Relapse Dynamics During Smoking Cessation:

Recurrent Abstinence Violation Effects and Lapse-Relapse Progression

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Smoking relapse is most often the end point of a process that unfolds over a period of days or weeks and is characterized by many intermittent lapses. According to Relapse Prevention theory, progression to relapse is driven by the Abstinence Violation Effect (AVE), a set of cognitive and emotional responses to lapsing that predisposes quitters to further lapses in an accelerating downward spiral. However, the dynamic relationship between lapse responses and relapse progression during smoking cessation has not been a focus of research. We used mixed-effect growth modeling and recurrent event survival analyses to investigate the way AVE-related lapse responses evolve over the course of a cessation attempt and prospectively influence subsequent lapse-relapse progression. Participants were 203 smokers who achieved abstinence and subsequently lapsed on one or more separate occasions. Using electronic diaries for Ecological Momentary Assessment, participants recorded their reactions to each lapse in real time. Findings revealed a great deal of variability between participants and from lapse-to-lapse in the severity of AVE responses, indicating that participants differed in the extent that their AVE responses intensified versus improved with each successive lapse. In turn, AVE response was found to explain subsequent lapse progression rates, above and beyond the predictive influence of other traditional explanatory variables. Results indicate that while participants' responses to the first lapse they experienced were unrelated to whether they ultimately relapsed, those who reported higher levels of self-efficacy following their first lapse had a slower rate of progression from each successive lapse to the next (HR=0.93, CI=0.89-0.97).Controlling for responses to

their initial lapse, we found that responses to each additional lapse influenced lapse progression rates, such that higher levels of both self-blame (HR=0.99, CI=0.98-0.99) and self-efficacy (HR=0.95, CI=0.92-0.99) were associated with slower progression to a subsequent lapse. Incremental increases in guilt from lapse-to-lapse were associated with slower progression to an additional lapse (HR=0.96, CI=0.92-0.99), while increasingly negative affective valence from lapse-to-lapse was associated with accelerated lapse progression (HR=1.05, CI=1.00-1.09). Results highlight the dynamic nature of lapse responses during smoking cessation, demonstrating the way psychological responses may drive progression from one lapse to the next.

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PREFACE

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1.0 Introduction: Background and Significance

Clinical data consistently indicate that attempts to change addictive behaviors most often end with full resumption of the problematic behavior pattern (i.e., relapse). Relapse necessarily begins with an initial episode of drug use (i.e., a lapse), yet relapse seldom occurs all at once (Brownell, Marlatt, Lichtenstein, & Wilson, 1986; McKay, Franklin, Patapis, & Lynch, 2006; Miller, 1996). This is particularly true during smoking cessation, as smoking relapse is most often the end point of a process that unfolds over a period of days or weeks and is characterized by many intermittent lapses.Over the course of this lapse-relapse process, the quitter attempts, with varying degrees of effort and success, to reestablish abstinence (i.e., achieve prolapse). While approximately 85-95% of lapsers ultimately relapse (e.g., Brandon, Tiffany, & Baker, 1986; Kenford, Fiore, Jorenby, Smith, Wetter, & Baker, 1994), many also successfully recover, especially when considering a time-limited cessation trial (e.g., Wileyto et al., 2005).

By far the dominant model for explaining why lapses progress to relapse is the relapse prevention model developed by Marlatt. According to the relapse-prevention model (Marlatt & Gordon, 1985), the primary determinants of whether an individual who has lapsed will either relapse or successfully reestablish abstinence are that person's cognitive and emotional responses to lapsing. Specifically, relapse is predicted to be more likely when lapses produce an abstinence violation effect (AVE), characterized by self-blame, reduced self-efficacy, and negative affect. This constellation of responses, coupled with the subjective effects of drug ingestion, is posited to predispose the person to further lapses, thus driving the lapse-relapse process in an accelerating downward spiral (Witkiewitz & Marlatt, 2004). Alternatively, when an individual manages to successfully resist a temptation to smoke, self-efficacy to cope with future temptations and maintain abstinence is expected to increase (Marlatt & Gordon, 1985). Thus,

the relapse prevention model predicts that AVE responses will fluctuate as individuals experience successive lapse and temptation events over the course of the lapse-relapse process.

Not all lapse episodes are expected to elicit an AVE response of equivalent magnitude. The relapse-prevention model assumes that greater AVE responses will occur when the associated lapse or temptation is attributed to internal, stable, and globalcausal factors (Marlatt & Gordon, 1985). These causal attributions are thought to determine the way individuals interpret the meaning and implications of a lapse. Whereas a lapse that is attributed to a momentary, context-specific, external cause (e.g., an unavoidable stressor) would be expected to elicit only a minimal AVE, the model predicts that a lapse which is interpreted as reflecting a lack of will-power would trigger a more powerful AVE and thereby increase the likelihood of relapse. Marlatt (Marlatt & Gordon, 1985) likens this reaction to the experience of cognitive dissonance as originally described by Festinger (1964). In both cases the individual is driven to resolve the perceived discrepancy between their intentions to maintain abstinence and their actual behavior (lapse), because this discrepancy produces an aversive cognitive and affective reaction.

The relapse prevention model can be contrasted with a number of alternative theoretical perspectives on the factors underlying lapse-relapse progression. For instance, incentive models (e.g., Shaham, Shalev, Lu, de Wit, & Stewart, 2003; Stewart & Wise, 1992) assume that small priming doses (i.e., such as those absorbed in lapses) of the drug from which an addict is abstaining can trigger relapse by reinstating conditioned self-administration of the drug. Yet there are competing explanations for why this may be the case. Priming doses of a drug may function as a pleasurable conditioned response and thus elicit an appetitive drive toward additional use via positive reinforcement (Stewart, de Wit, & Eikelboom, 1984). Negative reinforcement models (Baker et al., 2004), along with the opponent-process model (Siegel,

1989), posit that priming doses increase the likelihood that additional lapses will be used to overcome unpleasant craving and withdrawal. On the other hand, neural sensitization models posit that priming doses can reinstate self-administration entirely non-consciously, via neurocognitive motivation circuits that underlie previously conditioned procedural drug-use action schemes (Baker et al., 2004; Robinson & Berridge, 2003; Kalivas & Volkow, 2006). None of these models assign a central role to Marlatt's conceptualization of subjective abstinence violation effects during the lapse-relapse process.

Another alternative perspectiveon the lapse-relapse process isprovided by the self-control strength model developed by Baumeister and colleagues (Baumeister, Heatherton, & Tice, 2004; Muraven & Baumeister, 2000). According to this model, continuous self-regulation of emotion and behavior gradually reduces what are limited capacity self-control resources. Reductions in self-regulatory control are not permanent, because resources can be replenished when self-control is relinquished (e.g., during a lapse episode, while eating ice cream, or resting). Yet because self-control strength is regained more slowly than it is used, the self-control strength model predicts that self-control resources will progressively decline with continued self-control, even as the degree of decline is offset by intermittent periods of rest and recovery. Because lapsing during a cessation attempt may provide relief from exertion of self-control, doing so may actually allow recovery of self-control, and thereby protect against further lapsing, at least in the short term. On the other hand, in line with negative reinforcement accounts (Baker et al., 2004), the relief that lapses provide may increase the likelihood of additional lapses when control resources inevitably deplete. Fortunately for the quitter, this model assumes that those who repeatedly engage in cycles self-control depletion and recovery will progressively build self-control strength, like working-out to build muscle.

These alternative theoretical perspectives highlight the fact that we have a great deal to learn about quitters' cognitive and affective responses to lapse, and the way these responses are associated with relapse outcomes. The overarching goal of the proposed project is to examine the way psychological responses to lapse evolve over the lapse-relapse process, and the extent to which these responses drive the seemingly inevitable progression from lapse to relapse.The projectwill utilize EMA measures of the AVE obtained over the course of 7 weeks of monitoring as smokers struggled to avert relapse. The data were collected in the course of a randomized placebo-controlled clinical trial of nicotine patch for smoking cessation, but the efficacy of the pharmacological treatment is not the focus. The relapse-prevention model will be used as a theoretical framework for reviewing past research and forming new hypotheses regarding the mechanisms of lapse-relapse progression.

The project has two principal aims. The firstis to examineAVE responses to lapses that occur over the course of a voluntary cessation attempt. As such, this study will be the first to investigate the way AVE responses evolve over the course of the lapse-relapse process. Second, the proposed research will assess the extent to which AVE responses to lapse episodes influence lapse progression and relapse. Carefully modeling responses to lapse may improve our ability to predict variation in lapse-relapse progression, and thereby improve prediction of cessation success versus failure. In linking lapse responses to subsequent smoking across the lapse-relapse process, this project could provide a glimpse of the accelerating downward spiral thought to lead to relapse.

1.1 Studying the Abstinence Violation Effect

Although relapse-prevention theory posits a progressive process toward relapse, methodological limitations have often forced researchers to treat the AVE responses to the very

first lapse as an isolated event that directly influences relapse (cf., Collins, 1993). Given that smokers usually lapse many times before they relapse, over what is often an extended period of time, this approach to study of the AVE is especially limited when applied to smoking cessation. A smoker who experiences a severe AVE after an initial lapse might well recover from that AVE reaction and avoid relapse if he or she manages to maintain longer and longer periods of abstinence prior to subsequent slips. Conversely, a smoker who remains confident and optimistic after an initial lapse mightbecome demoralized afterseveral subsequent lapses, especially if they are attributed to personal weakness. It is therefore essential to look beyond the initial lapse and measure the way AVE responses fluctuate across multiple lapse episodes. Expanding our analysis in this way promises to provide valuable information regarding both the nature of AVE responses and their association with lapse-relapse outcomes during smoking cessation.

While there is preliminary evidence that initial AVE responses influence relapse (e.g., Baer, Kamarck, Lichtenstein, & Ransom, 1989; Curry, Marlatt, & Gordon, 1987), empirical support for the relapse-prevention model is hamperedby the fact that all studies have focused exclusively on responses to the first lapse that quitters experience, and thus fail to document the progressive aspect of the AVE discussed above. Moreover, an important methodological limitation of nearly all studies conducted to date is the use of retrospective self-report measures to assess these momentary AVE reactions long after they occur. In addition to the documented inaccuracies of retrospective recall (e.g., Shiffman, Hufford, et al., 1997), these measures are problematic because they are administered after respondents have already progressed to either prolapse or relapse. Knowledge of one's ultimate success or failure with cessation threatens to bias retrospective judgments about the determinants and implications of past lapses (Ross, 1989).

As such, it is hardly surprising that retrospective studies have found that smokers who have already relapsed are more likely than those who are still abstinent to recall low levels of post-lapse self-efficacy along with heightened levels of remorse. Such findings do not demonstrate a prospective, much less causal, link between the AVE and lapse-relapse progression.

The methodological limitations characteristic of most AVE studies are due, in part, to the inherent difficulty of measuring momentary, naturally occurring phenomena over extended periods of time. When individual quitters experience a lapse or temptation, they do so at random points throughout the day and night, making it difficult to assess AVE responses over the course of an entire cessation attempt. Probably the best way to accomplish this is via assessment methods that document momentary processes close to the time they occur (Bolger, Davis, & Rafaeli, 2003; Shiffman & Stone, 1994; Tennen & Affleck, 2003). Ecological momentary assessment (EMA),makes it possible to measure recurrent behaviors and subjective experience as they occur in naturalistic settings. Moreover, because EMA reduces the time-gap between response occurrence and assessment, it minimizes the influence of retrospective bias (Shiffman, Hufford, Hickcox, et al., 1997). Yet EMA methods have only recently begun to be utilized in studies of lapse-relapse progression (e.g., Shiffman, Ferguson, & Gwaltney, 2006; Shiffman & Kirchner, under review).

1.2 Abstinence Violation Effects and Lapse-Relapse Progression

As outlined above, the AVE is a multifaceted construct, comprised of both cognitive and affective elements. To the extent that a lapse in attributed to internal, dispositional factors, a constellation of responses is elicited that is characterized by self-blame, reduced self-efficacy, and negative affect. As a consequence, the likelihood of subsequent lapses is predicted to increase. The association between AVE responses and relapse has been examined across a wide

range of addictive behaviors (Marlatt & Donovan, 2005). Studieslinking AVE responses to alcohol use, other drug use, sexual risk-taking, and eating behavior have been published (e.g., Birke, Edelmann, & Davis, 1990; Collins & Lapp, 1991; Grilo & Shiffman, 1994; Laws, 1995; McKay, Rutherford, Alterman, and Cacciola, 1996; Miller, Westerber, Harris, & Tonigan, 1996;Walton, Castro, & Barrington, 1994).Despite its theoretical appeal and prevalence in other areas of health behavior change, however, a relatively small number of studies have examined the AVE during smoking cessation.

In most studies that have examined AVE responses during smoking cessation, the AVE has been conceptualized as an acute response to the initial lapse that quitters experience. Thus it is assumed that this single AVE response has consequences for the entire lapse-relapse process. In this regard, although it is a momentary response, the AVE is treated as a qualitative shift, similar to the throw of a switch, after which the individual precedes either to relapse or to recovery. From this perspective, the significance of the initial lapse response overshadows all those that follow. This approach to the study of the AVE fails to provide a complete test of Marlatt et al.'s (1985) original account, which allows for the fact that the first lapse that an individual experiences may elicit only a weak AVE response, and may therefore remain nothing but a single slip (p.158). Because many slips may occur that do not trigger an AVE powerful enough to trigger full-blown relapse, it follows that one must observe the entire cessation process to evaluate the degree to which AVE responses of different severities drive lapse-relapse progression. No study conducted to date has empirically examined the AVE in this way.

Some of the earliest research on AVE responses during smoking cessation used retrospective self-report measures to investigate the link between initial lapses and lapse attributions, decreased self-efficacy, and/or negative affect(e.g., Brandon et al., 1990; Condiotte

& Litchenstein, 1981; Curry, Marlatt, & Gordon, 1987). In one of the first studies of the AVE during smoking cessation, Condiotte and Litchenstein (1981) found that relapsing participants often reported that their initial lapse was due to internal factors, and that they had experienced decreased self-efficacy and feelings of guilt as a result. Similarly, Curry, Marlatt, and Gordon (1987) found that participants who had already relapsed attributed prior lapses to internal, global, and stable causal factors.While these findings are encouraging, their interpretation is obscured by the fact that both utilized retrospective designs. Although the authors suggest a link between lapses and AVE responses, an alternative interpretation of the findings is that participants' responses were post-hoc explanations for the fact that they had subsequently relapsed (e.g., "I relapsed, therefore I must have been too weak to avoid lapsing, and must have felt guilty").

To improve upon the limitations of retrospective design, Shiffman, Hickcox, Paty, Gnys, Kassel, & Richards (1996) conducted a prospective study of AVE responses to lapse during smoking cessation. Shiffman et al. (1996) used ecological momentary assessment methods to investigate the influence of abstinence violation effects, as well as a number of other predictors, on progression from an initial lapse to a second lapse, and from the second lapse to relapse. Results of this study indicated that AVE responses did not affect progression to relapse, although participants who reported that they felt like giving up after the first lapse progressed more rapidly to a second lapse. Lapses triggered by stress or mood (positive or negative) were associated with accelerated progression to relapse, whereas lapses that occurred during alcohol consumption or eating actually slowed progression to relapse down.

Researchers have also attempted to evaluate the predictions of the relapse-prevention model regarding the effects of resisted temptations on attributions of control over smoking and abstinence self-efficacy. Three studies have found evidence that supports the relapse-prevention

model, demonstrating that resisted temptations boosted ratings of abstinence self-efficacy(Garcia, Schmitz, & Doerfler, 1990; Schmitz, Rosenfarb, & Payne, 1993), and predicted a lower risk of lapsing (O'Connell et al., in press). However, two other studies have reported contradictory findings. First, Shiffman (1984) found that temptations actually resulted in reduced self-efficacy ratings. Second, Shiffman et al. (1997) reported that temptations had no effect on self-efficacy nor other AVE responses. These findings indicate that even when a lapse is averted, the experience of a strong temptation may sometimes be interpreted as cause for concern.

Although there aresome encouraging preliminary findings, the degree to which AVE responses are associated with relapse outcomesremains unclear. While some studies suggest that more intense AVE responses are associated with relapse (Baer, Kamarck, Lichtenstein, & Ransom, 1989; Garcia, Schmitz, & Doerfler, 1990; O'Connell & Martin, 1987;Schoeneman, Hollis, Stevens, Fischer, & Cheek, 1988), a number of others have found that initial AVE responses are unrelated to lapse-relapse outcomes (Brandon, Tiffany, Olefski, & Baker, 1990; Borland, 1990; Hall, Havassy, & Wasserman, 1990; Schoeneman, Stevens, Hollis, Cheek, &Fischer, 1988; Shiffman, Ferguson, & Gwaltney, 2006; Shiffman, Hickcox, Paty, et al., 1996; Shiffman, Hickox, Paty, et al., 1997). It is noteworthy that in contrast to the findings of many retrospective studies, the only prospective study conducted to date did not find a link between AVE responses to first lapse and relapse outcomes (Shiffman et al., 1996; Shiffman et al., 1997). Again, these findings suggest that inconsistent findings in the AVE literature are at least partially due to methodological design flaws.

Aside from the interpretive limitations associated with retrospective study design, it is difficult to draw convergent conclusions from the findings of studies conducted to date because

the AVE is often conceptualized and measured in different ways. There are currently neither measurement guidelines nor a standardized operational definition of what constitutes an AVE response. Smoking researchers have measured AVE responses with a variable range of measures related to causal attributions, self-efficacy, guilt, and/or negative affect. For example, whereas Curry, Marlatt, and Gordon (1987) restricted their conceptualization of the AVE to lapse attributions,Grove (1993) measured both attributions and self-efficacy,whileShiffman, Hickcox, et al. (1996) measured attributions, self-efficacy, guilt, and negative affect.As a consequence,the AVE construct may be too nonspecificfor reviewing findings from the research literature. Because studies have not measured the AVE the same way, it would be useful to consider the empirical findings regarding each component of the AVE separately. In this way we can assess the extent to which the findings from studies that measured common outcomes converge.

1.2.1 Abstinence Self-Efficacy

Self-efficacy (SE) refers to the degree to which an individual believes s/he is capable of performing a behavior in a specific situational context (Bandura, 1977; 1997). In the smoking literature, SE usually refers to confidence in one's ability to abstain from smoking. Unless otherwise specified, throughout the present manuscript SE refers to abstinence self-efficacy. SE has been established as a predictor of outcomes across a range of addictive behaviors (e.g., Baer, Holt & Lichtenstein, 1986; Sklar, Annis, & Turner, 1999; Sylvain, Ladouceur & Boisvert, 1997). Accordingly, SE theory has been incorporated into a number of prominent models of addictive behavior and behavior change (e.g., Ajzen, 1991; Baker et al., 2004; Witkiewitz & Marlatt, 2004; Niaura, 2000). Indeed, Marlatt (Marlatt & Gordon, 1985) contends that the perception of reduced SE following a lapse is the central tenet of the AVE construct.

Smoking a cigarette during a voluntary attempt to maintain abstinence has seemingly obvious implications for an individual's level of SE. Because the act of smoking directly contradicts intentions to maintain abstinence, violating abstinence should lead to a reduction in SE. According to the relapse prevention model, however, an individual is most likely to experience a reduction in SE when the lapse is attributed to internal, dispositional factors. Empirical findings generally confirm the predictions of the relapse prevention model regarding post-lapse SE.Several studies have reported that lapse attributions are related to SE (Curry, Marlatt, & Gordon, 1987; Eiser & Van der Plight, 1986; Eiser, Van der Plight, Raw, & Sutton, 1985; Grove, 1993; Hedeker& Mermelstein, 1996; Spanier, Shiffman, Maurer, Reynolds, & Quick, 1996; Weiner, 1974). For examples, both Curry, Marlatt, and Gordon (1987) and Spanier et al. (1996) observed that internal, stable, and global lapse attributions were inversely correlated with SE during cessation.

Like other AVE studies, those that examine the influence of post-lapse SE on relapse have measured SE before or after an initial lapse and use these responses to predict subsequent smoking relapse (e.g., Baer, Holt, & Lichtenstein, 1986; Godin, Valois, Lepage, & Desharnais, 1992;Norman, Conner, & Bell, 1999). The assumption implicitly underlying this approach is that SE responses are relatively stable, having a consistent effect on smoking behavior across environmental situations. However, empirical evidence and prominent models of addictive behavior suggest that SE should vary across both internal states and external contexts (e.g., Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Gwaltney, Shiffman, Balabanis, & Paty, 2005; Marlatt & Gordon, 1985). Especially during a cessation attempt, it is likely that recurrent lapse and temptation episodes have a powerful influence on quitters' perceptions of SE. Post-lapse variations in SE may thus explain much of the observed individual variation in lapse-

relapse outcome trajectories (e.g., Conklin et al., 2005). Yet, no studies have examined the way SE responses vary across a series of multiple lapse and temptation events, so this possibility remains untested.

A considerable number of studies have used retrospective methods to investigate the notion that post-lapse SE influences subsequent lapse-relapse progression (Baer, Holt & Lichtenstein, 1986; Colletti, Supnick, & Payne, 1985; DeClemente et al., 1991; Godin, Valois, Lepage, & Desharnais, 1992;McIntyre, Lichtenstein, & Mermelstein, 1983; Norman, Conner, & Bell, 1999; Velicer, DiClemente, Rossi, & Prochaska, 1990). Although it is difficult to interpret the findings of most of these studies because they rely on retrospective measures, some have demonstrated a prospective link between post-lapse SE and smoking behavior (e.g., Garcia, Schmitz, & Doerfler, 1990; Gwaltney, Shiffman, Balabanis, & Paty, 2005). For example, Garcia, Schmitz, and Doerfler (1990) found that self-monitored decreases in post-lapse SE and increases in post-temptation SE predicted later relapse. Likewise, in an electronic diary study, Gwaltney, Shiffman, Balabanis, and Paty (2005) found that drops in daily SE following a lapse predicted relapse outcomes.

Partially because it is difficult to measure AVE responses over the course of the entire lapse-relapse process, researchers investigating the AVE have restricted their analysis to the first lapse from abstinence that occurs, assuming that responses to this lapse are generally representative of those that follow The limitation of this approach is that it ignores within-subject variation in SE as quitters experience subsequent lapses and temptation episodes. Prospectively examining the way SE, as well as other AVE responses, evolve across the lapse-relapse process could provide a large amount of additional information regarding cognitive responses to lapse during cessation.

1.2.2 Negative Affect

Negative affect (NA) generally refers to an individual's subjective experience of negative emotion. A large number of empirical studies have sought to examine the influence of NA on smoking behavior (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Kassel, Stroud, & Paronis, 2003). Findings to date indicate that the link between NA and smoking is complex, with evidence demonstrating that the influence of NA varies considerably across the stages of smoking (Brown, Lejuez, Kahler, Strong, & Zvolensky, 2005; Kassel et al., 2003). However, because most studies have examined the degree to which NA precipitates initial lapses (Shiffman, 2005), the influence of NA on lapse-relapse progression remains poorly understood (e.g., Shiffman, Ferguson, & Gwaltney, 2006).

According to the relapse-prevention model, lapses are a demoralizing experience that can trigger a range of negative emotions. Because no research conducted to date has examined NA responses beyond the initial lapse, we know very little about the way momentary NA responses fluctuate as individuals experience additional lapse and temptation episodes. The relapse-prevention model predicts that NA responses will intensify with each successive lapse. Examining the way NA responses vary across these lapse events may partially account for both variation in lapse-relapse outcome trajectories, and variation in other more cognitive AVE responses, such as SE.

The relapse prevention model assumes that lapses often elicit general forms of NA, such as depression or anxiety, but the model also assumes that lapses can elicit more specific feelings of guilt or anger. It is currently unclear whether distinguishing between these variants of NA will clarify its role in lapse-relapse progression. Several studies have investigated the idea that lapses

trigger increases in different forms of NA(e.g., Brandon et al., 1990; Cinciripini, Wetter, Fouladi, et al., 2003; Schoeneman, Stevens, et al., 1988; Shiffman, Hickox, Paty, Gnys, Richards, & Kassel, 1997; Shiffman & Waters, 2004). For instance, Brandon et al. (1990) found that 76% of smokers attempting cessation reported feeling either depressed (50%), anxious (16%), or angry (10%) following their initial lapse. Similarly, in a study that utilized EMA methodology, Shiffman et al. (1997) reported thatinternally attributed lapses resulted in significant increases in NA, such that relative to temptation episodes, lapse episodes were associated with feelings of guilt and discouragement. While only a small number of studies have been conducted, it is noteworthy that only one (Shiffman et al., 1997) has contrasted lapse responses with responses to resisted temptations, and systematically examined the predictive link between lapse attributions and post-lapse NA.

Researchers have also attempted to examine the extent to which post-lapse NA exacerbates perceptions of reduced control and interferes with cognitive coping resources. The relapse-prevention modelassumes that one way post-lapse NA increases the likelihood of subsequent smoking is by undermining SE and the implementation of coping skills. These predictions are consistent with the larger literature on "hot" information processing (e.g., Damasio, 1998; Loewenstein & Lerner, 2003; Metcalfe & Mischel, 1999). For instance, laboratory studies demonstrate that affective states such as hunger, thirst, drug ingestion and addictive drug craving alter cognitive processing and thereby heavily influence behavioral responses (e.g., Ferguson & Bargh, 2004; Kirchner & Sayette, in press; Loewenstein, 1996; Slovic, Peters, Finucane, & MacGregor, 2005). Negative affect elicited by a lapse may affect smoking-related judgment and decision-making in a similar way, further undermining SE and reducing cognitive resources required to implement coping skills.

As predicted by the relapse-prevention model, clinical data provide empirical support for the notionthat NA reduces both SE and copingresponses (Cinciripini, Wetter, Fouladi, et al., 2003; Drobes, Meier, & Tiffany, 1994; Gwaltney, Shiffman, & Sayette, 2005; Rabois & Haaga, 2003; Shiffman, 2005). For example, utilizing EMA methodology, Gwaltney and colleagues (Gwaltney, Shiffman, & Sayette, 2005) found that momentary SE decreasedwhen momentary NA increased.Shiffman (2005) found that NA also interfered with coping, such that NA moderated the efficacy of coping in preventing lapses. It is interesting to note that findings of this sort explain why pretreatment SE judgments, made by smokers in an affectively neutral state, tend to be over inflated and sometimes fail to predict behavioral outcomes (e.g., Stuart, Borland, & McMurray, 1994; Baer, Holt, & Lichenstein, 1986). Efficacy judgments made by smokers who are currently experiencing NA may more accurately reflect their ability to deal with high-risk lapse situations, and may therefore correspond more closely with observed behavior.

Only a small number of studies have actually measured both post-lapse NA and smoking outcomes following a cessation attempt. These studies provide preliminary support for the notion that NA responses following an initial lapse are associated with subsequent relapse (e.g., Baer et al., 1987; Brandon et al., 1990; Condiotte & Litchenstein, 1981; O'Connell & Martin, 1987). For instance, Baer et al. (1987) interviewed participants in a smoking cessation program at 1, 2, and 3 months post-treatment. These researchers found that whereas lapse attributions and confidence ratings following lapses were not prospectively associated with relapse, guilt responses following lapse were associated with subsequent relapse outcomes. Of note, findings from the Baer et al. (1987) study highlight the limitations of a unidimensional conceptualization of the AVE construct. Contrasting these findings, both Hall, Havessy, & Wasserman (1990) and

Shiffman et al. (1997) found that NA had no association with smoking outcomes. It is clear that the potentially prospective link between post-lapse NA responses, lapse progression, and relapse remains an important focus for cessation research.

1.3 Overview of the Present Research

The AVE is posited to be an acute reaction to an internally attributed lapse, wherein responses associated with self-efficacy decrease and responses associated with negative-affect increase. Studies that have examined cognitive and affective responses to lapse provide mixed support for the core elements of the AVE, and highlight the need for additional research investigating the role played by the AVE in lapse-relapse progression. While a considerable number of studies have observed lapse responses consistent with the predictions of the relapse-prevention model, the degree to which AVE responses drive lapse-relapse progression remains unclear.

Review of the AVE literature reveals an exclusive reliance on responses to the initial lapse that quitters experience, while responses to all subsequent lapses are not studied. Although two prospective studies have been conducted (Shiffman, Hickcox, Paty, et al., 1996; 1997), they too only examined responses to the initial lapse. Because the AVE has always been measured cross-sectionally, we know especially little about some of the more subtle characteristics of the AVE. No studies have examined the time-course of AVE responses, such as the degree to which they intensify or abate over time. Given that quitters usually lapse multiple times before they relapse, and that AVE theory posits a progressive process, it seems misguided to ignore cognitive and emotional responses over the hours, days, and weeks that lie between the initial AVE response and study outcomes. Valuable information regarding lapse responses may be lost when the bulk of lapses that occur during the lapse-relapse process are not examined. For instance, because it

becomes increasingly difficult to deny personal responsibility for lapses as more and more occur, attributions of blame following a lapse episode may internalize as successive lapses occur. Consistent with relapse prevention theory, the degree to which quitters internalize attributions of blame may determine their psychological responses to lapse and ultimately their success with cessation. Yet the way attributions of blame evolve across the lapse-relapse process has not been systematically examined.

AVE research has also been plagued by the use of retrospective study design. Researchers investigating the predictions of the RP model have typically asked participants to retrospectively recall their responses to their initiallapse, and examined the association between these responses and relapse outcomes that have already occurred. As highlighted throughout this manuscript, these methods introduce serious interpretive limitations. Knowledge of one's success or failure with cessation threatens to bias retrospective judgments about the determinants and implications of past lapses (Ross, 1989). As a result, it is inappropriate to draw causal inference from such data.

This review of the AVE literature suggests that it would be useful to refine the way we conceptualize and examine the AVE during smoking cessation, as doing so may improve our ability to make sense of the lapse-relapse process. Instead of restricting our focus to responses that are directly associated with an initial lapse, an alternative approach is to think of the AVE as a dynamic cascade of responses that often leads to relapse. For example, one series of studies demonstrated that whereas immediate, event-driven decreases in SE did not predict progression to a subsequent lapse (Shiffman et al., 1996), day-to-day post-lapse SE was associated with accelerated lapse progression (Shiffman, Balabanis, Paty, Engberg, Gwaltney, & Liu, 2000). These data suggest that examining responses to the initial lapse alone may omit important

information about the processes underlying lapse-relapse progression. Examining momentary AVE responses following the multiple lapses that typically precede relapse mayelucidate these processes, and thereby improve our ability to predict lapse and lapse-relapse outcomes.

1.3.1 **Specific Aims**

The present research sought to evaluate responses to lapse episodes during abstinence and examine the association between these responses and concurrent lapse-relapse progression. The project had two specific aims. First, the project aimed to extend previous findings by documenting the degree to which AVE responses intensify or abate over multiple lapse episodes. The relapse-prevention model predicts that subsequent lapses should intensify AVE responses(Marlatt & Gordon, 1985; Schmitz, Rosenfarb, & Payne, 1993).Accordingly, AVE responses following a lapse should be greater in magnitude, and this difference should increase as additional episodes occur. To test these predictions in the proposed project, we will examine both the magnitude of responses to lapse, and the extent to which AVE responses increase and decrease, respectively, with each successive lapse. New approaches to modeling longitudinal data make it possible to carefully examine the way momentary responses evolve over time. Examining the extent to which AVE responses vary as a function of lapses during smoking cessation may provide key insights into the mechanisms of lapse-relapse progression.

Examining responses to lapse promises to improve our ability to explain individual variation in lapse-relapse progression, and thereby improve prediction of cessation success versus failure and guide treatment. Toward this end, the second aim of this project was to evaluate the association between quitters' cognitive and affective responses to lapse and subsequent lapse and relapse outcomes. To accomplish this, recurrent-event survival models

were used to evaluate the extent to which AVE responses influence the hazard of lapse and relapse events over the course of the lapse-relapse process.

1.3.2 **Hypotheses**

Aim 1: Evaluate AVE responses across the lapse-relapse process.

Hypotheses center around the idea that the AVE is characterized by a dynamic cascade of responses, rather than a single qualitative shift that occurs all at once. Magnifying the effects of any one lapse, we expected factors reflecting increased effort and commitment to abstinence would be associated with larger AVEs. These included longer periods of pre-lapse abstinence time and more resisted temptations to smoke prior to each lapse. Moreover, we expected that smoking more during a lapse would be associated with greater AVEs.

Consistent with relapse-prevention theory, each successive lapse episode is expected to undermine self-efficacy, while exacerbating negative-affect and guilt. Moreover, these responses should intensify to the degree that each lapse is attributed to internal weakness. This pattern of results would provide empirical support for the basic tenets of Marlatt's AVE model.

We also expected AVE responses to change as participants experienced each additional lapse. Overall, we hypothesized that AVE responses would progressively intensify over the course of the lapse-relapse process, such that as the number of lapses increased, so would the degree that each was attributed to internal weakness, and thus the degree that self-efficacy decreased and negative-affect and guilt increased.

Aim 2: Evaluate the association between AVEs and lapse-relapse progression.

As AVE responses intensify, the risk of subsequent lapses is expected to accelerate. The severity of the AVE response following each lapse is expected to predict the risk of a subsequent lapse, such that as the severity of AVE responses increases, the time before another lapse will decrease. As a result, AVE response intensity and lapse frequency should drive one another toward relapse. This pattern of results would represent the first empirical demonstration of a cascading downward spiral driven by cognitive and affective responses to lapse.

2.0 Method

2.1 Design and overview

The present project was based on an existing database (Shiffman, Ferguson, & Gwaltney, 2006; Shiffman, Scharf, et al., 2006). The parent study was a randomized, double-blind, placebo-controlled clinical trial of high-dose nicotine patch to help smokers quit smoking. Participant recruitment and data collection occurred between October 1997 and February 2000.

2.2 Participants

Participants were 305 smokers who quit for at least 24 hours while enrolled in a research smoking cessation clinic. These smokers were recruited via advertisements for smoking cessation treatment. Telephone interviews were used to determine their initial eligibility for the trial. To qualify for initial entry into the study, participants had to smoke at least 15 cigarettes per day, to have been smoking for at least 5 years, to be between the ages of 21 and 65, to be in good self-reported health, and to report high motivation and overall efficacy to quit during a screening interview (combined score of 150 on the sum of two 0-100 scales). Exclusion criteria were regular use of non-cigarette forms of tobacco, weight less than 110 lbs, specific medical

contraindications to NP use, other serious medical illness, history of recent alcohol and/or drug abuse or mental illness, current participation in a smoking cessation clinic or study, or use of bupropion hydrochloride within the last 2 months. Women who were or who were planning to become pregnant were also excluded. Smokers who were eligible, who passed a medical screening, and who signed an informed consent form were enrolled. Data from this sample have been used in other publications (e.g., Shiffman et al., 2006).

To be eligible for inclusion in the present analyses participants from the parent study had to have: (a) achieved initial abstinence (24 hours without smoking) on or after the Target Quit Day (TQD); and (b), experienced a lapse episode during the study period. A total of 203 smokers met both of these criteria. The sample was typical of a smoking cessation treatment cohort. Fifty-six percent of the participants were women and 84% were Caucasian. Participants averaged 38.94 years of age (SD=8.89) and had been smoking for 21.8 years (SD=9.0), smoking an average of 24.9 cigarettes per day (SD=8.9) at enrollment. Eighty-four percent reported at least one previous quit attempt, with an average 3 previous attempts (Table 1).

2.3 Procedure

Individuals interested in quitting smoking were followed prior to and during the first 7-weeks of a smoking cessation attempt. As they attempted to abstain from smoking, participants monitored their moment-to-moment experiences on palm-top computers, including their reactions to any smoking behavior.

Upon enrolling in the cessation program and providing informed consent, participants were trained in the use of a palm-top computer, and used theseelectronic diaries (EDs; Shiffman, Paty, Kassel & Hickcox, 1996; Stone & Shiffman, 1994) to monitor their smoking for two weeks prior to smoking cessation. Participants were also enrolled in cognitive-behavioral smoking cessation

treatment and were given a designated target quit day (TQD). The first full day of abstinence (i.e., no smoking for >=24 hr) was designated the Quit Day.

On their TQD, participants were randomized to receive either active or placebo patches and instructed to quit smoking completely. Assessments via EDs continued for six weeks after TQD,including recording of all smoking and temptation episodes. Participants were instructed to complete assessments following each lapse, and these cognitive and affective responses are the primary focus of the proposed project.In addition, at the end of each day, the ED asked participants whether they had smoked but had failed to report the lapse episode.While these end-of-the-day confessions did not include the momentary assessments of interest, and therefore cannot be included in the primary analyses of the AVE, they will be included in the overall lapse tally for each participant.

Cessation was biochemically validated, with participants seen at least weekly to verify their smoking status via carbon monoxide (CO) testing and to report on adverse events. Any participant who reported abstinence, but who had an expired air CO > 10 ppm was considered a smoker in the analyses with smoking assigned to the first day after the last clean CO.

The ED also audibly prompted participants at random intervals (Random Prompts) to complete assessments. The timing of the prompts was random with the constraint that no prompts were issued for 30 minutes after a lapse or temptation assessment. The audible prompt lasted for two minutes. Compliance with the assessment protocol was high (participants responded to Random Prompts within two minutes approximately 92% of the time; see Shiffman et al., 2006). Participants completed an average of 4.36 ± 1.38 random prompts per day during the post-quit period. More extensive details concerning the procedures used for this study can be found in Shiffman, Scharf, et al. (2006).

2.4 Nicotine-patch treatment

Participants were randomized on the TQD to receive either 35-mg transdermal nicotine patch (NP) or matched placebo. For the purpose of the present project NP treatment assignment will be considered a confounding variable and entered as a baseline control variable in all analyses. Patches were first applied upon waking the morning of TQD and every morning thereafter. EDs prompted participants to apply patches upon waking and asked for confirmation that they had been applied. Thirty percent more participants were randomized to active NP than to placebo NP. Randomization was stratified on the basis of reported smoking rate (split at 20 cigarettes/day) and ED-monitored baseline craving intensity (split at 5.84 on a 10-point scale). Individuals randomized to active NP treatment received 35 mg for 3 weeks, 21 mg for the next 2 weeks, and only placebo NP for 1 final week.

2.5 Cognitive-behavioral treatment

Participants received six sessions of cognitive-behavioral treatment in groups of 8 to 16. Two sessions were held prior to quit day, one on the designated quit day, and three during treatment. Treatment took a behavioral-psychoeducational approach with strong emphasis on providing a supportive group environment (e.g., Brown, 2003). Importantly, the treatment intervention specifically avoided discussion of the AVE.

2.6 Measures

2.6.1 Individual difference measures

Baseline individual difference measures administered during the screening session will be used to assess whether participants differ on traits that could influence the outcome measures of

interest. Specifically, measures related to demographic and smoking-related individual differences were examined. Demographic individual difference measures included data on age, gender, ethnicity, education, and income. To account for smoking patterns and nicotine dependency, daily smoking rateand number of past quit attempts were included, as well as degree of nicotine dependency, assessed with the Fagerstrom Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker & Fagerstrom, 1991). Each set of individual difference measures were entered in the analyses as covariate predictors.

2.6.2 Lapse and relapse outcomes

A lapse was defined as any smoking after initial abstinence (a period of 24 consecutive hours without smoking) has been achieved, ranging from a single puff to multiple cigarettes (Brownell et al., 1986; Shiffman et al., 1986), whereas a temptation was defined as a strong desire to smoke that was successfully resisted. Participants were instructed to report all lapse or temptation episodes on their ED as soon as possible after either occurred. As part of the lapse report protocol, participants reported the number of cigarettes they smoked during the lapse episode. As noted earlier,the ED also asked participants whether they had smoked but had failed to report the lapse episode at the end of each day. These lapses will be included in the tally of lapses for each participant.

Operational definitions of relapse were designed to distinguish between multiple lapses that occur in close proximity and the resumption of regular smoking. For the present project, we examined a relatively conservative definition of relapse, requiring smoking five cigarettes a day for three consecutive days (Shiffman et al., 1996; 2006). Relapse criteria will be determined by ED records of smoking, with the final lapse in each sequence marking the moment that relapse

occurred for each individual. When a relapse event was recorded, the ED was programmed to shift its assessment protocol, such that additional cigarette episodes were not considered "lapses" (as by definition lapses require a state of abstinence), and AVE assessments were no longer administered.

Timing of Lapses and Resisted Temptations. The timing and frequency of lapse and temptation events was expected to vary a great deal both within and between individuals. Participants experienced their initial lapse after different lengths of abstinence, and whereas some quitters experienced rapidly occurring lapses, others experienced isolated lapses spread over extended periods of time. Variations of this sort highlight the dynamic nature of the AVE, and the potential advantages of systematically examining the way AVE responses unfold during lapse-relapse progression. This variation may influence AVE responses, with increases in the rate of lapse and temptation events leading to increasingly severe AVEs. It was therefore be important to account for the timing and frequency of lapse and temptation events when modeling participants' AVE responses.

Quantity Smoked per Lapse. As described earlier, a lapse was defined as any smoking after initial abstinence was achieved, ranging from a single puff to multiple cigarettes. As a consequence, the number of cigarettes smoked during each lapse episode varied within and between participants. We expected that quantity smoked during a given lapse would influence cognitive and emotional reactions to that lapse. One possibility is that smoking a larger number of cigarettes during a lapse increased attributions of blame for the lapse episode, and thereby increased the severity of subsequent AVEs.

2.6.3 Psychological Responses to Lapse

Items administered to assess psychological responses to lapse were designed to capture the colloquial "spirit" of the AVE response, as derived from the literature. Elements of the AVE response included self-efficacy, encouragement, desire to give-up, negative affect, guilt, and self-blame. These items provide a detailed account of the AVE itself, and enabled a thorough examination of AVE theory during smoking cessation.

Self-efficacy (SE).During each lapse assessment, ED administered two items designed to measure aspects of self-efficacy. Participants rated each item on a 0 to 10 scale. Assessments were completed on-screen, one item at a time. First, a single-item measure of abstinence SEwas administered ("Confident in ability to abstain?"). In a previous study, changes in this item prospectively predicted relapse and the item was strongly correlated with a multi-item SE assessment (Shiffman et al., 2000), suggesting its validity as a measure of global momentary SE. To supplement this standard SE item, an item assessing optimism in their ability to successfully maintain abstinence ("Feel Encouraged?") was administered.

Desire to Give-Up.ED also administered an item assessing participants' desire to cease exerting control over smoking entirely ("Feel like giving up?"; 0=NO!!, 10=YES!!). This item was designed to capture the terminal phase of the AVE, which according to the relapse-prevention model is characterized by a perceived loss of control over resumption of regular use.

Affective Valence(AV). General negative affect was derived from an ED item that assessed the degree to which participants experienced negative versus positive affect after each

lapse. This single-item measure asked participants to rate their overall level of post-lapse affect, ranging from entirely negative to entirely positive ("Overall feeling at the end of slip/temptation?"; 0=Negative, 10=Positive). Because we were primarily interested in negative affective responses to lapse, this item was reverse scored prior to analyses, such that higher scores reflected increasingly less positive (for ratings ≤ the mid-point of the scale) or more negative (for ratings ≥ the mid-point of the scale) ratings of affect.

Guilt. Aside from general affective valence, ED assessed the extent to which participants felt guilty following the occurrence of each lapse episode. This single-item measure ("Feel Guilty?") was completed on-screen, with participants rating the item on a 0 to 10 scale that had anchors at the extremes (0=NO!!, 10=YES!!).

Self-Blame. Each assessment also included an item designed to measure the degree to which participants blamed themselves for the lapse episode. This single-item measure of self-blame asked participants to rate the degree to which they were responsible for the lapse on a scale from 0 to 10 ("Was the slip your fault?"). The scale had anchors at the extremes, with 0 associated with "NO!!" and 10 associated with "YES!!".

2.7 Data Analyses

Two separate data analysis strategies were used to address the specific aims of the present research. Because the focus of analysis is on many repeated observations clustered within participants, both analysis approaches incorporated mixed-effects, or the contribution of within-

and between-subject response variation. Mixed-effect models are well suited for the present analyses, as they readily handle variable numbers of within-subject observations (i.e., highly unbalanced data), as well as unequal intervals between assessments.

The first objective was to describe the way participants' AVE responses evolved across successive lapse episodes that occurred during the lapse-relapse process. To accomplish this, mixed-effects growth curve modeling was used to characterize the way participants responded to lapses, and to identify factors that account for this within-subject variation. The second objective of the present research was to investigate the degree to which AVE responses following each lapse influenced progression from one lapse to the next, and ultimately to relapse. To accomplish this,recurrent-events regression (i.e., parametric shared frailty survival analyses) were used to assess the extent to which AVE responses predictedthe rate of subsequent lapse occurrence, as well as the likelihood of relapse.

2.7.1 Mixed-effect growth curve modeling

Abstinence violation effects were expected to progressively intensify, such that each successive lapse episode would undermine SE and encouragement, while exacerbating self-blame, guilt, NA, and desire to give-up. Overall then, AVE responses following a lapse were expected to increase in magnitude as additional episodes occurred. To evaluate these predictions, mixed-effect growth curve modeling was used to characterize participants' AVE responses following each lapse and examine the extent to which responses systematically increased or decreased in severity with each successive episode. Growth curve modelingis an increasingly well-established system for analyzing longitudinal, repeated measures dataof this sort (Cohen et al., 2003). The term growth is used to imply a systematic kind of change.

Although change modeling would not be incorrect, the term change alone could refer to random variation, and is therefore less preferable when responses are expected to either increase or decrease systematically across observations. It is important to note that while responses are usually modeled over a specified unit of time, in the proposed analyses responses were modeled over the course of successive lapses, as these seem more theoretically relevant than the mere passage of time. Specifically, beginning with lapse number 1, the X-axis is composed of the numbered sequence of each additional lapse episode experienced during the study period.

Growth curve models can be estimated using either a mixed-effect, multilevel modeling framework (e.g., Bryk & Raudenbush, 1992), or a structural equation modeling (SEM) framework (e.g., Muthen & Curran, 1997). Mixed-effect modeling (i.e., multilevel modeling, hierarchical linear modeling, or random coefficient regression) is basically a hierarchical form of multiple regression, using maximum likelihood estimation (Littell, Milliken, Stroup, Wolfinger, & Schabenberger, 2006; Singer & Willet, 2003). Mixed-effectgrowth models group together repeated observations at the lower level of analysis (e.g., within-subject responses) within upper level between-subject factors (e.g., individual participants, treatment condition). The degree to which within-subject responses are correlated (i.e., autocorrelated) is specified in each model's covariance structure. Researchers can evaluate alternative covariance structures in order to find the one best suited for their data. Once a suitable covariance structure has been identified, independent between-subject predictor variablesentered at the higher-level of analysis (e.g., treatment condition) are used to account for variations in the value of lower-level regression coefficients and intercepts.

AVE response progression. Cognitive and affective responses assessed following each lapse event were the primary dependent variables. Regression coefficients estimated within each

model characterized within-subject variation in AVE responses across each lapse event recorded during the study period. The shape of these regression curves illustrates the direction and degree that AVE responses intensify with each successive episode. For instance, a linear regression function would indicate that responses increased or decreased at a constant rate following each successive lapse or temptation. Alternatively, a non-linear, quadratic regression function would indicate that the influence of lapse or temptation on AVE responses varied across the lapse process. For example, when examining AVE responses following a lapse, a positive quadratic parameter would indicate that response severity increased exponentially following each successive lapse. This means that instead of increasing at a constant, linear rate, the severity of AVE responses would have actually accelerated with each additional lapse episode.

2.7.2 Recurrent event survival analyses

As AVE responses intensified, the risk of subsequent lapses was expected to accelerate, and the risk of relapse was expected to increase. To evaluate these predictions, survival analyses were used to model the risk of an each successive lapse and relapse events, from the beginning of the study to the initial lapse, from each successive lapse to the next, and ultimately to relapse. Survival analyses assess changes in risk for an event by analyzing the incidence of the event over a specified period of time, referred to as the event's hazard. Recurrent events survival analyses extend this analysis approach to include changes in risk for events that occur multiple times (e.g., lapses).

In any survival analysis, the number of people at risk of experiencing either event during each unit of time is known as the risk set, while the proportion of the risk set who actually experience an event is known as the hazard function.Cases where an event of interest is not

observed before the end of the study were considered censored as of the last day of observation.This included cases where a subject dropped out of the study prematurely. Because it informed our examination of risk-per-unit time, any amount of time that participants were at risk and did not lapse or relapse was included in model estimation. Because continuous-time hazard functions represent the conditional probability of event occurrence per unit of time, they are rates rather than probabilities, and the logarithm of the hazard must be used as the dependent variable (because its values can exceed 1; Singer & Willet, 2003).

AVE responses at each lapse eventwere the primary AVE-related predictor variables. Specifically, AVE responses were incorporated as discrete time-varying covariates that are censored (i.e., change) following each lapse event. This means that AVE responses assessed following each episode were used to predict the likelihood of a subsequent lapse. To account for correlated observations due to repeated measures within subjects (i.e., recurrent lapse events), we used parametric shared-frailty models, which are the survival-data analog to mixed-effects (i.e., multilevel) regression models. Parametric survival models are preferable to semi-parametric models (e.g., Cox proportional hazards model) when specific predictions about time to failure are of interest, and parametric models more readily incorporate frailty (i.e., intraclass correlation) effects. We found a parametric Weibull distribution fit the data best, and compared results obtained by Weibull models with gamma shared frailty effects to Weibull models with frailties that followed an inverse-Gaussian distribution. Results indicated that each frailty distribution produced the same pattern of results. Accordingly, only results for models with gamma-distributed frailty effects are presented herein. HRs, representing increases in risk of lapse given a one-unit increase in a predictor variable, are the outcome statistic of primary interest.

2.7.3 Data Reduction

Two features of the study design and sampling procedure reduced the number of lapse responses included in the final sample. First, the probability that any one lapse episode was assessed decreased as additional episodes were recorded. As previously described, to reduce the burden placed on study participants, not all recorded lapse episodes triggered an AVE assessment. Moreover, the likelihood that an event would trigger an assessment decreased by about 2% with each additional episode. To compensate for the decreasing probability of assessment, we weighted of responses according to the sampling probability associated with their corresponding lapse number (Skinner & Holmes, 2003; Rabe-Hesketh & Skrondal, 2006). As a result, each lapse response had an equal influence on the analyses, regardless of whether it occurred early or later in the lapse-relapse process.

Second, because the number of lapse events recorded by each participant was highly variable, with a few subjects entering as many as n=125 lapses, we limited the scope of our analyses to a maximum of 20 lapses per individual participant. Fewer than 18% of participants recorded more than 20 lapse events, meaning that beyond lapse 20 the data become considerably sparse, and that responses from a relatively small number of participants would have an increasingly disproportionate influence on the analyses (Figure 1).

3.0 Results

3.1 Lapse and Relapse Outcomes

Of the 305 participants, 203 (67%) recorded at least one lapse episode. Within the context of our 20-lapse sample, participants experienced an average of 8.5 (*SD*=5.7) lapse episodes prior to either relapse or censoring. Altogether, participants recorded 1,834 lapse episodes through lapse 20 (Figure 1).Figure 1 presents the sequential distribution of all lapse episodes, illustrating the number of lapses that occurred first in the sequence n=186), second in the sequence (n=155), and so forth.

Only 28 participants (9% of total sample, 14% of lapsers) reached the 3-with-5 relapse criterion during the 7-week observation period (Table 1).The first day of relapsers' 3-day relapse sequence occurred an average of 18.5 (*SD*=9.2) days following an initial lapse.

The 175 (86%) participants who lapsed but did not relapse averaged 7.9 (*SD* = 5.7) lapses episodes each. While they managed to stay below the pre-established relapse threshold, about a third (n=58) of this lapse-no-relapse group smoked on at least 10 different occasions, and about a quarter (n=46) smoked on at least 15 separate occasions.

3.2 Within- versus Between-Person AVE Variation.

We first examined whether there was meaningful AVE variation either between- or within-participants, from one lapse episode to the next. We used an unstructured covariance structure that freely estimates each variance and covariance because it was found to fit the data better than a compound symmetry specification and equally as well as the more complex autoregressive structures. To estimate the relative amount of between- and within-subject AVE

variation, we constructed a set ofunconditional means models that assume AVE responses do not differ from one lapse episode to the next (Table 2). These models are referred to as unconditional means models because they only include an intercept coefficient that estimates mean AVE response severity. The overall fit of these means models, referred to as each model's deviance, is listed for each AVE response type in Table4. These deviance values were later used to evaluate the fit of more complex models, as described in Section 3.4.

To determine whether mean responses to lapse varied either between or within individual participants, we began by evaluating the significance of the intercept and residual variance coefficients in each unconditional means model. The variance of the intercept coefficient in each means model corresponds to the degree that mean AVE severity varied among participants in the study, while the residual variance corresponds to the amount of episode-to-episode variance within-subjects (Table 2). The significance of these coefficients can be evaluated with single parameter standard-score tests (Singer & Willet, 2003). For all AVEs, tests for each variance component revealed a significant level of between-person (zs>7.84, ps<.001) and within-person variation (zs>25.0, ps<.001) among lapse responses.

Next, to estimate the relative proportion of variance accounted for by between-subject differences, we calculated an intraclass correlation coefficient (ICC) for each AVE response. The ICC measures the relative degree that responses recorded by different participants are more discrepant from one another than those recorded by the same participant across occasions. The ICC ranges from 0.0 to 1.0, with scores greater than zero reflecting the degree that responses are increasingly clustered within subjects. Results indicate that between-subjects differences accounted for between 45% and 63% of AVE response variability across lapses (averaging 0.53; Table 2). This means that about 53% of the variance was due to differences among persons, with

the remaining 47% being due to variation among episodes within-persons. Together, these results indicate that the mean severity of AVE responses to lapse varied a great deal from one participant to the next, and within participants, from each lapse episode to the next.

3.3 Covariate Predictors of AVE Variation

We expected that a number of between- and within-subject variables might explain a significant proportion of AVE response variation. These included random assignment to active NRT, the timing of each lapse, the number of resisted temptations preceding each lapse, the amount smoked during each lapse episode, and the degree to which participants attributed the cause of each lapse internally. Univariate results are presented in Table 3 and described in the sections that follow. All covariates are included in the composite AVE models that follow.

3.3.1 Nicotine Replacement Therapy

Although effects of NRT were not a focus of the present project, NRT was included as a covariate in all analyses because assignment to active nicotine patch versus placebo could have influenced participants' lapse responses. NRT was entered as a level-2 predictor so that it might explain between-person variation in AVE responses.As previously reported (Gwaltney et al., 2005), results revealed a significant main effect on participants' self-efficacy ratings, such that assignment to active NRT was associated with increased levels of self-efficacy (β=0.66, p<.05; Table 3). Main effects of NRT assignment across all other AVE responses were non-significant. We also examined whether the effects of NRT have faded over time, as participants who received placebo NRT began to suspect they had not been assigned to active patch. Results

indicate that for all AVE responses NRT assignment did not have a differential effect on participants' early versus later lapse responses.

3.3.2 Relapse Outcomes

Although relapse outcomes are more naturally the focus of the survival analyses that follow, it was important to account for the fact that some participants relapsed while others did not when interpreting the growth curve modeling results. This is because the AVE responses of those who ultimately relapsed may have differed from those who did not along a number of dimensions. AVE responses experienced by relapsers may have been more severe, and/or intensified at a faster rate, than among those who remained abstinent. Results did not bear this out, however, indicating that AVE responses recorded by those who relapsed were not significantly different than those recorded by those who did not reach the 3-with-5 relapse threshold.

3.3.3 Timing of Lapses

Because the present analysis approach models AVE responses over successive lapses instead of over time, it was important to include the amount of time that elapsed prior to each lapse episode as a covariate predictor variable. Specifically, these analyses accounted for the amount of time each participant remained abstinent prior to an initial lapse, and the amount of time that elapsed from each lapse episode to the next.

After successfully achieving 24-hours of abstinence, participants' average time to an initial lapse was 7.64 days (Median=3.08 days; *SD*=9.8). Participants recorded their first lapse

after widely variable periods of abstinence, with initial abstinence periods ranging from 7.92 hours to 39.75 days. To investigate the influence of these individual differences in resistance to an initial lapse, we examined the extent to which maintaining abstinence for longer periods of time altered the severity of the AVE following participants' first lapse. Results indicate that initial abstinence time was unrelated to initial AVE ratings (*ps*>.2).

Following the initial lapse, across all subsequent lapses, the average interval between each lapse and the next was 1.9 days (*SD*=3.4), with a range from less than 1-hour to 37.04 days. Overall, the frequency of lapse events recorded by each participant accelerated over time, such that each additional lapse event occurred 4% faster than its predecessor (HR=1.04, 95% CI: 1.03 - 1.05). Figure 2 illustrates the time interval preceding each of the first 20 lapses. From inspection of Figure 2, it is apparent that the degree to which lapses accelerated was not constant (linear) over the course of the observation period; rather, beyond lapse 10 the average interval from one lapse to the next seems to level out at about 1 day. To evaluate this possibility, we entered a quadratic curvature term to the survival analysis. This revealed significant linear (HR=1.19, 95% CI: 1.14 - 1.23) and quadratic (HR=0.99, 95% CI: 0.99 - 0.99) effects, providing support for the idea that the hazard of each additional lapse increased quickly early in the lapse sequence but then significantly decreased toward the end of the sequence. To further examine the degree to which lapse acceleration varied at different points in the lapse process, we then divided the lapse sequence into segments. Across the first 5 lapses that participants recorded, each lapse occurred 23% faster than its predecessor (HR=1.23, 95% CI: 1.18 1.33), while across lapses 6 through 10 each lapse was 10% faster (HR=1.10, 95% CI: 1.02 - 1.19), and across lapses 11 through 20 lapses did not accelerate to a significant degree (HR=1.02, 95% CI: .98 - 1.05).

To examine the influence of the time interval from each lapse to the next, we estimated the extent to which each successive lapse latency altered the severity and direction of its corresponding AVE (Table 3). Results indicate that longer lapse latencies were associated with greater post-lapse self-efficacy (β=0.04, $p<.001$) and lower ratings of desire to give-up (β= -0.03, $p<.05$), while no other AVEs were affected ($ps>0.20$).

3.3.4 Resisted Temptations.

The average time from initial quitting to an initial temptation to smoke (M=2.1 days, SD=3.7) was not different than the average interval across all subsequent temptations (M=2.1 days, SD=3.8), although the frequency of temptations slightly accelerated over time (Figure 3). Even as the interval between temptations shortened, the temporal sequence of temptations and lapses suggests that participants were better able to resist temptations to smoke early in the lapse-relapse process. While we observed an average of 2.3 (SD=3.7) resisted temptations prior to the first lapse that participants reported, results indicate that the number of temptations proceeding additional lapses quickly decreased thereafter (β=-.11, $p<.001$). The number of resisted temptations preceding each lapse was not associated with any of the AVE response types ($ps>0.20$; Table 3).

3.3.5 Quantity Smoked per Lapse.

Participants smoked an average of 1.2 cigarettes during each lapse episode. Most lapse episodes included no more than a single cigarette (79%), while 12% included 2 cigarettes, and 9% included 3 or more cigarettes. The amount smoked per lapse varied both between ($z>7.9$,

$p<.001$) and within ($z>29.2$, $p<.001$) participants, with between-subject differences accounting for approximately 44% of variation in amount smoked per lapse. Moreover, the amount that participants smoked gradually increased as additional lapses occurred ($\beta=.04$, $p<001$), increasing by an average of about one-half cigarette by the end of the observation period. Results indicate that the degree to which amount smoked increased varied significantly between participants ($z=2.89$, $p<.01$). Figure 4 illustrates these findings, depicting the amount smoked per lapse for both the overall sample ("Fixed: Overall") and for each participant in the trial ("Random: Subjects").

To investigate the degree to which the quantity smoked altered AVE responses, we included the number of cigarettes smoked in each lapse episode as a time-varying covariate (Table 3). The value of this cigarette coefficient reflects the amount of change in each AVE response with each additional cigarette smoked during a lapse episode.Results indicated that as participants smoked more during each lapse episode their ratings of self-efficacy ($\beta= -0.25$, $p<.001$)and encouragement ($\beta= -0.28$, $p<.001$) decreased significantly,while desire to give-up ratings increased ($\beta= 0.24$, $p<.001$). Ratings of negative affect, guilt and self-blame were not associated with the amount that participants smoked during each lapse ($\beta s<0.05$).

3.3.6 Internal Lapse Attributions

As an initial test of the relapse prevention model, we examined the degree to which participants' internal lapse attributions were associated with the severity and direction of their other AVE responses. Results revealed a significant main effect on participants' ratings of guilt, such that higher levels of self-blame were significantly associated with higher ratings of guilt

(β=0.11, p<.001). Main effects of internal attributions across all other AVE responses were non-significant.

3.4 Modeling AVE Variation Across Lapses

To evaluate our hypotheses about AVE progression across successive lapses, we utilized a well-established model comparison technique that makes it possible to test hypotheses about longitudinal response variation (Singer & Willet, 2003). This technique involves comparing the relative fit, or deviance, of models that systematically incorporate regression parameters that describe participants' responses.These deviance-based comparisons offer a standardized method for evaluating goodness of fit, because the difference in deviance between two models (referred to as delta deviance or ΔD) follows achi-squaredistribution with degrees of freedom (*df*) equal to the number of parameters added to each model (Table 4). In other words, improvements in goodness-of-fit can be evaluatedas more parameters are added, and models can be compared.

In an initial step, we constructed a set of hierarchically nested multilevel models that describe the mean severity and variation (if any) of each participant's lapse responses. Regression coefficients estimated within each model characterize between- and within-subject variation in AVE responses across each lapse event experienced during the study period. The shape of these regression curves corresponds to the direction and degree that AVE responses changed following each successive episode.The relative fit of these models was used to select one model for each AVE response variable that provided the best fit to the observed data. These final models revealed whether each AVE systematically changed over the course of the observation period.

3.4.1 Linear Variation

We compared the fit of models that assumed AVE responses do not changeover lapses (i.e., Means Model in Table 4), to models that estimated the extent to which participants' AVE responses intensified or abated following each successive lapse (i.e., Linear: Fixed in Table 4). In addition to the intercept term in the means only models, these "growth" models included a linear slope coefficient. These linear slope terms imply that any observed change is constant across all lapse episodes.We then examined these linear slope coefficients in composite models that included the covariate predictors described in the previous sections. Figure 5 illustrates the direction and severity of participants' average linear AVE response variation across lapses. Together, results indicate that participants' self-efficacy progressively decreased (β= -.11, p<.01; Table 5), and affective valence became more positive (β= .15, p<.001; Table 8), while desire to give-up increased across successive lapse episodes(β=.05, p<.01; Table 6).Results for ratings of encouragement, guilt, and self-blame were not significant (Tables 7, 9, and 10).

To estimate the degree that linear changes in lapse varied from one participant to the next, in the next set of models we allowed the linear slope coefficient to vary freely between participants. In these models the linear slope coefficient was entered as a random, i.e., between-subjects effect. The fit of these random coefficient models was then compared to the fixed growth models above, which assume that AVE response variation is the same for all participants. For all lapse responses, treating the linear slope coefficient as a random effect produced a significant improvement in model fit, indicating that the degree the AVE increased or decreased with each subsequent lapse varied significantly between subjects (Linear: Random in Table 2). Composite results presented in Tables 5 through 10 (i.e., Variance Component "Lapse Sequence:

Linear") demonstrate that this between subject variation remains in models that include all of our covariate predictors.

3.4.2 Non-Linear Variation

In a final step, we evaluated whether AVE responses followed a nonlinear (curviliniear, quadratic) trend, progressively accelerating or decelerating as additional lapses occurred, becoming increasingly more or less severe. First, we examined the contribution of a fixed quadratic curvature coefficient that estimates overall nonlinear acceleration or deceleration (Quadratic: Fixed in Table 4). Averaged across participants, model comparison results (quadratic βs>.004, *p*s<.05) and inspection of participants' response curves (Figure 6) suggest that lapse-related self-efficacy and affective valence changed at a significantly accelerated rate early in the lapse sequence (i.e., < lapse 10) but leveled out thereafter. To confirm this pattern of nonlinear change, we tested whether change in self-efficacy and/or affective valence slowed or ceased beyond lapse 10 by fitting separate linear models spanning lapses 1 to 10 and lapses 11 to 20. Across lapses 1 to 10, results for self-efficacy indicated a significant downward slope (β> -0.10, *p*<.001), while results for affective valenceindicated a significantly positive slope (β> 0.09, *p*<.001). Across lapses 11 to 20, results for both self-efficacy (β<.003, *p*>.9) and affective valence (β<.000, *p*>.9) indicated that participants' responses did not vary.

To determine whether individual within-subject AVE responses followed a nonlinear trajectory, the quadratic coefficient was allowed to vary freely across each individual participant. These models treated both the linear slope coefficient and the quadratic curvature coefficient as random effects. The fit of these random quadratic change models was compared to the fixed quadratic change models (Quadratic: Randomin Table 4).It is apparent that treating the quadratic

coefficient as a random between-subject effect improved model fit for all lapse-related responses but guilt, indicating that the degree to which lapse responses changed at an accelerated or decelerated rate varied significantly across participants (Figure 6). From inspection of participants' individual regression curves, it appears that for all responses most response variation occurred early on in the lapse process, such that greatest jumps in severity occurred over the first 5 to 10 lapses participants recorded.

Taken together, results indicate that lapse responses varied significantly between-participants and across lapses. Averaged over the entire sample, participants reported decreasing levels of self-efficacy, increasingly positive affective valence, and increasing desire to give-up with each additional lapse (Figure 6). However, the average rate that participants' self-efficacy and affective valence changed appears to have slowed beyond lapse 10 (Figure 6). Importantly, we also observed a large amount of between-subject variability in all AVE response trajectories. With the exception of guilt responses, which increased or decreased linearly, ratings of self-efficacy, desire to give-up, encouragement, affective valence, and self-blame exponentially intensified or abated to varying degrees across participants.

3.5 Covariate Predictors of Lapse-Relapse Progression

Prior to evaluating the influence of participants' cognitive and affective responses to lapse on subsequent progression, we investigated a number of covariate predictors. Each was initially examined separately. The relative contribution of all significant predictors was then controlled in subsequent AVE analyses.

3.5.1 Nicotine Replacement Therapy

As reported elsewhere (Shiffman et al., 2006), NRT was found to be a significant predictor of relapse during the observation period, such that random assignment to active high-dose nicotine patch reduced the likelihood of relapse outcomes relative to placebo patch (HR=0.22, CI=0.09-0.53). Complementing this finding, the present results indicate that NRT assignment was also associated with slower lapse progression rates, such that the high-dose nicotine patch group progressing from one lapse episode to another more slowly then the placebo patch group (HR=0.51, CI=0.41-0.62).

3.5.2 Temptations.

In this step we evaluated whether the number of temptation episodes reported prior to each successive lapse was associated with lapse progression. The number of temptations successfully resisted since the previous lapse was associated with significantly slower lapse progression (HR=0.84, CI=0.80-0.88). Moreover, the degree to which resisted temptations protected against lapse magnified as additional lapses occurred (HR=0.98, CI=0.97-0.99).

3.5.3 Amount Smoked per Lapse.

When lapses did occur, the number of cigarettes smoked during each lapse episode was also a significant univariate predictor of progression to a subsequent lapse, such that smoking more cigarettes during a lapse was associated with significantly slower lapse progression (HR=0.93, CI=0.88-0.98).

3.6 Recurrent AVEs and Lapse-Relapse Progression

Recurrent event survival analyses were used to investigate the way AVE-related cognitive and affective lapse responses influenced lapse-relapse progression. Importantly, all AVE-related predictors were prospective, in that each predicted the likelihood of an additional lapse, or relapse. These included responses to the initial lapse that participants experienced, responses to the most recent lapse experienced, the slope of all responses preceding each successive lapse (cumulative slope), and the slope between each lapse response and its immediate predecessor (incremental slope). Results pertaining to each AVE-related predictor are described in the following sections.

3.6.1 Initial lapse responses.

As previously described, responses following an initial lapse from abstinence have traditionally been the focus of AVE research. To investigate the degree to which initial AVE responses were associated with relapse outcomes, we first examined whether participants' responses to the first lapse episode that they experienced predicted time to relapse. These analyses suggested that none of participants' initial AVE-related responses were significant predictors of relapse outcomes.

While they appeared unrelated to relapse outcomes, certain initial lapse responses were found to predict subsequent lapse-to-lapse progression rates. Specifically, participants who reported higher levels of SE following the first lapse experienced a slower rate of progression from each successive lapse to the next (HR=0.93, CI=0.89-0.97; Table 11), while those who reported higher levels of greater desire to give-up following their initial lapse had a faster lapse progression rate (HR=1.06, CI=1.02-1.10; Table 12). Initial ratings of self-blame (HR=1.01,

CI=0.97-1.06; Table 16), guilt (HR=0.99, CI=0.95-1.02; Table 15), AV (HR=0.99, CI=0.95-1.04; Table 14), andencouragement (HR=0.98, CI=0.95-1.01; Table 13) were not significant univariate predictors of subsequent lapse progression.

3.6.2 Recurrent lapse responses.

With responses to their initial lapse entered as covariate predictors, we next investigated the extent to which AVE ratings reported immediately after each successive lapse that participants experienced were associated with progression to a subsequent lapse. These analyses revealed that higher levels of post-lapse SE were associated with slower progression to a subsequent lapse (HR=0.95, CI=0.92-0.99; Table 11), while higher levels of desire to give-up were associated with faster progression to a subsequent lapse (HR=1.04, CI=1.00-1.07; Table 12). An interaction with lapse sequence number also emerged, indicating that the influence of post-lapse self-blame varied across successive lapses. Specifically, as more lapses occurred, higher levels of self-blame (HR=0.99, CI=0.98-0.99; Table 16) were increasingly associated with slower lapse progression. Recurrent ratings of guilt (HR=0.99, CI=0.95-1.02; Table 15), AV (HR=1.01, CI=0.96-1.03; Table 14), and encouragement (HR=0.98, CI=0.95-1.01; Table 13) did not predict subsequent lapse progression.

3.6.3 Lapse response dynamics.

Beyond the absolute level of response severity reported from episode to episode, we investigated whether the degree to which AVE responses increased or decreased in severity over the course of the observation period was associated with corresponding lapse progression rates.

First, we examined the slope of lapse responses preceding each successive lapse. This predictor is essentially a "moving" cumulative slope variable, in that it systematically incorporates each additional response value as participants experience additional lapses. The cumulative slope value at any point in the lapse process is then used to predict the likelihood of a subsequent lapse. Results indicate that the cumulative slope of participants' AVE-related lapse responses was not a significant prospective predictor of subsequent lapse progression (Tables 11 through 16).

Next, to investigate more immediate changes in AVE responses, we examined whether lapse-to-lapse shifts in AVE severity were prospectively associated with progression to an additional lapse. These analyses revealed that lapse-to-lapse increases in guilt (HR=0.96, CI=0.92-0.99; Table 15) and SE (HR=0.95, CI=0.91-0.99; Table 11) were both associated with slower lapse progression, while increasingly negativeaffective valencefrom lapse-to-lapse was associated with faster lapse progression (HR=1.05, CI=1.00-1.09). These results suggest that for responses related to guilt and negative affective valence, the proximal influence of incremental shifts in response severity were the strongest predictors of subsequent lapse progression.

3.6.4 Multivariate Composite Model.

To investigate the joint influence of participants' recurrent cognitive and emotional responses to lapse, we constructed a composite model that included all AVE response modalities across each successive lapse (Table 17). As in the univariate analyses described above, participants' responses to their initial lapse were entered as covariate predictors, along with the significant lapse-related variables identified earlier. Table 17 presents the hazard ratio estimates and significance levels for all predictors entered into this multivariate composite model. Results

confirm the pattern of results suggested by the univariate analyses. It is apparent from the interaction with lapse sequence number that self-blame was increasingly predictive of slower progression as more lapses occurred. Most striking are the results for the lapse-to-lapse slope estimates, indicating that incremental increases in SE and guilt were associated with slower progression, while increasingly negative affective valence from lapse-to-lapse associated with accelerated lapse progression.

4.0 DISCUSSION

Many smoking cessation studies have been conducted with the goal of identifying factors that influence cessation success versus failure. Perhaps the most common feature of these studies has been their reliance on a relapse threshold, operationalized as the point beyond which each participant is categorized as a relapser. The present project aimed to advance our understanding of factors that influence cessation success versus failure with two innovations to the study of the smoking cessation process. First, the project sought to shift focus from binary relapse outcomes to a dynamic process of recurrent lapse episodes recorded over the initial 7weeks of a cessation attempt. Second, the project investigated the role played by psychological responses in the lapse-relapse process, systematically examining the natural history and prospective influence of participants' immediate cognitive and affective lapse responses on subsequent lapse progression.

Hypotheses stem from the relapse prevention model (Marlatt & Gordon, 1985; Witkiewitz & Marlatt, 2004), which predicts that smoking cessation is a process that unfolds over time. Those who fail are expected to descend a "downward spiral" towards relapse, while those who lapse and recover climb their way to abstinence. It was hypothesized that lapses

attributed to internal, dispositional factors would trigger the AVE, a constellation of responses characterized by self-blame, reduced self-efficacy, and negative affect. As a consequence, the likelihood of subsequent lapses was predicted to increase.

Findings revealed a great deal of variability between participants and from lapse-to-lapse in the severity of theAVE response, indicating that participants differed in the extent that their AVE responses intensified versus improved with each successive lapse. In turn, variation in the AVE response was found to explain subsequent lapse progression rates, above and beyond the predictive influence of NRT and other traditional explanatory variables. Results highlight the predictive utility of fine-grained, momentary measures of both psychological and behavioral responses during cessation.

4.1 Relapse versus Recurrent Lapse Outcomes

Descriptive results from the present study document the quantity and frequency of lapse episodes recorded during the initial 7-weeks of a cessation attempt. The proportion of participants who reported a lapse (67%) is similar to those in previous reports (see Hughes, Keely, & Naud, 2004, for review). The proportion of those who lapsed and went on to relapse (14%) is somewhat lower than typically reported (e.g., Jarvis et al., 2003), but this is likely related to the fact that our follow-up for relapse was much shorter than usual, our threshold criterion for relapse (3 days with 5 cigarettes) was more conservative than most (7 days with 1 cigarette), those who never established 24-hr abstinence were removed from the pool of possible relapsers, and that we carefully distinguished between lapse episodes and relapse.

Immediately striking was the relatively large number of lapses recorded by the subset of participants who did not relapse within the observation period according to traditional binary

relapse thresholds. These recurrent lapse data highlight the relatively large amount of smoking behavior that occurs even among those who do not relapse within the constrains of a trial observation period. According to relapse prevention theory, each one of these lapse events represents a high-risk relapse situation after which each lapser will either become increasingly discouraged or remain confident and committed to cessation. The present project investigated whether psychological responses of this sort were associated with subsequent lapse-relapse progression.

4.2 Summary of AVE Modeling Results

4.2.1 Initial versus Recurrent AVE Response Variability

The AVE was originally conceptualized as a trait construct, representing a predisposition to attribute lapsing during self-imposed abstinence to personal weakness, yet the AVE is naturally tied to momentary circumstances (i.e., lapses and their precipitants), and should therefore vary over time. Accordingly, relapse prevention theory (Witkiewitz & Marlatt, 2004) predicts that AVE responses will vary both between individuals and across circumstances, as individuals find themselves in high-risk lapse situations. Previous AVE research has implicitly focused on the former, between-subject question, measuring the AVE after a single lapse and assuming that this initial response reflects each individual's propensity to experience the AVE following a (and indeed, any) lapse. The present study extended this approach to evaluate both individual differences in AVE severity and differences that occur from lapse-to-lapse. This approach provides a more thorough test of relapse prevention theory.

For all aspects of the AVE response, findings revealed significant between-person and within-person mean variation among AVE-like responses to lapses, indicating that the severity of AVE responses to lapse varied considerably from one participant to the next, and within participants, from lapse-to-lapse. As predicted by the relapse prevention model, individual participants did show differential reactivity to the lapses they experienced, while response severity also varied significantly across individual lapses. These data provide empirical support for the notion that the AVE is best conceptualized as a dynamic construct that evolves over the course of the lapse-relapse process, and suggests the importance of studying AVE responses across multiple lapses. The present analysis did not attempt to discern the individual difference factors that lead to variations in AVE responses. Findings suggest that we have much to learn about the complex role of psychological responses to lapses during cessation.

4.2.2 Nicotine Replacement Therapy

We were concerned that assignment to active nicotine patch versus placebo might partially explain variation in participants' lapse responses. Because they were protected against symptoms related to nicotine withdrawal, those on active patch may have been more likely to lapse in the presence of situational factors such as environmental stressors. Conversely, because they were nicotine deprived, participants on placebo patch may have been more likely to lapse in high withdrawal-based craving situations and therefore attribute their lapses to uncontrollable factors associated with their addiction to smoking. Thus, to the extent that active patch protected against internally attributed lapses, it may also have reduced the severity of participants' AVE responses. Results do not support this scenario, however, revealing non-significant main effects for NRT assignment across all AVE responses (Tables 5 through 10).

An alternative possibility was that effects of NRT assignment emerged later in the lapse-relapse process, as participants gradually deduced from their experience with quitting whether they had been placed in the active or placebo patch groups. Specifically, it is likely that a greater proportion of placebo patch participants came to believe that they had not received an active patch, and that they experienced increasingly severe AVE responses as a result. Consistent with this idea, Marlatt contends that when an individual believes the effects of a treatment have worn off or failed, lapses are more likely to be internally attributed and to reduce self-efficacy (Marlatt & Gordon, 1985, p.177). Observed results do not support this scenerio either, indicating that for all AVE responses NRT assignment did not have a differential effect on participants' early versus later lapse responses (Tables 5 through 10). In sum, analyses of NRT effects indicated that it would be appropriate to evaluate AVE responses recorded by participants in both of the randomly assigned NRT treatment groups.

4.2.3 Relapse Outcomes

Although relapse outcomes are of primary interest in the survival analyses that follow, it was important to account for the fact that some participants relapsed while others did not when interpreting the results of our growth curve modeling. This is because the AVE responses of those who ultimately relapsed may have differed from those who did not along a number of dimensions. For instance, AVE responses experienced by relapsers may have been more severe, and/or intensified at a faster rate, than among those who remained abstinent.

4.2.4 Timing of Lapse Episodes

We also sought to examine whether the amount of time each participant remained abstinent prior to an initial lapse, or the amount of time that elapsed from each lapse episode to the next, altered the psychological impact of each lapse. According to relapse prevention theory, AVE responses will intensify to the degree that the quitter is committed and exerts effort to maintain abstinence. It follows then that longer periods of abstinence should have been associated with greater AVEs (Shiffman et al., 1997).This may be because lapses occurring after longer periods of abstinence tend to be interpreted as ruining successful progress accumulated to that point.

Results regarding the time interval preceding each successive lapse did not support our hypotheses, indicating that longer intervals between lapses were associated with greater post-lapse self-efficacy ratings. No other AVE responses were affected, and results regarding the period of abstinence preceding an initial lapse indicated that the amount of time that initial abstinence was maintained did not affect ratings for any of the AVE responses.Thus, although it does not appear that there was a corresponding change in other AVE responses, these data indicate that longer time intervals between lapses actually bolstered self-efficacy, which, according to the relapse prevention model, should have protected participants from additional lapses. These findingsindicate that even after lapsing, participants were able to take stock in the success with abstinence they had achieved prior to the lapse. On the other hand, lapses occurring after shorter amounts of abstinence time elicited more severe AVE responses. These findings support the notion that lapses were not interpreted as isolated events with absolute consequences; rather, participants seem to have considered the amount of time preceding each lapse when interpreting their consequences.

4.2.5 Quantity Smoked per Lapse

Lapse episodes in the present study ranged from a single puff to multiple cigarettes. We hypothesized that smoking more cigarettes during a lapse episode would elicit increasingly severe AVE responses. Results indicate that as hypothesized, participants' ratings of self-efficacy and encouragement decreased significantly, while their ratings of loss-of-control increased to the extent that participants smoked more during each lapse episode. A straight forward explanation for this finding is that as the quantity smoked increased it became progressively more difficult for participants to reasonably deny the implications of the lapse episode moving forward (e.g., Kunda, 1990). For example, a single puff may have been more easily interpreted as a harmless slip than a couple of entire cigarettes, and the latter may have provided undeniable evidence that the lapser's ability to maintain abstinence was insufficient.

Ratings of negative affect, guilt and self-blame were not associated with the amount that participants smoked during each lapse. This pattern of results suggests that the amount smoked during a lapse episode affected participants' cognitive appraisal of each lapse, while affective responses and self-blame were unchanged. This seems to indicate that while the magnitude of a lapse effects confidence and desire to give-up, simply having a lapse at all, regardless of its size, leads to self-blame and guilt. Perhaps this is because the amount smoked during a lapse could be attributed to the lapser's degree of addiction, or to situational demands, both of which might be considered beyond the lapser's control. To the degree that a participant attributed a larger amount of smoking to factors out of his/her control, ratings of self-blame, and consequentially, guilt and negative affect may not have been affected.

4.2.6 Internal Lapse Attributions

According to relapse prevention theory, AVE severity is determined by the degree that a lapse is internally attributed. To assess internal attributions in the present study, participants were asked to rate the degree to which they bore personal responsibility for each lapse ("Was the lapse your fault?"). Results indicate that mean ratings of "fault" were consistently elevated and that there was less variation in ratings of fault than among other lapse responses (M = 8.0, SD = 2.3; Figure 6). Thus it appears that participants were generally inclined to acknowledge that they were responsible and therefore "at fault" for the lapse they just recorded. Perhaps this should not come as a surprise, as it may be too much to expect lapsers to excuse themselves from blame immediately after the occurrence of a lapse (e.g., Kunda, 1990).

In line with our predictions, modeling results revealed a significant association between participants' internal attributions and their ratings of guilt and desire to give-up, indicating that as internal attributions increased so did ratings of guilt, but that participants' desire to "give-up" actually decreased. Interestingly, these findings indicate that as participants took personal responsibility for a lapse and experienced guilt as a result, they simultaneously were less inclined to give up on their cessation attempt. This suggests that increases in personal agency and remorse may have actually served a protective role in the lapse-relapse process, a possibility we investigated further when examining lapse and relapse outcomes.

Aside from ratings of guilt and desire to give up, results of the present study indicated that the severity of participants' self-efficacy, encouragement, and affective valence ratings were not determined by their corresponding ratings of self-blame (Tables 3, 5 and 7). These findings seem to refute one of the tenets of relapse prevention theory, that psychological responses to lapse are secondary to attributions of personal responsibility.

Before drawing firm conclusions about the implications of the present results for relapse prevention theory, it is important to carefully consider how Marlatt and colleagues conceptualized the role of internal attributions in determining the AVE. Beyond acknowledging personal responsibility for a lapse, Marlatt et al. (1985) emphasized that it is important to consider the degree that a lapse is attributed to a stable, nonspecific lack of willpower (relative to their level of addiction; e.g., Marlatt & Gordon, 1985, p.179). This is because it is possible for a lapser to acknowledge a lapse was their "fault" in a particular case, while maintaining that the lapse did not reflect a more general inability to remain abstinent. In this case the lapse would not be expected to reduce self-efficacy nor elicit negative affect, because the lapser believes s/he could cope more effectively in the future. Accordingly, relapse prevention theory predicts that an AVE response is most likely when a lapse is attributed to a stable, nonspecific lack of willpower, whereas the AVE is less likely when the lapse was caused by a specific high-risk situation. This suggests the possibility that our measure of internal attributions may have been insufficiently specific, in that it did not allow participants to specify whether their lapse was due to a lack of willpower, a particularly difficult situation, or just an absentminded slip. In this sense, the present study did not provide a precise test of this element of relapse prevention theory, and results should not be interpreted as definitive evidence that internal lapse attributions do not determine the severity of self-efficacy, encouragement, and affect ratings. Our data suggest it would be useful for future studies to adopt a more multidimensional approach to the measurement of internal lapse attributions, especially when interest centers around the association between internal attributions and perceptions of self-efficacy.

4.2.7 Systematic Response Variation

A major aim of the present project was to investigate the degree to which participants' AVE responses following each lapse systematically intensified or abated as additional lapses occurred. It was hypothesized that AVE responses would intensify with additional lapses, effecting the expected downward spiral. AVE intensification was conceptualized as decreasing levels of self-efficacy coupled with increasing levels of self-blame and increasingly negative affective valence. Results regarding systematic change in AVE responses encompassed two separate levels of analysis: (fixed) systematic change across all participants and (random) systematic change across each participant's lapses responses. At the first, overall level of analysis, we sought to test the hypothesis that overall trends would reflect a general tendency for AVE responses to intensify. At the second level of analysis, between participants, we sought to determine whether individual participants' AVE responses intensified or abated to varying degrees as more lapses occurred. These between-subject analyses allowed us to determine whether there were subgroups of participants who followed different response trajectories, such as those who reported decreasing levels of AVE severity. These between-subject differences in AVE response variation could then be utilized as independent predictors of subsequent lapse progression.

Averaged across all participants, a couple of general trends in participants' lapse responses did emerge. First, as predicted by relapse prevention theory, it appears that participants' ratings of self-efficacy decreased while ratings of desire to give-up increased with each successive lapse that participants experienced. These data demonstrate for the first time that beliefs about ability to quit incrementally decline as lapses accumulate, rather than collapsing all at once. This finding suggests that perceptions of self-efficacy do not drop simply

because a person has a single lapse, but that self-efficacy may reflect a more detailed judgment based on each quitter's evaluation of their success thus far and their ability to continue at different points in the cessation process. This suggests that clinicians should think of the lapse process as an ongoing battle, and that interventions designed to slow quitters' slide down the slippery slope toward relapse may be beneficial.

Contrary to relapse prevention theory, a second overall trend also emerged, indicating that post-lapse ratings of affective valence actually became more positive with each successive lapse. Marlatt's AVE concept holds that lapses elicit increases in negative affect, thereby predisposing the lapser to additional lapses. Yet relapse prevention theory also posits a role for subjective drug effects (Witkiewitz & Marlatt, 2004), and one explanation for the present data is that affective valence became more positive over successive lapses because smoking provided relief. At the same time, it is possible that as participants proceeded farther into their cessation attempt lapses were less likely to occur in "high-risk" situations characterized by high levels of ambivalence and affect. As lapses became less high-risk and more routine it would follow that they would have been associated with lower distress about the lapse. Ultimately, our ability to draw firm conclusions regarding participants' ratings of negative versus positive affect is undermined by the fact that we used a uni-dimensional affective valence measure. This is because a single measure of general affect cannot neatly distinguish an increase in negative affect from a decrease in positive affect; by definition, both are opposite sides of the same coin. Nonetheless, results indicate that responses generally fell on the negative side of the affective continuum (see Mean: Intercept, Table 8), and while affect improved across lapses, mean ratings never crossed the mid-point of the scale (see Figures 5 and 6). Regardless, the present data provide only mixed support for the relapse prevention model: the predictions regarding self-

efficacy were upheld, while predictions regarding affective valence were not supported. Findings suggest that participants experienced mixed feelings during their quit attempt, characterized by both discouragement and relief. This indicates that relapse prevention interventions should sometimes focus on enhancing efficacy and motivation for continued abstinence more than they focus on improving affect.

Relapse prevention theory also predicts that guilt and self-blame would increase and encouragement would decrease over multiple, successive lapses. Averaged over all participants, results indicate that ratings of encouragement, guilt, and self-blame did not change as predicted. However, it does not appear that this was because ratings of encouragement, guilt, and self-blame did not vary at all. Rather, it appears that we did not observe overall effects for these responses because the degree to which they changed across lapses varied significantly from one participant to the next. When this occurs, subsets of subjects can offset one another and produce an overall trend that appears flat (Singer & Willet, 2003). Inspection of participants' individual regression curves bears this out (Figure 4), illustrating that participants' ratings of encouragement, guilt, and self-blame intensified and abated to different degrees, especially early on in the lapse process.

Results regarding systematic AVE response variation provide limited support for the predictions of the relapse prevention model, indicating that for the average participant, recurrent lapse episodes progressively undermined perceptions of self-efficacy, while exacerbating self-blame, guilt, and negative affect. However, this pattern of responses did not hold among all study participants. To the contrary, it appears that many participants reported decreasing levels of guilt, and self-blame, and that many actually reported increasing post-lapse self-efficacy and positive affective valence. According to relapse prevention theory, AVE response differences

should predict subsequent lapse progression. As such, we sought to determine whether relative differences in AVE response severity were prospectively associated with lapse-relapse progression using survival analyses.

4.3 Summary of Survival Analyses

4.3.1 Initial AVEs, Lapse Progression, and Relapse

Responses following an initial lapse from abstinence have traditionally been the focus of AVE research. Results of the present study indicate that participants' initial ratings of self-blame, encouragement, guilt, and affective valence were not significant univariate predictors of subsequent lapse progression or relapse outcomes. Only self-efficacy ratings from the initial lapse (i.e, greater self-efficacy and lower desire to give-up) predicted lapse progression, such that higher ratings of self-efficacy following the initial lapse, and lower desire to give-up, were associated with a slower progression from each lapse to the next, yet these ratings were not associated with relapse.

These findings replicate the results of the only other study that has used EMA to prospectively study lapse responses and cessation outcomes (Shiffman et al., 1996). Shiffman et al. (1996) concluded that initial AVE lapse responses did not prospectively influence the likelihood of relapse. Taken together, findings from both of these prospective studies indicate that initial lapse responses do not predict relapse outcomes, suggesting that their influence may be overshadowed by factors that emerge later in the cessation process. This is consistent with the idea that the AVE is a dynamic construct that evolves across the lapse process, rather than simply an individual difference factor that is established or capable of assessment at the first

lapse. Accordingly, the present study sought to identify and examine other more proximal influences on lapse-relapse progression.

4.3.2 Recurrent AVEs and progression

To learn more about the association between participants' AVE responses and their progression from lapse-to-lapse, a natural next step was to take a closer look at the successive AVE responses participants recorded immediately after each lapse. In this series of analyses, we used each one of these successive AVEs as a predictor of time to the next subsequent lapse. Importantly, all of these analyses controlled for the influence of participants' initial AVE responses, so they assess the extent to which each successive AVE predicts a subsequent lapse above and beyond the value of the initial AVE, and thus also independent of each subject's dispositional propensity to experience the AVE.If the severity of a given participant's AVE responses were determined entirely by his/her attributional style or otherwise preexisting vulnerability to experience the AVE, it follows that the severity of that participant's responses would vary to only a small degree from lapse to lapse, and that including these responses in our analyses would not significantly improve upon the predictive utility of his/her initial AVE response.

Controlling for responses to their initial lapse, results indicate that lower levels of post-lapse self-efficacy and higher levels of desire to give-up were associated with faster progression from each successive lapse to the next. These findings extend results of our previous work (Shiffman et al., 1996; 1997), and are consistent with predictions from relapse prevention theory, which holds that lapsing drive progression by undermining the lapser's confidence in his/her ability to resist smoking in the future. Results suggest that judgments quitters make about their

ability to maintain abstinence and their desire to give up immediately after they lapse bolster their ability to ward off subsequent lapses, perhaps by reducing the perception that another lapse is inevitable, or that coping with future lapses will require an overwhelming amount of effort. However, contrary to theory, none of the other recurrent AVE responses (AV, Guilt, discouragement) were significant univariate predictors of progression from one lapse to the next. The data also indicated that as more lapses occurred, higher levels of fault were increasingly associated with slower lapse progression. This is contrary to AVE theory. One possibility is that higher internal attributions protected against lapse progression for the same reason that we observed them to be associated with higher levels of guilt and lower desire to give-up; that is, it may be that greater internal attributions reflected a realization on the part of participants that they did not do enough to avert their previous lapse and need to exert additional effort. Thus, contrary to relapse prevention theory, blaming oneself for a lapse has a protective function, perhaps because it is motivating.

These results indicate that measuring ratings of self-efficacy and desire to give-up after each lapse improved their predictive utility over simply measuring responses to an initial lapse. In both cases we find that higher self-efficacy and lower desire to give-up were associated with slower lapse progression. Taken together, results support the hypothesis that self-efficacy ratings are inversely associated with lapse progression rates, but they do not support predictions regarding other AVE responses. The observed protective effect of self-blame was particularly unexpected, suggesting that internally attributed lapses that occurred later in the cessation process may have triggered a sort of "wake-up call" to participants that additional effort would be required to maintain abstinence. This protective effect is interesting when considered in light of the earlier finding linking self-blame to greater guilt and lower desire to give-up, although

ratings of guilt were not observed to have a protective effect in this series of analyses. Regardless, reduced desire to give-up was associated with slower lapse progression, and when considered along side results for internal attributions, these findings support the notion that increased self-blame may have protected against lapse progression by increasing motivation to quit successfully.

4.3.3 AVE response slopes and progression

Analyses examining the influence of each recurrent lapse response on progression to a subsequent lapse essentially treated each lapse as an independent, cross-sectional event. As such, this approach did not account for whether participants' responses had increased or decreased relative to their previous responses. As described earlier, examination of systematic response variation in the present study demonstrated that participants varied in the extent to which their lapses responses intensified or abated. We therefore sought to test the hypothesis that participants with increasingly severe AVE responses progressed more quickly to each subsequent lapse than those whose responses were stable or becoming less severe.

To investigate whether lapse progression rates were related to the degree that participants' lapse responses increased or decreased in severity over the course of the observation period, we used the slope of each participant's lapse responses to that point to predict the likelihood of a subsequent lapse. Results indicated that the cumulative slope of participants' AVE-related lapse responses was not a significant prospective predictor of a subsequent lapse. Post-hoc inspection of participants' responses suggested that this was because the cumulative slopes smoothed over lapse-to-lapse response fluctuations. As a result, the

direction of incremental shifts in AVE severity from lapse-to-lapse often did not correspond to the direction of participants' overall response trend (Figure 7).

To investigate more immediate changes in participants' responses from lapse-to-lapse, a second set of analyses tested whether shifts in AVE severity relative to the immediately-preceding lapse were prospectively associated with progression to an additional lapse ("Drops" and "Jumps" in Figure 7). These analyses revealed that increases in self-efficacy relative to the previous lapse was associated with slower lapse progression, while increasingly negative affective valence was associated with accelerated lapse progression. Consistent with our hypotheses, these findings support the notion that lower levels of self-efficacy and higher levels of negative affect predispose quitters to additional lapses on alapse-by-lapse basis. However, contrary to predictions, increases in guilt were actually found to protect against subsequent lapse progression. Especially because guilt is typically considered a form of negative affect, it was somewhat surprising to see increases in guilt having a preventive, rather than promoting, effect on lapse progression, while increases in more general negative affect increased the likelihood of lapse. However, considered along side the findings for self-blame described in the previous section, the present finding for guilt supports the idea that increasing levels of self-blame, and guilt may have reflected a kind of "wake-up call" phenomenon, whereby participants redoubled their cessation effort/resolve following lapses they regretted. Alternatively, these negative psychological responses could have served as "punishment" that deterred repetition of the lapse behavior.

Overall, results indicate that participants' responses to any single lapse were not as predictive as the relative degree that their responses changed from lapse-to-lapse. Moreover, the data demonstrate that participants' overall response trend was not nearly as predictive as the

incremental changes that occurred from one lapse to the next. In fact, the prospective effects of responses associated with increasing levels of negative affect were completely hidden until incremental lapse-to-lapse response variation was examined. It appears that for some AVE responses, and particularly those related to negative affective valence, the proximal influence of incremental "jumps" and drop-offs in response severity were stronger predictors of lapse progression than the cumulative slope of participants' lapse response history (Figure 5). This suggests that participants were especially sensitive to lapses that represented an immediate shift for better or for worse, regardless of whether the most recent shift was at odds with their overall response trajectory. These findings have potentially important implications for relapse prevention theory and practice, suggesting that interventions may have their most powerful effects when they target recurrent lapse responses at the time they occur, flexibly accounting for quitters' momentary responses to small failures and redemptions over the course of the cessation process.

4.4 Implications for Relapse Prevention Theory

The innovative methodology utilized by the present study provides both a rigorous test and a new perspective on relapse prevention theory. Results provide support for the notion that lapse-related self-blame, self-efficacy, and negative affect play a central role in lapse-relapse progression, yet the nature of the role played by these core AVE components did not always adhere to AVE theory.

A core assumption made by relapse prevention theory is that maladaptive abstinence violation effects occur when a lapser attributes the cause of the lapse internally. Results of the present study provide strong support for the notion that lapses during smoking cessation elicit internal attributions and self-blame. Moreover, we found that higher levels of self-blame

following a lapse are significantly associated with higher ratings of lapse-related guilt and decreased ratings of desire to give-up, supporting predictions of the relapse prevention model. Yet the present data do not indicate that self-blame ratings were associated with any of the other AVE components, including perceptions ofself-efficacy and affective valence. Particularly unexpected was that elevations in self-blame and guilt appear to have served a protective function, perhaps moving participants to re-double their efforts to avoid further lapses. Thus while relapse prevention theory traditionally assumes that lapse-related self-blame and guilt promote additional lapsing, the present data show that these negative responses actually serve to maintain abstinence.

Results regarding negative affective valence also provide mixed support for the tenets of the relapse prevention model. Contrary to model predictions, which hold that lapses elicit increases in negative affect, initial modeling results identified an overall trend indicating that ratings of affective valencebecame more positivewith each successive lapse episode. Interestingly, however, examination of incremental shifts in affective valence from lapse-to-lapse revealed that immediate jumps toward the negative end of the scale were significantly associated with acceleration to a subsequent lapse. Thus, it seems that immediate increases in negative affect precipitated lapse progression, as predicted by the relapse prevention model, even as participants followed an overall trend characterized by increasingly positive affect. This suggests the presence of two types of lapse events: those that became increasingly routine, producing a declining affective response, and those that represented a turn for the worse, producing an influx of negative affect and predisposing the lapser to accelerated progression.

Other core components of the AVE concept were found to behave as predicted by relapse prevention theory. Ratings of self-efficacy, for instance, were found to progressively decrease

with each successive lapse participants experienced, while desire to give-up progressively increased. In turn, decreases in self-efficacy ratings were prospectively predictive of accelerated lapse progression, while increased desire to give-up was predictive of accelerated progression. In their original account, Marlatt et al. (Marlatt & Gordon, 1985, p.181) explain that quitters' perceptions of their ability to maintain control over their abstinence is the central factor in the AVE concept. In this way, relapse prevention theory expanded upon Bandura's earlier conceptualization of self-efficacy (e.g., 1977). The present data provide empirical support for the central role of self-efficacy in the lapse-relapse process, demonstrating that self-efficacy ratings were sensitive to both between- and within-subject lapse-related variables, and that self-efficacy was a primary determinant of subsequent smoking behavior.

4.5 Limitations

4.5.1 Sample

Generalizability of the results of the present study may be limited by the fact that that participants were more motivated to quit than is typical in the general population. It is possible that less motivated and less confident quitters would have responded to lapses differently, and that the processes driving lapse progression would have been affected by different factors. Random assignment to active versus placebo nicotine patch may also limit the generalizability of results. It is unclear from the present study whether or not smokers who did not utilize nicotine replacement therapy during their quit attempt would have responded differently to lapses.Future research should investigate this possibility.

4.5.2 Length of Observation Period and Protocol Noncompliance

Relative to other smoking cessation trials, the 7-week observation period in the present study is short (e.g., 12 months follow-up; SRNT working group). The primary reason that the follow-up period was limited in this study was to reduce the burden imposed on participants. Nevertheless, clinical data suggest that a majority of lapses and relapses occur within the first month of an attempt. An extended observation period would have been beneficial for this study so that we could observe more relapse events, and draw firmer conclusions about relapse processes later in the cessation process.

Due to the burden of continuous ED monitoring, participants may also have been noncompliant and failed to record all smoking behavior. Although biochemical verification could not guarantee accurate reporting of individual cigarettes, it was helpful in verifying whether participants were being honest about their reported smoking status. Because data are time-stamped, noncompliance would have affected when lapse episodes were recorded. Failure to record smoking episodes at all could obscure the natural history of lapses.

4.5.3 Lapse and Relapse Definitions

A lapse was defined as any smoking after initial abstinence (a period of 24 consecutive hours without smoking) has been achieved, ranging from a single puff to multiple cigarettes. Participants were instructed to report all lapse episodes on their ED as soon as possible after either occurred. Allowing lapse episodes to encompass multiple cigarettes had the advantage of reducing participant burden, but a potential limitation of this approach was that the timing of lapse episodes may have varied between participants and/or over time.

Another definitional limitation is that our operational definition of relapse was necessarily arbitrary (Miller, 1996), conceptualized as the end-point of any 3-day period with at least 5-cigarettes smoked per day. This 3 with 5 relapse threshold has been observed to be more conservative than the 7-days with any smoking criterion used by most studies in the literature, in that the 3 with 5 criterion is reached by a smaller proportion of quitters than the 7-day criterion (Shiffman, *). Regardless, both of these relapse thresholds fall well short of resumption of participants' pre-quit, "normal" smoking rates, which have been shown to take months and maybe years to reestablish (e.g., Conklin et al., 2005).

4.6 Future Directions

Results of the present study suggest a number of directions for future work. First of all, it would be useful to carefully examine multivariate associations between each of the AVE response types examined in the present study. It may be that it would be informative to conceptualize the AVE as a single, latent construct, or some combination of latent factors that encompass the lower-level items analyzed herein. Exploratory factor analysis methods could be used to study multivariate response variation and identify latent AVE factors. These latent AVE factors could then be incorporated into a structural equation modeling framework and modeled over successive lapse episodes. Because it would restrict focus to a single dependent construct, this approach may actually simplify and thereby elucidate our understanding of AVE response variation.

Results of this study also highlight the fact that psychological responses to lapse are highly variable both between individual participants and across momentary circumstances. This suggests that it would be useful to formally identify subgroups of subjects who followed similar AVE response trajectories. Therefore, a potentially fruitful future direction for research would

be to utilize the increasingly well-established exploratory analysis procedure known as mixture-modeling, which makes it possible to identify clusters of subjects following similar growth trajectories in the context of a multilevel mixed-effect study design.

Regardless of whether participants are categorized as abstinent or relapsed, variation in the quantity and frequency of all post-cessation smoking behavior contains a wealth of information that can be related to explanatory variables of interest. For instance, studies examining daily smoking patterns have demonstrated that smoking cessation can be characterized as a dynamic series of shifts between periods of lapsing and periods of abstinence, and that variables influencing the incidence of lapses may not be the same as those influencing a return to abstinence (Conklin et al., 2005; Hoeppner et al., 2008; Swan & Denk, 1987; Wileyto, Patterson, Niaura, et al., 2005). These innovative approaches to the study of cessation outcomes embrace the complexity of the cessation process, rather than restricting focus to binary abstinence measures. The present project sought to extend this work with an even more fine-grained approach to the study of the cessation process, moving beyond daily summaries of smoking behavior to study the association between psychological responses and lapse behavior as each successive lapse was recorded in real time. Future work of this sort should investigate predictors of lapse-to-lapse response variation, including individual difference variables and the longitudinal effects of both pharmacological and behavioral treatment interventions.

Future research should also move toward reconciling seemingly incompatible theoretical accounts of lapse-relapse progression. A good example is the contrast between relapse prevention theory, incentive (Stewart & Wise, 1992), and incentive-sensitization (Robinson & Berridge, 1993)models. Whereas relapse prevention theory emphasizes subjective responses to lapse, both incentive models assume that the pharmacological effects of lapsing directly promote

relapse. Interestingly, however, empirical evidence increasingly shows that these alternative theories may not be as incompatible as they initially appear. Stimuli associated with drug-use have been shown to reinstate drug-seeking behavior in animals in the absence of the drug from which they are deprived (see Shaham et al., 2003, for review). A similar phenomenon has also been recently observed in humans, such that programmed lapses more than doubled the probability of subsequent smoking, regardless of whether the lapse involved nicotine-containing or denicotinized cigarettes (Juliano, Donny, Housmuller, & Stitzer, 2006). These findings indicate that stimulus factors and expectancies may account for "priming" effects better than the pharmacological effects of drug ingestion. On the other hand, Marlatt's recent reformulation of the relapse prevention model (Witkiewitz & Marlatt, 2004) acknowledges a greater role for pharmacologic factors such as nicotine withdrawal and the potentially reinforcing effects of lapsing. Taken together, developments in both relapse prevention and incentive-based models indicate that it would be useful to develop a more transdiciplinary framework for continued study of the lapse-relapse process. Doing so would allow us to contrast theoretical models that traditionally contradict one another, and may thereby improve our understanding of both treatment effects and the natural history of lapse-relapse progression during smoking cessation.

4.7 Conclusions

It is important to advance our understanding of the smoking cessation process, so that we might improve our ability to affect clinical outcomes. The present project investigated the role played by psychological responses in the lapse-relapse process, systematically examining the natural history and prospective influence of participants' immediate cognitive and affective lapse responses on subsequent lapse progression.

Methodologically, results of the present research highlight the advantages of embracing the complexity inherent to any self-imposed process of behavior change. Future studies that incorporate fine-grained, ecologically valid measures with dynamic longitudinal analysis techniques promise to reveal much more about the subtle processes that drive the link between lapse and relapse outcomes.

Findings provide considerable support for the tenets of relapse prevention theory, indicating that when abstinent smokers lapse during a cessation attempt they tend to blame themselves, their self-efficacy is undermined, and they experience heightened negative affect and guilt. Also consistent with relapse prevention theory, decreases in self-efficacy and increasingly negative affective valence were found to predispose quitters to additional lapses. Importantly, because we modeled these effects over recurrent lapse episodes, the present study was the first to empirically capture the "downward spiral" concept we have used to characterize the cessation process.

Of course, we also observed many participants climbing toward recovery on an every widening "upward spiral". Findings revealed a great deal of AVE response variability between participants and from lapse-to-lapse, indicating that participants differed in the extent that their AVE responses intensified versus improved with each successive lapse. In turn, AVE response variation was found to explain subsequent lapse progression rates, above and beyond the predictive influence of other traditional explanatory variables. Prospectively linking AVE responses to lapse progression in this way revealed some surprising results. In particular, elevated ratings of self-blame and guilt were actually found to protect against lapse progression, a finding that directly contradicts traditional assumptions of relapse prevention theory. We have suggested thatthe negative experience associated with heightened blame and guiltmay have

deterred repetition of lapse behavior, or that these responses may have produced a "wake-up call" phenomenon, whereby participants redoubled their resolve following lapses they regretted. Either way, these findings demonstrate that negative AVE responses may not always be a bad thing. To the contrary, negative AVE responses may be strongest among quitters who have maintained greater motivation to quit and consequentially experience higher levels of regret and remorse when lapses do occur. For this reason, it would be interesting for future studies to evaluate the effect of lapses on shifts in motivation to abstain (cf., Gibbons, Eggleston, & Benthin, 1997), as well as on attempted coping at the next high-risk challenge to maintain abstinence.

According to relapse prevention theory, each lapse in the lapse-relapse process represents a potential target for interventions designed to bolster coping resources and renew commitment to change. However, it is clear that the period following a lapse presents a challenging opportunity for intervention to prevent relapse, as indicated by the failure of initial attempts to apply relapse prevention theory clinically (Curry et al, 1989; Cooney & Kopel, 1980). Somewhat ironically, results of the present study suggest that the reason relapse prevention interventions have failed is that they are typically designed to alleviate AVEs. This is because reassuring those who lapse may inadvertently decrease their motivation to maintain abstinence. From a clinical perspective, this suggests that it may be misguided for treatment practitioners to focus exclusively on reassuring those who slip, always striving to reduce blame and guilt following a lapse. Rather, quitters may benefit from non-judgmental discussion about the intrinsic and extrinsic factors that precipitated their slip, with the goal of increasing intrinsic motivation for change. Such an approach would be consistent with the growing body of evidence supporting the efficacy of motivational enhancement interventions (e.g., Hettema, Steele, & Miller, 2005), the aim of

which is to promote behavior change by fostering ambivalence with the status quo. Findings from the present study also reveal that components of the AVE associated with progression to relapse may differ from those associated with recovery. This suggests that when we engage quitters with interventions during cessation, it will be important to carefully assess their current status and trajectory so that we can accurately target interventions designed to promote recovery versus prevent relapse.

Table 1. Participant Characteristics: Mean (SD) and N (%)

Variable	Abstinent (n=305)	Lapsed (n=203)	Relapsed (n=28)
Age	39.34 (9.19)	38.94 (8.89)	37.21 (8.35)
Cigarettes per Day	24.29 (8.89)	24.91 (8.94)	24.96 (8.07)
Years Smoking	21.95 (9.42)	21.82 (9.04)	20.79 (9.09)
Number of Previous Quits	3.20 (3.85)	2.83 (3.31)	1.68 (1.39)
FTND (0-10)	5.95 (1.94)	6.17 (1.90)	6.27 (1.64)
Gender: Female	170 (52.50)	117 (55.50)	21 (75.00)
Ethnicity: Caucasian	277 (85.50)	178 (84.40)	27 (96.43)
Education: Post HS	213 (65.70)	136 (64.50)	18 (64.29)
Married	165 (50.90)	102 (48.30)	10 (35.71)
Income: > 39,999	176 (54.30)	109 (51.70)	13 (46.43)
Treatment: Active Patch	188 (58.00)	119 (56.40)	6 (21.43)

Table 2. Unconditional Means Model for Each AVE (0 – 10 Scale)

Fixed Effects	Self-Blame	Self-Efficacy	Give-Up	Encouraged	Affective Valence	Guilt
Intercept (Mean)	**8.00	**6.73	**2.23	**4.80	**4.00	**7.18
SE	0.12	0.16	0.15	0.15	0.13	0.18
Random Effects						
Intercept Variance	**0.44	**0.75	**0.68	**0.68	**0.48	**0.86
SE	0.06	0.06	0.06	0.06	0.06	0.06
Residual Variance	**0.54	**0.45	**0.61	**0.66	**0.50	**0.63
SE	0.02	0.02	0.02	0.02	0.02	0.02
ICC	0.45	0.63	0.53	0.51	0.49	0.58

Note. Intercept Variance = Between-Subjects Variation. Residual Variance = Within-Subject Variation.ICC = Intraclass Correlation Coefficient.
** $p < .001$

Table 3. Covariate Predictors of AVE Variation(0 – 10 Scale)

Variable	Self-Blame	Self-Efficacy	Give-Up	Encouraged	Affective Valence	Guilt
NRT and Relapse Outcomes						
NRT: Active Patch	-0.08	0.84	-0.32	0.07	0.57	0.41
SE	0.25	0.32	0.31	0.31	0.25	0.36
p-value	0.75	*0.01	0.30	0.82	*0.02	0.25
Outcome: Relapsed	0.23	-0.99	0.56	-0.31	-0.61	0.05
SE	0.34	0.44	0.42	0.42	0.34	0.50
p-value	0.49	*0.02	0.18	0.47	0.08	0.92
Timing of Lapse Episodes (Days)						
Initial Abstinence	0.02	0.01	-0.01	-0.02	0.01	0.00
SE	0.01	0.02	0.02	0.02	0.02	0.02
p-value	0.09	0.51	0.66	0.38	0.73	0.87
Lapse-to-Lapse Intervals	-0.01	0.03	-0.02	0.00	0.01	-0.01
SE	0.01	0.01	0.01	0.01	0.01	0.01
p-value	0.61	***0.00	*0.04	0.77	0.38	0.59
Resisted Temptations, Amount Smoked, and Internal Attributions						
Pre-Lapse Temptations	-0.05	0.04	-0.06	0.04	0.02	-0.06
SE	0.03	0.03	0.03	0.03	0.03	0.03
p-value	~0.06	0.11	~0.06	0.19	0.49	0.08
Amount Smoked	0.04	-0.25	0.24	-0.28	0.00	-0.04
SE	0.07	0.05	0.07	0.06	0.05	0.07
p-value	0.54	***0.00	***0.00	***0.00	0.94	0.58
Internal Attribution	-	0.02	-0.04	-0.01	0.02	0.11
SE	-	0.02	0.03	0.03	0.02	0.03
p-value	-	0.33	0.13	0.80	0.38	***0.00

Table 4. Relative Fit of Models Estimating AVE Variation over Successive Lapses

Model Form (# Parameters)	Self-Blame	Self-Efficacy	Give-Up	Encouraged	Affective Valence	Guilt
Means Model: Deviance (3)	6393.81	6243.48	6775.36	6290.22	6808.61	6658.33
	Deviance Δ Values: Successive Improvements in Model Fit					
Linear Slopes: Fixed (4) [1]	-1.99	**-51.17	**-26.67	-0.07	**-13.56	*-7.81
Linear Slopes: Random (6) [2]	**-30.36	**-108.03	**-54.40	**-63.13	**-36.22	**-85.85
Quadratic: Fixed (7) [1]	-0.32	*-9.06	-3.41	-1.25	**-10.98	0.00
Quadratic: Random (10) [3]	**54.23	**-28.48	**-24.54	**-30.59	**44.45	-8.12

Note. Deviance Δ values follow a $\chi 2$ distribution with degrees of freedom (df) equal to the number of parameters added to each model. Significant $\chi 2$ values indicate that the addition of new parameters significantly improves the model's fit, which in turn indicates that the added parameters are meaningful. Deviance Δ values for fixed models are based on maximum likelihood estimation (ML). Deviance Δ values for random models are based on restricted maximum likelihood estimation (REML). ICC = Intraclass Correlation Coefficient.
[1]Deviance Δ $\chi 2$ df = 1; [2] Deviance Δ $\chi 2$ df = 2; [3] Deviance Δ $\chi 2$ df = 3
* $p < .01$; ** $p < .001$

Table 5. Composite Mixed-Effects Model: Self-Efficacy (0 – 10 Scale)

Fixed Effects	β	SE	*z*
Mean: Intercept	6.90	0.35	***19.89
NRT and Relapse Outcomes			
NRT: Active Patch	0.43	0.32	1.34
Outcome: Relapsed	-0.33	0.44	-0.74
Timing of Lapse Episodes (Days)			
Initial Abstinence Period	0.00	0.02	-0.22
Lapse-to-Lapse Intervals	0.03	0.01	**2.89
Resisted Temptations, Amount Smoked, and Internal Attributions			
Pre-Lapse Temptations	-0.03	0.03	-1.02
Amount Smoked per Lapse	-0.16	0.04	***-3.66
Internal Lapse Attribution	0.02	0.02	0.95
Growth over Lapse Sequence			
Lapse Sequence: Linear	-0.11	0.04	**-2.69
Lapse Sequence: Quadratic	0.00	0.00	1.66

Variance Components	Variance	SE	*z*
Mean: Intercept	5.053	0.711	***7.10
Lapse Sequence: Linear	0.117	0.031	***3.83
Lapse Sequence: Quadratic	0.000	0.000	**2.79
Within-Subjects Residual	1.881	0.079	***23.82

Note. Estimates the amount of change in satisfaction for a 1-point change in the predictor. When the predictor is categorical (e.g., NRT), it estimates the difference between groups. Covariance estimates not shown.
* p < .05; ** p < .01; *** p < .001

Table 6. Composite Mixed-Effects Model: Desire to Give-Up (0 – 10 Scale)

Fixed Effects	β	SE	*z*
Mean: Intercept	2.31	0.35	***6.64
NRT and Relapse Outcomes			
NRT: Active Patch	-0.07	0.32	-0.23
Outcome: Relapsed	0.24	0.44	0.55
Timing of Lapse Episodes (Days)			
Initial Abstinence Period	0.00	0.02	0.19
Lapse-to-Lapse Intervals	-0.02	0.01	-1.29
Resisted Temptations, Amount Smoked, and Internal Attributions			
Pre-Lapse Temptations	0.00	0.03	-0.11
Amount Smoked per Lapse	0.14	0.05	**2.87
Internal Lapse Attribution	-0.06	0.03	*-2.29
Growth over Lapse Sequence			
Lapse Sequence: Linear	0.05	0.01	**3.13

Variance Components	Variance	SE	*z*
Mean: Intercept	4.01	0.70	***5.70
Lapse Sequence: Linear	0.14	0.04	***3.42
Lapse Sequence: Quadratic	0.00	0.00	**2.94
Within-Subject Residual	2.81	0.12	***23.79

Note. Estimates the amount of change in satisfaction for a 1-point change in the predictor. When the predictor is categorical (e.g., NRT), it estimates the difference between groups. Covariance estimates not shown.
* p < .05; ** p < .01; *** p < .001

Table 7. Composite Mixed-Effects Model: Encouragement (0 – 10 Scale)

Fixed Effects	β	SE	*z*
Mean: Intercept	5.17	0.37	***13.95
<u>NRT and Relapse Outcomes</u>			
NRT: Active Patch	0.02	0.33	0.05
Outcome: Relapsed	-0.30	0.43	-0.69
<u>Timing of Lapse Episodes (Days)</u>			
Initial Abstinence Period	-0.02	0.02	-1.06
Lapse-to-Lapse Intervals	-0.01	0.01	-0.51
<u>Resisted Temptations, Amount Smoked, and Internal Attributions</u>			
Pre-Lapse Temptations	0.06	0.04	1.58
Amount Smoked per Lapse	-0.23	0.05	***-4.35
Internal Lapse Attribution	0.00	0.03	0.11
<u>Growth over Lapse Sequence</u>			
Lapse Sequence: Linear	0.00	0.02	0.23

Variance Components	Variance	SE	*z*
Mean: Intercept	5.96	0.93	***6.38
Lapse Sequence: Linear	0.16	0.05	***3.56
Lapse Sequence: Quadratic	0.00	0.00	**2.52
Within-Subject Residual	3.11	0.13	***23.79

Note. Estimates the amount of change in satisfaction for a 1-point change in the predictor. When the predictor is categorical (e.g., NRT), it estimates the difference between groups. Covariance estimates not shown.
* $p < .05$; ** $p < .01$; *** $p < .001$

Table 8. Composite Mixed-Effects Model: Affective Valence (0 – 10 Scale)

Fixed Effects	β	SE	*z*
Mean: Intercept	3.75	0.33	***11.35
NRT and Relapse Outcomes			
NRT: Active Patch	-0.43	0.27	-1.60
Outcome: Relapsed	0.36	0.36	1.02
Timing of Lapse Episodes (Days)			
Initial Abstinence Period	0.01	0.02	0.39
Lapse-to-Lapse Intervals	0.00	0.01	0.11
Resisted Temptations, Amount Smoked, and Internal Attributions			
Pre-Lapse Temptations	0.01	0.03	0.45
Amount Smoked per Lapse	-0.07	0.04	-1.60
Internal Lapse Attribution	-0.01	0.02	-0.28
Growth over Lapse Sequence			
Lapse Sequence: Linear	0.15	0.04	***3.33
Lapse Sequence: Quadratic	-0.01	0.00	**-2.99

Variance Components	Variance	SE	*z*
Mean: Intercept	4.45	0.69	***6.44
Lapse Sequence: Linear	0.14	0.03	***3.93
Lapse Sequence: Quadratic	0.00	0.00	***3.20
Within-Subject Residual	2.28	0.09	***24.49

Note. Estimates the amount of change in satisfaction for a 1-point change in the predictor. When the predictor is categorical (e.g., NRT), it estimates the difference between groups. Covariance estimates not shown.
* p < .05; ** p < .01; *** p < .001

Table 9. Composite Mixed-Effects Model: Guilt (0 – 10 Scale)

Fixed Effects	β	SE	*z*
Mean: Intercept	6.30	0.40	***15.94
NRT and Relapse Outcomes			
NRT: Active Patch	0.46	0.38	1.20
Outcome: Relapsed	0.25	0.52	0.48
Timing of Lapse Episodes (Days)			
Initial Abstinence Period	-0.01	0.02	-0.30
Lapse-to-Lapse Intervals	0.00	0.01	-0.15
Resisted Temptations, Amount Smoked, and Internal Attributions			
Pre-Lapse Temptations	-0.07	0.04	-1.95
Amount Smoked per Lapse	0.00	0.05	-0.05
Internal Lapse Attribution	0.11	0.03	***3.99
Growth over Lapse Sequence			
Lapse Sequence: Linear	-0.03	0.01	***-3.18

Variance Components	Variance	SE	*z*
Mean: Intercept	6.28	0.79	***7.91
Lapse Sequence: Linear	0.02	0.01	***4.23
Within-Subject Residual	3.00	0.12	***24.76

Note. Estimates the amount of change in satisfaction for a 1-point change in the predictor. When the predictor is categorical (e.g., NRT), it estimates the difference between groups. Covariance estimates not shown.
* p < .05; ** p < .01; *** p < .001

Table 10. Composite Mixed-Effects Model: Self-Blame (0 – 10 Scale)

Fixed Effects	β	SE	*z*
Mean: Intercept	7.84	0.23	***33.59
NRT and Relapse Outcomes			
NRT: Active Patch	-0.14	0.26	-0.51
Outcome: Relapsed	0.20	0.35	0.57
Timing of Lapse Episodes (Days)			
Initial Abstinence Period	0.02	0.02	1.59
Lapse-to-Lapse Intervals	0.01	0.01	0.64
Resisted Temptations, Amount Smoked, and Internal Attributions			
Pre-Lapse Temptations	-0.04	0.03	-1.37
Amount Smoked per Lapse	-0.03	0.05	-0.62
Internal Lapse Attribution	-	-	-
Growth over Lapse Sequence			
Lapse Sequence: Linear	0.01	0.01	1.03

Variance Components	Variance	SE	*z*
Mean: Intercept	4.47	0.57	***7.82
Lapse Sequence: Linear	0.18	0.04	***4.94
Lapse Sequence: Quadratic	0.00	0.00	***3.77
Within-Subject Residual	2.49	0.10	***23.97

Note. Estimates the amount of change in satisfaction for a 1-point change in the predictor. When the predictor is categorical (e.g., NRT), it estimates the difference between groups. Covariance estimates not shown.
* p < .05; ** p < .01; *** p < .001

Table 11. Recurrent event regression: Prospective Effects of Self-Efficacy on Lapse Progression

	Initial and Recurrent AVE	Lapse Number Interaction	Cumulative Slopes	Incremental Slopes
Predictor	HR	HR	HR	HR
Patch: Active	0.53***	0.54***	0.53***	0.57***
Temptations	0.69***	0.69***	0.63***	0.68***
Amount Smoked	0.88***	0.88***	0.88**	0.86***
Lapse Number	1.02**	1.020	1.030	1.03
Self-Efficacy				
Initial Lapse	0.93*	0.94*	0.940	0.93*
Successive Lapses	0.95**	0.94*	0.920	0.97
Lapse Number Interaction	-	1.000	1.000	1.00
Cumulative Slopes	-	-	0.990	-
Incremental Slopes	-	-	-	0.95*

Note. * p < .05; ** p < .01; *** p < .001

Table 12. Recurrent event regression: Prospective Effects of Desire to Give-Up on Lapse Progression

	Initial and Recurrent AVEs	Lapse Number Interaction	Cumulative Slopes	Incremental Slopes
Predictor	HR	HR	HR	HR
Patch: Active	0.50***	0.50***	0.46***	0.56***
Temptations	0.69***	0.69***	0.63***	0.68***
Amount Smoked	0.89***	0.89***	0.88**	0.87***
Lapse Number	1.02**	1.03**	1.05**	1.03**
Desire to Give-Up				
Initial Lapse	1.07**	1.07*	1.08*	1.07*
Successive Lapses	1.05**	1.05*	1.05	1.06
Lapse Number Interaction	-	1	1	1
Cumulative Slopes	-	-	0.94	-
Incremental Slopes	-	-	-	1.01

*Note.** p < .05; ** p < .01; *** p < .001

Table 13. Recurrent event regression: Prospective Effects of Encouragement on Lapse Progression

	Initial and Recurrent AVEs	Lapse Number Interaction	Cumulative Slopes	Incremental Slopes
Predictor	HR	HR	HR	HR
Patch: Active	0.52***	0.52***	0.50***	0.57***
Temptations	0.69***	0.69***	0.63***	0.68***
Amount Smoked	0.89***	0.89***	0.88**	0.87***
Lapse Number	1.02***	1.02	1.03	1.02
Encouragement				
Initial Lapse	0.96	0.96	0.97	0.98
Successive Lapses	0.97	0.96	0.93	0.95
Lapse Number Interaction	-	1	1	1
Cumulative Slopes	-	-	1.02	-
Incremental Slopes	-	-	-	1.02

Note. * p < .05; ** p < .01; *** p < .001

Table 14. Recurrent event regression: Prospective Effects of Affective Valence on Lapse Progression

	Initial and Recurrent AVEs	Lapse Number Interaction	Cumulative Slopes	Incremental Slopes
Predictor	HR	HR	HR	HR
Patch: Active	0.52***	0.52***	0.50***	0.58***
Temptations	0.68***	0.68***	0.63***	0.67***
Amount Smoked	0.89***	0.89***	0.88**	0.87***
Lapse Number	1.02**	1.04**	1.04*	1.04*
Negative Affect				
Initial Lapse	0.99	0.99	0.97	0.97
Successive Lapses	1.01	1.04	0.93	0.93
Lapse Number Interaction	-	1.00	1.00	1.00
Cumulative Slopes	-	-	0.96	-
Incremental Slopes	-	-	-	0.95*

Note. * p < .05; ** p < .01; *** p < .001

Table 15. Recurrent event regression: Prospective Effects of Guilt on Lapse Progression

	Initial and Recurrent AVEs	Lapse Number Interaction	Cumulative Slopes	Incremental Slopes
Predictor	HR	HR	HR	HR
Patch: Active	0.52***	0.52***	0.50***	0.58***
Temptations	0.68***	0.68***	0.62***	0.67***
Amount Smoked	0.89***	0.89***	0.88**	0.88***
Lapse Number	1.02**	1	1.05	1.03
Guilt				
Initial Lapse	1.02	1.02	1.04	1.01
Successive Lapses	0.99	0.97	1	1.03
Lapse Number Interaction	-	1	1	1
Cumulative Slopes	-	-	0.97	-
Incremental Slopes	-	-	-	0.96*

Note. * p < .05; ** p < .01; *** p < .001

Table 16. Recurrent event regression: Prospective Effects of Self-Blame on Lapse Progression

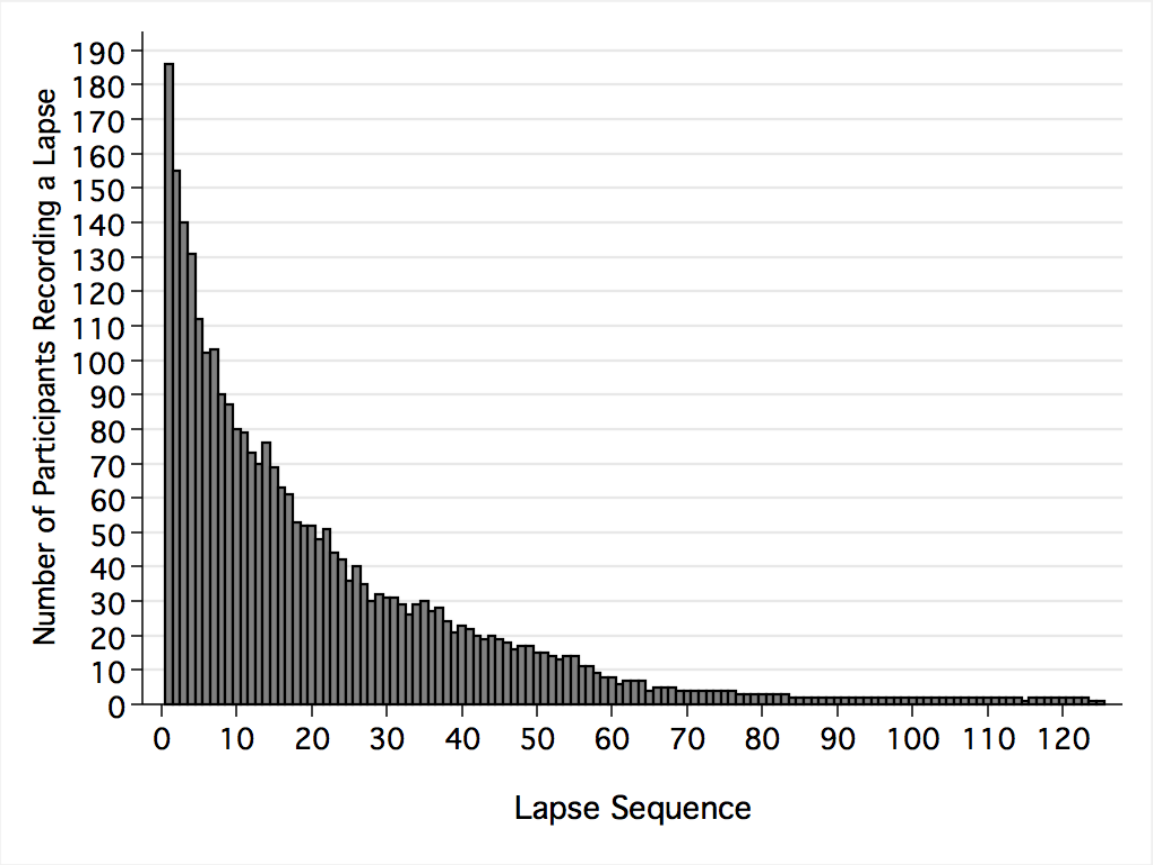
	Initial and Recurrent AVEs	Lapse Number Interaction	Cumulative Slopes	Incremental Slopes
Predictor	HR	HR	HR	HR
Patch: Active	0.53***	0.53***	0.51***	0.58***
Temptations	0.68***	0.68***	0.62***	0.66***
Amount Smoked	0.89***	0.89***	0.88**	0.87***
Lapse Number	1.02**	1.09**	1.13***	1.10**
Self-Blame				
Initial Lapse	1.02	1.02	1.06	1.03
Successive Lapses	1.01	1.06*	1.09	1.11*
Lapse Number Interaction	-	0.99*	0.99*	0.99*
Cumulative Slopes	-	-	1.02	-
Incremental Slopes	-	-	-	0.97

Note. * p < .05; ** p < .01; *** p < .001

Table 17. Recurrent event regression: Composite Model of Prospective Effects on Lapse Progression

Predictor	Hazard ratio	95% Confidence Interval	*p* value
Patch: Active	0.563	(0.435 -0.729)	0.000
Temptations	0.664	(0.590 -0.748)	0.000
Amount Smoked	0.864	(0.808 -0.924)	0.000
Initial Responses			
Fault	1.029	(0.978 -1.083)	0.269
Self-Efficacy	0.934	(0.866 -1.008)	0.079
Desire to Give-Up	1.053	(0.996 -1.113)	0.071
Encouragement	1.010	(0.960 -1.062)	0.696
Affective Valence	0.966	(0.913 -1.023)	0.231
Guilt	0.990	(0.938 -1.044)	0.711
Recurrent Responses			
Fault	1.067	(0.993 -1.146)	0.077
Self-Efficacy	0.988	(0.928 -1.053)	0.718
Desire to Give-Up	1.016	(0.975 -1.058)	0.451
Encouragement	0.983	(0.945 -1.022)	0.393
Affective Valence	1.084	(1.025 -1.146)	0.006
Guilt	1.058	(1.005 -1.115)	0.032
Lapse Number	1.095	(1.026 -1.169)	0.006
X Fault	0.992	(0.984 -1.000)	0.038
Incremental Slopes			
SE Increment	0.945	(0.903 -0.989)	0.015
AV Increment	0.938	(0.899 -0.979)	0.002
Guilt Increment	0.929	(0.891 -0.969)	0.001

Figure 1. Sequential Distribution of Lapse Episodes



Note. Lapses 1 through 20 are the focus of the present analyses.

Figure 2. Time interval (days) proceeding each lapse episode.

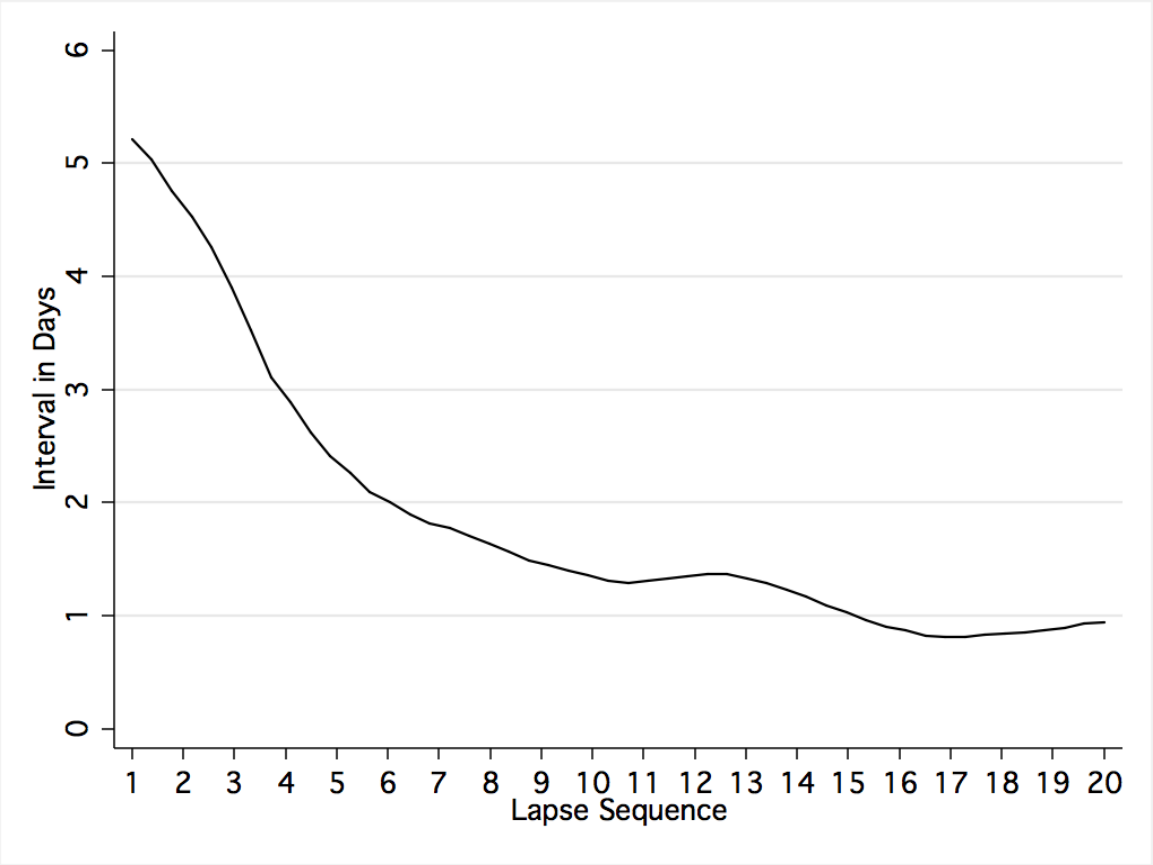


Figure 3. Resisted temptations preceding each lapse episode.

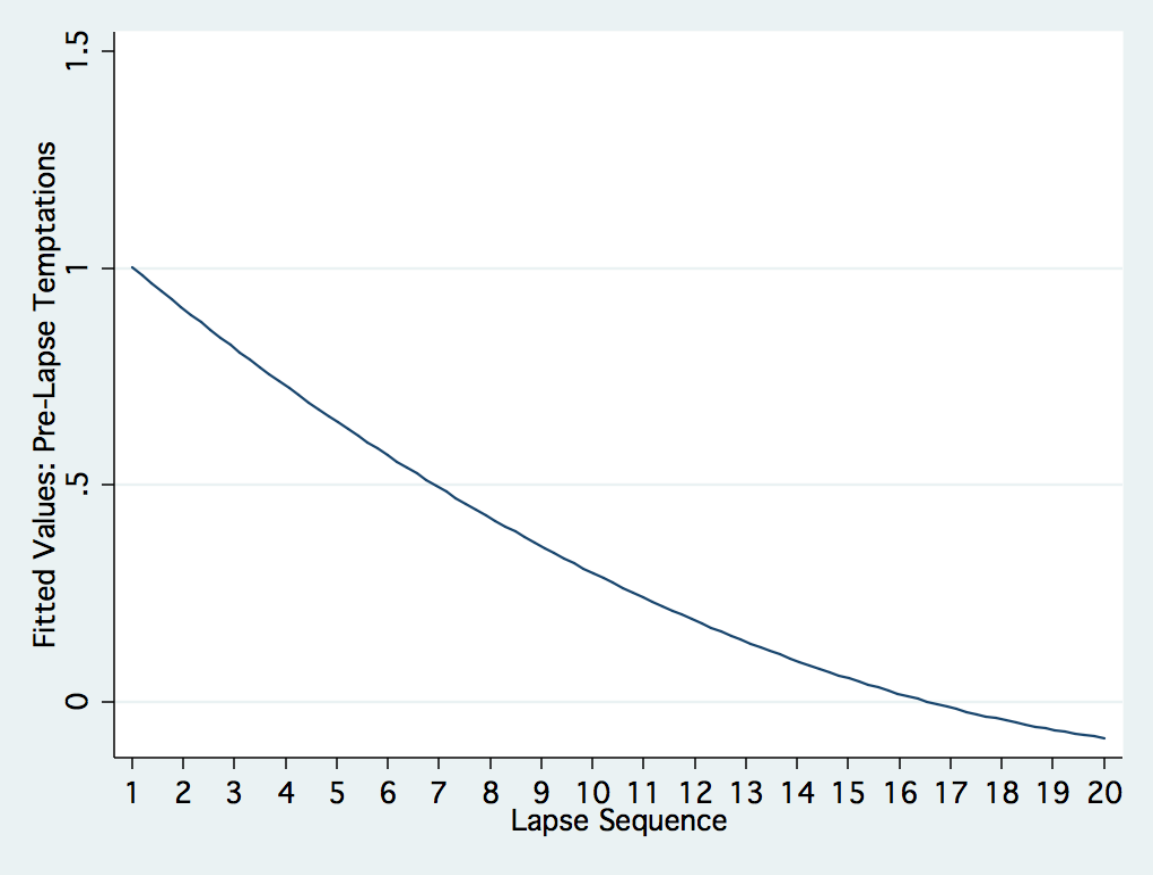


Figure 4. Number of Cigarettes Smoked per Lapse.

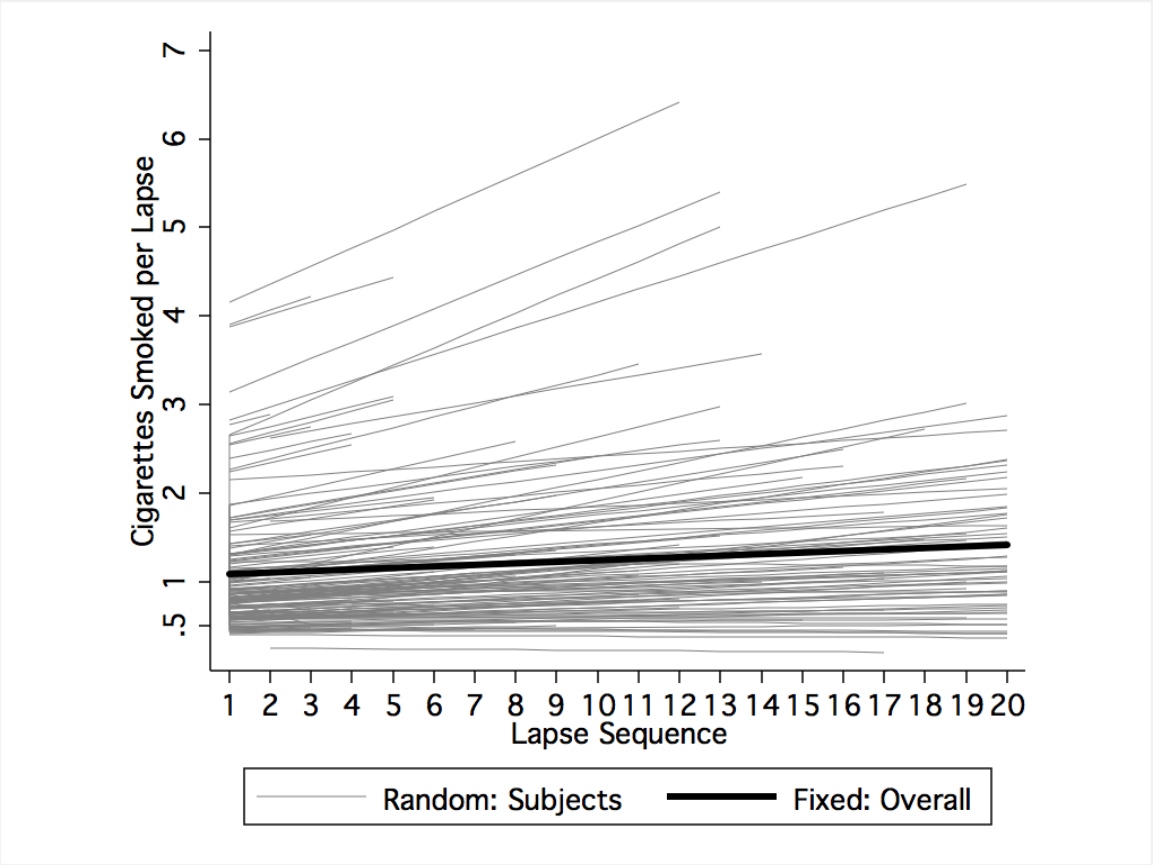


Figure 5. Overall, Fixed Trajectories for each AVE Response

Figure 6. Fixed and Random Fitted Values for each AVE response.

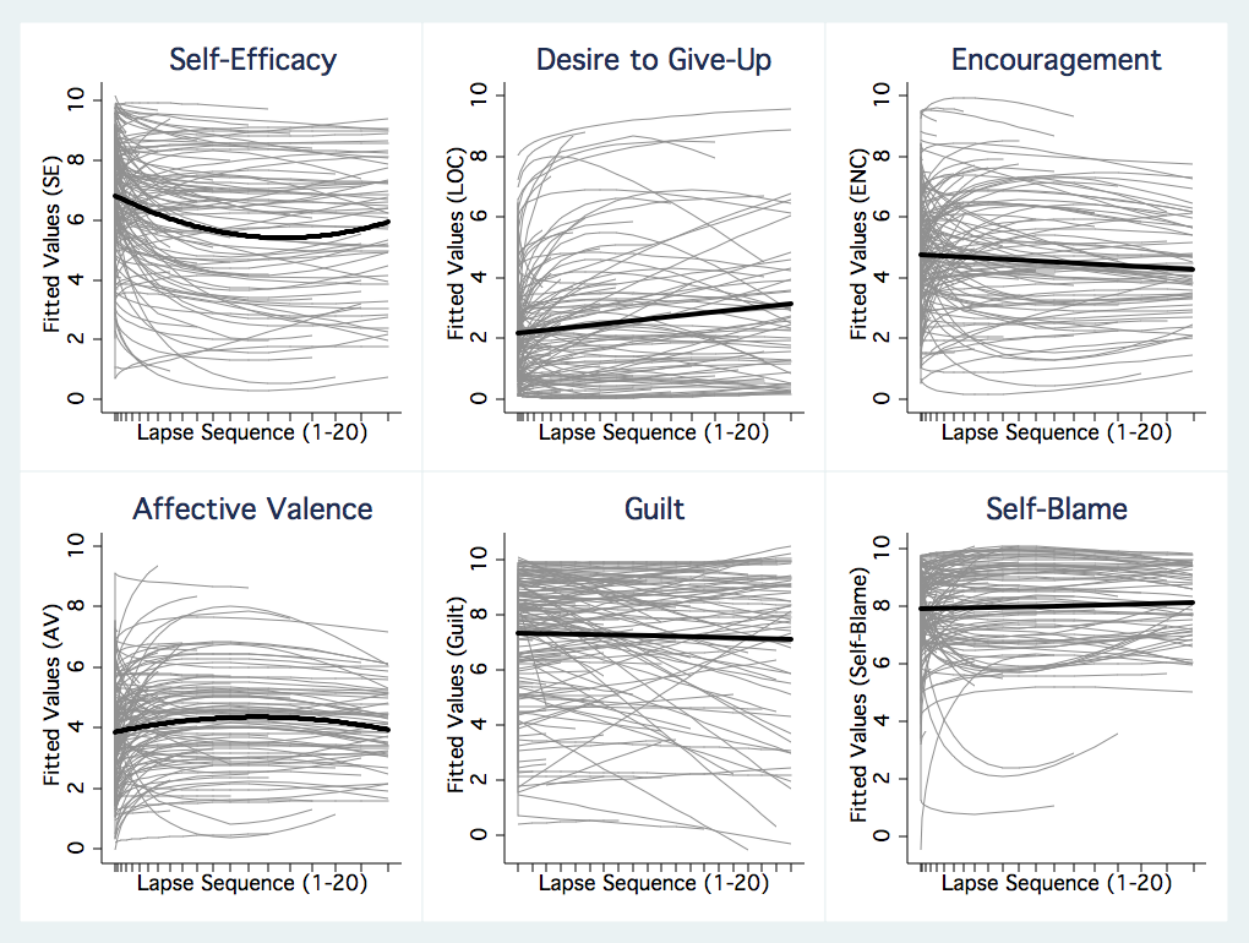
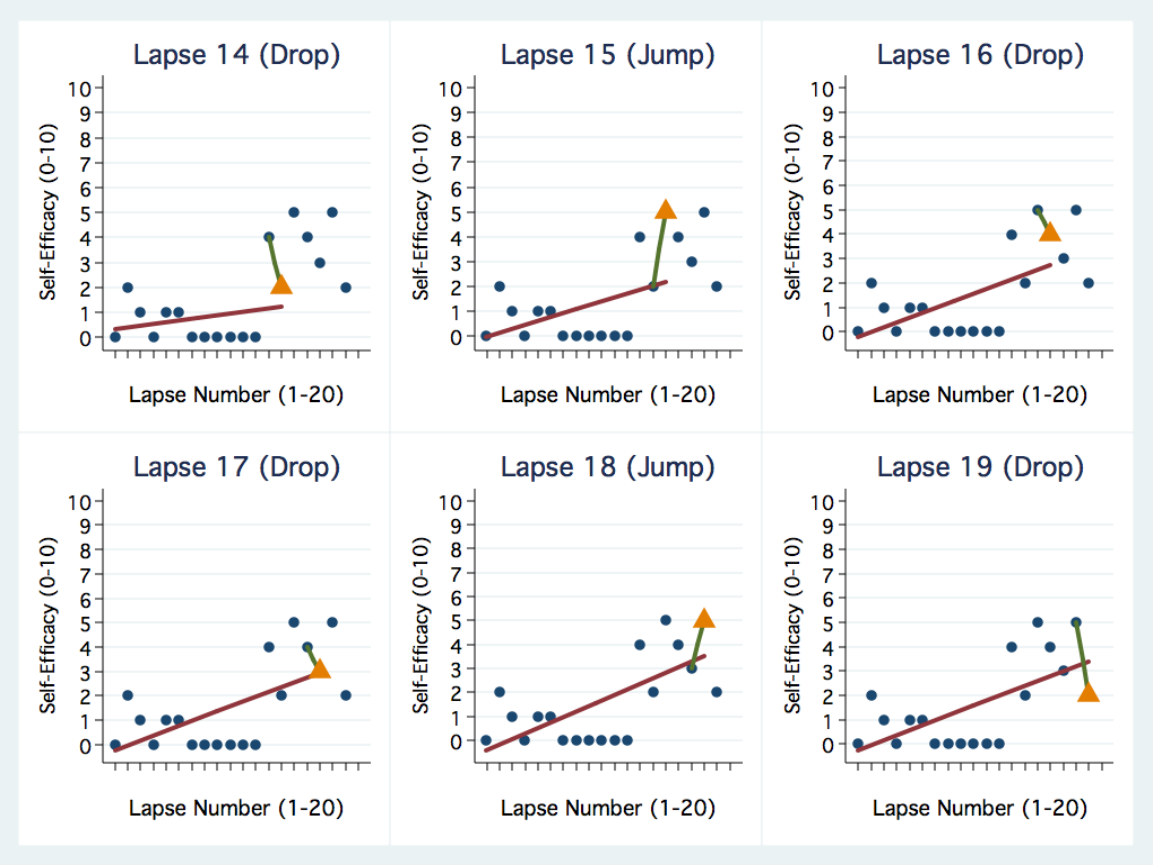


Figure 7. Prototypical Cumulative and Incremental Self-Efficacy Slopes.



Note. Panels indicate whether each incremental slope represented a "Drop" or a "Jump" relative to the constantly increasing cumulative slope. Cumulative and incremental slope values were used to prospectively predict time to the next successive lapse (i.e., lapses 15 through 20).

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