INDEPENDENT AND INTERACTIVE EFFECTS OF REAL-TIME RISK FACTORS ON LATER TEMPTATIONS AND LAPSES AMONG SMOKERS TRYING TO QUIT

By

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ABSTRACT OF THE DISSERTATION

Independent and interactive effects of real-time risk factors on later temptations and lapses among smokers trying to quit by KRYSTEN WILLIAMS BOLD

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Research on the proximal influences on smoking relapse has focused primarily on the independent effects of risk factors, yet relapse may also be governed by complex, interactive processes. The current study sought to expand our understanding of relapse mechanisms by identifying the independent and interactive effects of real-time risk factors on temptations and the ability to resist temptations in smokers during a quit attempt. This study was a secondary analysis of ecological momentary assessment data collected from 109 treatment-seeking smokers 4 times a day for 21 days following a quit attempt. All smokers received nicotine replacement therapy and smoking cessation counseling. Multinomial hierarchical linear models were used to evaluate ways momentary impulsiveness, affect, urge, cigarette exposure, alcohol use and their interactions predicted temptations and smoking up to 8 hours later. Level-one data comprised report-level predictors and outcomes nested within individuals at level-two. Results suggested temptations were predicted by higher momentary agitation, distress, and urge; and lower positive affect. The inability to resist temptations was predicted by prior smoking, higher distress, and recent alcohol use. There were significant interactions

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between level-one predictors that influenced the risk of temptations (positive affect x impulsiveness, urge x agitation, agitation x cigarette exposure, urge x cigarette exposure) and the odds of resisting a temptation (alcohol x impulsiveness). These results suggest studies of complex relationships between proximal risk factors may provide new information about relapse processes and inform smoking cessation interventions.

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Introduction

Tobacco use remains the leading cause of preventable death in the United States and poses a large public health burden (CDC, 2008; U.S. Department of Health and Human Services, 2004; Warner, Hodgson, & Caroll, 1999). More than half of current U.S. smokers attempt to quit each year (CDC, 2011), but relapse remains a central problem in the treatment of tobacco dependence. Despite smoking cessation aids currently available, roughly 95% of smokers who are able to achieve 24 hours of abstinence return to smoking within 3 months (CDC, 2011; Fiore et al., 2008). Understanding relapse processes is critical to identifying intervention targets and improving smoking cessation rates.

In particular, more research on proximal, phasic influences on lapse and relapse is needed. Such research may identify what happens in the moments before a person returns to smoking and what differentiates occasions in which smokers attempting to quit succeed in abstaining versus slip back into smoking. The determinants of temptations to smoke, or motivational lapses, may differ from those of behavioral lapses, or a return to smoking after quitting. This distinction may be clinically important for timing treatment and delivering effective interventions. Identifying factors that differentiate occasions in which smokers resist the urge to smoke and those in which they yield to temptation and smoke may help identify early warning signs of smoking.

Several studies have attempted to do this using ecological momentary assessment (EMA; Stone & Shiffman, 1994) data collected several times daily from smokers attempting to quit. Researchers have used EMA to identify proximal risk factors related to a first lapse (i.e., first instance of smoking following abstinence), and to contrast lapses

with temptations after quitting (i.e., situations in which a smoker was tempted but able to refrain from smoking) to isolate factors that relate specifically to the inability to resist the urge to smoke (Shiffman, 2009, Shiffman et al., 2007; Shiffman et al., 1996). This work suggests negative affect and urge differentiate these outcomes, however the extant studies compared only one lapse, temptation, and abstinent (control) event for each subject. Examining multiple smoking and temptation episodes post-quit may address this important question in a different way, especially if smoking cessation is a cyclical process where smokers alternate between smoking and abstinence until achieving stable abstinence (Baker et al., 2011; Prochaska & DiClemente, 1983). Each smoking opportunity after quitting is a critical choice point to smoke or abstain, and smokers who remain engaged in the process of quitting may have multiple periods of abstinence and smoking after a first lapse. Studying the antecedents to multiple temptation and smoking events after quitting, while controlling for smoking status, may enhance our understanding of the factors that influence smoking behavior more generally and promote improved intervention efforts.

Additionally, much of the research to date has focused on single relapse risk factors in isolation, but these cognitive and affective vulnerabilities may interact to influence smoking risk in the moment. A recent study by Lam and colleagues (2014) was among the first to investigate ways momentary relapse risk factors interact. Their results suggested negative affect, being around smokers, and consuming alcohol have additive effects on urge and lapse risk in a sample of female smokers. Additionally, urge significantly moderated negative affect and being around smokers such that these risk factors were more predictive of lapse in times of low compared to high urge. All risk factors and outcomes were examined concurrently in Lam et al. (2014); however, so the direction of the effect is unclear (i.e., low urge may be due to a recent lapse instead of the cause of smoking). Studying ways risk factors combine to influence smoking risk *before* smoking occurs may improve our understanding of relapse mechanisms and suggest opportunities to deliver just-in-time interventions.

The current project will fill existing gaps in the literature using time-lagged hierarchical linear modeling to examine ways momentary smoking risk factors (impulsiveness, affect, urge, cigarette exposure, and alcohol use) independently and interactively relate to later temptations and smoking within smokers during a quit attempt.

Impulsiveness

Impulsiveness may be an important factor related to the ability to refrain from smoking in the face of a temptation during a quit attempt. Research indicates that disinhibition (the ability to inhibit a response) relates to smoking status and heaviness (McClernon et al., 2008). Additionally, smokers who are more impulsive experience greater craving when nicotine-deprived (VanderVeen, Cohen, Cukrowicz, & Trotter, 2008), and relapse more quickly than do less impulsive smokers (Bickel, Odum, & Madden, 1999; Dallery & Raiff, 2007). Impulsiveness has often been conceptualized as a stable individual difference; although recent research indicates certain facets of behavioral impulsiveness are dynamic (Weafer, Baggott, de Wit, 2013); behavioral impulsiveness appears to depend on mood state (Weafer et al., 2013), nicotine deprivation status (Field, Santarcangelo, Sumnall, Goudie, & Cole, 2006; Mitchell, 2004), and stress exposure (Schepis, McFetridge, Chaplin, Sinha, & Krishnan-Sarin, 2011). Together, these results suggest that disinhibition may be a dynamic construct that is influenced by other risk factors and is meaningfully related to a smoker's ability to resist a temptation after quitting. Investigating momentary impulsiveness and its interaction with other risk factors (i.e., affect, craving, context) as an antecedent for temptations and smoking after quitting will provide new information about the processes driving smoking behavior.

Affect

The role of negative affect in smoking behavior has been widely investigated. The reformulated negative reinforcement model of drug motivation suggests escape from negative affect (such as by smoking to alleviate withdrawal) plays a central role in maintaining smoking behavior (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004). Over time, smoking may become a way to regulate general negative affect and stress, and smokers may be especially unable to resist a temptation when experiencing withdrawal, stress, or negative affect (Cooney, Litt, Cooney, Pilkey, Steinberg, & Oncken, 2007; Kassel, Stroud, & Paronis, 2003; Marlatt & Gordon, 1980; Shiffman, 1982). Indeed, research suggests that momentary negative affect differentiates the occurrence of temptations and lapses assessed with EMA reports (e.g., Shiffman et al., 1996). At present, it is unknown how positive affect relates to the occurrence of temptations and success resisting them. Recent evidence suggests that positive affect is also related to smoking motivation and behavior change. Lower levels of positive affect and greater decreases in positive affect prior to quitting predict smoking lapse independent of negative affect (Strong et al., 2009). There is also evidence that reductions in positive affect relate to urge intensity (Doran, Cook, McChargue, Myers, & Spring, 2008), which

may predict smoking. Furthermore, mediation analyses suggest certain smoking cessation treatments promote abstinence by enhancing positive affect or reducing withdrawal-related declines in positive affect (McCarthy et al., 2008; Piper et al., 2008). Accordingly, positive affect may have a protective role against smoking after quitting and may increase the odds of resisting a temptation without smoking. More research is needed to understand the role of momentary affect in smoking risk and ways these affective states interact with other factors to put a smoker at risk for imminent temptations or smoking. *Urge*

Craving or urge to smoke has been posited as a central motivating process of smoking behavior and relapse (Baker, Morse, & Sherman, 1987; Robinson & Berridge, 1993; Tiffany, 1990). The dynamic nature of this variable has been well-established, and evidence suggests that craving ratings differentially predict temptations and lapses after quitting (Shiffman et al., 1996). This suggests the intensity of momentary craving may affect a smoker's ability to resist a temptation to smoke. Even with targeted cessation aids designed to reduce urge intensity, roughly 65% of smokers relapse (Fiore et al., 2008). Thus, the relation between urge and smoking may be influenced by other momentary factors, such as affect, impulsiveness, or context. Studying how urge combines with other risk factors to influence a smoker's ability to resist later smoking temptations will provide new information about the phasic influences on smoking behavior.

Environment

The environmental context may also be an important factor that influences the occurrence of temptations and ability to resist temptations to smoke. Based on findings

from previous research (Shiffman et al., 1996), the current project will focus on the influence of cigarette exposure (i.e., being around smokers or having an opportunity to smoke) and alcohol consumption as predictors and moderators of the occurrence of temptations and smoking after quitting. Exposure to cigarettes or alcohol may synergistically combine with other momentary smoking risk factors (i.e., impulsiveness, affect, and urge) to increase the likelihood of experiencing temptations or smoking after quitting. Understanding how context interacts with these cognitive and affective risk factors may suggest intervention strategies to mitigate risk for temptations and lapses.

The current project will examine the independent and interactive influences of momentary impulsiveness, affect, craving, recent cigarette exposure, and alcohol consumption on the occurrence of later temptations and smoking. We will contrast times of strong temptation to times of abstinence without temptation to identify factors that put smokers at risk for temptations after quitting (motivational lapses). We will also examine factors that relate specifically to the inability to resist smoking (behavioral lapses) by contrasting times of smoking to strong temptations without smoking. We expect momentary impulsiveness, negative affect, urge, cigarette availability, and alcohol consumption to lead to motivational and behavioral lapses and positive affect to protect against these lapses. We will also examine the extent to which within-subjects effects vary as a function of trait levels of impulsiveness. We expect relations between momentary impulsiveness and smoking will be stronger for individuals higher in baseline impulsiveness compared to individuals lower in baseline impulsiveness. Lastly, we will examine two-way interactions between these level-one predictors to evaluate how combinations of certain risk factors change the risk of experiencing a temptation or

smoking after quitting. We expect synergistic effects of smoking-related drive states (i.e., high urge, impulsiveness, or negative affect), and we expect positive affect to protect against smoking risk in these high drive states. We also expect momentary impulsiveness, affect, and urge may be moderated by context such that exposure to cigarettes or recent alcohol use will increase the odds of smoking in these states.

Method

Participants

The proposed project is a secondary analysis of prospective longitudinal EMA data from daily smokers attempting to quit smoking. All participants reported being motivated to quit and received standard nicotine lozenge and counseling treatment. Subjects were 109 smokers recruited in central New Jersey via mass media advertisements for smoking cessation research participants. Eligibility criteria for participation included: being at least 18 years old; English literate; smoking at least 10 cigarettes per day for at least 6 months; having an expired breath carbon monoxide (CO) level of at least 8 parts per million; reporting motivation to quit smoking of at least 6 on a 10-point scale; reporting no health conditions that would contraindicate use of the nicotine lozenge (e.g., recent heart attack or heart surgery, heart disease, angina, irregular heartbeat, pregnancy or breastfeeding, past problems using the lozenge); reporting no serious psychiatric conditions (i.e., bipolar disorder or psychosis); not living with other study participants; and no current use of other forms of tobacco, smoking cessation treatments, marijuana, or other illegal drugs.

Procedure

Interested participants contacted the laboratory and were screened for eligibility over the telephone. Eligible participants were scheduled for a group orientation session at which the research procedures were explained and written informed consent was obtained. Participants completed baseline assessments and were trained to use the EMA device (Palm Z22 Palmtop computers, Palm Inc., Santa Clara, CA). Participants returned for once weekly visits for 5 weeks, beginning one week pre-quit and ending three weeks post-quit. At each visit, participants were given feedback about their EMA adherence. All participants received four smoking cessation counseling sessions (15 minutes each) during the first four visits and a 12-week course of nicotine lozenges to begin using on their quit day. Lozenge dose was tailored to individual smoking level; participants received 4mg lozenges if they reported smoking within 30 minutes of waking and 2mg lozenges if they reported smoking more than 30 minutes after waking. Subjects completed a follow-up telephone interview 3-months post-quit to assess smoking status. Expired breath carbon monoxide or collateral confirmation of 3-month point-prevalence abstinence was obtained.

As part of the larger project, computerized laboratory measures of impulsive decision-making (delay discounting, Johnson & Bickel, 2002) and impulsive action (Continuous Performance Test, CPT-II; Conners, 1985; Conners, 2004) were administered one week pre-quit, on the quit-date, and three weeks post-quit. This project extended prior research by developing brief, adapted versions of these validated measures using MiniCog software (MiniCog, Cambridge, MA) which were administered four times daily via palmtop computer for a period of 31 days (3 practice days, one week pre-quit, and three weeks post-quit). Delay discounting measures were not included in the subsequent analyses because of limited variability in the momentary measure. Results suggest impulsive decision-making assessed by delay discounting and impulsive action assessed by continuous performance test (CPT) may be related but distinct facets of impulsiveness (Weafer et al., 2013) that differentially relate to nicotine-seeking and nicotine use (Diergaarde et al., 2008). The current project will focus on impulsive action or disinhibition as a risk factor for temptations and smoking after quitting.

Baseline Assessments

Baseline measures were completed at orientation and at one week pre-quit. Participants completed self-report questionnaires to assess individual differences in nicotine dependence (WISDM: Wisconsin Inventory of Smoking Dependence Motives; Piper et al., 2004; and FTND: Fagerstom Test for Nicotine Dependence; Heatherton, Kozlowski, Frecker & Fagerstrom, 1991), smoking history (i.e., years smoked, maximum duration of past abstinence), and demographics. Participants also completed self-report questionnaires to assess baseline positive and negative affect (PANAS: Positive and Negative Affect Schedule; Crawford & Henry, 2004; Watson, Clark, & Tellegen, 1988) and withdrawal symptom severity (WSWS: Wisconsin Smoking Withdrawal Scale; Welsch et al., 1999).

Baseline trait impulsiveness was measured by the Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, & Barratt, 1995). Items are rated on a 4-point scale ranging from 1 (*rarely/never*) to 4 (*almost always/always*). Total scores on the BIS-11 range from 30 to 120 with scores over 72 indicating high impulsiveness (Stanford et al., 2009). The scale has shown adequate internal consistency (total scale Cronbach's alpha=.83,

subscale alphas=.59-.74) and test-retest reliability (total scale Spearman's rho=.83, subscales .61-.72) (Stanford et al., 2009).

Baseline behavioral impulsiveness, or disinhibition, was assessed using a modified version of Conners' Continuous Performance Test: CPT-II (Conners, 1985; Conners, 2004). This computerized task is designed to measure sustained attention and the ability to inhibit prepotent responses, a process critical in quitting smoking. In this task, the participant was instructed to press a key every time a letter appeared on a computer screen, except when the letter was an "X". On each trial, a single letter was presented in the center of the screen for 250 ms, followed by a variable inter-trial interval of 1, 2, or 4 seconds. The version of the task administered in this study differed from the Conners' CPT-II in that trials with varying inter-trial intervals were interspersed rather than blocked to better match the version of the task programmed in the EMA device described below. To enhance participant motivation and effort on the task, participants were given feedback about their responses (in terms of accuracy after each block of 60 trials) and were paid a bonus of up to \$7.20 according to their performance (\$0.02 per correct response on each of 360 trials). Responses were screened for inattention, whereby blocks were excluded when omission errors were greater than 5% in any block of 60 trials (this affected 5.4% of blocks). Percent commission errors serve as a measure of disinhibition. This task has been well-validated as a measure of impulsiveness and has been related to smoking cessation success (Krishnan-Sarin, 2007).

Ecological Momentary Assessments

Participants were prompted at four pseudo-random times each day to complete 5minute EMA reports. The alarms were set by the experimenter to take place at random times within four equal intervals in the waking day but had to be at least 30 minutes apart. The prompt signaled participants to answer questions of momentary positive and negative affect, withdrawal symptoms, craving, exposure to smoking cues and triggers, access to cigarettes, strong urge or temptation to smoke in the past 30 minutes, and recent smoking, and alcohol use. Participants were also instructed to complete a brief, modified version of the CPT-II task, described below. Participants were encouraged to respond to the prompt within 30 minutes. Participant response times ranged from 0-575 minutes after the investigator-initiated prompt, with the majority of responses (88%) completed within 60 minutes of the prompt. Participants were more likely to report elevated urge or positive affect and recent alcohol consumption, stress, strong urge or temptation, and smoking opportunity in reports that were considered "late" versus "on-time" (i.e., completed within 30 minutes following the prompt). Therefore, all completed reports were included in the analyses to avoid introducing systematic bias by excluding late reports.

Momentary Impulsiveness. Participants were instructed to initiate a 60-66-trial version of the CPT-II task following completion of the self-report items. Participants could earn \$.02 for each correct trial for up to 60 trials (\$1.20 total), and they could receive feedback about their accuracy and earnings as incentives to enhance performance. Momentary impulsiveness was measured by percent commission error, excluding reports in which omission errors were greater than 5% (this affected 15.6% of reports).

Momentary Affect. Items derived from the PANAS and WSWS were used in EMA reports to assess momentary affect and withdrawal symptoms. Confirmatory factor analysis suggested a best-fitting model with two correlated negative affect factors: *distress* comprised of items "sad or depressed", "distressed", and "upset"; and *agitation* comprised of items "impatient", "tense or anxious" and "restless"; and one correlated positive affect factor with items "I have felt enthusiastic" and "I have felt interested" (Bold & McCarthy, in preparation). Smokers rated each item based on their experience in the 15 minutes prior to the prompt on a scale from 1 (very slightly or not at all/disagree) to 5 (extremely/agree).

Momentary Urge. Urge to smoke was assessed by two questions "I have trouble getting cigarettes off my mind" and "I have been bothered by the desire to smoke a cigarette" rated from 1 (disagree) to 5 (agree). Participants rated their agreement with these items based on their experience in the 15 minutes preceding the prompt. Scores on these items were averaged as an index of momentary urge. These items were selected because they were the best performing items in previous research with similar EMA assessments of urge (McCarthy et al., 2008).

Cigarette Exposure. Recent cigarette exposure was assessed via EMA by asking whether or not smokers had an easy opportunity to smoke or had been with someone who was smoking in the last 15 minutes. Recent cigarette exposure was coded as 1=yes or 0=no if subjects responded "yes" to either question.

Recent Alcohol Use. Recent alcohol use was coded 1=yes and 0=no based on participants' response to a multi-choice EMA question, "Have you had anything to drink in the past two hours?" where they could select "alcohol" among other responses.

Smoking Status. Post-quit reports were coded into mutually-exclusive smoking states based on EMA responses. Reports were categorized as *Abstinent* reports (i.e., in which participants reported no smoking since the previous report and no strong

temptation in the past 30 minutes, 46.1% of reports post-quit), *Temptation* reports (i.e., in which participants reported a strong urge or temptation in the past 30 minutes and no smoking since the last report, 30.8% of reports post-quit), and *Smoking* reports (i.e., in which any smoking occurred since the previous report, 23.1% of reports post-quit). *Data Analysis*

A series of multilevel models were tested using Hierarchical Linear Modeling (HLM) version 7.01 software (Raudenbush, Bryk, & Congdon, 2007) to evaluate the independent and interactive effect of smoking risk factors at t_0 (impulsiveness, affect, urge, cigarette exposure, and alcohol) on smoking status up to 8 hours later (t_1). Reports were retained for analysis if the time between report t_0 and t_1 was at least 30 minutes (to prevent reduced variability due to response simultaneity) and no greater than 8 hours (to remove the influence of overnight time periods between time-lagged analyses, see Shiffman et al., 1996 for a discussion of the time-course of urges and lapses within a day). The final sample used for analyses included 109 participants and 4179 reports. Demographic characteristics of the 109 individuals included in the analyses are summarized in Table 1.

A multinomial outcome distribution was specified with *Temptation* as the reference group to allow efficient, simultaneous comparisons between *Abstinent* vs. *Temptation* outcomes and *Smoking* vs. *Temptation* outcomes. Although the model estimated the log odds of being untempted and abstinent (vs. tempted but abstinent) we will present the results in terms of risk of temptation (vs. abstinent) and smoking (vs. temptation) to facilitate interpretation. Therefore, the current results indicate the odds of experiencing a temptation (vs. untempted) or smoking (vs. tempted) as a function of

impulsiveness, affect, urge, and context at the previous report, controlling for recent smoking status (Figure 1). Smoking status at t_0 was coded as smoking or abstinent based on the reported number of cigarettes smoked since the prior EMA report. We also evaluated negative affect and urge as predictors in smoking vs. abstinent, untempted comparisons to see if our results replicated the findings of Shiffman et al. (1996).

Level-one data comprised individual report-level predictors (i.e., reports of momentary impulsiveness, negative and positive affect, craving, cigarette exposure, and recent alcohol use) nested within individuals at the second level. Report-level continuous predictors (i.e., impulsiveness, affect, urge) were centered around the group mean prior to entry in the models. Therefore, estimated model intercepts represent the probability of experiencing a temptation or smoking at the average levels of impulsiveness, affect, and urge at the prior report.

First, the main effects of each predictor were examined in separate multinominal models with one focal predictor. All models contained covariates controlling for smoking status, time between reports, and time since quit day. To verify linearity over smoking state and time, predictor by smoking status, predictor by time between reports, and predictor by time since quit day interactions were examined. Additionally, the assumption of linearity in the logit was evaluated for each continuous predictor. A best-fitting multiple predictor model was constructed and baseline covariates were tested and retained if they accounted for significant variance and improved model fit. Next, we examined whether baseline trait impulsiveness moderated relations between momentary impulsiveness and outcome by sequentially adding baseline self-report impulsiveness (BIS) and behavioral impulsiveness (CPT) as predictors of the level-one momentary

impulsiveness coefficients. Lastly, we examined two-way interactions between level-one predictors in separate models (impulsiveness, affect, urge, cigarette exposure, alcohol) to facilitate model convergence. These models contained the main and interactive effects and controlled for smoking status, time between reports, and time since quit day. Non-significant variables were pruned from the models. The models were initially set with a random intercept and random effects to allow regression coefficients to vary across individuals, as long as doing so improved model fit. Predictors were set to fixed if doing so permitted model convergence or improved overall model deviance (Raudenbush & Bryk, 2002), measured by a reduction in the -2 log likelihood value (Cohen, Cohen, West, & Aiken, 2003).

Results

Model Evaluation

All variables in the model were examined for normality. The distribution of momentary impulsiveness, measured by percent commission error, showed substantial skewness (*statistic*=2.27, *SE*=0.04) and kurtosis (*statistic*=10.18, *SE*=0.08) which was reduced to an acceptable level with a square-root transformation (*skewness*=1.82, *SE*=0.04; *kurtosis*=1.88, *SE*=0.09). Skewness and kurtosis values on other variables were within an acceptable range ($<\pm$ 2.0) so data transformations were not necessary. Missing data were handled using full-information maximum likelihood estimation. The assumption of linearity of the logit held for all continuously scaled predictors, so no further transformations were necessary.

There were no significant interactions between predictors of interest (impulsiveness, affect, urge, cigarette exposure, alcohol) and either recent smoking status or time between reports. There were significant interactions between time since quit day and agitation, distress, and urge. Further examination indicated the effects of agitation, distress, and urge on outcome were non-linear over time. The effect of most predictors appeared to be strongest in the first week post-quit compared to the following one to two weeks, with the exception of distress which appeared to have a slightly stronger relation to smoking vs. resisting a temptation in week 3 (Figure 2). Because the majority of the coefficients were most divergent from zero in the first week, subsequent models focused on the effects in this window by including a binary time covariate (0=week one post-quit, 1=weeks two-three post-quit).

The following predictors remained after pruning non-significant level-one and level-two variables in the multiple predictor model: baseline nicotine dependence (measured by the WISDM), recent smoking status (Y/N), time between reports (t₀ to t₁), time since quit day (0=week 1, 1=week 2-3), agitation, distress, urge, and recent alcohol consumption. The best-fitting multiple predictor model contained a random intercept; random coefficients for agitation, urge, and alcohol use; and fixed coefficients for all other predictors. This model fit significantly better than a model with these predictors set to fixed (χ^2 =61.83, *df*=6, *p*<.001), and setting other predictors to random did not significantly improve model fit.

Temptation vs. Abstinent

Single predictor (Table 2, top panel) and multiple predictor (Table 3, top panel) HLM models predicted the odds of experiencing a temptation (vs. being abstinent and untempted). The results indicate the odds of temptation were greatest in the first week after the quit-day. Consistent with our hypotheses, greater momentary agitation and urge predicted greater odds of being tempted up to 8 hours later (Figure 1). Single predictor analyses indicated higher distress was associated with increased risk of temptation while positive affect protected against temptations, yet these effects were not robust and were not significantly related to outcome in the multiple predictor model. Baseline nicotine dependence was the only significant level-two predictor, indicating smokers with higher nicotine dependence scores (as measured by the WISDM at baseline) were more likely to experience temptations post-quit. Other level-one and level-two predictors did not significantly relate to temptations and were dropped from the multiple predictor model. Level-two variables (nicotine dependence, baseline impulsiveness, baseline affect, baseline withdrawal rating) did not significantly moderate relations between momentary predictors and temptations.

Interactions between our focal level-one predictors (momentary impulsiveness, affect, urge, cigarette exposure, and alcohol) were examined. To plot these effects for continuously scaled constructs, log odds were calculated for one standard deviation below the mean, the average, and one standard deviation above the mean to reflect the effect at low, mid, and high values of the predictor and moderator.

There was a significant interaction between impulsiveness and positive affect in a model robust against misspecification (B=0.89, SE=0.44, t(2648)=2.00, p=.046). The model estimates for this interaction differed from the standard fixed effects model (B=0.89, SE=0.57, t(2648)=1.56, p=0.12) which assumes normally distributed level-two residual errors (Maas & Hox, 2004). Discrepancies between model estimates may indicate non-normality or misspecification of the covariance matrix possibly due to autocorrelation effects. When this occurs, the model which is robust against

misspecification is likely an improved estimate over the standard model (Hox, 2010; Maas & Hox, 2004). The results indicate high positive affect was more protective against temptations in states of low momentary impulsiveness (Odds ratio=0.71) compared to high momentary impulsiveness (Odds ratio=1.34) (Figure 3a).

Urge was significantly moderated by negative affect. Results revealed a significant synergistic relationship between urge and agitation (B=0.12, SE=0.06, t(3844)=1.98, p=.047), such that urge was more strongly related to later temptation in agitated states (Odds ratio range=5.94-60.28) compared to times when agitation was low (Odds ratio range=3.00-15.04) (Figure 3b).

Interactions between affect and context revealed a marginal interaction between agitation and cigarette exposure (B=0.24, SE=0.13, t(3844)=1.84, p=.065). Smokers were non-significantly more likely to experience a temptation following times when high agitation was coupled with recent exposure to smokers or an easy opportunity to smoke (Odds ratio=5.65) than in the absence of these triggers (Odds ratio=2.32) (Figure 3c). Cigarette exposure significantly moderated the relation between urge and experiencing temptations (B=0.32, SE=0.12, t(3844)=2.72, p=.006) such that urge severity was more positively related to temptation risk when cigarettes were available (Figure 3d). Specifically, smokers were almost 14.5 times more likely to report a strong temptation following times of heightened urge if cigarettes were available (Odds ratio=19.09) than if cigarettes were not available (Odds ratio=4.76).

Smoking vs. Temptation

Single predictor (Table 2, bottom panel) and multiple predictor (Table 3, bottom panel) models predicted the odds of smoking (vs. experiencing a temptation without

smoking). The results indicate the odds of smoking were greater 2-3 weeks post-quit compared to the first week after the quit-day. Additionally, the odds of smoking were higher when smoking occurred at the previous report and when there was more time between predictor and outcome reports (t_0 to t_1). Consistent with our hypotheses, higher distress and recent alcohol consumption predicted greater odds of smoking up to 8 hours later. Contrary to expectations, agitation was negatively related to this outcome such that higher agitation predicted lower odds of smoking, or increased odds of resisting a temptation without smoking. No other level-one or level-two covariates were significantly related to smoking.

Although momentary impulsiveness did not significantly relate to smoking, baseline impulsiveness was a significant moderator of this relationship. Baseline trait impulsiveness (measured by the BIS but not behavioral disinhibition) significantly moderated the relation between momentary impulsiveness and smoking, such that this relation was stronger for individuals with greater self-reported trait impulsiveness, compared to those with lower trait impulsiveness (Table 4). No other level-two variables (nicotine dependence, baseline affect, baseline withdrawal rating) significantly moderated relations between momentary predictors and smoking.

Interactions between level-one predictors (momentary impulsiveness, affect, urge, cigarette exposure, and alcohol) were examined. Results indicated a significant interaction between impulsiveness and context. Specifically, momentary impulsiveness was significantly more likely to lead to smoking if alcohol was recently consumed (Odds ratio=1.82) than if alcohol was not consumed (Odds ratio=0.30) (B=5.42, SE=2.83,

t(2648)=1.91, p=.050) (Figure 4). No other level-one interactions were significantly related to smoking.

Smoking vs. Abstinent

Additional analyses evaluated negative affect and urge as predictors of smoking versus abstinent, untempted events. Both agitation and distress were significant predictors of smoking (vs. being abstinent and untempted) in single predictor models (agitation: B=0.18, SE=0.08, t(106)=2.44, p=.016; distress B=0.34, SE=0.08, t(106)=4.26, p<.001), although only distress remained significant in a multiple predictor model controlling for other covariates such as nicotine dependence, previous smoking, time since the quit day, urge, and alcohol use (agitation: B=-0.08, SE=0.10, t(106)=-0.82, p=.411; distress: B=0.22, SE=0.10, t(3206)=2.15, p=.031). Additionally, urge was a significant predictor of smoking in single predictor (B=0.38, SE=0.08, t(106)=5.01, p<.001) and multiple predictor models (B=0.33, SE=0.08, t(106)=3.97, p<.001). Negative affect and urge did relate to smoking in this sample, although only distress differentiated the subjective desire to smoke and the behavioral action of smoking.

Discussion

The current project used time-lagged hierarchical linear modeling to evaluate the independent and interactive effects of momentary relapse risk factors (impulsiveness, affect, urge, cigarette exposure, and alcohol) on the occurrence of temptations (vs. abstinence without temptations) and smoking (vs. resisting temptations) up to 8 hours later in smokers trying to quit. The results suggest the proximal determinants of temptations and smoking differ. Additionally, there were significant interactions between cognitive, affective, and contextual states that meaningfully influenced risk. This

suggests relapse processes are governed not only by main effects of known risk factors but also by interactions between these dynamic states, and we may uncover new information about relapse by investigating more complex relations between proximal relapse risk factors.

This project evaluated the influence of key momentary variables on later temptation risk (vs. being abstinent and untempted). Contrary to expectations, momentary impulsiveness was not significantly related to temptations, and baseline trait impulsiveness did not moderate relations between momentary impulsiveness and temptations. Several risk factors were significantly related to temptation risk including greater agitation, distress, and urge; and lower positive affect. The effects of distress and positive affect were not robust and were non-significant in the multiple predictor model, however. There were also significant interactions between states that influenced temptation risk. Specifically, states of high impulsiveness reduced the protective effect of positive affect on temptation risk, and urge and agitation had synergistic effects on temptation risk. Although the main effects of cigarette exposure and alcohol use on temptation risk were non-significant, context significantly moderated affect and urge states. Agitation and urge were more strongly related to temptation risk when cigarettes were available.

This project also examined proximal risk factors of smoking (vs. resisting a temptation without smoking). Contrary to our expectations, the main effect of momentary impulsiveness on smoking risk was non-significant, although this relation was significantly stronger for individuals who reported higher trait impulsiveness at baseline. Surprisingly, higher momentary distress significantly predicted increased smoking risk

while higher agitation was significantly associated with resisting temptations without smoking. Urge did not differentiate smoking from experiencing a temptation, and there were no significant interactions between states that influenced smoking risk. Although cigarette exposure did not independently or interactively relate to smoking, alcohol use was a significant predictor of greater smoking risk. There was also a significant interaction between momentary impulsiveness and alcohol, such that impulsiveness was more strongly related to later smoking when alcohol was recently consumed.

Furthermore, examination of level-two predictors revealed that greater nicotine dependence was a significant predictor of temptations although it was unrelated to the ability to resist temptations without smoking. Thus, nicotine dependence may be a factor that puts certain smokers at risk for motivational lapses while the influence of specific states drives behavioral lapses. Interestingly, the effect of several of these state predictors was strongest for the first-week post-quit compared to the following two weeks. Additionally, smokers were less likely to experience temptations and more likely to smoke over time. Taken together, this suggests smokers reach a steady state within the first week or so of quitting. This is in line with other research suggesting that returns to smoking happen rapidly after quitting (Baker et al., 2007) and once smoking occurs post-quit, it often continues; in the current study, smokers were 3.5 times more likely to smoke than resist temptations when they recently smoked compared to times when they had abstained. This suggests there may be a critical post-quit window for intervention where achieving initial success resisting temptations enhances cessation success.

The current project provides new information about state impulsiveness, affect, urge, and context as risk factors for lapses during a quit attempt and ways these predictors

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differentiate motivational lapses (i.e., temptations) and behavioral lapses (i.e., smoking). Specifically, some factors such as agitation, urge, and nicotine dependence appear to set the stage for lapses by increasing the likelihood of experiencing strong temptations while other factors such as distress and alcohol use relate specifically to the inability to resist these temptations without smoking. The current results expand previous work by Shiffman and colleagues (1996) and indicate different facets of negative affect differentiate temptations and smoking while other factors, such as urge severity, do not distinguish these outcomes. Examining the effects of multiple risk factors in the same model allows us to identify unique indicators of temptation and smoking risk. It may be important to focus on separate triggers for preventing motivational and behavioral lapses because different factors may drive smoking once a temptation occurs.

It is also important to consider how these momentary variables combine to influence temptation and smoking risk. Examining only main effects may lead to incorrect conclusions that certain risk factors do not influence lapses. Although momentary impulsiveness and cigarette exposure did not have significant main effects on temptation and smoking risk, they combined with affective and environmental states to predict these outcomes. Investigating these interactions may provide new information about relapse mechanisms. For example, urge was significantly moderated by affective (i.e., agitation) and environmental (i.e., cigarette exposure) states. These synergistic effects may help explain smokers' high relapse rates despite use of treatments designed to modulate craving specifically (e.g., Covey et al., 2007, Croghan et al., 2007). These treatments may be less effective in dealing with urges and preventing temptations if smokers are also agitated or exposed to cigarettes or a smoking opportunity. We need to update our models of relapse to include more complex relationships and interactions between momentary risk factors in order to better understand the dynamic processes driving relapse.

These findings could have important implications for smoking cessation treatments. The cognitive, affective, and contextual factors examined here led to temptations and the inability to resist smoking in this sample of motivated treatmentseekers. This risk was apparent despite the fact that all smokers received nicotine replacement therapy and counseling. Counseling sessions focused on identifying triggers, finding ways to modulate stress or craving (i.e., relaxation, nicotine replacement), and avoiding high-risk situations (i.e., being around other smokers, drinking alcohol), yet the current results suggest these strategies may need to be strengthened to effectively reduce temptation and smoking risk. For example, psychoeducational efforts to avoid high-risk situations such as cigarette and alcohol exposure may be critical to help smokers resist a temptation to smoke. Although alcohol use was endorsed in relatively low quantity (drinks per drinking day M=3.5, SD=3.8) and frequency post-quit (about 20% of days, 2.2% of reports) in this sample, any alcohol use was related to significantly greater odds of smoking, especially in impulsive states. Thus, advising smokers more effectively to avoid any alcohol use while quitting smoking may help prevent relapse.

It may also be important to target interoceptive triggers (i.e., agitation and distress) to the same extent as these external triggers. Smokers were encouraged to conduct self-monitoring of their moods to identify potential triggers for smoking, although the current findings suggest this was not done effectively enough to mitigate risk. This may indicate smokers would benefit from encouragement to self-monitor more

closely or from additional interventions designed to increase their awareness of and ability to modulate their affective states. Furthermore, this study identified important interactions between momentary internal and external risk factors which may suggest coping strategies designed to modulate internal risk (i.e., reduce negative affect or increase positive affect) may be less effective in certain contexts such as when cigarettes are readily available. Thus, strategies to prevent risk (i.e., through avoidance and stimulus control; Leventhal & Cleary, 1980) could be more helpful than those designed to mitigate exposure to risk once it occurs. These results also suggest some risk factors (such as nicotine dependence and baseline impulsiveness) may be identifiable at the outset of quitting, which may provide opportunities for interventions tailored to these at-risk individuals.

All participants received the same treatment in this study so the current data cannot address specific effects of counseling or nicotine replacement therapy on temptation risk or the ability to resist smoking; however, this study identified important relations between risk factors and temptations and smoking that could be used to guide future intervention research. It will be important to examine ways our currently available treatments modify both the independent and interactive effects of these risk factors on temptations and the ability to resist temptations.

There are several important limitations to the current study. Although the current project adds to the literature suggesting EMA technology may be a useful method to study relations between proximal risk factors and smoking outcome during a quit attempt (Shiffman, 2009; Stone & Shiffman, 1994), there are several limitations to this assessment method. First, the results are subject to reporting bias due to missing

responses to EMA prompts and particular compliance issues with the momentary impulsiveness measure. Smokers may be especially unlikely to respond to experimenter prompts in times of high stress, strong temptation, or when smoking which may reduce variance and limit our ability to identify critical, proximal risk factors for temptations and smoking. Additionally, there is limited evidence regarding the optimal assessment timeframe to capture the critical mechanisms of temptations and smoking. We attempted to control for lag between predictor and outcome by limiting the time between reports, yet longer time between reports remained a significant predictor of smoking. This may suggest there are other influential risk factors for smoking which were not identified with this method or in this time-frame. Additionally, if there is systematic missingness (as described above), we may be missing critical information from a report that should have occurred between the predictor and outcome reports that better predicts smoking behavior. Future research may benefit from more frequent EMA assessment or use of complementary research methodologies (i.e., laboratory studies) to identify the most proximal mechanisms driving smoking and provide information about the temporal resolution necessary to capture the phenomena of interest. Furthermore, there is limited information on the psychometric properties of brief self-report EMA measures and brief behavioral measures such as the momentary disinhibition task used in this study. There may be unique challenges to assessing in-vivo disinhibition including reduced attention to the task, and future research would benefit from examining the construct validity of these measures and replicating these results in additional samples. Lastly, we had limited power to detect interactions between level-two and level-one variables due to our small sample size at level two. It may be important to consider how other baseline and

personality variables influence the relationship between momentary risk factors and smoking outcome in future studies.

Despite these limitations, the current study provides new information about key relapse risk factors and their relation to temptations and smoking behavior after quitting. These risk factors were detectable up to 8 hours in advance of temptations and smoking, suggesting potential targets for near- or real-time interventions. Additionally, this study is among the first to document ways momentary impulsiveness, affect, urge, and context combine to modify short-term risk for temptations and smoking. Examining complex models that take into account interactions between proximal risk factors may be a critical step in improving our understanding of relapse processes in order to develop more effective smoking cessation interventions.

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References

- Baker, T.B., Mermelstein, R., Collins, L.M., Piper, M.E., Jorenby, D.E., Smith, S.S., ... Fiore M.C. (2011). New methods for tobacco dependence treatment research. *Annals of Behavioral Medicine*, *41*(2), 192-207.
- Baker, T. B., Morse, E., & Sherman, J. E. (1987). The motivation to use drugs: A psychobiological analysis of urges. In P. C. Rivers (Ed.), *The Nebraska* symposium on motivation: Alcohol and addictive behavior (pp. 257–323). Lincoln: University of Nebraska Press.
- Baker, T.B., Piper, M.E., McCarthy, D.E., Bolt, D.M., Smith, S.S., Kim, S., ... Toll, B.A. (2007). Time to first cigarette in the morning as an index of ability to quit smoking: Implications for nicotine dependence. *Nicotine & Tobacco Research*, 9(4), S555-S570.
- Baker, T. B., Piper, M. E., McCarthy, D. E., Majeskie, M. R., & Fiore, M. C. (2004). Addiction motivation reformulated: An affective processing model of negative reinforcement. *Psychological Review*, 111, 33-51.
- Bickel, W. K., Odum, A. L., & Madden, G. J. (1999). Impulsivity and cigarette smoking: Delay discounting in current, never, and ex-smokers. *Psychopharmacology* 146, 447-454.
- Bold, K.W., & McCarthy, D.E. (in preparation). Assessing the factor structure and time invariance of affect measured with electronic momentary assessment.
- Centers for Disease Control and Prevention (2008). Smoking-attributable mortality, years of potential life lost, and productivity losses—United States, 2000-2004. *Morbid Mortal Weekly*, *57*, 1226-1228.
- Centers for Disease Control and Prevention. (2011). Quitting Smoking Among Adults— United States, 2001–2010. *Morbidity and Mortality Weekly Report*, 60, 1513– 1519.
- Cohen, J., Cohen, P., West, S.G., & Aiken, L.S. (2003). *Applied multiple* regression/correlation analysis for the behavioral sciences (3rd ed.). Mahwah, NJ: Erlbaum.
- Conners, C. K. (1985). The computerized continuous performance test. *Psychopharmacology Bulletin, 21, 891-892.*
- Conners, C. K. (2004). Conners' Continuous Performance Test II (CPTII) for Windows Technical Guide and Software Manual. New York: Multi-Health Systems, Inc.
- Cooney, N.L., Litt, M.D., Cooney, J.L., Pilkey, D.T., Steinberg, H.R., & Oncken, C.A. (2007) Alcohol and tobacco cessation in alcohol-dependent smokers: Analysis of real-time reports. *Psychology of Addictive Behaviors*, 21(3), 277–286.
- Covey, L.S., Glassman, A.H., Jiang, H., Fried, J., Masmela, J., LoDuca, C., ... Rodriguez, K. A. (2007). Randomized trial of bupropion and/or nicotine gum as maintenance treatment for preventing smoking relapse. *Addiction*, 102, 1292-1302.
- Crawford, J. R. & Henry, J. D. (2004). The Positive and Negative Affect Schedule (PANAS): Construct validity, measurement properties and normative data in a large non-clinical sample. *British Journal of Clinical Psychology*, 43, 245–265.
- Croghan, I.T., Hurt, R.D., Dakhil, S.R., Croghan, G.A., Sloan, J.A., Novotny, P.J., ... Loprinzi, C.L. (2007). Randomized comparison of a nicotine inhaler and bupropion for smoking cessation and relapse prevention. *Mayo Clinic*

Proceedings, 82, 186-195.

- Dallery, J., & Raiff, B. R. (2007). Delay discounting predicts cigarette smoking in a laboratory model of abstinence reinforcement. *Psychopharmacology*, *190*, 485-496.
- Diergaarde, L., Pattij, T., Poortvliet, I., Hogenboom, F., de Vries, W., Schoffelmeer, A.N., & De Vries, T.J. (2008). Impulsive choice and impulsive action predict vulnerability to distinct stages of nicotine seeking in rats. Biological Psychiatry, 63, 301–308.
- Doran, N., Cook, J., McChargue, D., Myers, M., & Spring, B. (2008). Cue-elicited negative affect in impulsive smokers. *Psychology of Addictive Behaviors*, 22(2), 249-256.
- Field, M., Santarcangelo, M., Sumnall, H., Goudie, A., & Cole, J. (2006). Delay discounting and the behavioral economics of cigarette purchases in smokers: The effects of nicotine deprivation. *Psychopharmacology*, 186, 255–263.
- Fiore, M. C., Jaén C. R., Baker T. B., Bailey, W.C., Benowitz, N.L., Curry, S.J., ...Wewers, M.E. (2008). Treating Tobacco Use and Dependence: 2008 Update. Clinical Practice Guideline. Rockville, MD: U.S. Department of Health and Human Services. Public Health Service.
- Heatherton, T. F., Kozlowski, L. T., Frecker, R. C., & Fagerström, K. O. (1991). The Fagerström Test for Nicotine Dependence: A revision of the Fagerström Tolerance Questionnaire. *British Journal of Addiction*, 86, 1119–1127.
- Hox, J.J. (2010). *Multilevel analysis: Techniques and applications* (2nd ed.). New York, NY: Routledge.
- Johnson, M. W., & Bickel, W. K. (2002). Within-subject comparison of real and hypothetical money rewards in delay discounting. *Journal of the Experimental Analysis of Behavior*, 77, 129-146.
- Kassel, J. D., Stroud, L. R., & Paronis, C. A. (2003). Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychological Bulletin*, 129(2), 270-304.
- Krishnan-Sarin, S., Reynolds, B., Duhig, A. M., Smith, A., Liss, T., McFetridge, A., ... Potenza, M. N. (2007). Behavioral impulsivity predicts treatment outcome in a smoking cessation program for adolescent smokers. *Drug and Alcohol Dependence*, 88, 79-82.
- Lam, C.Y., Businelle, M.S., Aigner, C.J., McClure, J.B., Cofta-Woerpel, L., Cinciripini, P.M., & Wetter, D.W. (2014). Individual and combined effects of multiple highrisk triggers on postcessation smoking urge and lapse. *Nicotine & Tobacco Research*, 16(5) 569-575.
- Leventhal, H., & Cleary, P. D. (1980). The smoking problem: A review of the research and theory in behavioral risk modification. *Psychological Bulletin*, 88, 370–405.
- Maas, C.J.M., & Hox, J.J. (2004). Robustness issues in multilevel regression analysis. *Statistica Neerlandica*, 58(2), 127-137.
- Marlatt, G. A., & Gordon, J. R. (1980). Determinants of relapse: Implications for the maintenance of behavior change. In P. O. Davidson, & S. M. Davidson (Eds.). *Behavioral medicine: Changing health lifestyles*. New York: Bunner/Mazel.
- McCarthy, D. E., Piasecki, T. M., Lawrence, D. L., Jorenby, D. E., Shiffman, S., &

Baker, T. B. (2008). Psychological mediators of bupropion sustained-release treatment for smoking cessation. *Addiction*, *103*, 1521–1533.

- McClernon, F.J., Kollins, S.H., Lutz, A.M., Fitzgerald, D.P., Murray, D.W., Redman, C., & Rose, J.E. (2008). Effects of smoking abstinence on adult smokers with and without attention deficit hyperactivity disorder: results of a preliminary study. *Psychopharmacology*, 197, 95-105.
- Mitchell, S. H. (2004). Effects of short-term nicotine deprivation on decision-making: Delay, uncertainty, and effort discounting. *Nicotine and Tobacco Research*, *6*, 819-828.
- Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt Impulsiveness Scale. *Journal of Clinical Psychology*, *6*, 768–774.
- Piper, M.E., Federman, B.E., McCarthy, D.E., Bolt, D.M., Smith, S.S., Fiore, M.C., & Baker T.B. (2008). Using meditational models to explore the nature of tobacco motivation and tobacco treatment effects. *Journal of Abnormal Psychology*, 117(1), 94-105.
- Piper, M. E., Piasecki, T. M., Federman, E. B., Bolt, D. M., Smith, S. S., Fiore, M. C., & Baker, T. B. (2004). A multiple motives approach to tobacco dependence: The Wisconsin Inventory of Smoking Dependence Motives (WISDM-68). *Journal of Consulting and Clinical Psychology*, 72, 139–154.
- Prochaska, J. O. & DiClemente, C. C. (1983). Stages and processes of self-change of smoking: Toward an integrative model of change. *Journal of Consulting and Clinical Psychology*, 51(3), 390-395.
- Raudenbush, S. W. & Bryk, A. S. (2002). Hierarchical linear models: Applications and data analysis methods. Newbury Park: Sage Publications.
- Raudenbush, S., Bryk, A., & Congdon, R. (2007). *HLM for Windows (Version 6.04)*. Lincolnwood IL: Scientific Software International.
- Robinson, T. E. & Berridge, K. C. (1993). The neural basis of drug craving: An incentive sensitization theory of addiction. *Brain Research. Brain Research Reviews*, 18, 247–291.
- Schepis, T.S., McFetridge, A., Chaplin, T.M., Sinha, R., & Krishnan-Sarin. (2011). A pilot examination of stress-related changes in impulsivity and risk taking as related to smoking status and cessation outcome in adolescents. *Nicotine and Tobacco Research*, 13, 611-615.
- Shiffman, S. (1982). Relapse following smoking cessation: A situational analysis. *Journal of Consulting and Clinical Psychology*, 50(1), 71-86.
- Shiffman, S. (2009). Ecological Momentary Assessment (EMA) in studies of substance use. *Psychological Assessment*, 21, 486-497.
- Shiffman, S., Balabanis, M.H., Gwaltney, C.J., Paty, J.A., Gnys, M., Kassel, J.D., ... Paton, S.M. (2007). Prediction of lapse from associations between smoking and situational antecedents assessed by ecological momentary assessment. *Drug and Alcohol Dependence*, 91, 159-168.
- Shiffman, S., Paty, J.A., Gnys, M., Kassel, J.A., & Hickcox, M. (1996). First lapses to smoking: Within subjects analysis of real-time reports. *Journal of Consulting and Clinical Psychology*, 64(2), 366-379.
- Stanford, M. S., Mathias, C. W., Dougherty, D. M., Lake, S., Anderson, N. E., & Patton, J. H. (2009). Fifty years of the Barratt Impulsiveness Scale: An update and

review. Personality and Individual Differences, 47, 385-395.

- Stone, A.A., & Shiffman, S. (1994). Ecological momentary assessment (EMA) in behavioral medicine. *Annals of Behavioral Medicine*, *16*(3), 199-202.
- Strong, D.R., Kahler, C.W., Leventhal, A.M., Abrantes, A.M., Lloyd-Richardson, E., Niaura, R., & Brown, R.A. (2009). Impact of bupropion and cognitive-behavioral treatment for depression on positive affect, negative affect, and urges to smoke during cessation treatment. *Nicotine & Tobacco Research*, 11(10), 1142-1153.
- Tiffany, S. T. (1990). A cognitive model of drug urges and drug use behavior: Role of automatic and nonautomatic processes. *Psychological Review*, 97, 147–168.
- U.S. Department of Health and Human Services. *The health consequences of smoking: A report of the surgeon general.* Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2004.
- VanderVeen, J. W., Cohen, L. M., Cukrowicz, K. C., & Trotter, D. R. M. (2008). The role of impulsivity on smoking maintenance. *Nicotine and Tobacco Research*, 10, 1397-1404.
- Warner, K.E., Hodgson, T.A., & Caroll, C.E. (1999). Medical costs of smoking in the United States: Estimates, their validity, and their implications. *Tobacco Control*, 8, 290-300.
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology*, 54(6),1063-1070.
- Weafer, J., Baggott, M.J., & de Wit, H. (2013). Test-retest reliability of behavioral measures of impulsive choice, impulsive action, and inattention. *Experimental* and Clinical Psychopharmacology, 21(6), 475-481.
- Welsch, S. K., Smith, S. S., Wetter, D. W., Jorenby, D. E., Fiore M. C., & Baker, T. B. (1999). Development and validation of the Wisconsin smoking withdrawal scale. *Experimental and Clinical Psychopharmacology*, 7 (4), 354–361.

Figure Captions

Figure 1. Model of hypothesized relations between predictors (t0) and smoking outcome (t1).

The model was fit to test the hypothesized effects of impulsiveness, affect, urge, cigarette exposure, and alcohol use at t_0 on smoking behavior t_1 , while controlling for recent smoking, time between reports, and time since the quit day using HLM analysis. Odds ratios and p values are indicated only for variables of primary interest that were significantly associated with smoking outcomes (p < .05). Odds ratios are shown for the results of the multiple predictor models with the exception of distress and positive affect predicting temptation risk (indicated by dashed lines) which were significant in single predictor models controlling for smoking status, time between reports, and time since the quit day but were not significant in the multiple predictor analysis.

Figure 2. Time since quit day X agitation, distress, and urge interaction effects.

Moderating effects of time since quit day on relations between agitation, distress, and urge and experiencing temptations or smoking up to 8 hours later. Each line represents the regression coefficient for the predictor at each time point: week 1, week 2, week 3. The coefficients for most of these effects (with the exception of distress and smoking vs. temptation) were most divergent from zero (indicating stronger effects) in the first week post-quit compared to the following one to two weeks.

Figure 3a-d. Level one interaction effects predicting log odds of experiencing a strong temptation vs. being abstinent and untempted.

Interaction effects of positive affect x impulsiveness, urge x agitation, agitation x cigarette exposure, and urge x cigarette exposure on the log odds of experiencing a temptation up to 8 hours later. To plot these effects for the continuously scaled constructs, log odds were calculated from low, mid, and high range values representing one standard deviation below the mean, the average, and one standard deviation above the mean on these constructs, respectively. Impulsiveness values reflect square root percent commission errors on a momentary behavioral disinhibition task. Log odds can be exponentiated to derive odds ratios of experiencing a temptation (e^x).

Figure 4. Alcohol x impulsiveness interaction effect predicting log odds of smoking vs. resisting a temptation to smoke.

Moderating effects of alcohol use within the past 2 hours (t_0) on the relation between momentary impulsiveness (t_0) and the log odds of smoking up to 8 hours later (t_1). Low, mid, and high range impulsiveness values (measured by square root percent commission errors on a momentary behavioral disinhibition task) represent one standard deviation below the mean, the average, and one standard deviation above the mean of momentary impulsiveness, respectively. Log odds can be exponentiated to derive odds ratios of smoking (e^x).

| Variable | Value | n (%) | | |
|--------------------------------|-------------------------|-------------|--|--|
| Gender (N=109) | Female | 52 (47.7%) | | |
| Race/Ethnicity | Hispanic | 5 (4.6%) | | |
| (N=107) | White | 72 (67.3%) | | |
| | African-American | 25 (23.4%) | | |
| | Asian, Pacific Islander | 6 (5.6%) | | |
| | American Indian | 1 (0.9%) | | |
| | Other | 3 (2.8%) | | |
| Marital Status | Married | 41 (37.7%) | | |
| (N=109) | Never married | 31 (28.4%) | | |
| | Divorced | 13 (11.9%) | | |
| | Cohabitating | 10 (9.2%) | | |
| | Separated | 8 (7.3%) | | |
| | Widowed | 6 (5.5%) | | |
| Education | < High school graduate | 1 (0.9%) | | |
| (N=109) | High school graduate | 27 (24.8%) | | |
| | Some college | 47 (43.1%) | | |
| | College degree | 34 (31.2%) | | |
| Employment Status | Employed for wages | 59 (54.1%) | | |
| (N=109) | Self-employed | 15 (13.8%) | | |
| | Unemployed <1 year | 13 (11.9%) | | |
| | Unemployed >1 year | 8 (7.3 %) | | |
| | Homemaker | 3 (2.8%) | | |
| | Student | 11 (10.1%) | | |
| | Retired | 5 (4.6%) | | |
| | Disabled | 9 (8.3%) | | |
| Household Income | < \$25,000 | 35 (32.7%) | | |
| (N=107) | \$25,00-\$49,999 | 19 (17.8%) | | |
| | \$50,000-\$74.999 | 22 (20.6%) | | |
| | >\$75.000 | 31 (28.9%) | | |
| Variable | | M (SD) | | |
| Age (N=109) | | 45.0 (12.0) | | |
| Cigarettes smoked p | er day (N=109) | 18.6 (6.7) | | |
| Previous quit attempts (N=109) | | 4.3 (9.5) | | |
| Baseline FTND Score | re (N=109) | 5.3 (2.0) | | |

Table 1. Demographic characteristics of the final sample (N=109)

| Fixed Effect | Coefficient | Standard Error | T-ratio | Approx. df | p- value |
|--|-------------|-------------------|---------|---------------|-------------|
| Temptation vs. Abstinent | | | | | |
| Control Variables | | | | | |
| Recent smoking (prior to t_0 : Y/N) | -0.08 | 0.16 | -0.50 | 3850 | 0.616 |
| Time between reports $(t_0 \text{ to } t_1)$ | 0.02 | 0.02 | 0.58 | 3850 | 0.560 |
| Time since quit day (0=week 1; 1=week 2-3) | -1.10 | 0.10 | -10.12 | 3850 | <.001 |
| Single Focal Predictor Main Effects | | | | | |
| Impulsiveness ^a | -0.18 | 0.36 | -0.50 | 2652 | 0.617 |
| Agitation ^b | 0.37 | 0.08 | 4.19 | 106 | <.001 |
| Distress | 0.26 | 0.07 | 3.51 | 3848 | <.001 |
| Positive affect ^b | -0.22 | 0.08 | -2.50 | 106 | 0.014 |
| Urge ^b | 0.54 | 0.08 | 6.19 | 106 | <.001 |
| Cigarette exposure | 0.12 | 0.14 | 0.86 | 3848 | 0.392 |
| Alcohol ^b | -0.52 | 0.48 | -1.08 | 106 | 0.283 |
| Smoking vs. Temptation | | | | | |
| Control Variables | | | | | |
| Recent smoking (prior to t ₀ : Y/N) | 1.23 | 0.14 | 8.41 | 3850 | <.001 |
| Time between reports $(t_0 \text{ to } t_1)$ | 0.14 | 0.03 | 4.28 | 3850 | <.001 |
| Time since quit day (0=week 1; 1=week 2-3) | 0.74 | 0.12 | 5.80 | 3850 | <.001 |
| Single Focal Predictor Main Effects | | | | | |
| Impulsiveness ^a | 0.11 | 0.42 | 0.26 | 2652 | 0.789 |
| Agitation ^b | -0.18 | 0.10 | -1.90 | 106 | 0.059 |
| Distress | 0.08 | 0.08 | 1.02 | 3848 | 0.306 |
| Positive affect ^b | 0.06 | 0.10 | 0.64 | 106 | 0.518 |
| Urge ^b | -0.16 | 0.09 | -1.66 | 106 | 0.098 |
| Cigarette exposure | 0.04 | 0.16 | 0.27 | 3848 | 0.786 |
| Alcohol ^b | 1.36 | 0.46 | 2.96 | 106 | 0.004 |

Table 2. Single focal predictor main effects for all predictors (t_0) on smoking outcome within 8 hours (t_1)

Coefficients represent relations between the predictor (t_0) and log odds of experiencing a temptation or smoking (t_1) controlling for recent smoking, time between reports, and time since quit day.

a. Momentary impulsiveness data available for *N*=2930 reports.

b. Random coefficient. All other predictors were treated as fixed to facilitate model parsimony and convergence.

| Fixed Effect | Coefficient | Standard Error | T-ratio | Approx. df | p- value |
|--|-------------|-------------------|---------|---------------|-------------|
| Temptation vs. Abstinent | | | | | |
| Intercept ^a | -0.06 | 0.24 | -0.22 | 105 | 0.820 |
| Baseline Nicotine Dependence ^b | 0.04 | 0.02 | 2.54 | 105 | 0.013 |
| Recent smoking (prior to t ₀ : Y/N) | -0.19 | 0.16 | -1.22 | 3206 | 0.223 |
| Time between reports $(t_0 \text{ to } t_1)$ | 0.02 | 0.02 | 0.79 | 3206 | 0.428 |
| Time since quit day (0=week 1; 1=week 2-3) | -0.77 | 0.12 | -6.59 | 3206 | <.001 |
| Agitation ^a | 0.20 | 0.09 | 2.18 | 106 | 0.031 |
| Distress | -0.05 | 0.10 | -0.52 | 3206 | 0.600 |
| Urge ^a | 0.48 | 0.08 | 5.42 | 106 | <.001 |
| Alcohol ^a | -0.46 | 0.52 | -0.90 | 106 | 0.367 |
| Smoking vs. Temptation | | | | | |
| Intercept ^a | -0.94 | 0.23 | -4.04 | 105 | <.001 |
| Baseline Nicotine Dependence ^b | -0.00 | 0.02 | -0.21 | 105 | 0.834 |
| Recent smoking (prior to t ₀ : Y/N) | 1.24 | 0.14 | 8.37 | 3206 | <.001 |
| Time between reports $(t_0 \text{ to } t_1)$ | 0.14 | 0.03 | 4.18 | 3206 | <.001 |
| Time since quit day (0=week 1; 1=week 2-3) | 0.56 | 0.13 | 4.22 | 3206 | <.001 |
| Agitation ^a | -0.28 | 0.10 | -2.70 | 106 | 0.008 |
| Distress | 0.26 | 0.10 | 2.58 | 3206 | 0.010 |
| Urge ^a | -0.14 | 0.10 | -1.40 | 106 | 0.164 |
| Alcohol ^a | 1.31 | 0.49 | 2.66 | 106 | 0.009 |

Table 3. Trimmed multiple predictor HLM model of the effects of predictors (t_0) on smoking outcome within 8 hours (t_1)

Multiple predictor HLM model including level one and level two covariates. Nonsignificant variables were pruned from the model.

a. Random coefficient. All other predictors were treated as fixed to facilitate model parsimony and convergence.

b. Baseline nicotine dependence measured by the total score on the WISDM-68.

| Fixed Effect | Coefficient | Standard Error | T-ratio | Approx. df | p- value |
|--|-------------|-------------------|---------|---------------|-------------|
| Temptation vs. Abstinent | | | | | |
| Intercept ^a | -0.04 | 0.24 | -0.14 | 103 | 0.886 |
| Baseline Nicotine Dependence ^b | 0.04 | 0.02 | 2.31 | 103 | 0.023 |
| Recent smoking (prior to t ₀ : Y/N) | -0.20 | 0.18 | -1.10 | 2018 | 0.268 |
| Time between reports $(t_0 \text{ to } t_1)$ Time since quit day (0=week 1; | 0.03 | 0.03 | 0.96 | 2018 | 0.334 |
| 1=week 2-3) | 0.77 | 0.14 | 5.72 | 2018 | <.001 |
| State Impulsiveness | -0.16 | 0.38 | -0.45 | 2018 | 0.651 |
| Baseline Trait Impulsiveness ^c | 0.01 | 0.04 | 0.14 | 2018 | 0.888 |
| Agitation ^a | 0.23 | 0.10 | 2.32 | 104 | 0.022 |
| Distress | 0.00 | 0.10 | 0.03 | 2018 | 0.973 |
| Urge ^a | 0.39 | 0.08 | 4.58 | 104 | <.001 |
| Alcohol ^a | -0.46 | 0.59 | -0.78 | 104 | 0.433 |
| Smoking vs. Temptation | | | | | |
| Intercept ^a | -1.12 | 0.24 | -4.56 | 103 | <.001 |
| Baseline Nicotine Dependence ^b | 0.00 | 0.02 | -0.24 | 103 | 0.804 |
| Recent smoking (prior to t ₀ : Y/N) | 1.40 | 0.18 | 7.97 | 2018 | <.001 |
| Time between reports $(t_0 \text{ to } t_1)$ Time since quit day (0=week 1; | 0.15 | 0.04 | 3.68 | 2018 | <.001 |
| 1=week 2-3) | 0.50 | 0.16 | 3.18 | 2018 | 0.002 |
| State Impulsiveness | 0.09 | 0.44 | 0.21 | 2018 | 0.833 |
| Baseline Trait Impulsiveness ^c | 0.12 | 0.04 | 2.78 | 2018 | 0.005 |
| Agitation ^a | -0.18 | 0.12 | -1.52 | 104 | 0.131 |
| Distress | 0.22 | 0.12 | 1.82 | 2018 | 0.069 |
| Urge ^a | -0.14 | 0.11 | -1.29 | 104 | 0.199 |
| Alcohol ^a | 1.64 | 0.56 | 2.94 | 104 | 0.004 |

Table 4. HLM analysis of the interactive effects of baseline trait impulsiveness x momentary impulsiveness on smoking outcome up to 8 hours later

a. Random coefficient. All other predictors were treated as fixed to facilitate model parsimony and convergence.

b. Baseline nicotine dependence measured by the total score on the WISDM-68.

c. Baseline trait impulsiveness measured by the total score on the BIS.

Figure 1.

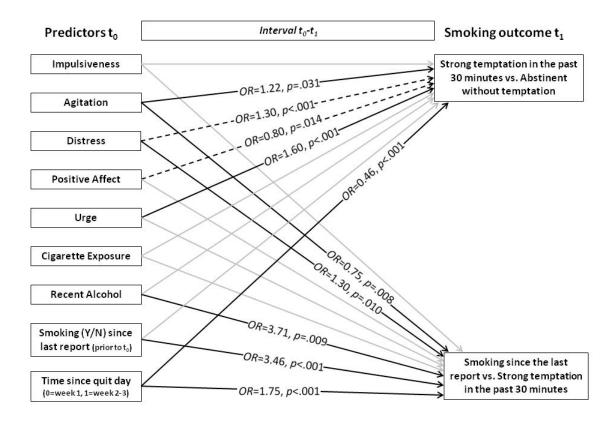


Figure 2.

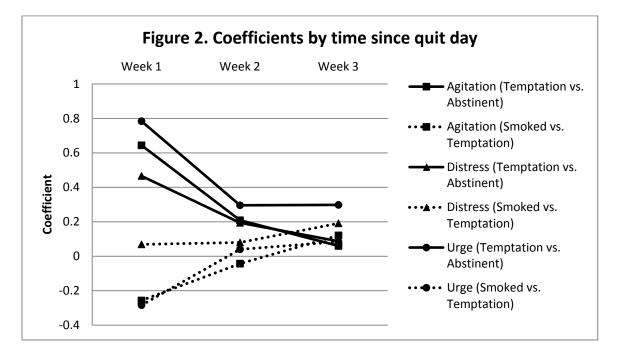


Figure 3.

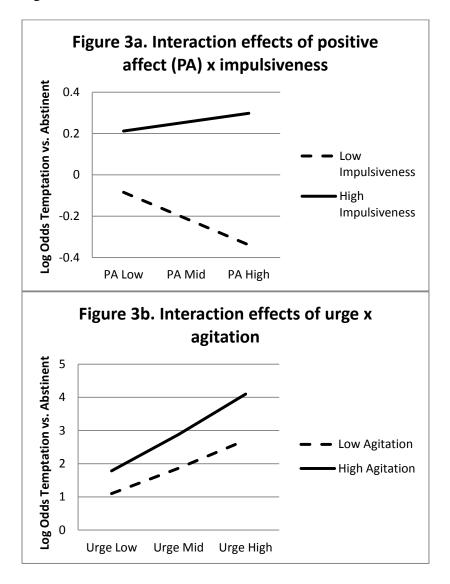


Figure 3 continued.

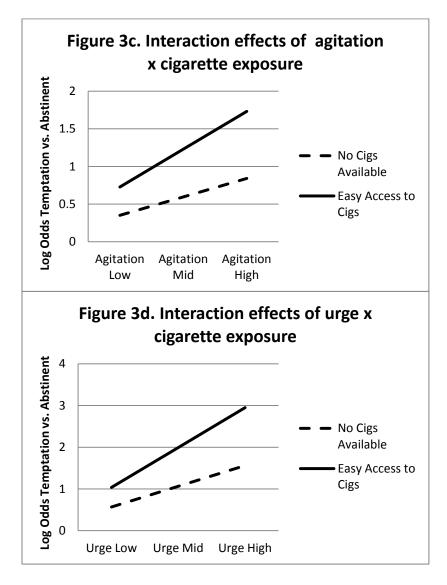


Figure 4.

