggest that initial smoking lapses can have a negative effect on subsequent smoking outcomes. In addition, this effect is not entirely due to pharmacological effects of nicotine and other internal and situational factors play a role.

Taken together, the overall conclusion is that regardless of the treatment modality employed, smoking lapses within the first several days of abstinence are a common phenomenon. Moreover, smokers who tend to lapse early are at a higher risk for subsequent relapse. It is plausible that there are unique individual factors that differentiate this group of high-risk smokers from those who are able to successfully abstain from smoking either permanently or for longer periods of time. Therefore, in the



next section will review some of the predictors found to contribute to early smoking lapse and relapse.

### *Predictors of Early Smoking Lapse/Relapse*

#### *Withdrawal Symptoms*

Smoking cessation is associated with a number of negative withdrawal symptoms such as anxiety, irritability, difficulty concentrating, hunger, impatience, restlessness, and cravings for tobacco (Hughes, 1992; Hughes, Gust, Skoog, Keenan, & Fenwick, 1991; Hughes & Hatsukami, 1986). While the majority of these symptoms tend to remit within a month following cessation, others such as hunger and cravings tend to persist for up to six months or longer (Hughes, Gust, Skoog, Keenan, & Fenwick, 1991).

Pharmacological theories of smoking relapse posit that withdrawal symptoms are a hallmark of nicotine dependence and play an instrumental role in smoking maintenance and relapse behavior (Benowitz, 1983; Benowitz, 1992; Gilman, Goodman, Rall, & Murad, 1985; Henningfield & Goldberg, 1988; Schachter, 1978; Shiffman, 1991; Stolerman & Shoaib, 1991). Specifically, they hypothesize that withdrawal symptoms are the direct result of neural adaptations to chronic levels of nicotine in the body that once removed cause a disruption in the adapted symptoms. As a result, the individual experiences a cluster of aversive symptoms that are directly related to the inability of some smokers to successfully quit. In particular, it is hypothesized that these symptoms contribute to smoking due to the individual's desire to avoid and/or reverse smoking withdrawal symptoms.

While smokers will often attribute their cessation failures to aversive withdrawal symptoms (Cummings, Jaen, & Giovino, 1985; O'Connell & Martin, 1987; Wise, 1988),

empirical studies specifically examining the role of these symptoms in smoking relapse have been equivocal. For example, Patten and Martin (1996) conducted a review of 15 prospective studies and found that neither individual withdrawal symptoms or the total frequency and severity of symptoms served as a consistent predictor of smoking cessation or relapse. In fact, only six of the studies reviewed evidenced a significant association between withdrawal symptoms and either early-or long-term abstinence (Covey, Glassman, & Stetner, 1990; Gritz, Carr, & Marcus, 1991; Gunn, 1986; Persico, 1992; Robinson, Pettice, & Smith, 1992; West, Hajek, & Belcher, 1989), while the other nine did not (Hall, Hall; Hughes, Hughes et al, Hughes and Hatsukami, Kenford, Fiore, Jorenby, Smith, Wetter, & Baker, 1994; Norregaard, Stitzer & Gross, Swan and Denk). In studies where an association was found it tended to be related to short-term as opposed to long-term outcomes. However, this is not entirely surprising since it is consistent with the finding that the majority of withdrawal symptoms endure for up to a month. Therefore, it is plausible to assume that this initial period would be the highest risk for withdrawal related relapse. Patten and Martin (1996) note that a number of important methodological inconsistencies across studies may help explain these equivocal findings. For example, measurement issues relating to the construct validity of the symptom inventories, reliance on total severity scores as opposed to individual symptoms, and variability in duration between assessment periods may all contribute to the inconsistent results across studies. Another important limitation involves sampling. With the exception of Hughes (1992), all the other studies cited used individuals who were seeking smoking treatment interventions. However, the majority of smokers who do attempt to quit smoking, do so their own. In addition, research has shown that those individuals

who enter treatment programs are distinct from those who self-quit (Fiore et al., 1990; Lichtenstein & Hollis, 1992), therefore generalizability of these results across the majority of smokers is limited.

Piasecki and colleagues (1998) noted an important limitation in previous withdrawal symptom research was the lack of attention to individual variability and the assumption that an aggregate pattern of withdrawal symptoms is applicable to any given smoker. In a series of studies they noted dramatic heterogeneity in individual symptom profiles and found that mean symptom elevation and the trajectory of withdrawal symptoms were significantly associated with smoking relapse in both self-quitters and those using the nicotine patch (Piasecki 1998; 2000). In particular, individuals displaying atypical withdrawal profiles characterized by late peaks and unremitting symptoms were more likely to relapse. The authors noted that perhaps smokers who initially remain abstinent but then suffer from increasing withdrawal symptoms may resume smoking as a way to decrease their symptoms. In a later study using more advanced statistical models, Piasecki et al (2003a) examined reports of smoking withdrawal in 893 smokers and found that those smokers who lapsed evidenced a more aversive symptoms pattern and tended to have higher than expected withdrawal symptoms on the day they lapsed. In addition, they found a high degree of variability in terms of the severity, trajectory and variability of withdrawal symptoms, and these dimensions were predictive of both smoking lapse and relapse (Piasecki et al 2003b).

The overall conclusion of the literature on withdrawal symptoms and relapse remains unclear. While anecdotally many smokers report withdrawal symptoms precipitating smoking lapses, empirical evidence on the subject has been equivocal and

methodological issues obscure the findings. In addition, Piasecki and colleagues note the dramatic heterogeneity between individual withdrawal symptoms and trajectories, and stress the limitations of using aggregate measures of withdrawal symptoms. In particular, they found that those smokers who displayed withdrawal profiles characterized by late peaks and unremitting symptoms were more likely to relapse. In addition, as will be discussed in the next section, recent work has focused on the role of affective withdrawal symptoms on smoking relapse. Therefore, there seems to be some sort of relationship between withdrawal symptoms and relapse however the mechanisms underlying this association need to be further elucidated.

#### *Negative Affect-specific Withdrawal Symptoms*

As discussed above, smoking withdrawal is associated with a number of unpleasant symptoms including negative affective states (i.e. anxiety, irritability, frustration, dysphoria; American Psychiatric Association, 1994). Recent work has suggested that these affective withdrawal symptoms are critical to understanding the relationship between withdrawal symptoms and subsequent relapse, especially during the first couple weeks of abstinence when withdrawal symptoms are at their peak. For example, Piasecki, Kenford, Smith, Fiore, and Baker (1997) found that negative affect was a better predictor of relapse than physical dependence. Specifically, they found that measures of physical dependence did not contribute incremental validity in predicting relapse relative to measures of negative affect. In a later study attempting to replicate these findings in a group of self-quitters, Piasecki and colleagues (2000) found that variability in negative affect demonstrated a more potent relationship to smoking cessation outcome than variability in other withdrawal related factors (i.e. urge,

sleep/energy). These results are consistent with other research that has found that affective items contribute most of the reliable variance in withdrawal measures (Welsch et al., 1999). Finally, Kenford and colleagues (2002) found that post-quit negative affect (as measured by history of depression and negative affect during the first week of quitting) was a more robust predictor of smoking relapse at six month follow-up than measures of physical dependence. Therefore, these studies demonstrate that affective withdrawal symptoms play an important role in motivating individuals to return to smoking.

These findings on the role of affective withdrawal symptoms and relapse are consistent with the idea that state negative affect is widely believed to play an important role in the maintenance and relapse of smoking behavior. For example, a number of people report smoking during situations where they feel stressed or upset (Shiffman, 1993). In addition, results from retrospective studies indicate that smokers cite relief from negative affect as a core reason for smoking (Brandon & Baker, 1991; Piper et al., 2004). However, it is important to note that empirical evidence that nicotine actually works to reduce negative affect is equivocal. In some studies nicotine has been shown to reduce negative affect in the context of challenging and/or stressful tasks (Gilbert & Spielberger, 1987; Gilbert, Robinson, Chamberlin, & Spielberger, Pomerleau & Pomerleau, 1987). However, other studies that used naturalistic methods of data collection to get smoking antecedents in real-time have not found an consistent relationship between smoking and affective states (Delfino, Jamner, & Whalen, 2001; Shapiro, Jamner, Davydov, & Porsha, 2002; Shiffman et al., 2002; Shiffman, Paty, Gwaltney, & Dang, 2004).

Although the overall findings examining the purported reliance on cigarettes as a means to cope with stress and negative affect states are unclear, it has been shown that those individuals who do tend to smoke more frequently in negative affect situations and are less effective in managing these mood states are more likely to relapse (Abrams et al., 1987; Kamarck & Lichtenstein, 1988; O'Connell & Martin, 1987; O'Connell & Shiffman, 1988). Incidentally, several studies do indicate that negative affect (i.e. anxiety, anger, depression) serves as an immediate antecedent of smoking relapse (Baer, Kamarck, Lichtenstein, & Ransom, 1989; Baer & Lichtenstein, 1988; Bliss, Garvey, Heinold, & Hitchcock, 1989; Brandon et al., 1986; Brandon, Tiffany, Obremski, & Baker, 1990; Marlatt & Gordon, 1980; Shiffman, 1982). For example, Shiffman & Waters (2004) found increases in negative affect hours before a smoking lapse occurred. Moreover, it has been found that lapses in negative affect situations are more likely to lead to complete relapses (O'Connell & Martin, 1987). However, many of these studies have been retrospective in nature, and one study found that individuals attribute stress to smoking relapse retrospectively, but not prospectively, highlighting the importance of measurement periods (Hall, Harvey, & Wasserman, 1990). Therefore, a study Shiffman, Paty, Gnys, Kassel, & Hickcox (1996) sought to examine factors related to smoking temptation and lapse in real-time using palm pilots. They found that negative affect was able to discriminate between lapses, temptations, and random situations, with reported levels of negative affect being highest for lapses, followed by temptations, and finally random situations. Furthermore, about 20% of first smoking lapses occurred during moments when negative affect was particularly high (i.e. 2.5 SDs above the mean). In addition, when these same lapsed smokers were followed-up 12 weeks later and asked to

retrospectively describe the characteristics of their lapse they tended to exaggerate estimates of negative affect during the lapse, suggesting that affective states tend to be remembered more by smokers than other factors (Shiffman, Hufford, Hickcox, Paty, Gnys, & Kassel, 1997).

In summary, there appears to be a relationship between negative affect and smoking relapse particularly through its role in affect-related withdrawal symptoms. However, it is important to note that many individuals experience these symptoms of negative affect and are able to resist the urge to smoke and go to achieve abstinence.

### *Depression*

In addition to general levels of negative affect, depression has also been found to be an important predictor of smoking behavior. In general, smokers have been shown to have higher rates of comorbid depression compared to nonsmokers (Breslau, Kilbey & Andreski, 1991; Brown, Lewinsohn, Seely, & Wagner, 1996; Kendler, Neale, MacLean, Heath, Eaves, & Kessler, 1993) and tend to report higher rates of past major depressive episodes (Glassman, Covey, Stetner, & Rivelli, 2001; Ginsburg, Hall, Reus, & Munoz, 1995; Tsoh et al., 2000). In addition, individuals with a past history of depression are at an increased risk for developing depressive symptoms upon smoking cessation (Borrelli et al., 1996; Covey et al., 1997; Niaura, Britt, Borrelli, Shadel, Abrams, & Goldstein, 1999). One study found that smoking cessation may lead to a major depressive episode in about 15% of individuals with a past history of major depression (Kahler et al., 2002). On a related note, smokers with past major depression are more likely to report greater increases in negative mood following a quit attempt (Ginsburg et al., 1995). For example, Hall, Muñoz, and Reus (1990) found that smokers with past major depressive

disorder were more likely to endorse higher levels of anger, confusion and depression compared to non-past MDD subjects, and these subjects were more likely to have relapsed at six-month follow-up. However, despite this apparent association between past major depression and smoking relapse, treatment studies designed to address this proposed vulnerability have demonstrated inconsistent results (Hall, Muñoz, & Reus, 1994; 1996; 1998). One study using smokers with a past history of major depression compared a standard smoking cessation program to one augmented with cognitive-behavioral therapy for depression (CBT-D; Brown, Khaler, et al., 1991). They found that both treatments produced similar abstinence rates and that the CBT-D component did not significantly increase the odds of quitting. However, it is important to note that secondary analyses, did find that smokers with recurrent past major depression (i.e., two or more past major depressive episodes) and heavy smokers who received CBT-D had significantly higher abstinence rates than those receiving ST. Therefore, these results suggest that the association between relapse and major depression may not be as clear-cut as believed. In fact, a recent meta-analysis of 15 smoking cessation studies concluded that a lifetime history of major depression does not appear to be an independent risk factor for cessation failure in smoking cessation treatment (Hitsman et al., 2003). Instead, it may be the current levels of depressive symptomatology, rather than a history of major depression that is predictive of smoking outcomes.

There is compelling evidence to suggest that current levels of depressive symptoms serve as a more reliable predictor of smoking cessation failure (Anda, Williamson, Escobedo, Mast, Giovini, & Remington, 1990; Breslau, Kilbey, & Andreski, 1991; Glassman, Helzer, Covey, Cottler, Stetner, Tipp et al., 1990; Glassman 1993;



Hanna, Faden, & Dufour, 1994; Niaura, Britt, Shadel, Goldstein, Abrams, & Brown, 2001; Zuckerman, Amaro, Bauchner, & Cabral, 1989). For example, one study found that mean POMS depression scores prior to entering a smoking cessation treatment were significantly higher in individuals who were subsequently unable to quit compared to those who could quit (Rausch, Nicholson, Lamke, & Matloff, 1990). In addition, Kinnunen and colleagues (1996) found that those smokers with high depressive symptoms were less likely to be abstinent at a three-month follow-up compared to those smokers with low baseline depressive symptoms. Finally, a study by Blondal et al. (1999) found that independent of treatment condition, high depressive symptom smokers showed a decreased likelihood of abstinence at 6-week, 3-month and 6-month follow-ups. There are a number of explanations to help account for the poor outcome in smoking cessation in individuals with major depression. For example, these individuals begin their quit attempt with higher levels of negative mood, report greater elevations in negative mood and overall withdrawal intensity during the first week of quitting, and exhibit more anger and depression during the first week of quitting compared to individuals without depression (Covey, Glassman, & Stetner, 1990; Ginsberg, Hall, Reus & Muñoz, 1995; Hall, Muñoz & Reus, 1994).

In conclusion, depression appears to play an important role in both smoking behavior and cessation outcome. However, studies focusing on past history of major depressive episodes have been equivocal. Rather, it appears that baseline levels of depressive symptomatology may serve as a more reliable predictor of smoking relapse.

*Anxiety Sensitivity and Panic Disorder*

A number of studies have demonstrated an association between smoking and panic disorder. For example, individuals with panic are more likely to be smokers compared to normal controls and individuals with other forms of mental illness (Amering, Bankier, Berger, Griengl, Windhaber, & Katschnig, 1999; Lasser, Boyd, Woolhandler, Himmelstein, McCormick, & Bor, 2000; Pohl, Yeragani, Balon, Lycaki, & McBride, 1992). In addition, a number of prospective studies have shown that smoking can increase the risk in developing panic attacks and panic disorder (Breslau & Klein, 1999; Isensee, Wittchen, Stein, Hofler, & Lieb, 2003; Johnson, Cohen, Pine, Klein, Kasen, & Brook, 2000). It appears that panic attacks also play an important role in smoking relapse. In particular, one study found that smokers who reported a history of nonclinical panic attacks were more likely to endorse shorter quit attempts than those without panic attacks (Zvolensky, Lejuez, Khaler, & Brown, 2004). In addition, these smokers also reported more anxiety related withdrawal symptoms (i.e. anxiety, restlessness, difficulty concentrating, and irritability) but not other types of symptoms during their last quit attempt compared to the nonpanic group.

In understanding the mechanism involved in this association, increased attention has been paid the construct of anxiety sensitivity (AS), a dispositional trait-like cognitive vulnerability that has been shown to longitudinally predict panic disorder (Schmidt, Lerew, & Jackson, 1997). Anxiety sensitivity is defined as the fear of anxiety-related sensations which are thought to come from the belief that these sensations have harmful physical, psychological, or social consequences. It is typically measured using the Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986), a brief 16-item self-report questionnaire where respondents rate the degree to which they fear the

negative consequences of anxiety symptoms. Although the precise role of anxiety sensitivity in smoking related behavior is unclear, it is hypothesized that this construct may be elevated in a certain subgroup of smokers resulting in distress over the pharmacological or nonpharmacological induced interoceptive sensations associated with smoking and withdrawal. Individuals who are high on anxiety sensitivity and also lack adaptive coping strategies may turn to smoking as a means of escaping these unpleasant interoceptive states (Kassel & Shiffman, 1997; Zvolensky, Schmidt et al., 2003).

Brown, Kahler, Zvolensky, Lejuez, & Ramsey (2001) looked at the role of anxiety sensitivity as measured by the ASI in a group of 60 smokers with a history of major depression. They found that anxiety sensitivity scores were positively correlated with smoking as a means to reduce negative affect but not with any other reasons. These results are similar to other studies which found that smokers with elevated levels of anxiety sensitivity report smoking as a means to manage their mood (Comeau, Stewart, & Loba, 2001; Novak, Burgess, Clark, Zvolensky, & Brown, 2003; Stewart, Karp, Phil, & Peterson, 1997). Other studies have focused on the potential relationship between anxiety sensitivity and nicotine expectancies. For example, one study looked at the association between smoking outcome expectancies and three lower order factors on the ASI (Zvolensky, Feldner, Leen-Feldner, Bonn-Miller, McLeish, & Gregor, 2004). They found that physical and mental incapacitation were significantly associated with outcome expectancies for negative affect reduction and negative personal consequences. In a follow-up study, Zvolensky, Bonn-Miller, Feldner, Leen-Feldner, McLeish, & Gregor (2006) found that both physical and mental incapacitation concerns were associated with greater negative affect reduction smoking motives, but not other motives (i.e. Addictive,

Stimulation, Relaxation, and Sensorimotor). Moreover, these factors were associated lower levels of self-confidence about remaining abstinent when emotionally distressed. These findings persisted even after controlling for nicotine dependence, average number of cigarettes smoked per day, and gender.

In terms of smoking cessation, an early study by Zvolensky, Feldner, Eifert, & Brown (2001) looked at anxiety related emotional reactivity to 20% carbon dioxide enriched challenge, a procedure which has been shown to elicit a panic-related arousal state. They found that elevated emotional reaction to this challenge was negatively correlated with duration of smoking cessation abstinence among smokers with no history of panic related psychopathology. In addition, Brown and colleagues (2001) found that higher scores on the ASI were predictive of increased risk of smoking lapse during the first week of abstinence. In a related study, Zvolensky, Baker, Leen-Feldner, Bonn-Miller, Feldner, & Brown (2004) examined the associations between anxiety sensitivity and retrospective nicotine withdrawal symptoms in a group of 127 habitual smokers. They found that anxiety sensitivity predicted the severity of retrospectively recalled withdrawal symptoms during the first week of their latest quit attempt. Therefore, these results seem to suggest that individuals who are elevated in anxiety sensitivity may have difficulty quitting due to their inability to tolerate the withdrawal symptoms associated with smoking cessation.

Although the research in the area of anxiety sensitivity and smoking is quite new, preliminary results do suggest that individuals with elevated levels of anxiety sensitivity may smoke as a means to manage negative moods, expect that smoking will alleviate negative affect, and may have more difficulty quitting smoking during the first critical

weeks due to their inability to tolerate the anxious withdrawal symptoms associated with smoking cessation.

### *HPA Reactivity/Cortisol*

Research in smoking behavior has become increasingly focused on the effects of nicotine abstinence on the biobehavioral systems involved in the stress response. In particular, the hypothalamic-pituitary-adrenal axis (HPA), and more specifically its primary hormone cortisol have been shown to play an important function in smoking relapse both through their effects on withdrawal symptoms and ability to effectively deal with stress. A number of studies have demonstrated that acute nicotine intake causes an elevation in cortisol concentrations (e.g. Gilbert, Dibb, Plath, & Hyane, 2000; Kirschbaum, Wust, Strasburger, 1992; Mendelson, Sholar, Goletiani, Siegel, & Mello; 2005; Pomerleau & Pomerleau, 1990a; Seyler, Pomerleau, Fertig, Hunt, & Parker, 1986; Thakore, Berti, Dinan, 1999; Wilkins, Carlson, Van Vunakis, Hill, Gritz, & Jarvik, 1982; Winternitz & Quillen, 1977). In addition, although the findings are less consistent, there is also evidence of increased basal levels of cortisol in habitual smokers (Canal, Colomina, Domingo, & Domenech, 1997; del Arbol 2000; Field, Colditz, Willet, Longcope, & McKinlay, 1994). As a result, it is reasonable to assume that abstinence from cigarette smoking would be associated with a rebound decrease in cortisol levels. In fact, a number of studies do support this hypothesis and demonstrate a sharp decrease in cortisol concentrations following even a short abstinence from nicotine (Pomerleau, Pomerleau, & Marks, 2000; Meliska, Stunkard, Gilbert, Jensen, & Martinko, 1995). Moreover, there is evidence that these changes in cortisol levels persist for extended periods of time (i.e., up to six weeks) following smoking cessation (Gilbert et al., 1999;

Puddey, Vandongen, Beilin, & English, 1984; Steptoe & Ussher, 2006). However, it is also important to note that not all studies have found this effect (al'Absi, Amunrund, & Wittmers, 2002; Benowitz, Kuyt & Jacob, 1984; Hughes, Arana, Amori, Stewart, & Workman, 1988; Pickworth & Fant, 1998; Teneggi et al., 2002). However, many of these studies were limited by methodological issues (e.g. measurement and sampling issues) and the overall consensus in the literature supports the contention that nicotine withdrawal is followed by a decrease in cortisol concentrations both long and short term.

As noted earlier, smoking cessation is associated with a number of unpleasant withdrawal symptoms such as dysphoria, anxiety, irritability, difficulty concentrating, restlessness, appetite dysregulation, weight gain, and insomnia (American Psychiatric Association, 1994; Hughes, 1992, Hughes, Gust, Skoog, Keenan, & Fenwick, 1991; Hughes and Hatsulami, 1986). Incidentally, this symptom profile is similar to that experienced during episodes of acute stress (Hughes 1992, Selye, 1976). This has led to the hypothesis that the sudden drop in cortisol following abstinence from smoking may serve as a marker of intensity of withdrawal symptoms and dependence, and can potentially lead to an increased risk of relapse possibly as a way to self-medicate and ameliorate these unpleasant symptoms (al'Absi, Hatsukami, Davis & Wittmers, 2004; Piasecki, Jorenby, Smith, Fiore, & Baker, 2003). A number of studies seem to support the hypothesis that cortisol levels following nicotine abstinence are associated with withdrawal symptoms both in the immediate period following cessation (Cohen, al'Absi, & Collins, 2004), and up to several weeks post-quit (Frederick et al., 1998; Ussher et al, 2006). In addition, it appears that the delta (i.e. change) between baseline cortisol and post-quit levels is particularly important in determining severity of distress

(Frederick et al., 1998). In addition, al'Absi and colleagues (2004) looked at the role of cortisol levels on mood states and withdrawal symptoms during the first 24-hours of abstinence in predicting early smoking relapse (i.e. < one week). They found that although all smokers experienced significant withdrawal symptoms and changes in diurnal cortisol levels, early relapsers evidenced a greater decline in morning cortisol concentrations along with greater withdrawal symptoms, cravings, and distress compared to those who maintained abstinence. Therefore, although there have only been a small handful of studies that have directly examined the association between cortisol and symptoms of distress and withdrawal, it appears that the cortisol decrease following smoking abstinence may represent a marker of intensity of withdrawal effects and contribute to risk of early relapse.

Finally, it has been found that stress is an important risk factor for smoking relapse (e.g. Cohen & Lichenstein, 1990; Shiffman et al., 1996; Wills et al., 2002). Because cortisol and the HPA axis are heavily implicated in the stress response it is hypothesized that the chronic effects of nicotine may inhibit the HPA axis from responding adequately to other challenges (Al'Absi, Wittmers, Erickson, Hatsulami, & Crouse, 2003). In addition, because smoking cessation is associated with a decrease in cortisol concentrations, and cortisol production is an important component of the stress response this decline may make it difficult for the individual to mount an effective stress response which can leave an individual vulnerable to relapse in the absence of other possible coping mechanisms. There is some evidence to suggest that abstinence from smoking causes a hyporesponsiveness towards stress in habitual smokers (al'Abis 2003) though this finding has not been consistently demonstrated (al'Absi, Amunrud, &

Wittmers 2002). In addition, there is some evidence to suggest that these alterations in the stress response contribute to or mediate the stress-withdrawal symptoms associated with smoking cessation and relapse. In particular, al'Absi and colleagues (2005) examined the extent to which cardiovascular and cortisol response to stress during initial nicotine withdrawal predicted early relapse. They had smokers who had been abstinent for 24-hours complete a series of psychological stressors (e.g. public speaking and cognitive challenges) and found that men (but not women) who relapsed within four weeks evidenced an attenuated cortisol response to stress compared to male abstainers. In addition, those who relapsed also showed reduced blood pressure response to stress, exaggerated withdrawal symptoms and mood deterioration. Moreover, these responses predicted relapse even when controlling for baseline smoking and psychological measures, suggesting that an altered stress response at least partially due to changes in cortisol concentrations in men (but not necessarily women) is associated with an increased vulnerability for smoking relapse. Therefore, although the literature is scant, there is some suggestion that the decline in cortisol concentrations following nicotine withdrawal may interact with a hyporesponsiveness to stress that can be predictive of early relapse.

Overall, the results of numerous studies provide evidence to suggest that the HPA axis, particularly cortisol plays an important role in smoking relapse. Cortisol has been shown to increase following acute and habitual nicotine intake. In addition, there is evidence that smoking cessation is associated with a rebound decrease in cortisol which may serve as a marker of dependence and be associated with withdrawal symptoms and subsequent smoking relapse. Furthermore, there is a nascent literature that shows a



relationship between this decrease in cortisol levels, withdrawal symptoms, hyporesponsiveness to stress and subsequent smoking relapse.

### *Distress Tolerance*

Distress tolerance is broadly defined as the ability to experience and withstand negative psychological states (Linehan, 1993). Although its roots are in the area of affect regulation in borderline personality disorder (BPD), in recent years it has been subject to increased attention in the area of substance use and relapse. As we have discussed, smoking relapse is associated with a number of unique predictors, however in some individuals it may not be the presence of these risk factors specifically, but rather the individual's ability to tolerate them during a quit attempt that determines early smoking relapse. For example, an individual's ability to withstand the initial psychological and physical stress and discomfort associated with smoking cessation and everyday life may be a key determinant in whether or not that individual remains abstinent or lapses back into smoking as a way to ameliorate these feelings of discomfort.

Eisenberger's (1992) theory of learned industriousness provides a conceptual framework for researchers to examine the relationship between an individual's ability to tolerate discomfort and smoking cessation outcome. Learned industriousness theory posits that the amount of effort (i.e. aversive, subjective experience that occurs when information processing, physical motion, or both are obstructed or fatigued) that an individual is willing to exert is dependent upon the degree of aversiveness associated with the task, which is a function of prior learning history. Therefore, learned industriousness is a learning based theory of motivation that posits that individuals with a history of being rewarded for high effort will be more likely to persist at an effortful task

than these with a history of being rewarded for low effort who will regard the task as being highly aversive. Specifically, quitting smoking can be viewed as a high-effort task that often times requires several attempts before being successful. Moreover, ex-smokers must utilize a variety of cognitive and behavioral coping skills in order to successfully avoid smoking and effectively deal with the withdrawal symptoms, cravings, and negative affect that often accompanies early nicotine withdrawal. According to the learned industriousness theory, those ex-smokers with a history of being rewarded for high effort will be more likely to persist in employing these alternative coping skills compared to those with reward histories of low effort. Moreover, those individuals who exhibit high levels of effort may be more likely to delay the immediate reinforcement of smoking (i.e. the pharmacological aspects of smoking) in exchange for the delayed but long-term reinforcement of good health.

There have been several studies which demonstrated a relationship between the ability to tolerate discomfort and smoking cessation. For example, a series of studies by Hajek and colleagues (Hajek, 1991; Hakeck, Belcher, & Stapleton, 1987; West et al., 1989) found that breath-holding duration, a measure of physical discomfort was significantly correlated with duration of subsequent smoking abstinence. However, Quinn, Brandon and Copeland (1996) were the first to apply the theory of learned industriousness (as defined by task persistence) to smoking relapse. Fifty-two heavy smokers (i.e. >20 cigarettes per day) and 57 nonsmokers participated in two psychologically distressing tasks, the anagram persistence task (APT) and the mirror tracing persistence task (MTPT). The anagram persistence task (APT; Eisenberger & Leonard, 1980) requires subjects to complete a series of difficult anagrams, while the

mirror-tracing persistence task (MTPT) is a stressor task that requires the subject to trace the outline of a geometric figure while viewing it through a mirror. Results indicated that nonsmokers were more persistent than smokers on the APT and the MTPT, and that histories of drug and alcohol abuse were inversely related to task persistence. However, it is important to note that this study used a cross sectional design so the results are correlational in nature.

Because prospective or experimental designs are necessary to determine the direction of causality, Brown, Lejuez, Kahler, & Strong (2002) looked at the role of distress tolerance in predicting early smoking relapse in a group of 32 current smokers. Based on their retrospective reporting of past quit attempts, participants were divided into two groups; those smokers who had completed at least one sustained quit attempt lasting three months or longer (delayed relapsers), and those who had failed to quit smoking for more than 24 hours (immediate relapsers). All participants were exposed to a mental arithmetic stressor (PASAT) and two physical challenges consisting of a breath holding task and inhalation of carbon dioxide enriched air. These tasks were performed during both a normal ad libitum smoking day and following a 12-hour smoking abstinence. Results indicated that immediate relapsers endorsed higher levels of baseline depressive symptoms, a greater tendency to react to stress with negative affect, and higher increases in dysphoria and the urge to smoke following the 12-hour nicotine deprivation. Furthermore, immediate relapsers exhibited less behavioral persistence compared to delayed relapsers. These results suggest that these individuals may be particularly vulnerable to early smoking relapse due to increased emotional distress in response to smoking abstinence combined with a tendency not to persist in the face of physical or

psychological discomfort. In a second study (Lejuez, Zvolensky, Leen, & Feldner, 2001), task persistence on the PASAT was compared to retrospective reports of previous smoking cessation attempts ranging from several hours to six months. Longest cessation duration was positively associated with task persistence even after controlling for nicotine dependence.

While these studies provide good initial evidence that distress tolerance plays a role in smoking relapse, prospective studies are necessary to determine the predictive ability of distress tolerance. A study by Brandon and colleagues (2003) used the same methods employed in the Quinn and Brown (1996) study but utilized a longitudinal design. All subjects participated in a pretreatment assessment of task persistence using the APT and MTPT before receiving a six session cognitive-behavioral smoking cessation treatment and a nicotine replacement therapy. Following the treatment, subjects were divided into three groups; treatment non-completers, those who completed treatment but relapsed, and those who remained abstinent until follow-up. They found that task persistence on the MTPT (but not the APT) predicted smoking abstinence across 12 months above and beyond other significant predictors such as gender, nicotine dependence, negative affect, and self-efficacy. In a more recent study, Brown and colleagues (2004) administered the PASAT, breath-holding and CO<sub>2</sub> challenge tasks to a group of 81 smokers who were attempting to quit smoking on their own. They found that although about 20% of the subjects were able to remain abstinent during the full 28-day follow-up, almost 65% lapsed within the first week. In addition, those individuals who lapsed and were not able to remain abstinent demonstrated a lower level of persistence on the two physical stressors (i.e. breath holding and CO<sub>2</sub>), but not on the PASAT.

Moreover, proportional hazard models showed that those individuals low in task persistence had a 2.94 time greater risk of lapsing over the course of the 28-day follow up compared to those with high task persistence, and 1.75 times greater risk compared to those with average levels of persistence.

Although distress tolerance is a relatively new construct with respect to smoking research, results from a handful of studies suggest that it plays an important role in early smoking relapse. Its effects have been demonstrated both retrospectively and prospectively, and persist regardless of whether the individual self-quits or enlists in a formal smoking treatment program. However, it is important to note that these various studies are all limited to some degree. For example, the early studies on distress tolerance were cross sectional in nature and considered past quit attempts, while the prospective studies focused on self-quitters (Brown et al., 2006). In addition, these later studies tended to be limited to assessing psychological distress tolerance. As a result, they failed to include other measures of distress tolerance and other relevant variables (e.g. anxiety sensitivity, cortisol) which help provide a more comprehensive picture of distress tolerance and its unique relationship to relapse (Brandon et al., 2005).

#### Statement of Problem

Despite increased awareness of the dangers of smoking and broad-based public health efforts to promote smoking cessation, over 44.5 million Americans continue to smoke regularly (CDC, 2005b). Although smoking treatments significantly improve the success of quitting when compared to self-quitting, relapse rates are still high. In addition, many individuals who attempt to quit, lapse within a few days, and many of these individuals ultimately resume smoking and are not able to recover to achieve abstinence. Therefore,

it is imperative to understand the significance of the early smoking lapse and isolate factors specific to this subgroup of individuals that makes it difficult for them to quit smoking. Although a number of variables such as withdrawal symptoms, negative affect and anxiety sensitivity have been found to play a role in early smoking relapse, the exact mechanisms underlying these factors that contribute to relapse is still unclear. One particular hypothesis is that it is not necessarily the presence of any of these individual factors, but rather how the individual reacts to, and tolerates them that may be a key contributing factor of relapse. Therefore, for some individuals it may be an inability to tolerate the withdrawal symptoms and negative affect associated with smoking cessation that leads to the initial smoking lapse and subsequent relapse as a way to cope with or ameliorate their distress. This threshold for tolerating physical and psychological stress is known as distress tolerance.

While early evidence has suggested that distress tolerance is associated with duration of smoking quit attempts, to date, no study has examined the effects of distress tolerance across physical, psychological, and biological domains on a number of other relapse predictors (e.g. negative affect, anxiety sensitivity and withdrawal symptoms) in determining smoking outcome. Therefore, the following study looked at the role of these variables in predicting smoking outcome in a group of 58 smokers participating in a smoking cessation study. Specifically, each participant completed a battery of distress tolerance measures including two psychological distress tolerance tasks, two physical challenges, a self-report measure of distress tolerance, and a measure of HPA reactivity. Participants then received the Nicoderm CQ nicotine patch in conjunction with an 8-week group smoking cessation treatment. They were assessed weekly to determine smoking

abstinence. As such, this study provides a comprehensive picture of the role of distress tolerance in an ecologically valid way by using both behavioral and NRT smoking treatment components as indicated by Fiore and colleagues (2000). Results from this study have important treatment implications in terms of identifying smokers at high risk for early smoking relapse and developing novel pharmacological and psychosocial interventions to target this at-risk group of smokers.

### Hypotheses

1. Early lapsers will evidence higher levels of negative affect (as measured by state negative emotionality, depressive symptoms and anxiety sensitivity) compared to delayed lapsers.
2. Early lapsers will exhibit higher levels of negative affect-related withdrawal symptoms in response to smoking abstinence compared to delayed lapsers.
3. Early lapsers will demonstrate lower levels of distress tolerance (as measured by task persistence and salivary cortisol) in response to a series of four laboratory challenge procedures and a self-report measure compared to delayed lapsers.
4. Early lapsers will not differ from delayed lapsers in baseline state positive emotionality and withdrawal symptoms not related to affect
5. Using a regression approach, distress tolerance across its various components will evidence incremental validity beyond negative affect-related withdrawal symptoms, depression, and anxiety sensitivity in the prediction of early smoking lapse.
6. Self-report, behavioral and biological indices of distress tolerance will demonstrate convergent validity.



## Chapter 2: Methods and Procedure

### Participants

Participants consisted of 58 adults with elevated depressive symptoms ( $M$  BDI at baseline = 9.89;  $SD$  = 7.55) who were motivated to quit smoking. Participants were recruited as part of a larger study examining a novel behavioral activation treatment program for depressed smokers. The final sample included 35 males and 23 females with a mean age of 44.7 years ( $SD$ =11.87). The sample was predominately African American (75.4%) and Caucasian (19.3%), with 1.8% Hispanic/Latino, 1.8% Native American, and 1.8% other. With respect to education, 8.8% of participants completed some high school or less, 26.4% completed high school/GED, 45.6% finished at least some college or technical school, 5.3% graduated college, and 14.1% had at least some graduate education. In terms of employment status, 42.8% of the sample was employed, 32.2% were unemployed, 9% were students, and 14.3% were retired. Finally, with respect to smoking history, participants smoked an average of 17.4 ( $SD$ =8.5) cigarettes a day, had been smoking an average of 23.7 ( $SD$ =12.24) years, and had tried to quit 3.8 ( $SD$ =3.5) times.

### Screening Measures

#### *Phone Screen*

All participants completed a brief ten minute phone screen. In addition to general demographic information, the screener also included questions related to smoking behavior, current use of any psychotherapy treatment or psychotropic medications, current use of nicotine replacement therapy or other tobacco products, and current

medical conditions. During this phone screen, participants were also administered the Beck Depression Inventory-II (BDI) to assess for elevated depressive symptoms. In order to be eligible for the larger treatment outcome study, participants needed to score a 12 or higher on the BDI during their phone screen. However, it is important to note that for the current study, BDI scores were not of particular interest and did not serve as an exclusionary variable. Therefore, although many participants subsequently endorsed lower scores when they were given the BDI a few weeks later at baseline, this drift is not of concern for the current study.

### *Beck Depression Inventory-II*

The Beck Depression Inventory-II (Beck, Steer, & Brown, 1996) is a 21 item self report measure that assesses depressive symptoms over the past week. Questions are rated on a 4-point likert scale where a value of 0 means that the statement does not describe the individual at all and a value of 3 means that it describes them very well. The individual's total score is then used to determine the existence and severity of the depression. According to the BDI-II, a score of 0-13 indicates minimal depression, 14-19 is mild depression, 20-28 is considered moderate depression, and 29-63 indicates severe depression.

The BDI-II has been shown to be a reliable and well validated tool for the assessment of depressive symptoms. Internal reliability is high, ranging from .89 to .93 in student samples (Beck, Steer, & Brown, 1996; Steer & Clark, 1997; Whisman, Perez, & Ramel, 2000) to .89-.92 in psychiatric samples (Beck, Steer, & Brown, 1996; Steer, Rissmiller, & Beck, 2000). Internal consistency of the BDI-II is good with a Cronbach alpha around 0.85 (Ambrosini et al., 1991). In terms of validity, the BDI-II displays good

criterion validity, with patients diagnosed with major depression scoring significantly higher on the BDI-II than those without depression (Arnau, Meagher, Norris, & Bramson, 2001). In addition, the BDI-II has evidenced good discriminant and convergent validity in that it correlates more strongly with measures of depression compared to measures of anxiety (Beck, Steer, & Brown, 1996).

#### *Structured Clinical Interview for DSM-IV (SCID)*

During intake, all participants were administered the Structured Clinical Interview for DSM-IV-non-patient version (SCID-NP; First, Spitzer, Gibbon, & Williams, 1995) to assess for current DSM-IV Axis I disorders present during the past year. The SCID-NP is a clinician administered semi-structured clinical interview that provides coverage of all major DSM-IV Axis I disorders. Because the measure is so comprehensive, the format of the SCID-NP utilizes a screener to determine which specific modules (each representing a distinct class of disorders) need to be covered, along with various skip-out instructions which allow the interviewer to leave a particular module once it becomes clear that the individual does not meet criteria for that particular diagnosis.

Studies on the psychometric properties of the SCID-NP have shown the measure to have good reliability and validity. In terms of the reliability of the SCID-IV, test-retest reliability was found to range from poor (.35 for dysthymic disorder) to excellent (.78 for PTSD) for the various disorders (Zanarini et al., 2000). In terms of validity, the SCID-IV has been compared to a best-estimate diagnosis using the LEAD standard (Spitzer, 1983) which includes a longitudinal assessment conducted by an expert diagnostician using all available data (e.g. family interviews, medical records, behavioral observations). Two

studies utilizing an approximation of this standard found superior validity of the SCID compared to standard clinical interviews (Basco et al., 2000; Kranzler et. al., 1996).

### *Medical History*

Participants also completed a brief medical history questionnaire that focused primarily on potential contraindications for using the nicotine patch (e.g. significant medical illness, cardiovascular illness, pregnancy, etc...). In addition, all participants meet with the study physician for a brief physical to determine whether or not they were eligible to receive the nicotine patch. In this sample there were no participants excluded from the study for medical reasons.

### *Procedures*

#### *Baseline Assessment*

All participants who were deemed eligible following the initial phone screen were scheduled to complete the baseline assessment. Upon arrival for their baseline session, written informed consent was obtained and a trained graduate student administered the SCID-NP (Axis I), to determine if any DSM-IV psychopathology exclusion criteria were met. Specifically, participants were excluded if they endorsed criteria for any current DSM-IV disorder or met criteria for any psychoactive substance use or dependence (excluding nicotine) within the last six months prior to coming to the study. Individuals who were not eligible for the study were thanked for their participation, provided with mental health referrals if necessary, and given a self-help booklet on smoking cessation prepared by the National Cancer Institute (Clearing the Air; USDHHS, 1995).

All laboratory tasks were conducted by the experimenter. Prior to the initiation of the experimental tasks, baseline salivary cortisol readings were obtained. The participant then engaged in each of the four distress tolerance tasks described in detail below. These tasks included two psychological (i.e. PASAT, MTPT-C) and two physical (i.e. breath holding and Cold Pressor) stressors. In order to increase motivation, participants were told that their performance on each task would influence the amount of money they could earn for the study and as such, they were instructed to try and do their best. However, in reality this was not the true and all participants received the same compensation regardless of performance. The order of the presentation of the various tasks was counterbalanced across participants to control for order effects. Upon completion of the stressor tasks, the participants completed a variety of self-report measures. Finally, 20 minutes after completion of the last stressor task, the participants provided a second salivary cortisol sample to assess for HPA reactivity to stress. Participants were paid \$20 at the conclusion of the study.

#### *Smoking Cessation Treatment*

Following their initial baseline assessment, participants completed one of two eight-week smoking cessation treatment groups. Although both groups included standard behavioral treatment components (e.g. coping skills, social contracting, relapse prevention, relaxation), the experimental group also integrated elements of behavioral activation modified for smoking (i.e. pleasurable activity setting in line with a smoke-free lifestyle). In addition participants in both groups received eight weeks of the Nicoderm CQ transdermal nicotine patch. There were no significant differences between treatment groups and smoking outcome ( $p > .05$ ), therefore in the subsequent analyses treatment

condition was collapsed across groups. Attrition was fairly high throughout treatment, with a drop out rate of almost 40% by quit day (week 4), and 46% drop out rate at the end of treatment (week 8). These rates are consistent with other smoking cessation treatment studies which find high levels of attrition between baseline and initiation of treatment (30%-50%), with rates tapering off as treatment progresses (Curtin, Brown, & Sales, 2000).

### Laboratory Tasks

#### *Paced Auditory Serial Addition Task (PASAT)*

All participants completed a modified computerized version of the Paced Auditory Serial Addition Task (PASAT; Diehr, Heaton, Miller & Grant, 1998; Lejuez, Kahler, & Brown, 2001), an addition task that has been shown to increase participants' stress levels (Brown, Lejuez, Kahler, & Strong, 2002; Daughters, Lejuez, Bornoalova, Kahler, Strong, & Brown, 2005; Daughters, Lejuez, Kahler, Strong, & Brown, 2005; Deary, Ebmeier, MacLeod, Dougall, Hepburn). During the task, numbers were flashed sequentially across a computer screen and participants were instructed to add the current number to the previously presented number. They were then told to click on the correct sum using a keyboard provided on the computer screen. The participant received one point for each correct answer with the total number of points earned displayed in a box on the right-hand side of the screen. The task consisted of three levels which progress in terms of difficulty. The first level of the PASAT lasted one minute and provided a three-second latency between number presentations (i.e. low difficulty) while the second level lasted for two minutes level and provided a two-second latency (i.e. medium difficulty). During the third and final level, the latency between number presentations was one-

second (i.e. high difficulty). The third level lasted for up to seven minutes with the participant having the option to terminate the level at any time by clicking a quit button provided on the computer screen. Distress tolerance was measured as the latency in seconds to task termination. In addition, the number of points that the participant accrued over the course of the task was recorded to control for proficiency/skill on task persistence. Finally, the experimental administration of a six item dysphoria scale occurred before the beginning of the task and at the end of the final level of the PASAT to determine if the task increased psychological stress. This scale consisted of six single-item ratings that were designed to assess moment-to-moment levels of anxiety, irritability, discomfort, and frustration (Brown et al., 2002). A more detailed discussion of this scale is provided later.

#### *Computerized Mirror-Tracing Persistence Task (MTPT-C)*

The Computerized Mirror-Tracing Persistence Task (MTPT-C; Strong, Lejuez, Daughters, Marinello, Kahler, & Brown, 2005) is a computerized version of the Mirror Tracing Persistence Task (MTPT; Quinn, Brandon, & Copeland, 1996). For the MTPT-C, participants were instructed to trace a dot along the lines of various shapes using the computer mouse. However, to make the task similar to the original, the mouse was programmed to move the red dot in the opposite direction than that showed on the screen (i.e. like a mirror). Therefore, when the participant moved the mouse down, the red dot would move up, and so forth. In order to increase the degree of difficulty and frustration, each time the participant moved the mouse out of the lines or stopped moving the mouse for more than two seconds, a loud buzzer sounded and the red dot moved back to the beginning of the shape. Similar to the PASAT, there were three rounds of the

MTPT-C with each shape presented progressing in difficulty. The first two rounds lasted one minute each while the third round of the MTPT-C lasted up to seven minutes.

Participants were instructed that they had the option to terminate the task at any point during the third round by pressing on the space bar. As with the PASAT, distress tolerance was measured by the latency in seconds to task termination. Additionally, the number of errors per second (i.e., number of times the participant had to return to the starting position during the task divided by the task time) was recorded to control for the effects of skill on persistence. Finally, the participant completed the dysphoria scale at both the beginning and end of the MTPT-C to determine if the task caused an elevation in psychological distress. The original written version of the MTPT has been shown to increase stress (Matthews & Stoney, Tutoo, 1971) and has demonstrated good reliability ( $\alpha = .92$ ; Brandon et al., 2003).

#### *Physical Challenge: Breath Holding Task*

Breath holding is a common task used to assess for physical distress tolerance and has been found to be predictive of length of time to smoking relapse (Brown et al., 2002; Hajek, Belcher, & Stapleton, 1987). During this task participants were instructed to take a deep breath and hold it for as long as they can. They were then asked to notify the experimenter when they began to feel uncomfortable by holding up a sign that signified they were feeling discomfort. However, the participants were instructed to continue holding their breath beyond that point of initial discomfort for as long as possible.

Distress tolerance was measured as the latency in seconds between when the participant began to feel uncomfortable and when they finally let out their breath. This procedure is



safe and has been used in a previous large-scale study looking at distress tolerance in substance users (Daughters et al., 2005).

*Pain Challenge: Cold Pressor Task (CPT)*

The cold pressor task is a commonly used measure of pain that involves having the participant submerge their hand in a bucket of freezing cold water (0-2 degree Celsius) a stimulus which produces a gradual escalation of pain (Shumate & Worthington, 1987; Willoughby, Hailey, Mulkana, & Rowe, 2002). Similar to the breath holding task, the participant was instructed to notify the experimenter when they began to feel uncomfortable by holding up a sign that indicated so. However, they were told to continue to keep their hand immersed in the cold water for as long as possible. Distress tolerance was measured as the latency in seconds between when the participant began to feel uncomfortable and when they finally terminated the task by taking their hand out of the water.

*Self Report Measures*

*Measures of Smoking History, Nicotine Dependence*

*Smoking History.* Smoking history was assessed at baseline using the smoking history and current status indices agreed upon by a National Cancer Institute consensus panel (Proceedings of the National Working Conference on Smoking Relapse, 1986). This included information such as: the rate of smoking, preferred brand of cigarette, nicotine content of preferred brand of cigarette, history and duration of previous quit attempts, number of household smokers, and age of onset of habitual smoking behavior.

*Fagerstrom Test for Nicotine Dependence (FTND).* The Fagerstrom Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991)

served as a measure of nicotine dependence. The FTND has shown good internal consistency, a single dimension factor structure, and a positive relationship with degree of nicotine intake as assessed by saliva cotinine (Heatherton et al., 1991). The FTND is considered to be the standard instrument in the field for measuring nicotine dependence.

### *Distress Tolerance*

The distress tolerance scale (DTS; Simons & Gaher, 2005) is a self-report measure of emotional distress tolerance. The questionnaire consists of 15 items designed to assess the individual's ability to tolerate emotional distress, their subjective appraisal of distress, degree to which their attention is absorbed by negative emotions, and regulation efforts to alleviate distress. Individual items are rated on a 5-point likert scale with higher scores being indicative of higher distress tolerance. Data on the psychometric properties of the DTS show that the measure demonstrates adequate reliability and validity. For example, the DTS exhibits good internal consistency with an alpha of .89 (Simon & Gaher, 2005). In addition, the measure exhibited good test-retest reliability ( $r = .61$ ) over the course of a six month period (Simon & Gaher, 2005). In terms of validity, the DTS demonstrated expected associations with other measures of affective functioning supporting its convergent and discriminant validity. Criterion validity was established by evidence of significant negative correlations with measures of substance abuse coping, but not enhancement motives (Simon & Gaher, 2005).

### *Affective States*

*Beck Depression Inventory-II (BDI)*. The Beck Depression Inventory-II was used to assess for current elevations in depressed mood. A brief summary of the scale and its psychometric properties have been described earlier.

*Positive and Negative Affect Schedule.* For a baseline measure of positive and negative affect, this study used the 20-item Positive and Negative Affect Schedule (PANAS; Watson, Clark & Tellegen, 1988). This instrument has shown good internal consistency and discriminant and convergent validity (Crawford & Henry, 2004).

*Revised Anxiety Sensitivity Index (ASI-R).* Anxiety sensitivity was measured via self-report using the Revised Anxiety Sensitivity Index (ASI-R; Taylor & Cox, 2001). The ASI-R is a 36-item self-report scale that uses a 5-point likert scale (0=very little to 4= very much) to assess fear of anxiety related symptoms. Anxiety sensitivity is measured across four factors including: fear of respiratory sensations, fear of publicly observable anxiety reactions, fear of cognitive dyscontrol, and fear of cardiovascular sensations. The ASI-R has been used extensively and has sound psychometric properties. In particular it has been found to have excellent internal consistency (alpha = 0.94) and adequate validity (Deacon, Abramowitz, Woods, & Tolin, 2003) in a nonclinical college sample.

#### *Nicotine Withdrawal Symptoms*

Withdrawal symptoms following smoking cessation were assessed weekly throughout treatment. Symptoms were measured using the Shiffman-Jarvik Smoking Withdrawal Questionnaire (Shiffman & Jarvik, 1976). This self-report measure consists of 25 items rated on a 7-point likert scale. This questionnaire has been used in previous smoking studies and has been shown to be a reliable and sensitive measure of the duration, severity, frequency, and temporal variability of nicotine withdrawal symptoms.

#### *Outcome Measures of Distress Tolerance*

##### *Task Persistence*

Task persistence served as a behavioral measure of distress tolerance on each of the four laboratory challenge tasks and was measured as latency in seconds to task termination.

### *Dysphoria*

A series of single-item questions rated on a Likert scale ranging from 0 (none) to 100 (extreme) were used to assess moment-to-moment levels of anxiety, irritability, discomfort, and frustration (Brown et al., 2002). These items were selected on the basis of their relevance for sensitivity to psychological distress. A total score was derived by summing the score on each item to obtain measurement of general.

### *Biological Measure*

*Cortisol.* In order to examine the role of biological arousal, an indices thought to be related to distress intolerance, salivary cortisol samples were obtained at baseline and 20 minutes after the final laboratory challenge. The baseline sample of cortisol gave an indication of baseline cortisol/HPA functioning while the 20 minute sample provided information on peak HPA reactivity. HPA reactivity was defined as the difference in cortisol levels between the two collection points. Cortisol samples were immediately frozen and sent to Salimetric Inc (State College, PA) for cortisol level determination by radioimmune assay.

### Measures of Smoking Status

#### *Timeline Follow-back Calendar*

A self report measure of smoking status was obtained from participants on their quit day and during their 1, 2, and 4-week post-quit day follow-ups. Specifically, all subjects completed a timeline follow-back (TLFB) calendar at each follow-up session to

assess for any cigarette use since the previous follow-up. In addition, for those individuals who did not remain abstinent, the TLFB was used to assess the time to first smoking lapse. The TLFB procedure has demonstrated good reliability and validity with adult alcoholics (Sobell & Sobell, 1979; 1980; 1996) and has recently been validated for the assessment of adult cigarette use (Brown et al., 1998).

#### *Biochemical Verification of Smoking Status*

For cases of self-reported abstinence a carbon monoxide analysis of breath samples was used to verify abstinence (Jarvis, et al., 1987). Expired air carbon monoxide levels were assessed using the Bedfont Micro 4-Smokerlyzer carbon monoxide monitor. Detected values above the cutoff score of 8ppms were considered an indication of recent smoking.

## Chapter 3: Results

### Relationships among Distress Tolerance Measures

Descriptive statistics for each distress tolerance and affect measure as well as the correlations among these measures are discussed below and these correlations are presented in Table 1.

#### *Psychological Distress Tolerance*

Overall, individuals persisted on the MTPT-C for an average of 297.5 seconds ( $SD = 144$ ). The mean level of dysphoria increased following the MTPT-C ( $M = 30.1$ ;  $SD = 28.3$ ) compared to the assessment prior to the task ( $M = 14.6$ ;  $SD = 18.5$ ;  $t(57) = -5.68$ ,  $p < .001$ ], suggesting the task was considered psychologically stressful by the participants.

To control for skill on the MTPT-C, the number of errors per second (EPS) was calculated by dividing the MTPT-C time by the number of errors. There was a significant relationship between EPS and MTPT-C duration, such that individuals with fewer EPS tended to persist on the task longer than those with more EPS [ $r(58) = -.339$ ,  $p < .01$ ]. Given this finding, EPS was used as a covariate in further analyses to partial out the effect of skill on persistence.

With respect to the PASAT, individuals persisted on the task for an average of 286 seconds ( $SD = 160.3$ ). The mean level of dysphoria at the start of the task was 16.8 ( $SD = 16.4$ ) and at the end of the task was 30.8 ( $SD = 24.6$ ). The overall difference in level of dysphoria following administration of the task was statistically significant [ $t(56) = -5.64$ ,  $p < .001$ ], suggesting that the task was psychologically stressful to the participants. In order to control for skill on the PASAT the number of correct responses

on the second level was used. There was no significant relationship between the number of correct responses and PASAT duration ( $p > .05$ ).

Correlations were then calculated to determine the relationship between the two psychological distress tolerance tasks. As hypothesized, the MTPT-C and the PASAT were significantly correlated even after controlling for EPS on the MTPT-C,  $r(54) = .383$ ,  $p < .01$ .

#### *Physical Distress Tolerance*

Overall, participants persisted with the Breath Holding task (BH) for an average of 38.6 seconds ( $SD = 15.3$ ). The difference score was calculated by subtracting the time at which the participant first began to feel discomfort from their overall breath holding duration. The mean difference score for BH was 12.4 seconds ( $SD = 7.7$ ). With respect to the Cold Pressor task (CP), the average amount of time they persisted on the task was 56.8 seconds ( $SD = 48.8$ ), and the difference score was 25.6 seconds ( $SD = 31.9$ ).

Correlations were then calculated to determine the relationship between the difference scores on the two physical distress tolerance tasks, and as hypothesized the two tasks were significantly correlated,  $r(56) = .633$ ,  $p < .01$ .

#### *Physiological Distress Tolerance*

Cortisol samples were obtained pre-and-post distress tolerance tasks and a difference level was obtained by subtracting the pre-task levels from the post-task levels to get a measure of cortisol change as a result of the tasks. The mean cortisol difference level was  $-.02$  ( $SD = .05$ ). The cortisol difference level was significantly positively correlated with MTPT-C quit time,  $r(53) = .387$ ,  $p < .01$  and negatively correlated with the CP difference score,  $r(51) = -.278$ ,  $p < .05$ . Specifically, these findings indicate that

the greater the cortisol difference (i.e. the greater the spike in cortisol following the four behavioral tasks), the longer the subject persisted on the MTPT-C, and the less time they persisted on the Cold Pressor.

#### *Distress Tolerance Scale Self-Report*

The overall score on the DTS was 3.3 ( $SD = .82$ ). With respect to the four subscales, the means were as follows: Tolerance 2.9 ( $SD = 1$ ), Regulation 3.2 ( $SD = 1$ ), Appraisal 3.5 ( $SD = .87$ ), and Absorption 3.5 ( $SD = 1$ ). As expected the four DTS subscales were all significantly correlated with one another (see Table 1).

#### *Relationships among Affective Self-Reports and Distress Tolerance Measures*

The relationship among affective self-reports measures and distress tolerance is also presented in Table 1. There were no significant relationships between the self-report measures and measures of psychological or physiological distress tolerance. There were several significant correlations between affective and physical distress tolerance measures. There were significant negative correlations between the BH difference score and negative affect withdrawal symptoms ( $r = -.39, p < .05$ ), and the PANAS PA scale ( $r = -.31, p < .05$ ). There was also a positive relationship between BH difference score and non affect-related withdrawal symptoms ( $r = .53, p < .01$ ). Finally, there was also a significant relationship between the CP difference score and non-affect related withdrawal symptoms ( $r = .39, p < .05$ ). With respect to the DTS, there were significant negative correlations between the DTS total score and the BDI ( $r = -.47, p < .01$ ) and the ASI ( $r = -.58, p < .01$ ). The BDI and the ASI-R were also negatively correlated with each of the four DTS subscales (see Table 1). These negative correlations suggest that as the BDI and ASI scores increased, scores on the DTS decreased.



A number of the affective self-report measures were significantly correlated with one another. Specifically, the BDI was correlated with the ASI-R ( $r = .41, p < .01$ ) and the PANAS NA scale ( $r = .48, p < .01$ ). The ASI-R was also correlated with the PANAS NA scale ( $r = .34, p < .05$ ). Finally, negative withdrawal symptoms was negatively correlated with non-affect related withdrawal symptoms ( $r = -.63, p < .01$ ).

### Smoking Status

Smoking abstinence was analyzed using a categorical measurement of early smoking lapse (< 3 day). Participants were separated into groups based upon those who endorsed having a smoking lapse within three days of quitting (early lapsers;  $n=34$ ) and those who abstained from cigarettes for three days or longer (delayed lapsers;  $n=24$ ). These categories were chosen based on findings from the smoking cessation literature which has found that many individuals who attempt to quit smoking lapse within the first few days of their quit attempt (e.g. Gulliver, Hughes, Solomon, & Dey, 1995; Hughes, Keely, & Naud, 2004). Table 2 presents the frequency distribution for the number of days until first lapse.

A separate set of analyses was initially planned to compare those participants who remained abstinent at least 30 days to those who relapsed. However, only six of the 58 participants (~10%) endorsed not smoking for at least one month which resulted in a sample too small to yield adequate power.

### Mean Differences between Early and Delayed Lapsers

#### *Demographic and Smoking History*

Table 3 presents differences between early and delayed lapsers with regards to age, gender, ethnicity, annual household income, employment status, education, number

of years smoking, current number of cigarettes, and number of past quit attempts. Annual household income was assessed using an 11-point likert scale ranging 1 to 11 with each level representing \$10,000 increments, such that 1 represented \$0-\$9,999 and 11 represented \$100,000 or more. These ratings were then separated into three groups for ease of analyses. With respect to employment status, individuals were categorized as employed if they endorsed either full or part-time working status. These individuals who endorsed being unemployed, a student, homemaker, or retired were categorized as unemployed. Finally, education level was assessed using a 10-point likert scale where 1 represented no education and 10 represented a graduate or professional degree. Again, for ease of analysis participants were divided into three categories which included some high school education or lower, high school graduate/GED, and some college and beyond.

There were no significant differences between early and delayed lapsers on any demographic variables. With respect to smoking history, there was a significant between group difference on current number of cigarettes smoked ( $p < .05$ ), with early lapsers endorsing smoking more cigarettes per day at baseline than delayed lapsers. Therefore, this variable was controlled for in subsequent analyses.

#### *Affective Self-report Measures*

Mean differences between early and delayed lapsers on affective self-report measures are presented in Table 4. There were no significant between group differences on measures of general negative and positive affect, depression, anxiety sensitivity, and withdrawal symptoms.

#### *Distress Tolerance Measures*

Group differences between the various measures of distress tolerance are presented in Table 4 and Figures 1-4. There were no significant between group differences on any of the DTS scales, MTPT-C and cortisol difference levels. However, there were significant differences on the PASAT,  $F(1, 55) = 9.12, p < .01$ , CP difference score,  $F(1, 53) = 6.53, p < .05$ , and BH difference score  $F(1, 53) = 4.26, p < .05$ , such that early lapsers displayed shorter task persistence on these measures compared to delayed lapsers.

#### *Predictors of Smoking Abstinence*

To determine the extent to which each significant distress tolerance measure predicts smoking outcome, a logistic regression analysis was conducted and results are presented in Table 5. Prior to analyses, all non-dichotomous variables were centered as way of reducing multicollinearity. In the first step of the model gender and average number of cigarettes smoked per day were entered. Although there was no significant difference between the two groups on gender, it was included in the model because it has been shown to be related to length of smoking abstinence (Wetter et al., 1999). The second step of the model included PASAT quit time and a composite score of physical distress tolerance (PHYSICAL) that was created by adding the z scores of the BH and CP difference scores. Overall, the first step of the model was not significant,  $X^2(2) = 4.67, p > .05$ , suggesting that the gender and number of cigarettes smoked per day do not reliably predict smoking status. Upon entering the distress tolerance measures in the second step, the overall model was significant,  $X^2(2) = 19.52, p < .001$ , and was able to correctly classify smoking status for over 69% of the participants. Within this completed model the PHYSICAL composite score (Wald = 4.84,  $p = .028$ ; OR = .515; 95% CI = .285-

.930), and PASAT quit time (Wald = 6.67,  $p = .01$ ; OR = .994; 95% CI = .989-.998) were both significantly related to smoking status

Table 1

*Means, standard deviations and intercorrelations among distress tolerance measures and affect self-reports*

Measure	M(SD)	1	2	3	4	5	6	7	8	9	10.	11	12	13	14	15	16
1. MTPT-C	297.5 (144)	---	<b>.36**</b>	.25	.12	<b>.29*</b>	.20	.24	<b>.27*</b>	.25	<b>.38**</b>	-.26	-.23	-.12	-.00	.03	.13
2. PASAT	286 (160.3)		---	.041	.10	-.03	-.06	-.11	.02	.06	-.06	-.10	-.20	-.07	-.07	-.11	-.13
3. BH_diff	12.5 (7.7)			---	<b>.63**</b>	.13	.12	.17	.10	.02	.14	-.04	-.19	.13	<b>-.39*</b>	<b>-.31*</b>	<b>.53**</b>
4. CP_diff	25.6 (32)				---	.24	.19	.24	.19	.13	<b>-.28*</b>	-.05	-.19	.19	-.34	-.22	<b>.39*</b>
5. DTS_Total	3.3 (.82)					---	N/A	N/A	N/A	N/A	-.07	<b>-.47**</b>	<b>-.58**</b>	-.07	-.17	.23	.30
6. DTS-Tol	2.9 (1)						---	<b>.44**</b>	<b>.56**</b>	<b>.56**</b>	-.00	<b>-.32*</b>	<b>-.40**</b>	-.12	-.29	.13	.33
7. DTS-Reg	3.3 (1)							---	<b>.65**</b>	<b>.49**</b>	-.08	<b>-.28*</b>	<b>-.57**</b>	.04	-.09	.05	.19
8. DTS-App	3.6 (.87)								---	<b>.71**</b>	-.01	<b>-.44**</b>	<b>-.49**</b>	-.14	-.08	.19	.23
9. DTS-Absorp	3.6 (1)									---	-.11	<b>-.52**</b>	<b>-.45**</b>	-.06	-.08	<b>.36*</b>	.28
10. Cort_diff	-.02 (.05)										---	.03	.03	-.03	.06	-.18	.07
11. BDI	9.89 (7.55)											---	<b>.41**</b>	<b>.48**</b>	.03	-.08	-.01
12. ASI	20.6 (11.9)												---	<b>.34*</b>	.03	.06	-.01
13. PANAS NA	15.8 (5.85)													---	-.01	.07	.09
14 Neg. Withdrawal	44.1 (7.31)														---	.20	<b>-.63**</b>
15. PANAS PA	32.8 (8.55)															---	-.15
16. Non-Affect. Withdrawal	20.1 (5.65)																---

\* p<.05; \*\* p<.01

Table 2:

*Frequency distribution table for number of days to first smoking lapse*

Days to First Lapse	Frequency	Percent	Cumulative Percent
0	34	58.6	58.6
3	8	13.8	72.4
7	7	12.1	84.5
14	2	3.4	87.9
21	1	1.7	89.7
≥28	6	10.3	100
Total	58	100	---

Table 3.

*Means and standard deviations of demographics and smoking history variables among the entire sample as well as group differences between early and delayed lapsers*

Variable	Overall Sample	Early Lapsers	Delayed Lapsers	Statistic	Cohen's d
Age	44.7 (11.8)	44.8 (11.9)	44.7 (11.9)	$t(56) = .141, p = 7.08$	.008
Gender (% Male)	60.3%	50%	75%	$\chi^2(1) = 3.67$	
Ethnicity (% African American)	74.4%	76.7%	75%	$\chi^2(1) = 2.88$	
Total Household Income				$\chi^2(2) = 8.91$	
\$0-\$39,999	64.8%	67.7%	60.8%		
\$40,000-\$79,999	24%	25.8%	21.7%		
\$80,000+	11.1%	6.4%	17.3%		
Employment Status (% Unemployed)	41%	37.5%	45.8%	$\chi^2(1) = 7.08$	
Education Level				$\chi^2(2) = 8.07$	
Some High School or Lower	8.7%	15.1%	0%		
High School Graduate/GED	26.3%	30.3%	20.8%		
Some College/Technical School/College Graduate	64.9%	54.5%	79.1%		
Length of Smoking (Months)	284.7 (146.9)	307.2 (139.2)	253.4 (154.5)	$t(56) = .364, p = .549$	.365
Number of Cigarettes Smoker per Day at Baseline	17.4 (8.5)	18.3 (10.4)	16 (4.7)	$t(56) = 6.54, p = .013^*$	.285
Number of Time Attempted to Quit	3.8 (3.6)	3.7 (3.9)	3.9 (3)	$t(56) = .272, p = .604$	-.05

\* $p < .05$

Table 4.

*Means, standard deviations, and group differences on self-report and distress tolerance measures between early and delayed lapsers*

Measure	Early Lapsers		Delayed Lapsers		F	Partial Eta Squared
	Mean	SD	Mean	SD		
BDI Total Score	10.96	8.11	8.45	6.62	1.33	.025
ASI Total Score	22.69	11.92	17.78	11.54	2.38	.045
PANAS NA Scale	15.80	6.16	15.95	5.52	.001	.000
Negative Withdrawal Sxs	47.33	5.72	42.71	7.60	2.48	.084
PANAS PA Scale	33.83	8.53	31.38	8.58	1.50	.030
Non-Affect Withdrawal Sxs	17.89	4.46	21	5.94	1.79	.062
DTS Total Score	3.27	.83	3.42	.83	.361	.007
DTS Tolerance	2.85	.98	3.17	1.12	1.27	.023
DTS Regulation	3.16	1.08	3.40	.99	.885	.016
DTS Appraisal	3.59	.92	3.56	.81	.048	.001
DTS Absorption	3.56	1.13	3.60	1.06	.000	.000
MTPT-C	288	146.84	311.13	141.83	.296	.005
PASAT	236.53	173.45	359.21	104.40	<b>9.12**</b>	.144
CP Difference Score	17.28	20.93	36.75	40.37	<b>6.53*</b>	.110
BH Difference Score	11.09	7.04	14.30	8.32	<b>4.26*</b>	.074
Cortisol Difference Score	-.014	.062	-.018	.040	.085	.002

\*p<.05, \*\*p<.01



Figure 1.

*Mean task persistence (seconds) between early and delayed lapsers on the PASAT and*

*MTPT-C tasks*

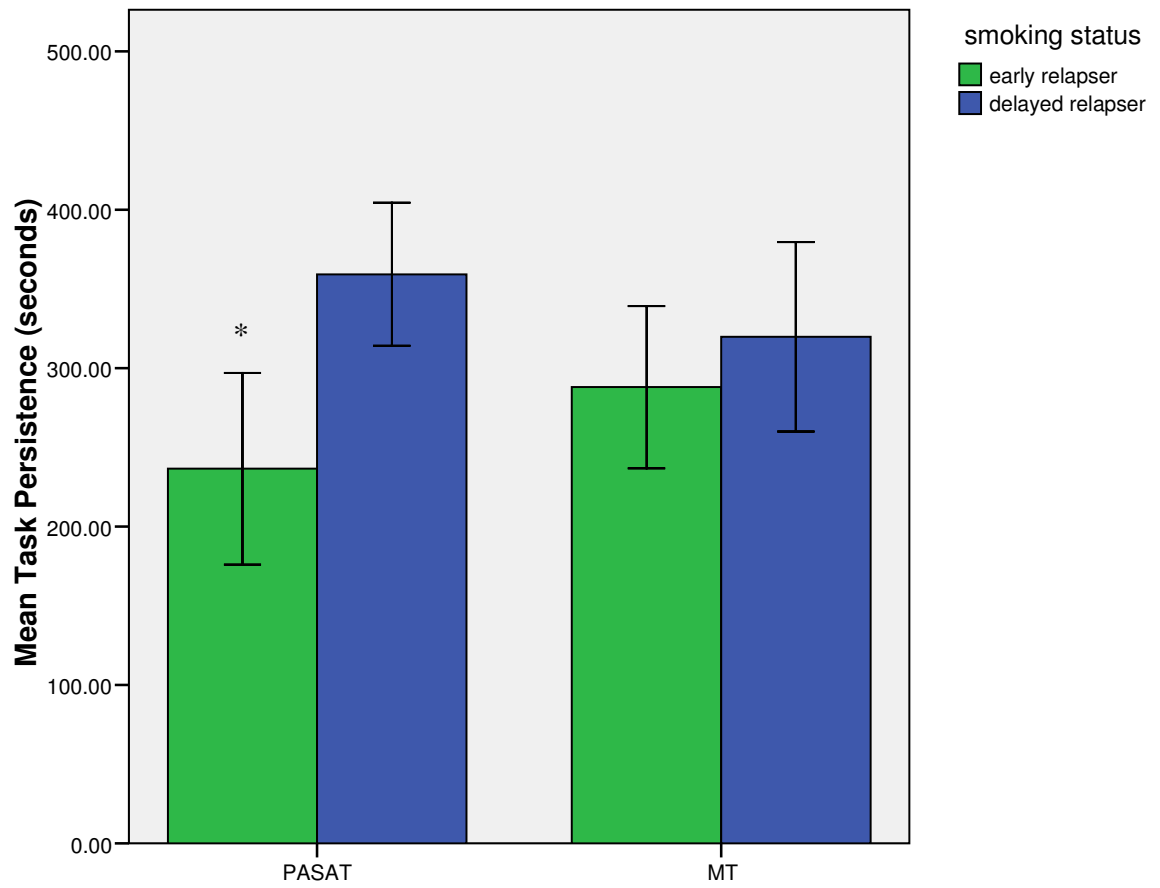


Figure 2.

*Mean difference score (seconds) between early and delayed lapsers on the BH and CP*

*tasks*

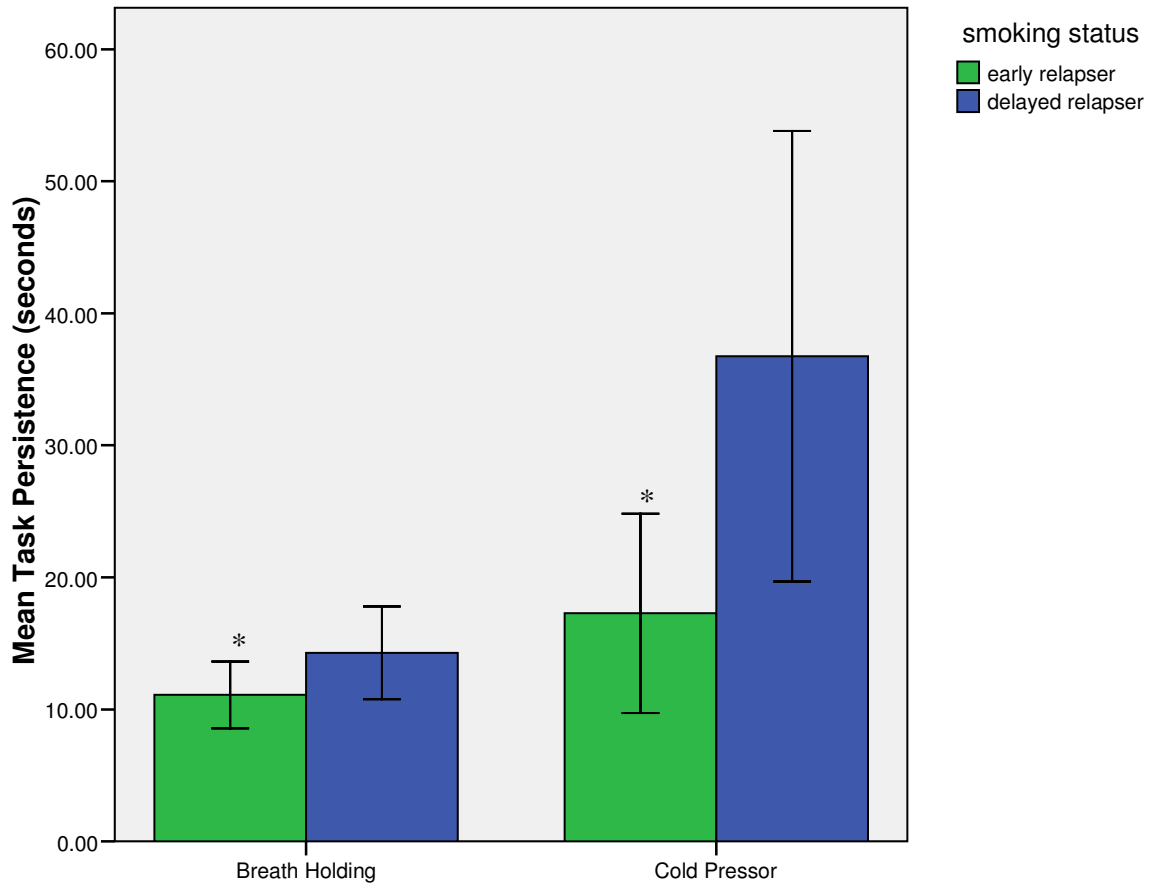


Figure 3.

*Mean scores between early and delayed lapsers on the DTS (total score and subscales)*

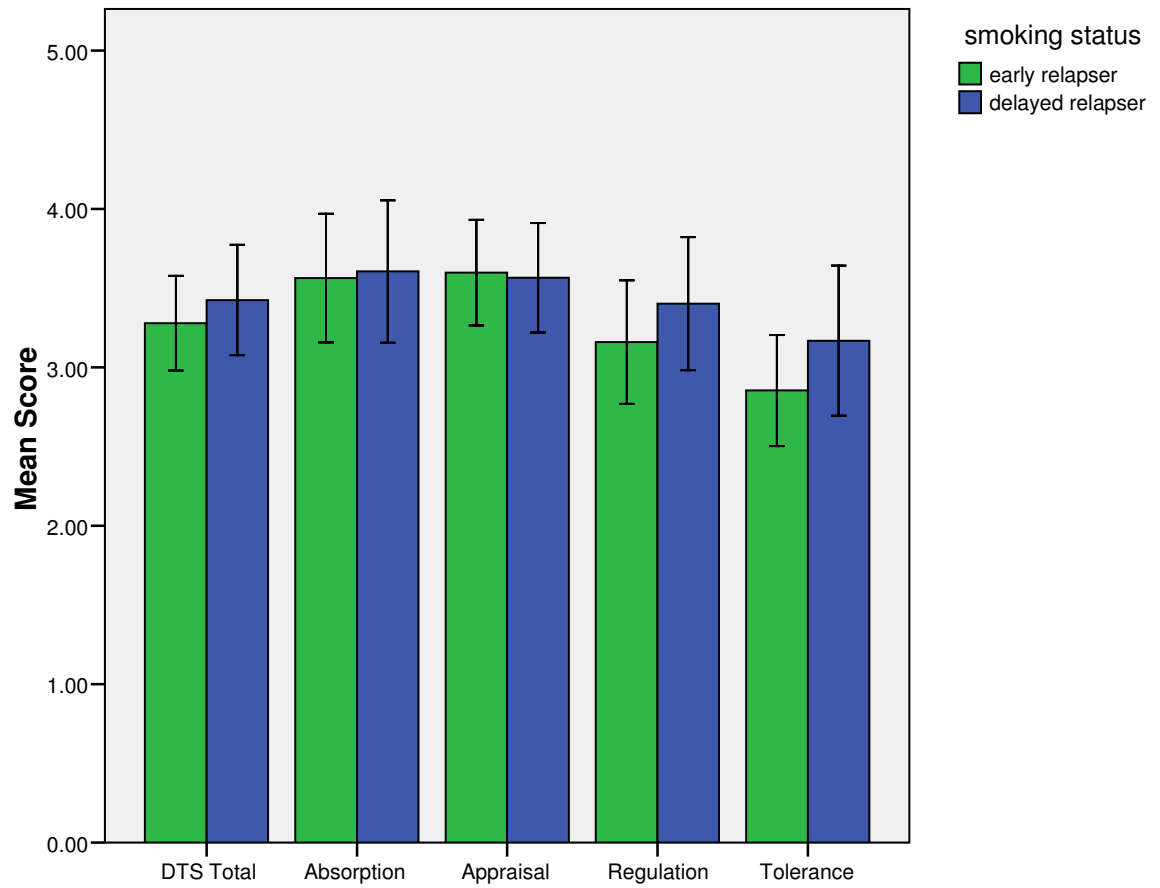


Figure 4.

*Mean salivary cortisol differences scores between early and delayed lapsers*

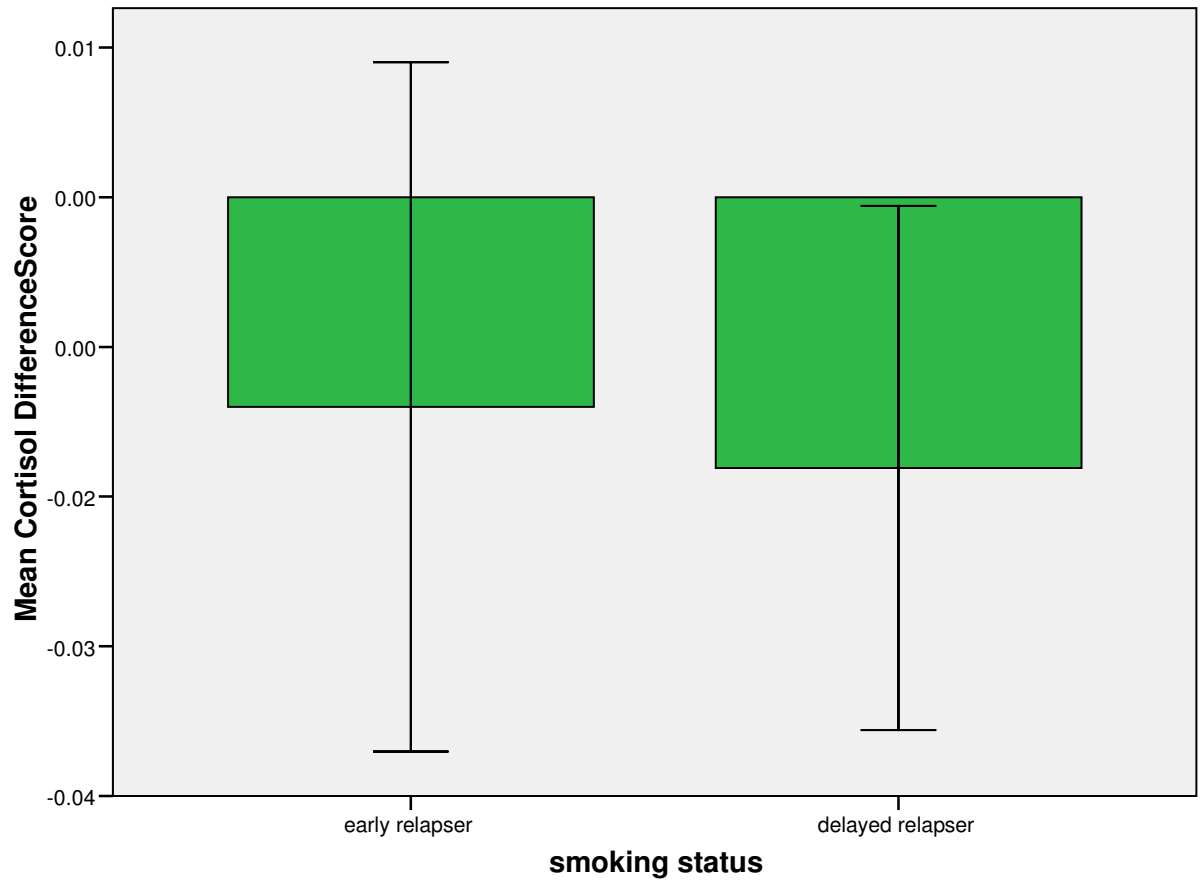


Table 5.

*Logistic regression analysis of immediate and delayed lapsers with covariates gender and average number of cigarettes smoked per day, and relevant measures of distress tolerance*

<b>Predictor</b>	<b>B</b>	<b>SE (B)</b>	<b>Wald <math>\chi^2</math></b>	<b>Odds Ratio</b>	<b>95% CI</b>
<b>Step 1</b>					
Gender	1.045	.601	3.025	2.84	.876-9.240
# Cigarettes/Day	.043	.038	1.270	1.04	.968-1.126
<b>Step 2</b>					
Gender	.785	.686	1.312	2.19	.572-8.40
# Cigarettes/Day	.093	.049	3.59	1.09	.997-1.20
PASAT	-.006	.002	<b>6.67*</b>	.994	.989-.998
PHYSICAL	-.664	.302	<b>4.84**</b>	.515	.285-.930

Note: CI = Confidence Intervals; \*p =.010; \*\*p=.02

## Chapter 4: Discussion

Despite its well documented dangers and a vast public health campaign, over 20% of Americans continue to smoke (CDC, 2005b). Many smokers who attempt to quit, lapse within a few days, and many of these individuals ultimately resume smoking. As such, it is imperative to understand the factors unique to early smoking relapse. Although variables such as withdrawal symptoms, negative affect and anxiety sensitivity may play a role in early smoking lapse, the mechanisms underlying these factors are not fully understood. One particular hypothesis is that it may not be the presence of any of these distress-related variables specifically, but rather how the individual reacts to and tolerates them that ultimately leads to smoking lapse. This threshold for tolerating physical and psychological stress is known as distress tolerance. The following study prospectively examined the role of these variables in predicting smoking outcome in a group of 58 smokers participating in an eight-week smoking cessation treatment.

### *Distress Tolerance*

It was hypothesized that early lapsed smokers would evidence lower psychological, physical, self-report, and biological levels of distress tolerance compared to delayed lapsed smokers. Results from this study provide partial support for this hypothesis. As predicted, early lapsed smokers evidenced lower levels of physical distress tolerance compared to delayed lapsed smokers. Moreover, this relationship remained significant even after accounting for other factors in this study related to early smoking lapse (i.e. gender, number of cigarettes per day). These positive findings are consistent with other studies which have found a relationship between behavioral tasks of physical distress tolerance and smoking abstinence using both cross sectional and prospective designs (Brown, Lejuez, Kahler, & Strong, 2002;

Hajeck, 1991; Hakeck, Belcher, & Stapleton, 1987; West et al., 1989). However, the majority of studies which included a measure of physical distress tolerance have been limited to respiratory challenges such as breath holding and CO<sub>2</sub> inhalation. To date, only one other study has included the cold pressor task as a measure of physical distress tolerance (Daughters et al., 2005). Specifically, they found that the cold pressor task was not related to early treatment drop-out from a residential drug treatment facility. Aside from studying a different substance, this study only included those individuals who had been through detoxification or had been abstinent from drugs for at least 72 hours. Therefore, this sample was potentially biased towards individuals who demonstrated an ability to tolerate withdrawal symptoms long enough to make it into the facility. On the contrary, the current study examined smokers prospectively through the early phase of their quit attempt when physical withdrawal symptoms would presume to be at their highest level (Cummings, Giovino, Jaen, & Emrich, 1985).

With respect to the relationship between psychological distress and early smoking lapse, findings were mixed. While there was a significant difference between early and delayed lapsers on the PASAT, there were no significant differences on the MTPT-C. Similar to the findings on physical distress tolerance, this relationship with the PASAT provided incremental validity above and beyond other smoking related factors. While it was expected that both measures of psychological distress tolerance would predict time to smoking lapse, other studies have also found inconsistent results when using multiple measures of psychological distress tolerance (Brandon et al., 2003; Stipelman et al., 2007). For example, Brandon and colleagues (2003) found that persistence on the MTPT predicted smoking abstinence, while performance on a second measure of psychological

distress tolerance (the APT) did not. There have been a few studies which have found a relationship between the PASAT and retrospective reports of smoking cessation (Brown, Lejuez, Kahler, & Strong, 2002; Lejuez, Zvolensky, Leen, & Feldner, 2001). Interestingly, the one study that looked prospectively at smokers across a self-quit attempt failed to find a relationship between the PASAT and smoking lapse/relapse (Brown et al., 2004). However, it is important to note that this study followed participants across an unaided self-quit attempt. It could be that without pharmacological aid, the physical withdrawal symptoms during a quit attempt may become more prominent (and ultimately lead to lapse) than the psychological withdrawal symptoms associated with smoking cessation.

Contrary to expectations, persistence on the MTPT-C was not predictive of time till smoking lapse (though findings were in the right direction). This runs contrary to other studies which have found the MTPT to be related to smoking status (Quinn, Brandon, and Copeland, 1996), treatment initiation and drop out (Brandon et al., 2003), and sustained smoking abstinence (Brandon et al., 2003; Stipelman et al., 2007). One potential explanation for the lack of findings may have to do with the fact that this study used a computerized version of the MTPT rather than the paper version employed in other studies. The nature of the computer program is such that it is virtually impossible to be successful on the task (as opposed to the paper version which is difficult, but not impossible). Therefore, participants may have realized during the task that they had no chance of being successful which could have influenced how hard they tried and how long they persisted on the task. Specifically, participants could have terminated the task slightly quicker than they would have if they had thought they could be successful.



One of the novel inclusions in this study was the use of the Distress Tolerance Scale (DTS), a self-report measure designed to assess emotional distress tolerance. Contrary to our expectations, there were no group differences between early and delayed lapsers on the DTS total score or any of its four subscales (Tolerance, Appraisal, Regulation, and Absorption). While this was the first study which included a self-report measure specifically designed to assess distress tolerance, results from other studies which have used self-report measures of “persistence” have been equivocal (Etter et al., 2003; Sabol et al., 1999, Steinberg et al., 2007). For example, Steinberg and colleagues (2007) used two questions related to persistence from the Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987) to compare smoking behavior in a large sample of adolescents. They found higher persistence scores in nonsmokers compared to current smokers, and in those adolescents planning to quit versus those with no plans. However, in this study persistence scores were not related to prior successful quit attempts. These findings suggest that self-reports of distress tolerance and task persistence may not measure the construct in the same way as behavioral tasks. In addition, individuals may have difficulty accurately reporting how persistent they are, whereas a behavioral task is able to produce a more objective measure.

It has been suggested that frequent and prolonged stimulation of the HPA-axis as a result of cigarette smoking may lead to reduced responsiveness of the system during times of stress (Kirschbaum, Strasburger, and Langkar, 1993). Given that quitting smoking is viewed by many as a stressful experience, this attenuation of the stress response system may leave the individual ill equipped to handle the quit attempt. As such, it was hypothesized that early lapsers would evidence lower salivary cortisol

difference scores following the distress tolerance tasks compared to delayed lapsers. However, contrary to our expectations, we did not find evidence of this relationship. There were a number of methodological limitations that could explain this lack of findings. In particular, cortisol concentrations are influenced by a variety of factors apart from stress (i.e. smoking, natural diurnal variation, food, infection, etc...) that were not controlled for due to methodological constraints on data collection. Therefore, it is plausible that more controlled collection of cortisol levels may have yielded different results. However, it is also important to note that the literature is still unclear regarding the relationship between stress, smoking, and cortisol. While a number of studies have shown attenuated cortisol levels in smokers during times of stress (Gilbert et al., 1996; Kirschbaum, Strasburger, & Langkrar, 1993; Rohleder & Kirschbaum, 2006), others have actually found the opposite result (Pomerleau & Pomerleau, 1990; Roy, Steptoe, & Kirschbaum, 1994). Moreover, all of these studies compared smokers to nonsmokers and it may be that within smokers there is not enough variation to yield between group differences.

#### *Affect Related Variables*

A number of studies have suggested a potential relationship between early smoking lapse and depression (Rausch, Nicholson, Lamke, & Matloff, 1990 ), negative affect (Kenford et al., 2002), and to a lesser extent withdrawal symptoms (Piasecki et al, 2003a) and anxiety sensitivity (Brown, Kahler, Zvolensky, Lejuez., & Ramsey, 2001). Therefore, it was hypothesized that early lapsers would endorse higher levels of overall negative affect and affect related withdrawal symptoms compared to delayed lapsers. Contrary to our expectations, although findings were generally in the expected direction,

there were no significant differences between early and delayed lapsers on any affect related variables measured. In order to be eligible for the larger treatment study, participants were required to evidence slightly elevated BDI symptoms (mean= 9.89). Furthermore, due to their significant correlation with one another, these increased BDI scores likely also lead to elevated NA PANAS scores. Therefore, it is possible that this inclusion criteria lead to a lack of between group variability on these variables. With respect to anxiety sensitivity, findings in the literature have been equivocal, and the lack of relationship in this study is consistent with others which have also failed to find a direct relationship between anxiety sensitivity and duration of smoking abstinence (Brown, Lejuez et al., 2001). However, it is also important to note that ASI-R scores in this sample were fairly low across both groups which may have precluded any differences. These same inconsistent findings have also been documented in negative affect related withdrawal symptoms (Patten & Martin, 1996). The hypothesis that positive emotionality and withdrawal symptoms not related to affect (e.g. are you thinking of cigarettes more than usual?) would not differ between early and delayed lapsers was supported.

It has been suggested that there is a relationship between distress tolerance and the various affect related variables described above (Brown, Lejuez, Kahler, & Strong, 2002). In particular, it may not be the presence of any of these factors per say, but rather how the individual responds to them that determines smoking lapse. Therefore, it was hypothesized that distress tolerance would be significantly correlated with these affect variables. Results from this study partially support this hypothesis. Both the BDI and the ASI-R were significantly negatively correlated with the DTS and its subscales, such that

as BDI and ASI-R scores increased, DTS scores decreased. This makes conceptual sense given the evidence suggesting that many individuals smoke as way to alleviate symptoms of depression and anxiety (Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001; Lerman et al., 1996). This may be particularly true for those smokers who have a difficult time experiencing and tolerating distressing emotions. Interestingly, the BDI and ASI-R were not significantly related to any other distress tolerance measures.

Also consistent with expectations, negative withdrawal symptoms were negatively correlated with Breath Holding difference score, suggesting that the more negative withdrawal symptoms the individual experienced on quit day, the less time they persisted holding their breath after experiencing discomfort during the behavioral task. Interestingly, there was no relationship with the Cold Pressor task or the self report and psychological measures of DT. It could be that the type of physical discomfort caused by breath holding is more comparable to the type of negative physical withdrawal symptoms commonly experienced by smokers (e.g. headache, sweating, tingling), as opposed to the Cold Pressor which is simply a basic physical pain task. Therefore, for the purposes of this study it is possible that the Breath Holding task was more ecologically valid than the Cold Pressor. The lack of findings with respect to the psychological distress tolerance tasks was surprising, especially given the fact that many negative withdrawal symptoms are related to psychological discomfort (e.g. anxiety, irritability). In terms of non-affect related withdrawal symptoms there was a significant positive relationship with both the Breath Holding and Cold Pressor difference scores, suggesting that more non-affect related withdrawal symptoms (e.g. do you feel awake? Is your appetite smaller than usual?) an individual experienced, the longer they persisted beyond the point of

discomfort on the two physical tasks. Similar to the negative withdrawal symptoms, there were no significant findings on the self report or psychological distress tolerance measures.

Finally, the PA PANAS score was positively correlated with the DTS Absorption scale, such that the greater the number of positive feelings checked off on the PANAS, the less likely the participant was to endorse feeling absorbed or functionally impaired in the face of distressing emotions. This makes intuitive sense in that an individual who generally experiences positive emotions is likely to be someone who is able to cope effectively with distress and not let their negative moods overcome them. Surprisingly, the positive score on the PANAS was negatively correlated with the BH difference score, suggesting that the more positive mood items an individual endorsed, the less likely they were to persist with holding their breath beyond the point of discomfort. The reason for this discrepant finding is unclear and future examination is needed to determine if this inverse relationship actually exists or is merely an aberrant finding. Contrary to expectations, there were no significant findings on the negative PANAS items and measures of distress tolerance. Overall, these findings are suggestive of some association between affect related variables and distress tolerance, however, the exact nature of that relationship remains unclear.

### *Convergent Validity*

One limitation in the distress tolerance literature has been the lack of consistency in the method of measurement of distress tolerance across studies. While the majority of studies have used behavioral assessment tasks (as opposed to self-report and biological indicators) there is still considerable variability in the specific type of behavioral task

employed. However, given that all these methods purport to measure distress tolerance, we hypothesized that we would find evidence of convergent validity across all domains of distress tolerance assessed. One of the novel methodological elements of the current study was the use of multiple measures of psychological and physical distress tolerance. As hypothesized, we found high correlations within the psychological (PASAT, MTPT-C) and physical (BH, CP) distress tolerance tasks suggesting adequate construct validity. However, we did not find significant correlations between the psychological and physical distress tolerance measures suggesting that the two types of measures are not redundant and may be capturing a different aspect of the distress tolerance construct.

With regard to the DTS self report, as expected the DTS total score and its subscales were significantly correlated, suggesting that each of the subscales is capturing a related (though distinct) aspect of the distress tolerance construct (Simons & Gaher, 2005). When comparing the DTS to other measures of distress tolerance, there was a relationship between the MTPT-C and the DTS total score and DTS appraisal score, such that the more time one persisted on the MTPT-C, the more likely they reported greater ability to withstand and accept emotional/psychological distress. While it makes sense that a self report measure of psychological distress tolerance would be related to behavioral measure of psychological distress tolerance, this finding was surprising given that the PASAT, and not the MTPT-C actually predicted time to smoking lapse. One anecdotal possibility to account for these findings has to do with the nature of the two tasks. While the PASAT is certainly a frustrating task, it is a working memory task that requires the ability to do simple math in your head quickly and accurately. As such, individuals who have a weakness in math may have gone in with the attitude that they

were going to perform poorly on the task no matter what. On the other hand, the MTPT-C consists of tracing a shape, which is a seemingly less difficult task. Therefore, it is possible that when participants had a difficult time completing the MTPT-C they felt especially frustrated and ashamed at their level of distress because tracing a shape felt like a task they should have been successful on. Unfortunately the current study did not measure attitudes towards the given tasks so there is no way of knowing if their perceptions of the task influenced their level of frustration. None of the other distress tolerance measures were significantly related to the DTS. With respect to the physical distress tolerance tasks this lack of findings was not surprising given that the DTS is specific to emotional and psychological distress and does not include questions related to tolerance of physical discomfort.

Finally, the cortisol difference score, which served as a biological indicator of distress tolerance, was significantly related to the MTPT-C and the Cold Pressor difference score. However, the directionality was actually reversed for each of the tasks, such that the greater the cortisol difference score the *longer* the subject persisted on the MTPT-C, and the *less* time they persisted on the Cold Pressor. As mentioned above, a handful of studies have found that smokers exhibit an increase in cortisol levels (possibly attenuated, but still elevated) following a psychological stress task (Kirschbaum, Strasburger, & Langkrar, 1993; Rohleder & Kirschbaum, 2006; Roy, Steptoe, & Kirschbaum, 1994). Given these findings, it is logical to presume that the longer the participants persisted on the MTPT-C (a psychological stressor), the greater the expected elevation in cortisol level. However, the contrary finding with respect to the Cold Pressor task is more puzzling. The literature examining the role of smoking and stress on cortisol

response has been largely limited to the effects of a psychological stressor. The one study examining the effects of physical stress and cortisol was conducted in nicotine dependent rabbits, where they found an increase in corticosterone following the application of a physical restraint stressor (Morse, 1989). However, the effects of physical stress on cortisol levels in adult smokers is unclear. For example, it is possible that initial experience of physical pain causes an immediate spike in cortisol levels that tends to decrease and habituate over time if the pain stimulus persists. Unfortunately, the current study only assessed for cortisol levels at baseline and following the completion of all four behavioral tasks. As such, it is impossible through this study to tease apart the unique variability in smoker's cortisol levels following either a psychological or physical stressor.

It is also interesting to note that the mean cortisol difference score was  $-.02$ , suggesting that a majority of the participants (33 out of 54) actually had a decrease in cortisol levels following the completion of the four behavioral tasks. Although this finding was surprising, there is no clear consensus in the literature regarding the effects of stress on cortisol levels in smokers with some studies finding an elevation in cortisol levels (Pomerleau & Pomerleau, 1990b; Roy, Steptoe, & Kirschbaum, 1994) and others finding no effect or a blunted response (Gilbert, Stunkard, Jensen, Detwiler, & Martinko, 1996; Kirschbaum, Strasburger, & Langkrar, 1993).

#### Limitations and Future Directions

There are a number of noteworthy limitations in the current study. First, with respect to the sample, the majority of participants (~75%) in this study were African Americans. While certainly this is an important and underrepresented population to



study, the lack of heterogeneity precludes generalizability to the general population. Therefore, future research on this topic should include a more ethnically representative sample. Moreover, the majority of participants came from a relatively low SES which is potentially important given the financial compensation associated with the project. In order to be included in the study, participants needed to express a high degree of motivation to quit smoking. While the mean level of motivation in the sample was 8.8 (out of 10), there is reason to suspect that at least a handful of individuals entered the larger treatment outcome study for the financial incentives associated with participating. Therefore, it is unclear if all participants really made a concerted effort to try and quit smoking. Indeed, there was a significant correlation between income level and smoking such that, as income level went up so did number of days abstinent. While there are a number of factors besides motivation which may account for this difference (Gilman, Abrams, & Buka, 2003; Manfredi, Cho, Young, & Crittenden, 2007), future studies should apply more rigorous assessment of motivation to ensure that participants are truly ready to try and quit.

On a related note, the smoking cessation treatment used in this current study yielded very low quit rates. Research has suggested that a combination of a pharmacological agent and behavioral treatment produces the most favorable quitting outcome with quit rates ranging from 21-67%, depending upon treatment and abstinence assessment points used (Ingersoll & Cohen, 2005). However, within this sample, over 70% of participants lapsed within the first week and only about 10% of the sample remained abstinent at one month. Research has shown that smokers with current elevated levels of depression tend to fare worse than nondepressed smokers on quitting (Rausch,

Nichinson, Lamke, & Matloff, 1990), Moreover, consistent with the population used in this study, this effect has even been documented in smokers with fairly low subclinical levels of depression (Niaura, et al., 2001). However, it is likely that elevated depressive symptoms are not enough to solely account for the poor outcome in this study. As discussed above, it is possible that motivation could have played a role in these low relapse rates. On a related note, there is some evidence to suggest that low income African Americans have lower quit rates compared to other populations (Fiore et al., 1996; King, Polednak, Bendel, Vilsaint, & Nahata, 2004). Finally, it is possible that part of the low quit rate could be attributable to the choice of NRT used in this study. For convenience and its benign side effect profile, the Nicoderm CQ nicotine patch was the pharmacological aid employed in this treatment. While the Nicotine patch provides a passive steady flow of nicotine throughout the day, it does not provide an immediate burst of nicotine to help during a craving. Therefore, it is possible that another type of NRT used alone or in conjunction with the patch would have yielded better results (Foulds, Steinberg, & Williams, 2006).

The current study only assessed smoking abstinence for one month after the quit date. Distress tolerance has been conceptualized to be an important component of *early* smoking lapse based on premise that the initial weeks of quitting smoking are characterized by uncomfortable withdrawal symptoms and a general elevation in stress associated with the quit process (Brown, Lejuez, Kahler, Strong, & Zvolensky, 2005). As such, those individuals who exhibit an inability to tolerate this discomfort are more vulnerable to give in to the immediate negative reinforcement provided by smoking. However, there is some available evidence to suggest that distress tolerance may also be

relevant to more long term smoking lapses and may not be specific to early lapses (Brandon et al., 2003; Stipelman et al., 2007). Therefore, future studies need to include more long term follow-up to gain a more complete understanding of the role of distress tolerance across the quitting process.

One of the strengths of the current study was the use of multiple methods and multiple measures of the distress tolerance construct. The majority of studies investigating the role of distress tolerance (or related constructs such as persistence or learned industriousness) in smoking have included only one method of assessment (e.g. behavioral tasks, self reports, etc...). However, each type of measure has its own inherent set of advantages and limitations. As such, it will be important for future studies to include a range of distress tolerance measures in order to develop a more reliable and valid assessment battery for measuring this construct that can be compared across studies. For example, behavioral assessments are the most common measurement of distress tolerance and are an easy way to obtain objective information regarding one's ability to tolerate discomfort. However, behavioral tasks have the disadvantage of being influenced by other factors such as motivation or experiment demand characteristics (Meyer et al., 2001). Self-report questionnaires are a cheap and efficient method for gathering data on large samples. In addition, they are able to assess for emotional aspects of distress tolerance (e.g. depression, anxiety) that may be important to smoking lapse and can not be measured using experiential paradigms (Simons & Gaher, 2005). On the other hand, self-report questionnaires measuring behaviors and/or internal states are subject to a number of weaknesses including possible lack of insight or understanding of the questions (Lucas & Baird, 2006) and response biases such as careless responding,

omissions, extremity bias and socially desirable responding (Paulhus, 1991). Finally, this was one of the first studies which included a biological indicator of distress tolerance (salivary cortisol). In order to gain a comprehensive understanding of distress tolerance it is important to elucidate the role that various physiological mechanisms play in regulating stress and discomfort. However, one of the major disadvantages in collecting biological measures are the significant number of confounds that are difficult to control for and the sensitivity required to ensure a clean sample. Despite these issues, it is important that future studies provide a more clear understanding of the role of the HPA axis, as well as other potentially important biological indices such as heart rate variability and blood pressure.

Lastly, future studies are needed to develop a more integrative model of distress tolerance. The lack of convergent validity found across presumed measures of distress tolerance suggests that it may not be a simple unitary construct. Rather, it appears that distress tolerance can be conceptualized across a number of domains including physical, psychological, physiological, and emotional. More work will need to be done to develop a clearer understanding of the distress tolerance construct and how it is related to smoking cessation both as a unique predictor and a moderator of other smoking related variables.

### Implications

Despite their limited generalizability, results from this study have potential implications for the conceptualization and treatment of cigarette smoking. Specifically, these findings add to the growing body of literature suggesting that there are unique differences between those smokers who lapse back to smoking early compared to those

who are able to go on and sustain longer term quit attempts. Although the distress tolerance literature is still in its early stages, as a more clear definition of the construct emerges it may make sense to provide smokers looking to quit with a distress tolerance paradigm to determine their potential risk for early smoking lapse.

Despite the early stages of this line of research, these findings have possible implications for the way in which cigarette smoking is treated. By and large, current behavioral smoking cessation treatments have focused on a handful of components such as relapse prevention, support, and motivational interviewing (Fiore et al 2000). While these strategies are certainly important aspects of treatment, the results from this study suggest that at least for some smokers, it may be advantageous to also include treatment elements aimed at increasing one's distress tolerance. Brown and colleagues (2005) have actually outlined such a treatment program that would include standard behavioral strategies in conjunction with exposure and acceptance based elements designed to increase the individual's distress tolerance. Specifically, the authors propose that prior to their quit date, smokers would engage in systematic and repeated periods of abstinence designed to help them habituate and develop coping skills to manage the unpleasant withdrawal symptoms associated with smoking cessation. Secondly, the authors suggest that a distress tolerance treatment for smoking should also utilize acceptance based strategies which can improve cognitive flexibility and teach the individual to be willing to accept discomfort on the way to achieving their nonsmoking goal. Consistent with Brown and colleagues (2005), research should be conducted to design and test novel smoking cessation treatment programs that address the issue of distress tolerance.

## Bibliography

- Abrams, D.B., Monti, P.M., Pinto, R.P., Elder, J.P., Brown, R.A., & Jacobus, S.I. (1987). Psychosocial stress and coping in smokers who relapse or quit. *Health Psychology, 6*, 289-303.
- al'Absi, M., Amunrud, T., & Wittmers, L.E. (2002). Psychophysiological effects of nicotine abstinence and behavioral challenges in habitual smokers. *Pharmacology, Biochemistry and Behavior, 72*, 707-716.
- al'Absi, M., Hatsukami, D., Davis, G.L., & Wittmers, L.E. (2004). Prospective examination of effects of smoking abstinence on cortisol and withdrawal symptoms as predictors of early smoking relapse. *Drug and Alcohol Dependence, 73*, 267-278.
- al'Absi, M., Hatsukami, D., & Davis, G.L. (2005). Attenuated adrenocorticotrophic responses to psychological stress are associated with early smoking relapse. *Psychopharmacology, 181*, 107-117.
- al'Absi, M., Wittmers, L.E., Erickson, J., Hatsukami, D., & Crouse, B. (2003). Attenuated adrenocortical and blood pressure response to psychological stress in ad libitum and abstinent smokers. *Pharmacology, Biochemistry and Behavior, 74*, 401-410.
- Alberg, A.J., Patnaik, J.L., Mays, J.W., Hoffman, S.C., Gitchell, J., Comstock, G.W. et al. (2005). Nicotine replacement therapy use among a cohort of smokers. *Journal of Addictive Diseases, 24*, 101-113.
- Ambrosini, P.J., Metz C., Bianchi M.D., Rabinovich H., & Undie A.(1991). Concurrent validity and psychometric properties of the Beck Depression Inventory in outpatient adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry 30*, 51-57.
- American Psychiatric Association (1994). Diagnostic and statistical manual of mental disorders 4<sup>th</sup> ed. Washington (DC): *American Psychiatric Association*.
- Anda, R.F., Williamson, D.F., Escobedo, L.G., Mast, E.E., Giovino, G.A., & Remington, P.L. (1990). Depression and the dynamics of smoking. A national perspective. *Journal of the American Medical Association, 264*(12), 1541-1545.
- Arnau, R. C., Meagher, M. W., Norris, M. P. & Bramson, R. (2001). Psychometric evaluation of the Beck Depression Inventory–II with primary care medical patients. *Health Psychology, 20*, 112-119.
- Ascher, J.A., Cole, J.O., Colin, J.N., Feighner, J.P., Ferris, R.M., Fibiger, H.C., et al.

- (1995). Bupropion: a review of its mechanism and antidepressant activity. *Journal of Clinical Psychiatry*, 56, 395-401.
- Amering, M., Bankier, B., Berger, P., Griengl, H., Windhaber, J., & Katschnig, H. (1999). Panic disorder and cigarette smoking behavior. *Comprehensive Psychiatry*, 40, 35-38.
- Baer, J.S., Kamarck, T., Lichtenstein, F., & Ransom, C.C. (1989). Prediction of smoking relapse: analyses of temptations and transgressions after initial cessation. *Journal of Consulting and Clinical Psychology*, 57, 623-627.
- Baer, J., & Lichtenstein, E. (1988). Classification and prediction of smoking relapse episodes: an exploration of individual differences. *Journal of Consulting and Clinical Psychology*, 56, 104-100.
- Bandura, A. (1997). *Self-efficacy: the exercise of control*. New York: Freeman
- Bartecchi, C.E., Mackenzie, T.D., & Schrier, R.W. (1994). The human costs of tobacco use, part II. *The New England Journal of Medicine*, 330, 975-980.
- Basco, MR, Bostic, JQ, Davies, D, Rush, A.J., Witte, B., Hendrickse, W., et al. (2000). Methods to improve diagnostic accuracy in a community mental health setting. *American Journal of Psychiatry* 157:1599-1605.
- Beck, A. T., Steer, R.A. & Brown, G. (1996). *Beck Depression Inventory II manual*. San Antonio, TX: The Psychological Corporation.
- Benowitz, N.L. (1983). Pharmacological aspects of cigarette smoking and nicotine addiction. *New England Journal of Medicine*, 19, 1318-1330.
- Benowitz, N.L. (1992) Cigarette smoking and nicotine addiction. *Medical Clinics of North America*, 76, 415-437.
- Benowitz, N. L., Kuyt, F., & Jacob, P. (1984). Influence of nicotine on cardiovascular and hormonal effects of cigarette smoking. *Clinical Pharmacology and Therapeutics*, 36, 74-81.
- Bliss, R., Garvey, A., Heinold, J., & Hitchcock, J.L. (1989). The influence of situation and coping on relapse crisis outcomes after smoking cessation. *Journal of Consulting and Clinical Psychology*, 57, 443-449.
- Blondal, T., Gudmundsson, L. J., Tomasson, K., Jonsdottir, D., Hilmarsdottir, H., Kristjansson, F., Nilsson, F., & Bjornsdottir, U. S. (1999). The effects of fluoxetine combined with nicotine inhalers in smoking cessation--a randomized trial. *Addiction*, 94, 1007-1015.

- Borrelli, B., Niaura, R., Keuthen, N.J., Goldstein, M.G., DePue, J.D., Murphy, C., et al. (1996). Development of major depressive disorder during smoking cessation treatment, *Journal of Clinical Psychiatry*, 57, 534-538.
- Brandon, T., & Baker, T.B. (1991). The smoking consequences questionnaire: the subjective utility of smoking in college students. *Psychological Assessment*, 3, 484-491.
- Brandon, T., Collins, B., Juliano, L., & Lazev, A.B. (2000). Preventing relapse among former smokers: a comparison of minimal interventions through telephone and mail. *Journal of Consulting and Clinical Psychology*, 55, 780-782.
- Brandon, T.H., Herzog, T.A., Juliano, L.M., Irvin, J.E., Lazev, A.B., & Simmons, V.N. (2003). Pretreatment task persistence predicts smoking outcome. *Journal of Abnormal Psychology*, 112, 448-456.
- Brandon, T.H., Tiffany, S.T., Obremski, K.M., & Baker, T.B. (1990). Postcessation cigarette use: the process of relapse. *Addictive Behaviors*, 15, 105-144.
- Breslau, N., Johnson, E.O., Hirpi, E., & Kessler, R. (2001). Nicotine dependence in the United States. *Archives of General Psychiatry*, 58, 810-816.
- Breslau, N., Kilbey, M., & Andreski, P. (1991) Nicotine dependence, major depression, and anxiety in young adults. *Archives of General Psychiatry*, 48, 1069-1074.
- Breslau, N., & Klein, D. F. (1999). Smoking and panic attacks: An epidemiologic investigation. *Archives of General Psychiatry*, 56, 1141-1147.
- Breslau, N., Kilbey, M.M., Andreski, P. (1994). DSM-III-R nicotine dependence in young adults: prevalence correlates and associated psychiatric disorders. *Addiction*, 89. 743-754.
- Brown, R. A., Burgess, E. S., Sales, S. D., Whiteley, J. A., Evans, D. M., & Miller, I. W. (1998). Reliability and validity of a smoking timeline follow-back interview. *Psychology of Addictive Behaviors*, 12(2), 101-112.
- Brown, R.A., Herman, K.C., Ramsey, S.W., & Stout, R.L. (1998). Characteristics of smoking cessation participants who lapse on quit date. Paper presented at the First International Conference for the Society for Research on Nicotine and Tobacco, Copenhagen, Denmark.
- Brown, R. A., Kahler, C. W., Zvolensky, M. J., Lejuez, C. W., & Ramsey, S. E. (2001). Anxiety Sensitivity: Relationship to Negative Affect Smoking and Smoking Cessation in Smokers with Past Major Depressive Disorder. *Addictive Behaviors*, 26, 887-899.



- Brown, R. A., Lejuez, C. W., Kahler, C. W., & Strong, D. (2002). Distress tolerance and duration of past smoking cessation attempts. *Journal of Abnormal Psychology*, 111, 180-185.
- Brown, R.A., Lejuez, C.W., Strong, D.R., Kahler, C.W., Niaura, R., Carpenter, L., et al. (2004, February). Distress tolerance in response to physical and psychological stressors: relationship to smoking cessation among adult self-quitters. Paper presented at the Annual meeting of the Society for Research on Nicotine and Tobacco, Scottsdale, Arizona.
- Brown, R.A., Lewinsohn, P.M., Seely, J.R., & Wagner, E.F. (1996). Cigarette smoking, major depression, and other psychiatric disorders among adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35, 1602-1610.
- Butler, C.C., Rollnick, S., Cohen, D., Bachmann, M., Russell, I., & Stott, N. (1999). Motivational consulting versus brief advice for smokers in general practice: a randomized trial. *British Journal of General Practice*, 49, 611-616.
- Canals, J., Colomina, M.T., Domingo, J.L., & Domenech, E. (1997). Influence of smoking and drinking habits on salivary cortisol levels. *Journal of Personality and Individual Differences*, 23, 593-599.
- Centers for Disease Control (1993). Smoking-attributable mortality and years of potential life lost -United States, 1990. *Morbidity and Mortality Weekly Report*, 42(33), 645-8. (PDF-22K). Accessed: May, 2006.
- Centers for Disease Control (2002a). Annual smoking-attributable mortality, years of potential life lost, and economic costs-United States, 1995-1999. *Morbidity and Mortality Weekly Report*. 51(14), 300-303. (PDF-22k). Accessed: May, 2006.
- Centers for Disease Control (2002b). Cigarette smoking among adults – United States 2000. *Morbidity and Mortality Weekly Report*, 51 (29). (PDF-22k). Accessed: May 2006.
- Centers for Disease Control (2003). 2003 Chartbook on trends in the health of Americans. (PDF-119k). Accessed: May, 2006.
- Centers for Disease Control (2005a). Attributable mortality, years of potential life lost, and productivity losses- United States, 1997-2001. 54(25), 625-628. (PDF-256k). Accessed May: 2006.
- Centers for Disease Control (2005b). Cigarette smoking among adults- United States, 2004. *Morbidity and Mortality Weekly Report*. 54(44), 1121-1124. (PDF-22k). Accessed: May 2006.

- Chornock, W. M., Stitzer, M. L., Gross, J. & Leischow, S. (1992). Experimental model of smoking re-exposure: Effects on relapse. *Psychopharmacology (Berlin)*, 108, 495-500.
- Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants: A proposal. *Archives of General Psychiatry*, 44, 573-588.
- Cohen, L.M., al'Absi, M., & Collins, F.L. (2004). Salivary cortisol concentrations are associated with acute nicotine withdrawal. *Addictive Behaviors*, 29, 1673-1678.
- Colby, S.M., Monti, P.M., Barnett, N.P., Rohsenow, D.J., Weissman, K., Spirito, A., et al. (1998). Brief motivational interviewing in a hospital setting for adolescent smoking: a preliminary study. *Journal of Consulting and Clinical Psychology*, 66, 574-578.
- Comeau, N., Stewart, S.H., & Loba, P. (2001). The relations of trait anxiety, anxiety sensitivity, and sensation seeking to adolescents' motivations for alcohol, cigarette, and marijuana use. *Addictive Behaviors*, 26, 803-825.
- Cook, M. R., Gerkovich, M. M., O'Connell, K. A., & Potocky, M. (1995). Reversal theory constructs and cigarette availability predict lapse early in smoking cessation. *Research in Nursing & Health*, 18, 217-224.
- Covey, L. S., Glassman, A. H., & Stetner, F. (1990). Depression and depressive symptoms in smoking cessation. *Comprehensive Psychiatry*, 31, 350-354.
- Crawford, J.R., & Henry, J.D. (2004). The positive and negative affect schedule (PANAS): construct validity, measurement properties and normative data in a large non-clinical sample. *British Journal of Clinical Psychology*, 43, 245-265.
- Cummings, K. M., Giovino, G., Jaen, C. R., & Emrich, L. J. (1985). Reports of smoking withdrawal symptoms over a 21 day period of abstinence. *Addictive Behaviors*, 10, 373-381.
- Cummings, K.M., Jaen, C., & Giovino, G. (1985). Circumstances surrounding relapse in a group of recent ex-smokers. *Preventive Medicine*, 14, 195-202.
- Curry, S., Marlatt, G., & Gordon, J. (1987). Abstinence violation effect: validation of an attributional construct with smoking cessation. *Journal of Consulting and Clinical Psychology*, 59, 318-324.
- Curry, S.J., & McBride, C.M. (1994). Relapse prevention for smoking cessation: review and evaluation of concepts and interventions. *Annual Review of Public Health*, 15, 345-366.
- Curtin, L., Brown, R. A., & Sales, S. D. (2000). Determinants of attrition from cessation

treatment in smokers with a history of major depressive disorder. *Psychology of Addictive Behaviors*, 14, 134-142.

Daughters, S.B., Lejuez, C.W., Bornovalova, M.A., Khaler, C., Strong, D., & Brown, R. (2005). Distress tolerance as a predictor of early treatment dropout in a residential substance abuse treatment facility. *Journal of Abnormal Psychology*, 114, 729-734.

Daughters, S.B., Lejuez, C.W., Kahler, C., Strong, D., & Brown, R. (2005). Psychological distress tolerance and duration of most recent abstinence attempt among residential treatment seeking substance abusers. *Psychology of Addictive Behaviors*, 19, 208-211.

Deacon, B.J., Abramowitz, J.S., Woods, C.M., & Tolin, D.F. (2003). The anxiety sensitivity index-revised: psychometric properties and factor structure in two nonclinical samples. *Behaviour Research and Therapy*, 41, 1427-1449.

Deary, I. J., Ebmeier, K. P., MacLeod, K. M., Dougall, N., Hepburn, D. A., Frier, B. M. et al. (1994). PASAT performance and the pattern of uptake of –super (99m)Tc-exametazime in brain estimated with single photon emission tomography. *Biological Psychology*, 38, 1-18.

Del Arbol, J.L., Raya Munoz, J., Ojeda, L., Lopez Cascales, A., Rico Irlas, J., Miranda, M.T., et al. (2000). Plasma concentrations of beta-endorphin in smokers who consume different numbers of cigarettes per day. *Pharmacology, Biochemistry and Behavior*, 67, 25-28.

Delfino, R.J., Jamner, L.D., & Whalen, C.K. (2001). Temporal analysis of the relationship of smoking behavior and urges to mood states in men versus women. *Nicotine and Tobacco Research*, 3, 235-248.

Diehr, M. C., Heaton, R. K., Miller, W., & Grant, I. (1998). The paced auditory serial addition task (PASAT): Norms for age, education, and ethnicity. *Assessment*, 5, 375-387.

Doherty, K., Kinnunen, T., Militello, F. S., & Garvey, A. J. (1995). Urges to smoke during the first month of abstinence: Relationship to relapse and predictors. *Psychopharmacology*, 119, 171-178.

Donovan, D. (1996). Marlatt's classification of relapse precipitants: is the Emperor still wearing clothes? *Addiction*, 91(Suppl). S131-S137.

Eisenberger, R. (1992). Learned industriousness. *Psychological review*, 99, 248-267.

Eisenberger, R., & Leonard, J.M. (1980). Effects of conceptual task difficulty on generalized persistence. *American Journal of Psychology*, 93, 285-298.

- Etter, J. F., Pelissolo, A., Pomerleau, C., & De Saint-Hilaire, Z. (2003). Associations between smoking and heritable temperament traits. *Nicotine and Tobacco Research*, 5(3), 401-409.
- Fagerstrom, K.O. (1988). Efficacy of nicotine chewing gum: a review. *Progress in Clinical and Biological Research*, 261, 109-128.
- Ferry, L.H. (1999). Non-nicotine pharmacotherapy for smoking cessation. *Tobacco Use Cessation*, 26, 653-659.
- Field, A.E., Colditz, G.A., Willett, W.C., Longcope, C., & McKinlay, J.B. (1994). The relation of smoking, age, relative weight, and dietary intake to serum adrenal steroids, sex hormones, and sex hormone-binding globulin in middle-aged men. *Journal of Clinical Endocrinology and Metabolism*, 79, 1310-1316.
- Fiore, M.S., Bailey, W.C., Cohen, S.T., Dorfman, S.F., Goldstein, M.G., & Gritz, E.R. (2000). Treating tobacco use and dependence: clinical practice guideline. Rockville, MD: US Department of Health and Human Services, Public Health Service.
- Fiore, M.C., Novotny, T.E., Pierce, J.P., Giovino, G.A., Hatziendreu, E.J., Newcomb, P.A., et al. (1990). Methods used to quit smoking in the United States: do cessation programs help? *Journal of the American Medical Association*, 263, 2760-2765.
- Fiore, M.C., Smith, S.S., Jorenby, D.E., & Baker, T.B. (1994). The effectiveness of the nicotine patch for smoking cessation: a meta-analysis. *Journal of the American Medical Association*, 271, 1940-1947.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1995). *Structured Clinical Interview for DSM-IV Axis I Disorders*. New York: New York State Psychiatric Institute
- Foulds, J., Steinberg, M. B., & Williams, J. M. (2006). Developments in pharmacotherapy for tobacco dependence: Past, present and future. *Drug and Alcohol Review*, 25, 59-71.
- Frederick, S.L., Reus, V.I., Ginsberg, D., Hall, S.M., Munoz, R.F., & Ellman, G. (1998). Cortisol and response to dexamethasone as predictors of withdrawal distress and abstinence success in smokers. *Biological Psychiatry*, 43, 525-530.
- Garvey, A.J., Bliss, R.E., Hitchcock, J.L., Heinold, J.W., & Rosner, B. (1992). Predictors of smoking relapse among self-quitters: a report from the Normative Aging Study. *Addictive Behaviors*, 17, 367-377.

- Gilbert, D.G., Dibb, W.M., Plath, L.C., & Hiyane, S.G. (2000). Effects of nicotine and caffeine, separately and in combination, on EEG topography, mood, heart rate, cortisol, and vigilance. *Psychophysiology*, 37, 583-595.
- Gilbert, D.G., McClernon, F.J., Rabinovich, N.E; Dibb, W.D., Plath, L.C., Hiyane, S., et al. (1999). EEG, physiology, and task-related mood fail to resolve across 31 days of smoking abstinence: Relations to depressive traits, nicotine exposure, and dependence. *Experimental and Clinical Psychopharmacology*, 7, 427-443.
- Gilbert, D. G., & Spielberger, C. D. (1987). Effects of smoking on heart rate, anxiety, and feelings of success during social interaction. *Journal of Behavioral Medicine*, 10, 629-38.
- Gilbert, D.G., Robinson, J.H., Chamberlin, C.L., & Spielberger, C.D. (1989). Effects of smoking/nicotine on lateralization of EEG while viewing a stressful movie. *Psychophysiology*, 26, 311-320.
- Gilbert, D.G., Stunkard, M.E., Jensen, R.A., Detwiler, F.R., & Martinko, J.M. (1996). Effects of exam stress on mood, cortisol, and immune functioning: Influences of neuroticism and smoker-non smoker status. *Journal of Personality and Individual Differences*, 21, 235-246.
- Gilman, S. E., Abrams, D. B., & Buka, S. L. (2003). Socioeconomic status over the life course and stages of cigarette use: Initiation, regular use, and cessation. *Journal of Epidemiology & Community Health*, 57, 802-808.
- Gilman, A.G., Goodman, L.S., Rall, T.W., & Murad, F. (1985). Goodman and Gilman's, *The Pharmacological Basis of Therapeutics* (7<sup>th</sup> Ed.). New York: Macmillan.
- Ginsberg, D., Hall, S. M., Reus, V.I., & Muñoz, R. F. (1995). Mood and depression diagnosis in smoking cessation. *Experimental and Clinical Psychopharmacology*, 3, 389-395.
- Glassman, A. H., Covey, L. S., Dalack, G. W., Stetner, F., Rivelli, S. K., Fleiss, J. et al. (1993). Smoking cessation, clonidine, and vulnerability to nicotine among dependent smokers. *Clinical Pharmacology and Therapeutics*, 54, 670-679.
- Glassman AH, Helzer JE, Covey LS, Cottler LB, Stetner F, Tipp JE, et al. Smoking, smoking cessation, and major depression. *Journal of the American Medical Association* 1990;264(12):1546-9.
- Gonzales, D., Nides, M., Ferry, L., Kustra, R.P., Jamerson, B.D., Segall, N., et al. (2001). Bupropion SR as an aid to smoking cessation in smokers previously treated with bupropion: a randomized placebo-controlled study. *Clinical Pharmacology and Therapeutics*, 69, 438-444.

- Gonzales, D., Nides, M., Ferry, L., Kustra, R.P., Jamerson, B.D., Segall, N., et al (2002). Retreatment with bupropion: results from 12-month follow-up. Society for Research on Nicotine and Tobacco, 8<sup>th</sup> Annual Meeting, Savannah, GA.
- Gritz, E. R., Carr, C. R., & Marcus, A. C. (1991). The tobacco withdrawal syndrome in unaided quitters. *British Journal of Addiction*, 86, 57-69.
- Gunn, R. C. (1986). Reactions to withdrawal symptoms and success in smoking cessation clinics. *Addictive Behaviors*, 11, 49-53.
- Hajek, P. (1991). Individual differences in difficulty quitting smoking. *British Journal of Addiction*, 86, 555-558.
- Hajek, P., Belcher, M., & Stapleton, J. (1987). Breath-holding endurance as a predictor of success in smoking cessation. *Addictive Behaviors*, 12, 285-288.
- Hajek, P., & Stead, L.F. (2006). Aversive smoking for smoking cessation. *The Cochrane Database of Systematic reviews*, CD000546.
- Hajek, P., West, R., Foulds, J., Nilsson, F., Burrows, S., Meadow, A. (1999). Randomized comparative trial of nicotine polacrilex, a transdermal patch, nasal spray, and inhaler. *Archives of Internal Medicine*, 159, 2033-2038.
- Hall, D. (1987). Factors to consider in research on prevention of relapse in cigarette smoking. *Psychological Reports*, 60, 967-974.
- Hall, S. M., Havassy, B. E., & Wasserman, D. A. (1990). Commitment to abstinence and acute stress in relapse to alcohol, opiates, and nicotine. *Journal of Consulting and Clinical Psychology*, 58, 175-181
- Hall, S.M., Ginsberg, D., & Jones, R.T. (1986). Smoking cessation and weight gain. *Journal of Consulting and Clinical Psychology*, 54, 342-346.
- Hall, S. M., Muñoz, R. F., & Reus, V. I. (1994). Cognitive-behavioral intervention increases abstinence rates for depressive-history smokers. *Journal of Consulting and Clinical Psychology*, 62, 141-146.
- Hanna, E. Z., Faden, V. B., & Dufour, M. C. (1994). The motivational correlates of drinking, smoking, and illicit drug use during pregnancy. *Journal of Substance Abuse*, 6, 155-167.
- Heatherton, T. F., Kozlowski, L. T., Frecker, R. C., & Fagerstrom, K. O. (1991). The Fagerstrom test for nicotine dependence: A revision of the Fagerstrom Tolerance Questionnaire. *British Journal of Addiction*, 86, 1119-1127.
- Henningfield, J.E., & Goldberg, S.R. (1988). Pharmacologic determinants of tobacco

- self-administration by humans. *Pharmacology, Biochemistry, & Behavior*, 30, 221-226.
- Hettema, J., Steele, J., & Miller, W.R. (2005). Motivational Interviewing. *Annual review of Clinical Psychology*, 1, 91-111.
- Hughes, J. R. (1992). Tobacco withdrawal in self-quitters. *Journal of Consulting and Clinical Psychology*, 60, 689-697.
- Hughes, J. R., Arana, G., Amori, G., Stewart, F., & Workman, R. (1988). Effect of tobacco withdrawal on the dexamethasone suppression test. *Biological Psychiatry*, 23, 96-98.
- Hughes, J.R., Gust, S.W., & Pechacek, T.F. (1987). Prevalence of tobacco dependence and withdrawal. *American Journal of Psychiatry*, 144, 205-208.
- Hughes, J. R., Gust, S. W., Skoog, K., Keenan, R. M., & Fenwick, J. W. (1991). Symptoms of tobacco withdrawal. *Archives of General Psychiatry*, 48, 52-59.
- Hughes, J.R., & Hatsukami, D. (1986). Signs and symptoms of tobacco withdrawal. *Archives of General Psychiatry*, 43, 289-294.
- Hughes, J.R., Keely, J., & Naud, S. (2004). Shape of the relapse curve and long-term abstinence among untreated smokers. *Addiction*, 99, 29-38.
- Hughes, J.R., Stead, L.F., & Lancaster, T. (2003). Antidepressants for smoking cessation (Cochrane review). In: *The Cochrane Library*, Issue 2, Oxford: Update Software.
- Hurt, R.D., Sachs, D.P., Glover, E.D., Offord, K.P., Johnston, J.A., Dale, L.C., et al. (1997). A comparison of sustained-release bupropion and placebo for smoking cessation. *New England Journal of Medicine*, 337, 1195-1202.
- Ingersoll, K., & Cohen, J. (2005). Combination treatment for nicotine dependence: State of the science. *Substance Use & Misuse*, 40, 1923-1943.
- Irvin, J., Bowers, C., Dunn, M. ET AL (1999). Efficacy of relapse prevention: a meta-analytic review. *Journal of Consulting and Clinical Psychology*, 67, 563-570.
- Isensee, B., Wittchen, H., Stein, M.B., Hofler, M., Lieb, R. (2003). Smoking increases the risk of panic: findings from a prospective community study. *Archives of General Psychiatry*, 60, 692-700.
- Jaffee, J.H. (1985). Drug addiction and drug abuse. In: A.G. Gilman, L.S. Goodman, T.W. Rail, and F. Murad (Eds.). *Goodman and Gilman's The Pharmacological Basis of Therapeutics*, 7<sup>th</sup> Edition. New York: MacMillan. Pages 532-581.

- Jarvis, M. J., Tunstall-Pedoe, H., & Feyerabend, C. e. a. (1987). Comparison of tests to distinguish smokers from nonsmokers. *American Journal of Public Health*, 77, 1435-1438.
- Johnson, J.G., Cohen, P., Pine, D.S., Klein, D.F., Kasen, S., & Brook, J.S. (2000). Association between cigarette smoking and anxiety disorders during adolescence and early adulthood. *Journal of the American Medical Association*, 284, 2348-2351.
- Jorenby, D.E., Leischow, S.J., Nides, M.A., Rennard, S.I., Johnston, J.A., Hughes, A.R. et al. (1999). A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. *New England Journal of Medicine*, 340, 685-691.
- Juliano, L.M., Donny, E.C., Houtsmuller, E.J., Stitzer, M.L. (2006). Experimental evidence for a causal relationship between smoking and relapse. *Journal of Abnormal Psychology*, 115, 166-173.
- Kahler, C.W., Brown, R.A., Ramsey, S.E., Niaura, R., Abrams, D.B., Goldstein, M.G. et al. (2002). Negative mood, depressive symptoms, and major depression after smoking cessation treatment in smokers with a past history of major depressive disorder. *Journal of Abnormal Psychology*, 111, 670-675.
- Kamarack, T.W., & Lichtenstein, E. (1988). Program adherence and coping strategies as predictors of success in a smoking treatment program. *Health Psychology*, 7, 557-574.
- Kassel, J.D., & Shiffman, S. (1997). Attentional mediation of cigarette smoking's effect on anxiety. *Health Psychology*, 16, 359-368.
- Kendler, K. S., Neale, M. C., MacLean, C. J., Heath, A. C., Eaves, L. J., & Kessler, R. C. (1993). Smoking and major depression: A causal analysis. *Archives of General Psychiatry*, 50, 36-43.
- Kenford, S.L., Smith, S.S., Wetter, D.W., Jorenby, D.E., Fiore, M.C., & Baker, T.B. (2002). Predicting relapse back to smoking: contrasting affective and physical models of dependence. *Journal of Consulting and Clinical Psychology*, 70, 216-227.
- Kenford, S.L., Fiore, M.C., Jorenby, D.E., Smith, S.S., Wetter D., & Baker, T.B. (1994). Predicting smoking cessation: who will quit with and without the nicotine patch, *Journal of the American Medical Association*, 271, 589-594.
- Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., Walters, E. E. (2005). Prevalence, severity, and comorbidity of twelve-month DSM-IV disorders in the National Comorbidity Survey Replication (NCS-R). *Archives of General Psychiatry*, 62, 617-627.



- King, G., Polednak, A., Bendel, R. B., Vilsaint, M. C., & Nahata, S. B. (2004). Disparities in smoking cessation between African Americans and Whites: 1990-2000. *American Journal of Public Health*, 94, 1965-1971.
- Kinnunen, T., Doherty, K., Militello, F. S., & Garvey, A. J. (1996). Depression and smoking cessation: Characteristics of depressed smokers and effects of nicotine dependence. *Journal of Consulting and Clinical Psychology*, 64, 791-798.
- Kirschbaum, C., Strasburger, C.J., & Langkrar (1993). Attenuated cortisol response to psychological stress but not to CRH or ergometry in young habitual smokers. *Pharmacology, Biochemistry and Behavior*, 44, 527-531.
- Kirschbaum, C., Wust, S., & Strasburger, C.J. (1992). "Normal" cigarette smoking increases free cortisol in habitual smokers. *Life Sciences*, 50, 435-442.
- Kranzler H.R., Kadden R.M., Babor T.F., Tennen, H., & Rounsaville, B.J. (1996). Validity of the SCID in substance abuse patients. *Addiction* 91, 859-868.
- Lasser, K., Boyd, J.W., Woolhandler, S., Himmelstein, D.U., McCormick, D., & Bor, D.H. (2000). Smoking and mental illness. *Journal of the American Medical Association*, 284, 2606-2610.
- Lejuez, C. W., Kahler, C. W., & Brown, R. A. (in press). A Modified Computer Version of the Paced Auditory Serial Learning Task (PASAT) as a Laboratory-Based Stressor. *Journal of Behavior Therapy and Experimental Psychiatry*, 26(4), 290-293.
- Lejuez, C. W., Zvolensky, M. J., Leen, E., & Feldner, M. (2001). Persistence on a psychological stressor as a predictor of longest smoking cessation duration. Manuscript in preparation.
- Lerman, C., Audrain, J., Orleans, C.T., Boyd, R., Gold, K., Main, D., & Caporaso, N. (1996). Investigation of mechanisms linking depressed mood to nicotine dependence. *Addictive Behaviors* 21, 9-19.
- Lichtenstein, E., Glasgow, R. E., & Abrams, D. B. (1986). Social support in smoking cessation: In search of effective interventions. *Behavior Therapy*, 17, 607-619.
- Lichtenstein, E., & Hollis, J.F. (1992). Patient referral to a smoking cessation program: who follows through? *Journal of Family Practice*, 34, 739-744.
- Linehan, M.M. (1993). Cognitive-behavioral treatment of borderline personality disorder. New York, NY, US: Guilford Press
- Lucas, R. E., & Baird, B. M. (2006). Global Self-Assessment. In M. Eid, & E. Diener (Eds.), *Handbook of multimethod measurement in psychology* (pp. 29-42). Washington, DC: American Psychological Association.

- Manfredi, C., Cho, I. Y., & Crittenden, K. S. (2007). A path model of smoking cessation in women smokers of low socio-economic status. *Health Education Research*, 22, 747-756.
- Marlatt, G.A. & Gordon, J.R. (1980). Determinants of relapse: implications for the maintenance of behavior change. In P.O. Davidson, & S.M. Davidson (Eds.), *Behavioral medicine: Changing health lifestyle* (pp. 410-452). New York: Brunner/Mazel.
- Marlatt, G.A., & Gordon, J.R. (Eds) (1985). *Relapse Prevention: Maintenance Strategies in the Treatment of Addictive Behaviors*. New York: Guilford Press.
- Matthews, K.A., & Stoney, C.M. (1988). Influences of sex and age on cardiovascular response during stress. *Psychosomatic Medicine*, 50, 46-56.
- McGinnis, J., & Foege, W.H. (1993). Actual causes of death in United States. *Journal of American Medical Association*, 270, 2207-2212.
- Meliska, C.J., Stunkard, M.E., Gilbert, D.G., Jensen, R.A., & Martinko, J.M. (1995). Immune function in cigarette smokers who quit for 31 days. *Journal of Allergy and Clinical Immunology*, 95, 901-905.
- Mendelson, J.H., Sholar, M.B., Goletiani, N., Siegel, A., & Mello, N. (2005). Effects of low-and-high nicotine cigarette smoking on mood states and HPA axis in men. *Neuropsychopharmacology*, 30, 1751-1763.
- Meyer, G. J., Finn, S. E., Eyde, L. D., Kay, G. G., Moreland, K. L., & Dies, R. R. (2001). Psychological testing and psychological assessment: a review of evidence and issues. *American Psychologist* 56, 128-165.
- Morse, D.E. (1989). Neuroendocrine responses to nicotine and stress: Enhancement of peripheral stress responses by the administration of nicotine. *Psychopharmacology (Berlin)*, 98, 539-543.
- Niaura, R., Britt, D.M., Shadel, W.G., Goldstein, M., Abrams, D.B., & Brown, R. (2001). Symptoms of depression and survival experience among three samples of smokers trying to quit. *Psychology of Addictive Behaviors*, 15, 13-17.
- Niaura, R., Britt, D.M., Borelli, B., Shadel, W.G., Abrams, D.B., & Goldstein, M.G. (1999). History and symptoms of depression among smokers during a self-initiated quit attempt. *Nicotine and Tobacco Research*, 1, 251-257.
- Norregaard, J., Tonnesen, P., & Peterson, L. (1993). Predictors and reasons for relapse in smoking cessation with nicotine and placebo patches. *Preventive Medicine*, 22, 261-271.

- Novak, A., Burgess, E.S., Clark, M., Zvolensky, M.J., & Brown, R.A. (2003). Anxiety sensitivity, self-reported motives for alcohol and nicotine use and level of consumption. *Journal of Anxiety Disorders*, 17, 165-180.
- Ockene, J., Emmons, K., Mermelstein, R., ET AL (1995). Relapse and maintenance issues for smoking cessation. *Health Psychology*, 19, 17-31.
- O'Connell, K.A., & Martin, E.J. (1987). Highly tempting situations associated with abstinence, temporary lapse, and relapse among participants in smoking cessation programs. *Journal of Consulting and Clinical Psychology*, 55, 367-371.
- O'Connell, K.A., & Shiffman, S. (1988). Negative affect and smoking relapse. *Journal of Substance Abuse*, 1, 25-33.
- Patten, C. A., & Martin, J. E. (1996). Does nicotine withdrawal affect smoking cessation? Clinical and theoretical issues. *Annals of Behavioral Medicine*, 18, 190-200.
- Paulhus, D. L. (1991). Measurement and control of response bias. In J. P. Robinson, P. R. Shaver, & L. S. Wrightsman (Eds.), *Measures of personality and social psychological attitudes, Vol. 1* (pp. 17-59). San Diego, CA: Academic Press.
- Persico, A. M. (1992). Predictors of smoking cessation in a sample of Italian smokers. *International Journal of the Addictions*, 27, 683-695.
- Peterson, R. A., & Reiss, S. (1992). *Anxiety Sensitivity Index Manual* (2nd ed.). Worthington, OH: International Diagnostic Systems.
- Peto, R., Lopez, A.D., Boreham, J., Thun, M., & Heath, C.Jr. (1992). Mortality from tobacco in developed countries: indirect estimation from national vital statistics. *Lancet*, 339, 1268-1278.
- Piasecki, T.M., Jorenby, D.E., Smith, S.S., Fiore, M.C., & Baker, T.B. (2003a). Smoking withdrawal dynamics: I. Abstinence distress in lapsers and abstainers. *Journal of Abnormal Psychology*, 112, 3-13.
- Piasecki, T.M., Jorenby, D.E., Smith, S.S., Fiore, M.C., & Baker, T.B. (2003a). Smoking withdrawal dynamics: II. Improved tests of withdrawal-relapse relations. *Journal of Abnormal Psychology*, 112, 14-27.
- Piasecki, T. M., Kenford, S. L., Smith, S. S., Fiore, M. C., & Baker, T. B. (1997). Listening to nicotine: Negative affect and the smoking withdrawal conundrum. *Psychological Science*, 8, 184-189.
- Piasecki, T. M., Niaura, R., Shadel, W. G., Abrams, D., Goldstein, M., Fiore, M. C. et al.

- (2000). Smoking withdrawal dynamics in unaided quitters. *Journal of Abnormal Psychology*, 109, 74-86.
- Pickworth, W.B., & Fant, R.V. (1998). Endocrine effects of nicotine administration, tobacco and other drug withdrawal in humans. *Psychoneuroendocrinology*, 23, 131-141.
- Pohl, R., Yeragani, V.K., Balon, R., Lycaki, H., & McBride, R. (1992). Smoking in patients with panic disorder. *Psychiatry Research*, 43, 253-262.
- Pomerleau, O.F., & Pomerleau, C.S. (1990a). Dexamethasone attenuation of the cortisol response to nicotine in smokers. *Psychopharmacology (Berl)*, 101, 284-286
- Pomerleau, O.F. & Pomerleau, C.S. (1990b). Cortisol response to a psychological stressor and/or nicotine. *Pharmacology, Biochemistry and Behavior*, 36, 211-213.
- Pomerleau, O.F., Pomerleau, C.S., & Marks, J.L. (2000). Abstinence effects and reactivity to nicotine during 11 days of smoking deprivation. *Nicotine and Tobacco Research*, 2, 149-157.
- Proceedings of the National Working Conference on Smoking Relapse. (1986). *Health Psychology*, 5 (Supplement).
- Puddey, I.B., Vandongen, R., Beilin, L.J., & English, D. (1984). Haemodynamic and neuroendocrine consequences of stopping smoking- a controlled study. *Clinical Experimental Pharmacology and Physiology*, 11, 423-426.
- Quinn, E. P., Brandon, T. H., & Copeland, A. L. (1996). Is task persistence related to smoking and substance abuse? The application of learned industriousness theory to addictive behaviors. *Experimental and Clinical Psychopharmacology*, 4, 186-190.
- Rausch, J.L., Nicholson, B., Lamke, C., & Matloff, J. (1990). Influence of negative affect on smoking cessation treatment outcome: a pilot study. *British Journal of Addiction*, 85, 929-933.
- Robins, L.N., Helzer, J.E., & Przybeck, T. (1986). Substance abuse in the general population. In J. Barrett, & R.M. Rose (Eds.). *Mental Disorders in the Community: Progress and Challenge, Proceedings of the American Psychopathological Association* (pp. 9-31). New York, NY: Guilford Press.
- Robinson, M. D., Pettice, Y., Smith, W. A. (1992). Buspirone effect on tobacco withdrawal symptoms: A randomized placebo-controlled trial. *Journal of the American Board of Family Practice*, 5, 1-9
- Rohleder, N., & Kirschbaum, C. (2006). The hypothalamic-pituitary-adrenal (HPA) axis

- in habitual smokers. *International Journal of Psychophysiology*, 59, 236-243.
- Roy, M.P., Steptoe, A., & Kirschbaum, C. (1994). The association between smoking status and cardiovascular and cortisol stress responsivity in healthy young men. *International Journal of Behavioral Medicine*, 1(3), 264-283.
- Sabol, S. Z. Nelson, M. L., Fisher, C., Gunzerath, L., Brody, C. L., et al. (1999). A genetic association for cigarette smoking behavior. *Health Psychology*, 18, 7-13.
- Schachter, S. (1978). Pharmacological and psychological determinants of smoking. *Annals of Internal Medicine*, 88, 104-114.
- Schmidt, N.B., Lerew, D.R., & Jackson, R.J. (1999). Prospective evaluation of anxiety sensitivity in the pathogenesis of panic: replication and extension. *Journal of Abnormal Psychology*, 108, 532-537.
- Schneider, N.G., Olmstead, R., Mody, F.V., Doan, K., Franzon, M., Jarvik, M.E., et al. (1995). Efficacy of a nicotine nasal spray in smoking cessation: a placebo-controlled, double-blind trial. *Addiction*, 90, 1293-1306.
- Schneider, N.G., Olmstead, R., Nilsson, F., Mody, F.V., Franzon, M., & Doan, K. (1996). Efficacy of a nicotine inhaler in smoking cessation: a double-blind, placebo-controlled trial. *Addiction*, 91, 1293-1306.
- Seyle H. *The stress of life*. New York: McGraw-Hill. 1976.
- Seyler, Jr., L.E., Pomerleau, O.F., Fertig, J.B., Hunt, D., & Parker, K. (1986). Pituitary hormone response to cigarette smoking. *Pharmacology, Biochemistry and Behavior*, 24, 159-162.
- Shapiro, D., Jamner, L.D., Davydov, D.M., & Porscha, J. (2002). Situations and moods associated with smoking in everyday life. *Psychology of Addictive Behaviors*, 16, 342-345.
- Shiffman, S. (1982). Relapse following smoking cessation: a situational analysis. *Journal of Consulting and Clinical Psychology*, 50, 71-86.
- Shiffman, S. (1991). Refining models of dependence: variations across persons and situations. *British Journal of Addiction*, 86, 611-615.
- Shiffman, S. (1993). Assessing smoking patterns and motives. *Journal of Consulting and Clinical Psychology*, 50, 71-86.
- Shiffman, S., Balabanis, M.H., Paty, J.A., Gwaltney, C.J., Liu, K.S., Gnys, M., et al. (2000). Dynamic effects of self-efficacy on smoking lapse and relapse. *Health Psychology*, 19, 315-323.

- Shiffman, S., Di Marini, M.E., & Pillitteri, J.L. (2005). The effectiveness of nicotine patch and nicotine lozenge in very heavy smokers. *Journal of Substance Abuse Treatment*, 28, 49-55.
- Shiffman, S., Dresler, C.M., & Rohay, J.M. (2004). Successful treatment with a nicotine lozenge of smokers with prior failure in pharmacological therapy. *Addiction*, 99, 83-92.
- Shiffman, S., Gwaltney, C.J., Balabanis, M.H., Liu, K.S., Paty, J.A., Kassel, J.D., et al. (2002). Immediate antecedents of cigarette smoking: An analysis from ecological momentary assessment. *Journal of Abnormal Psychology*, 111, 531-545.
- Shiffman, S., Hickcox, M., Paty, J.A., Gnys, M., Kassel, J.D., & Richards, T.J. (1996). Progression from a smoking lapse to relapse: prediction from abstinence violation effects, nicotine dependence, and lapse characteristics. *Journal of Consulting and Clinical Psychology*, 64, 993-1002
- Shiffman, S., Hickcox, M., Paty, J.A., Gnys, M., Richards, T., & Kassel, J.D. (1997). Individual differences in the context of smoking lapse episodes. *Addictive Behaviors*, 22, 797-811
- Shiffman, S., Hickcox, M., Paty, J., Gnys, M., Kassel, J.D., & Richards, T.J. (1997). The abstinence violation effect following smoking lapses and temptations. *Cognitive Therapy and Research*, 21, 497-523.
- Shiffman, S., Hufford, M., Hickcox, M., Paty, J.A., Gnys, M., & Kassel, J.D. (1997). Remember that? A comparison of real-time versus retrospective recall of smoking lapses. *Journal of Consulting and Clinical Psychology*, 65, 292-300.
- Shiffman, S., & Jarvik, M.E. (1976). Smoking withdrawal symptoms in two weeks of abstinence. *Psychopharmacology*, 30, 35-39.
- Shiffman, S., Paty, J.A., Gnys, M., Kassel, J.A., & Hickcox, M. (1996). First lapse to smoking: within-subjects analysis of real-time reports. *Journal of Consulting and Clinical Psychology*, 64, 366-379.
- Shiffman, S., Paty, J.A., Gwaltney, C.J., & Dang, Q. (2004). Immediate antecedents of cigarette smoking: an analysis of unrestricted smoking patterns. *Journal of Abnormal Psychology*, 113, 166-171.
- Shiffman, S., Scharf, D.M., Shadel, W.G., Gwaltney, C.J., Dang, Q., Paton, S.M., et al. (2006). Analyzing milestones in smoking cessation: illustration in a nicotine patch trial in adult smokers. *Journal of Consulting and Clinical Psychology*, 74, 276-285.

- Shiffman, S., & Waters, A.J. (2004). Negative affect and smoking lapses: a prospective analysis. *Journal of Consulting and Clinical Psychology*, 72, 192-201.
- Shumate, M., & Worthington, E.L. (1987). Effectiveness of components of self-verbalization training for control of cold pressor pain. *Journal of Psychosomatic Research*, 31(3), 301-310.
- Silagy, C., Lancaster, T., Stead, L., Mant, D., & Fowler, G. (2002). Nicotine replacement therapy for smoking cessation. *Cochrane Database System Review*, 4, CD000146.
- Silagy, C., Mant, D., Fowler, G., & Lodge, M. (1994). Meta-analysis on efficacy of nicotine replacement therapies in smoking cessation. *Lancet*, 353, 139-142.
- Simons, J.S. & Gaher, R.M. (2005). The distress tolerance scale: development and validation of a self-report measure. *Motivation and Emotion*, 29, 83-102.
- Smith, S. S., Jorenby, D. E., Fiore, M. C., Anderson, J. E., Mielke, M. M. & Beach, K. E. (2001). Strike while the iron is hot: Can stepped-care treatments resurrect relapsing smokers? *Journal of Consulting and Clinical Psychology*, 69, 429-439.
- Sobell, L. C., & Sobell, M. B. (1996). *Timeline followback: A calendar method for assessing alcohol and drug use*. Toronto, Canada: Addiction Research Foundation.
- Sobell, L. C., & Sobell, M. B. (1980). Convergent validity: An approach to increasing confidence in treatment outcome conclusions with alcohol and drug abusers. In L. C. Sobell, M.B. Sobell & E. Ward (Eds.), *Evaluating alcohol and drug abuse treatment effectiveness: Recent advances* (pp. 177-183). New York: Pergamon Press.
- Sobell, L. C., & Sobell, M. B. (1979). Validity of self-reports in three populations of alcoholics. *Journal of Consulting and Clinical Psychology*, 46, 901-907.
- Soria, R., Legido, A., Escolano, C., Lopez Yeste, & Montoya, J. (2006). A randomised controlled trial of motivational interviewing for smoking cessation. *British Journal of General Practice*, 56, 768-774.
- Spanier, C.A., Shiffman, S., Maurer, A., Reynolds, W., & Quick, D. (1996). Rebound following failure to quit smoking: the effects of attributions and self-efficacy. *Experimental and Clinical Psychopharmacology*, 4, 191-197.
- Spitzer, R.L. (1983). Psychiatric diagnosis: Are clinicians still necessary? *Comprehensive Psychiatry*, 24, 399-411
- Steer, R. A. & Clark, D. A. (1997). Psychometric characteristics of the Beck Depression Inventory-II with college students. *Measurement & Evaluation in Counseling & Development*, 30, 128-136.

- Steer, R. A., Rissmiller, D. J. & Beck, A. T. (2000). Use of the Beck Depression Inventory–II with depressed geriatric inpatients. *Behaviour Research & Therapy*, 38, 311-318.
- Step toe, A., & Ussher, M. (2006). Smoking, cortisol and nicotine. *International Journal of Psychophysiology*, 59, 228-235.
- Stewart, S.H., Karp., J., Pihl, R.O., & Peterson, R.A. (1997). Anxiety sensitivity and self-reported reasons for drug use. *Journal of Substance Abuse*, 9, 223-240.
- Steinberg, M. L., Krejci, J. A., Collett, K. Brandon, T. H., Ziedonis, D. M., et al. (2007). Relationship between self-reported task persistence and history of quitting smoking, plans for quitting smoking, and current smoking status in adolescents. *Addictive Behaviors*, 32, 1451-1460.
- Stipelman, B. A., Bornovalova, M., Brown, R., Kahler, C., Strong, D., Zvolensky, M., & Lejuez, C. W. (2007). *Distress tolerance as a predictor of early smoking relapse*. Paper presented at the 69th annual meeting of the College on Problems of Drug Dependence, Quebec, Canada.
- Stitzer, M.L., & Gross, J. (1988). Smoking relapse: the role of pharmacological and behavioral factors. In O.F. Pomerleau, & C.S. Pomerlau (Eds.) *Nicotine replacement: A critical evaluation*. New York: Alan R. Liss Inc.
- Stolerman, I.P. & Shoaib, M. (1991). The neurobiology of tobacco addiction. *Trends in Pharmacological Sciences*, 12, 467-473.
- Strong, D.R., Lejuez, C.W., Daughters, S., Marinello, M., Kahler, C.W., & Brown, R.A. (2003). The Computerized Mirror Tracing Task Version 1. *Unpublished Manual*
- Swan, G.E., & Denk, C.E. (1987). Dynamic models for the maintenance of smoking cessation: event history analysis of late relapse. *Journal of Behavioral Medicine*, 10, 527-554.
- Tashkin, D., Kanner, R., Bailey, W., Buist, S., Anderson, P., Nides, M. et al. (2001). Smoking cessation in patients with chronic obstructive pulmonary disease: a double blind placebo-controlled randomized trial. *Lancet*, 357, 1571-1575.
- Taylor, S., & Cox, B.J. (1998). An expanded Anxiety Sensitivity Index: Evidence for a hierarchic structure in a clinical sample. *Journal of Anxiety Disorders* 12, 463–483.
- Teneggi, V., Tiffany, S.T., & Squassante, L. (2002) Smokers deprived of cigarettes for 72 hours: Effect of nicotine patches on craving and withdrawal. *Psychopharmacology*, 164, 177-187.



- Thakore, J.H., Berti, C., & Dinan, T.G. (1999). Diurnal variation of nicotine-induced ACTH and cortisol secretion in non-smoking healthy volunteers. *Human Psychopharmacology*, 14, 179-183.
- Tutoo, D.N. (1971). Psychodiagnostic applications of the mirror tracing test. *Indian Educational Review*, 6, 293-303.
- USDHHS (1995). *Clearing the Air: How to quit smoking ... and quit for keeps* (No. NIH publication no. 95-1647): Public Health Service, National Institutes of Health, National Cancer Institute.
- US Department of Health and Human Services (1994). Preventing Tobacco Use Among Young People: A Report of the Surgeon General. Washington, DC, US Government Printing Office.
- Ussher, M., West, R., Evans, P., Steptoe, A., McEwen, A., Clow, A., et al. (2006). Reduction in cortisol after smoking cessation among users of nicotine patches. *Psychosomatic Medicine*, 68, 299-306.
- Velicer, W.F., Diclemente, C. C., Rossi, J. S., & Prochaska, J. O. (1990). Relapse situations and self-efficacy: An integrative model. *Addictive Behaviors*, 15, 271-283.
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, 54, 1063-70.
- Welsch, S., Smith, S., Wetter, D., Jorenby, D., Fiore, M., & Baker, T. (1999). Development and validation of the Wisconsin Smoking Withdrawal Scale. *Experimental and Clinical Psychopharmacology*, 7, 354-361.
- West, R. J., Edwards, M., & Hajek, P. (1998). A randomised controlled trial of a "buddy" system to improve success at giving up smoking in general practice. *Addiction*, 93, 1007-1011.
- West, R. J., Hajek, P., Belcher, M. (1989). Severity of withdrawal symptoms as a predictor of outcome of an attempt to quit smoking. *Psychological Medicine*, 19, 981-985.
- Westman, E.C., Behm, F.M., Simel, D.L., & Rose, J.E. (1997). Smoking on the first day of a quit attempt predicts long-term abstinence. *Archives of Internal Medicine*, 157, 335-340.
- White, W.D., Crockford, D., & Patten, S. (2005). A randomized, open-label pilot

- comparison of gabapentin and bupropion SR for smoking cessation. *Nicotine & Tobacco Research*, 7, 809-813.
- Wilkins, J.N., Carlson, H.E., Van Vunakis, H., Hill, M.A., Gritz, E., & Jarvik, M.E. (1982). Nicotine from cigarette smoking increases circulating levels of cortisol, growth hormone, and prolactin in male chronic smokers. *Psychopharmacology (Berl)*, 78, 305-308.
- Willoughby, S. G., Hailey, B. J., Mulkana, S., & Rowe, J. (2002). The effect of laboratory-induced depressed mood state on responses to pain. *Behavioral Medicine*, 28(1), 23-31
- Wills, T.A., Sandy, J.M., & Yaeger, A.M. (2002). Stress and smoking in adolescence: A test of directional hypothesis. *Health Psychology*, 21, 122-130.
- Winternitz, W.W., & Quillen, D. (1977). Acute hormonal response to cigarette smoking. *Journal of Clinical Pharmacology*, 17, 389-397.
- Wise, R.A. (1988). The neurobiology of craving: implications for the understanding and treatment of addiction. *Journal of Abnormal Psychology*, 97, 118-132.
- Zahler, C., Halbert, R., DuBois, R., George, D., & Nonikov, D. (2004). Smoking related diseases: the importance of COPD. *International Journal of Tuberculosis and Lung Disorders*, 8, 1423-1428.
- Zanarini MC, Skodol AE, Bender D, Dolan R, Sanislow C, Schaefer E, et al. (2000). The Collaborative Longitudinal Personality Disorders Study: reliability of axis I and II diagnoses. *Journal of Personality Disorders*, 14(4):291-299.
- Zhu, S.H., Stretch, V., Balabanis, M., Rosbrook, B., Sadler, G., & Pierce, J.P. (1996). Telephone counseling for smoking cessation: effects of single-session and multiple-session interventions. *Journal of Consulting and Clinical Psychology*, 64, 202-211.
- Zuckerman, B., Amaro, H., Bauchner, H., & Cabral, H. (1989). Depressive symptoms during pregnancy: relationship to poor health behaviors. *American Journal of Obstetrics and Gynecology*, 160, 1107-1111
- Zvolensky, M.J., Baker, K.M., Leen-Feldner, E.W., Bonn-Miller, M.O., Feldner, M.T., & Brown, R.A. (2004). Anxiety sensitivity: association with intensity of retrospectively-rated withdrawal symptoms and motivation to quit. *Cognitive and Behavioural Therapy*, 33, 114-124.
- Zvolensky, M.J., Bonn-Miller, M.O., Feldner, M.T., Leen-Feldner, E., McLeish, A.C., &

- Gregor, K. (2006). Anxiety sensitivity: concurrent associations with negative affect smoking motives and abstinence self-confidence among young adult smokers. *Addictive Behaviors*, 31, 429-439.
- Zvolensky, M.J., Feldner, M.T., Eifert, G.H., & Brown, R.C. (2001). Affective style among smokers: understanding anxiety sensitivity, emotional reactivity, and distress tolerance using biological challenge. *Addictive Behavior*, 26, 901-915.
- Zvolensky, M.J., Feldner, M.T., Leen-Feldner, E., Bonn-Miller, M.O., McLeish, A.C., & Gregor, K. (2004). Evaluating the role of anxiety sensitivity in smoking outcome expectancies among regular smokers. *Cognitive Therapy and Research*, 28, 374-486.
- Zvolensky, M.J., Lejuez, C.W., Kahler, C.W., & Brown, R.A. (2004). Panic attack history and smoking cessation: an initial examination. *Addictive Behaviors*, 29, 825-830.
- Zvolensky, M.J. & Schmidt, N.B., & Stewart, S.H. (2003). Panic disorder and smoking. *Clinical Psychology: Science and Practice*, 10, 29-51.