Psychopharmacology (2018) 235:2001–2012
https://doi.org/10.1007/s00213-018-4898-4

ORIGINAL INVESTIGATION

Momentary changes in craving predict smoking lapse behavior: a laboratory study

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Received: 14 December 2017 / Accepted: 5 April 2018 / Published online: 27 April 2018

Abstract

Rationale Current research on factors that predict smoking lapse behavior is limited in its ability to fully characterize the critical moments leading up to decisions to smoke.

Objectives We used a validated and widely used experimental analogue for smoking lapse to assess how moment-to-moment dynamics of craving relate to decisions to smoke.

Methods Heavy smokers ($N = 128$, M age = 35.9) participated in a 50-min laboratory delay to smoking task on 2 consecutive days, earning money for each 5 min they remained abstinent or ending the task by choosing to smoke. Participants rated craving and negative affect levels immediately prior to each choice. Participants were randomized to smoking as usual ($n = 50$) or overnight abstinence ($n = 50$ successfully abstained, $n = 22$ failed abstaining) prior to session 2. Discrete-time hazard models were used to examine craving and negative affect as time-varying predictors of smoking.

Results Higher craving levels prior to smoking opportunities predicted increased risk of smoking. When controlling for craving levels, incremental increases in craving predicted increased smoking risk. Increases in negative affect incrementally predicted increased smoking risk at session 2 only. Smokers who failed to abstain were at a higher risk of smoking than those who successfully abstained, whereas abstinent and non-abstinent smokers did not differ in smoking risk.

Conclusions Findings demonstrate an extension of the smoking lapse paradigm that can be utilized to capture momentary changes in craving that predict smoking behavior. Evaluations of nuanced craving experiences may inform clinical and pharmacological research on preventing smoking lapse and relapse.

Keywords Smoking · Lapse · Relapse · Craving · Cessation

The first smoking lapse during a cessation attempt is a robust predictor of smoking relapse (Brandon, Tiffany, Obremski, and Baker 1990; Shiffman, Paty, Gnys, Kassel, and Hickcox 1996). Extant research on smoking lapse has been generated to (1) characterize key person or situational variables that cause people to lapse or progress to full relapse (Shiffman et al. 1996; Shiffman and Waters 2004; McKee 2009) and (2) improve existing interventions to target these factors (McCallion and Zvolensky 2015; Shiffman, Kassel, Gwaltney, and McChargue 2005). Some programmatic research has sought to identify risk factors for lapse in smokers' natural environments through the use of ecological momentary assessment (EMA; Shiffman et al. 1996; Shiffman and Waters 2004). One major advantage of EMA studies is their ability to assess risk factors more proximally to smoking outcomes (Serre, Fatseas, Swendsen, and Auriacombe 2015). However, EMA data collection often occurs several minutes or hours prior to a lapse (Serre et al. 2015; Shiffman and Waters 2004). Such assessments therefore provide a crucial, but partial, characterization of factors pertinent to decisions to smoke.

Laboratory models of smoking lapse provide a useful analogue to prospectively evaluate proximal predictors of smoking behavior (McKee, Krishnan-Sarin, Shi, Mase, and O'Malley 2006; McKee 2009). In the smoking lapse paradigm, smokers engage in a brief abstinence period in which they are repeatedly offered opportunities to smoke or delay smoking for a small monetary reward (McKee 2009). Giving

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in and choosing to smoke provides an analogue of an initial smoking lapse. The duration of smokers' abstinence and the relationship of that duration to relevant smoking risk factors can then be examined (McKee 2009). Research employing the smoking lapse paradigm has demonstrated that several real-world risk factors for smoking lapse increase the likelihood of choosing to smoke in the paradigm, including alcohol consumption (Kahler et al. 2014; McKee et al. 2006), stress induction (McKee et al. 2011), self-control depletion (Heckman et al. 2017), and longer durations of abstinence (Heckman et al. 2017; McKee, Weinberger, Shi, Tetrault, and Coppola 2012). These findings validate the utility of this paradigm for examining risk factors for smoking lapse behavior. Although the smoking lapse paradigm cannot fully recreate the conditions under which smokers lapse or relapse in the natural environment, it offers a procedure to access and understand motivational factors involved in decisions to smoke after a period of abstinence.

No prior research using the smoking lapse paradigm has assessed potential risk factors for smoking throughout the duration of the task. This is a substantial limitation, as reactions in the moments leading up to when a smoker decides to smoke or remain abstinent may be critical in determining whether smoking occurs. One important risk factor for smoking lapse behavior is craving (Drummond 2001). Several theories of substance dependence propose that craving is a prominent aspect of drug use motivation (e.g., Marlatt 1985; Wikler 1948). Theories of craving based on classical conditioning predict that cues become associated with the psychoactive effects of the drug or the drug's withdrawal syndrome over time; subsequently, these cues can elicit conditioned craving responses (cue-elicited craving) that then act as a trigger for relapse even after long periods of abstinence (e.g., Stewart, de Wit, and Eikelboom 1984; Wikler 1948). Alternatively, cognitive theories suggest that the relationship between craving and drug use will necessarily depend on mediating factors, including drug expectancies (Marlatt 1985), drug availability (Tiffany 1990), and affect (Baker, Piper, McCarthy, Majeskie, and Fiore 2004). Despite its prominent role in nearly all theories of substance dependence, craving and drug use are not always tightly coupled (Gass, Motschman, and Tiffany 2014; Wray, Gass, and Tiffany 2013). Although more recent theories of substance dependence have been modified to explain this finding (e.g., Baker et al. 2004), we still have a limited understanding of the role of craving in motivating smoking lapse.

A major challenge in research on craving and its influence on lapse behavior is its variability in intensity over time (Drummond 2001; Tiffany and Wray 2012). To date, cumulative evidence from EMA studies supports a positive association between craving and smoking behavior, with craving providing greater predictive value when assessed in closer temporal proximity to smoking lapse (Serre et al. 2015). Although EMA assessments provide more detailed depictions of craving fluctuations than singular timepoint assessments, craving is a highly variable phenomenon (Wray, Gass, and Tiffany 2013). Therefore, more nuanced measures of craving may provide greater insight into its motivational relevance (Tiffany and Wray 2012). While very frequent assessments may be prohibitive in EMA research due to substantial participant burden, the smoking lapse paradigm offers a practical method to dissect the dynamics of cue-elicited craving and its influence on smoking risk. Learning how the dynamics of craving lead to decisions to smoke or remain abstinent could allow researchers to more effectively target these factors in smoking interventions.

Previous studies of craving within the smoking lapse paradigm have assessed craving levels prior to the onset of the task and/or after participants have made a decision to smoke, but not during the task itself (e.g., Heckman et al. 2017; McKee et al. 2011). In these studies, higher pre-task craving levels were associated with shorter latencies to smoke (Aquirre, Madrid, and Leventhal 2015; Kahler et al. 2014; Heckman et al. 2017; McKee et al. 2011; Roche et al. 2014; Zvolensky, Farris, Guillot, and Leventhal 2014; though cf. McKee et al. 2006, 2012). Some studies have found decreases in craving immediately after deciding to smoke in the paradigm (McKee et al. 2011, 2012), suggesting a role for craving in motivating smoking behavior. However, previous studies did not assess changes in craving immediately prior to smoking opportunities, which could reveal motivational dynamics critical to decisions to smoke.

The present study evaluated the dynamics of craving over the duration of an abstinence period and its association with smoking risk. Strongly dependent smokers engaged in a brief abstinence period using a modified version of the smoking lapse paradigm (McKee 2009). Participants rated their craving and negative affect immediately prior to making a choice to smoke or delay smoking for a small monetary reward. We sought to examine relationships between craving and smoking risk, in addition to change in craving as an incremental predictor of smoking risk. Increases in craving have been associated with smoking behavior in other studies (Shadel et al. 2011; Shiffman et al. 1997), though it is unclear to what extent *momentary* changes in craving may increase or decrease smoking risk during a period of abstinence. Further, negative affect is a consistent correlate of craving (Baker et al. 2004) and predictor of real-world lapse behavior (Shiffman and Waters 2004; Witkiewitz and Villarroel 2009). Thus, evaluations of dynamic changes in negative affect can provide additional information about the interplay between craving and negative affect and their potential joint influence on smoking risk.

In light of previous research demonstrating associations between pre-task craving and latency to smoke, we hypothesized that higher craving levels just prior to smoking opportunities would increase the risk of smoking. Further, we

hypothesized that momentary increases in craving would increase the risk of smoking, as escalating levels of craving may promote greater impetus to smoke. In a second session, we additionally evaluated the effect of abstinence on smoking risk. Based on prior studies with abstinence manipulations, we expected that abstinent smokers would have an increased risk of smoking relative to non-abstinent smokers (Heckman et al. 2017; McKee et al. 2012).

Methods

Participants

This experiment was part of a larger study examining an implicit measure of craving-related processes which included a cue reactivity procedure and cigarette purchase task (CPT; Jacobs and Bickel 1999) that are not fully discussed here (Germeroth and Tiffany 2017).

Eligibility criteria

Ages 18 to 65, smoking ≥ 15 cigarettes per day (CPD), on average, in the past month, smoking at current level for ≥ 1 year, not attempting to cut down or quit smoking, expired carbon monoxide (CO) level ≥ 10 ppm, other tobacco use ≤ 5 times in the past month, no diagnosis of drug dependence (other than nicotine) in the past year, not pregnant, and no medical issues affecting fine motor functioning (e.g., arthritis). Due to the possibility of random assignment to the abstinent condition, participants were required to indicate that they were confident in their ability to maintain overnight abstinence. As part of the larger study, equal sample sizes of successfully abstinent and non-abstinent smokers were required; therefore, a greater number of participants were recruited and assigned to the abstinent condition. Participants included 128 smokers who attended session 1 and 122 participants who also attended session 2 (Table 1).[1] Participants who failed to achieve overnight abstinence ($N = 22$) were retained in the sample as a separate group from non-abstinent ($N = 50$) and successfully abstinent participants ($N = 50$).

Measures

Craving was assessed using the four-item Craving Questionnaire (Carter and Tiffany 2001). Participants responded to each item for how they felt "right now," on a 1 (*strongly disagree*) to 7 (*strongly agree*) Likert scale, with anchors counterbalanced across participants (Nicholls, Orr, Okubo, and Loftus 2006). Negative affect was assessed using four items from the Mood Form (Diener and Emmons 1984), which were modified to match the Craving Questionnaire in word count and number of syllables ("All I feel right now is depressed or blue," "At this time, I feel worried or anxious," "I feel frustrated," and "I feel unhappy"). Negative affect items were assessed on a 1 (*not at all*) to 7 (*extremely*) Likert scale (anchors counterbalanced across participants). Reliability, calculated from session 1, for the craving (trial 1, $\alpha = .92$) and negative affect items (trial 1, $\alpha = .90$) were both high. Craving items were presented prior to negative affect items.

Nicotine dependence level was assessed with the 12-item Nicotine Addiction Taxon Scale (NATS; Goedeker and Tiffany 2008) and the Fagerström Test for Nicotine Dependence (FTND; Fagerström, Heatherton, and Kozlowski 1990). Current smoking level was measured with a 28-day Timeline Followback assessment (Sobell and Sobell 1996). Annual household income was measured on a 12-point Likert scale (1 ≤ $10,000, 12 ≥ $150,000). Smoking history variables, including age of first cigarette, years smoking, and number of previous quit attempts, were assessed with individual items in a smoking history questionnaire.

Procedure

Participants provided consent and completed a baseline CO assessment. At each session, experimenters collected a single cigarette of the participants' preferred brand, which was used in the delay to smoking task. Participants completed baseline measures of craving and negative affect, followed by a cue reactivity procedure (Germeroth and Tiffany 2017).

The delay to smoking task began approximately 1 h after the start of the session. Participants were presented with a half cigarette (McKee et al. 2006) displayed on a glass plate, a lighter, and an ashtray on a table in view of the participant. (A half, relative to whole, cigarette was used to increase the number of participants who would delay smoking, thereby resulting in variability in latency to smoke and allowing us to examine predictors of decisions to smoke.) The experimenter explained that the task could last up to 50 min and that participants would need to remain in the lab for an additional 30 min after the task was completed. The experimenter explained that every 5 min, they would be prompted via instructions presented on the computer monitor to make a choice between smoking half a cigarette and earning an amount of money that would be paid in cash immediately after the task ended. The experimenter described how the amount of money earned for each delay would decrease over the task, and as soon as participants chose to smoke, they could no longer earn additional money.

On a computer monitor, participants viewed the amount of money they could earn for delaying smoking for the next 5 min and the total amount of money earned thus far. After each 5 min delay, participants were prompted to "think about

[1] The six participants missing from session 2 either did not return for the session ($n = 5$) or a computer malfunction resulted in missing data for session 2.

Table 1 Characteristics of smokers in total sample and by abstinence condition

	Session 1 sample	Session 2 sample ($N = 122$)		
Demographics	($N = 128$)	Non-abstinent ($N = 50$)	Abstinent ($N = 50$)	Failed abstinence ($N = 22$)
% Male	70.3%	66.0%	68.0%	81.8%
Age (years)	35.9 (11.8)	37.5 (12.8)	35.9 (11.5)	32.5 (9.5)
Household income (USD)	$33,750 ($21,370)	$32,800 ($30,200)	$23,600 ($23,500)	$30,900 ($25,600)
% High school education or higher	89.3%	92.0%	90.0%	81.8%
Race/ethnicity				
% Caucasian	87.5%	90.0%	88.0%	90.9%
% Black or African American	10.9%	10.0%	10.0%	4.5%
% Asian	0.0%	0.0%	0.0%	0.0%
% Other race	1.6%	0.0%	2.0%	4.5%
% Hispanic or Latino	7.0%	4.0%	12.0%	4.5%
Smoking characteristics				
CPD	20.7 (6.6)	21.5 (6.7)	20.8 (6.0)	20.9 (6.0)
Age first cigarette (years)	14.5 (3.5)	14.6 (3.3)	14.1 (3.9)	14.9 (3.5)
Years smoking	21.5 (11.8)	22.9 (12.6)	21.9 (12.0)	17.6 (8.4)
NATS total	19.5 (2.3)	19.4 (2.5)	19.7 (2.2)	19.4 (2.3)
FTND total	5.9 (1.8)	6.1 (1.8)	5.8 (1.8)	5.6 (1.8)
% ≥ 1 quit attempt	75.8%	86.0%	68.0%	77.3%
Time since last cigarette (min)[†]	17.8 (18.0)	15.0 (12.0)	20.0 (22.0)	13.0 (11.0)
Baseline CO level (ppm)	32.1 (14.4)	31.6 (13.7)	35.8 (15.7)	27.1 (12.6)
Session 2 CO level (ppm)	–	$31.1\ (14.5)_a$	$10.6\ (6.0)_b$	$21.7\ (11.9)_c$

Table 1 displays sample characteristics with means and standard deviations or percentages. USD = U.S. dollar; CPD = cigarettes per day; NATS = Nicotine Addiction Taxon Scale; FTND = Fagerström Test for Nicotine Dependence; CO = carbon monoxide; ppm = parts per million. [†] Time since last cigarette was measured at the start of session 1. Participants who attended session 2 did not differ on any demographic or baseline characteristics across abstinence conditions, $ps > .05$. Consistent with the abstinence manipulation, smokers who were non-abstinent had a higher CO level at session 2 than smokers who successfully abstained or failed overnight abstinence, $ps \leq .002$. Smokers who were successfully abstinent had a lower CO level at session 2 than smokers who failed abstaining, $p < .001$. Subscripts indicate significant mean differences within the row at $p < .05$.

whether you want to earn $ (current interval dollar amount) or smoke a half cigarette right now." Participants completed the craving and negative affect questionnaires and then chose to smoke or earn money by pressing one of two response box keys. If participants chose to smoke the half cigarette, they discontinued the task and were instructed to smoke the cigarette as they normally would. Participants could remain abstinent for up to 50 min across 10 trials and earn a maximum of $5.50 ($1.00 for the first 5 min delay, reduced by $0.10 on each choice thereafter).

After the delay to smoking task, participants were required to wait 30 min. During this time, participants completed questionnaires on their smoking history, nicotine dependence, and demographics. At the end of session 1, participants were randomly assigned to overnight abstinence or non-abstinence by drawing a "non-abstinent" or "abstinent" indicating paper from a container. Participants in the non-abstinent condition were instructed to smoke as they normally would until session 2. Participants randomized to the abstinent condition were instructed to smoke as usual for the remainder of their session 1 day, but to refrain from smoking and using any other nicotine/tobacco products upon waking the following day. They were instructed that they would earn $25 for remaining abstinent which would be confirmed by CO assessment. Session 2 procedures were identical to session 1 but did not include post-task questionnaires. Session 2 abstinence was defined as a ≥ 50% CO level reduction from the CO level sample provided at session 1 (Day, Kahler, Spillane, Metrik, and Rohsenow 2014; Rhodes and Hawk 2016; VanderVeen, Cohen, and Watson 2013).

Participants received $75 for attending both sessions, $5 for arriving on time, $25 for maintaining overnight abstinence (abstinent-only group), and the money remaining from the delay to smoking task. Participants did not know the outcome of the CO level abstinence confirmation until the end of session 2.

Resources

DirectRT Precision Timing software (Empirisoft Corporation, New York, NY) and button boxes were used for the computerized delay to smoking task.

Data analytic strategy

Discrete-time hazard models (Cox survival analyses) were used to examine predictors of choosing to smoke. In these analyses, we evaluated whether higher craving, higher negative affect, and greater increases in craving and negative affect would predict increased risk of smoking over time. On each trial, participants who chose to smoke were assigned a value of 1 = *smoked*, or, if they chose to remain abstinent, a value of 0 = *abstinent*. Participants rated their craving and negative affect on every trial up until choosing to smoke and were right-censored if they chose to remain abstinent throughout the entire task. Sex, income, time since last cigarette, nicotine dependence (NATS total), and baseline expired CO level were used as covariates in these models. At session 2, abstinence condition was included as a three-level dummy-coded variable but time since last cigarette was not included.

All predictors except sex (male = 0) and abstinence condition (1 = non-abstinent, 2 = successfully abstinent, 3 = failed abstinence) were converted to *z*-scores to allow for comparison of hazard ratios across predictors. Craving and negative affect levels were standardized within the timepoint they were assessed, as each trial included different participants (i.e., all those who had not yet smoked). Analyses were performed using PROC PHREG in SAS version 9.4. Craving and negative affect were first examined in separate models as time-varying predictors of smoking. This allowed us to evaluate whether higher craving or negative affect levels, just prior to smoking opportunities, increased the risk of smoking. Change in craving and negative affect levels were then entered into their respective models as time-varying predictors. Change in craving and negative affect scores were calculated by subtracting values of the preceding trial from the current trial; thus, change scores captured both increases (positive scores) and decreases (negative scores) in craving and negative affect within the 5-min period prior to a smoking opportunity. We employed this operationalization of change scores because it allowed us to capture both large and rapid surges in craving and negative affect that may increase smoking risk and decreases in craving that may reduce smoking risk. Given that our primary study aim was to examine effects of momentary craving changes on proximal smoking risk, we reasoned that this operationalization, as opposed to within-subject mean difference change scores or other methods, best captured the construct of interest.

Significant hazard ratios > 1 indicated that greater increases in craving or negative affect (or alternatively, smaller decreases in craving or negative affect) predicted an *increased* risk of smoking over time; significant hazard ratios < 1 indicated that greater increases in craving or negative affect predicted a *decreased* risk of smoking. After examining craving and negative affect and their change in separate models, a combined model was used to examine their unique contributions to predicting smoking. Session 2 analyses were identical to session 1 models with abstinence condition included as a covariate. Predictors were considered significant at $p < .05$. Session 1 and 2 data met normality assumptions for survival analyses.

Results

Session 1

Overall, 98 participants (76.6%) chose to smoke during the task (median time to smoking = 31.3 min). Of those, eight participants (8.2%) chose to smoke on the first trial and did not delay smoking (Fig. 1a). Craving was highly inter-correlated across the 10 trials, rs = .71–.97, ps < .0001. Negative affect was also highly inter-correlated, rs = .61–.98, ps < .0001. Craving and negative affect were moderately to highly correlated across trials, rs = .34–.66, ps ≤ .01. Table 2 presents means and standard deviations.

Craving and negative affect levels When examined in separate models, both higher craving and negative affect levels immediately prior to smoking opportunities predicted increased risk of smoking, $\chi^2(1, N = 128) = 75.71$, $p < .0001$, $HR = 3.98$, 95% C.I. = 2.92–5.44, and $\chi^2(1, N = 128) = 23.15$, $p < .0001$, $HR = 1.68$, 95% C.I. = 1.36–2.08, respectively. However, only the effect of craving level on smoking risk remained significant in a combined model, such that a one *SD* increase in craving was associated with a more than fourfold increase in smoking risk, $HR = 4.10$ (Table 3, model 1).

Craving and negative affect levels and change When accounting for craving level, change in craving incrementally predicted increased risk of smoking, $\chi^2(1, N = 120) = 53.76$, $p < .0001$, $HR = 2.16$, 95% C.I. = 1.76–2.66. When accounting for negative affect level, change in negative affect did not predict smoking risk, $\chi^2(1, N = 120) = 2.18$, $p = .14$, $HR = 1.16$, 95% C.I. = 0.95–1.41. When accounting for negative affect variables, craving level and change each uniquely predicted increased risk of smoking (Table 3, model 2). A 1 *SD* increase in craving level was associated with a more than threefold increase in smoking risk, $HR = 3.63$, whereas a 1 *SD* increase in change in craving was associated with a more than twofold increase in smoking risk, $HR = 2.23$. When accounting for craving variables, however, the effect of negative affect level on smoking risk became non-significant, $HR = 0.96$. The effect of change in negative affect also remained non-significant, $HR = 0.92$.

Other covariates Higher nicotine dependence was associated with *decreased* risk of smoking in all models, HRs = 0.52 to 0.77, ps < .04. However, when nicotine dependence was the

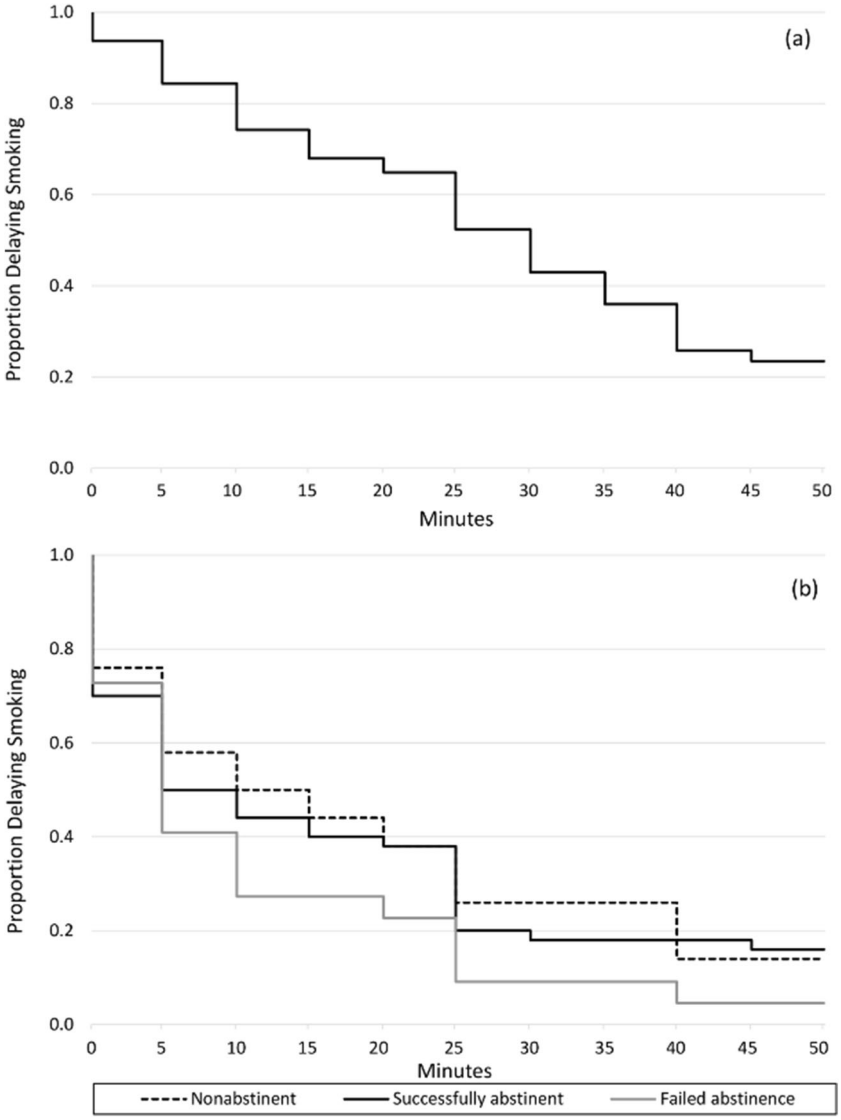

Fig. 1 **a** Survival curve displays the total proportion of participants ($N = 128$) choosing to delay smoking at each timepoint (0, 5, 10, 15, 20, 25, 30, 35, 40, and 45 min) they were provided an opportunity to smoke across the 50-min delay to smoking task at session 1. **b** Survival curves display the proportion of participants choosing to delay smoking across the 50-min delay to smoking task at session 2. Separate survival curves are displayed for non-abstinent smokers ($N = 50$), successfully abstinent smokers ($N = 50$), and smokers who failed to achieve overnight abstinence ($N = 22$)

only predictor in the model, it was not associated with smoking risk, $HR = 0.88$, $p = .24$. Household income was inconsistently associated with smoking risk, and when it was associated, higher income levels predicted increased risk of smoking, HRs = 1.13 to 1.26, ps = .02 to .27. Sex, baseline CO level, and time since last smoked were not predictive of smoking risk, ps > .05.

Session 2

A total of 105 participants (86.0%) chose to smoke during the task at session 2 (median time to smoking = 15.0 min). Of those, 33 participants (31.4%) chose to smoke on the first trial (Fig. 1b). Craving was highly inter-correlated across the 10 trials, rs = .56–.97, ps < .004. Negative affect was also highly inter-correlated, rs = .65–.97, ps < .001. Craving and negative affect were modestly to highly correlated across trials, r-s = .11–.62, ps < .0001 to .66. Lower correlations at session 2 likely reflect smaller sample sizes and fewer observations, particularly on later trials. Among non-abstinent smokers, latency to smoke was significantly correlated across sessions, $r = .75$, $p < .0001$. However, non-abstinent participants chose to smoke significantly sooner at session 2 ($M = 18.60$, $SD = 18.10$) than session 1 ($M = 26.50$, $SD = 16.94$), $t(49) = 4.51$, $p < .0001$.

Craving and negative affect levels Craving and negative affect level effects replicated session 1 such that, in separate models, higher craving and negative affect levels were associated with increased risk of smoking, $\chi^2(1, N = 122) = 41.01$, $p < .0001$, $HR = 2.45$, 95% C.I. = 1.86–3.22, and $\chi^2(1, N = 122) = 20.36$, $p < .0001$, $HR = 1.68$, 95% C.I. = 1.34–2.10, respectively. Similarly, when examining craving and negative affect levels as unique predictors, only craving predicted smoking risk. A 1 SD increase in craving was associated with a more than two-fold increase in smoking risk, $HR = 2.20$ (Table 3, model 3).

Table 2 Means and standard deviations of craving and negative affect measures across the delay to smoking task

Variable	Trial number									
	1	2	3	4	5	6	7	8	9	10
Session 1										
Craving	3.8 (2.0)	4.2 (2.0)	4.3 (2.0)	4.3 (1.9)	4.3 (1.9)	4.6 (2.0)	4.5 (2.1)	4.2 (2.0)	4.4 (2.1)	4.2 (2.0)
Negative affect	2.2 (1.5)	2.4 (1.7)	2.4 (1.5)	2.3 (1.4)	2.4 (1.5)	2.6 (1.5)	2.5 (1.7)	2.2 (1.6)	2.3 (1.6)	2.3 (1.6)
Sample size (*N*)	128	120	108	95	87	83	67	55	46	33
Session 2										
Craving										
Non-abstinent	3.4 (2.1)	4.0 (2.1)	3.7 (2.1)	3.6 (2.0)	3.8 (2.1)	3.4 (1.9)	3.4 (2.1)	3.6 (2.1)	4.0 (2.2)	3.1 (1.9)
Abstinent	5.6 (1.5)	5.8 (1.5)	5.4 (1.7)	5.4 (1.8)	5.4 (1.5)	5.1 (1.8)	4.4 (2.0)	4.3 (2.1)	4.0 (2.1)	4.1 (2.3)
Failed abstinence	5.2 (1.9)	4.9 (2.0)	3.8 (2.0)	3.8 (2.1)	3.8 (2.2)	3.9 (1.8)	4.8 (2.8)	4.3 (3.9)	5.1 (2.7)	2.5 (--)
Negative affect										
Non-abstinent	2.3 (1.7)	2.3 (1.4)	2.3 (1.4)	2.0 (1.3)	2.3 (1.7)	1.9 (1.2)	2.0 (1.2)	2.1 (1.2)	2.2 (1.4)	2.4 (1.4)
Abstinent	2.8 (1.5)	3.4 (1.8)	3.0 (1.7)	2.9 (1.9)	2.7 (1.8)	2.6 (1.7)	2.4 (1.2)	2.4 (1.3)	2.1 (1.1)	2.3 (1.3)
Failed abstinence	2.4 (1.4)	2.6 (1.5)	1.9 (1.1)	2.3 (1.3)	2.1 (1.2)	1.5 (0.6)	1.9 (0.9)	2.1 (1.2)	2.0 (0.7)	1.3 (--)
Sample size (*N*)	122	89	63	53	48	43	25	24	24	17

Table 2 displays means and standard deviations of craving and negative affect levels across the 10 trials of the delay to smoking task at session 1 and session 2. Higher values indicate stronger craving or negative affect (range = 1 to 7).

Craving and negative affect levels and change Also replicating session 1 results, when accounting for craving levels, change in craving incrementally predicted increased risk of smoking, $\chi^2(1, N = 89) = 21.13$, $p < .0001$, $HR = 1.78$, 95% C.I. = 1.39–2.28. In contrast to session 1, when accounting for negative affect levels, change in negative affect also incrementally predicted increased risk of smoking, $\chi^2(1, N = 89) = 14.64$, $p = .0001$, $HR = 1.55$, 95% C.I. = 1.24–1.95. When accounting for negative affect variables, craving level and change each uniquely predicted increased risk of smoking (Table 3, model 4). A 1 *SD* increase in craving level was associated with a 95% increased risk of smoking, $HR = 1.95$, whereas a 1 *SD* increase in change in craving was associated with a 73% increased risk of smoking, $HR = 1.73$. The direction of these effects replicated the findings of session 1. When accounting for craving variables, the effect of change in negative affect on smoking risk was also significant, $HR = 1.40$, such that a 1 *SD* increase in change in negative affect was associated with a 40% increased risk of smoking. This finding contrasted session 1 results.

Abstinence Abstinence condition was a significant predictor of smoking risk after accounting for craving and negative affect levels. Participants who attempted but failed to achieve abstinence had a more than twofold increased risk of smoking compared with participants who successfully abstained, $HR = 2.27$ (Table 3, model 3). This effect remained significant when accounting for both craving and negative affect levels and change, $HR = 2.94$ (Table 3, model 4). Participants who successfully abstained did not differ in smoking risk compared with non-abstinent participants, HRs = 1.36 and 1.23.

Other covariates Nicotine dependence, household income, sex, and baseline CO level were not predictive of smoking risk in any models, $ps > .05$.

Discussion

This study examined the dynamics of craving over the duration of a delay to smoking task, including craving and craving change as proximal predictors of smoking. Higher craving levels immediately prior to smoking opportunities were associated with an increased risk of smoking, and this effect was not accounted for by levels of negative affect. These findings extend results of previous studies using the smoking lapse paradigm that found higher pre-task craving predicted decreased latency to smoke (Heckman et al. 2017; McKee et al. 2011; Roche et al., 2014). Among previous studies, only one study examined smoking choice behavior using survival analyses that appropriately modeled censored data of smokers who remained abstinent throughout the entire task (Roche et al., 2014). Compared to that study, we found stronger relationships between craving and smoking when assessing craving as an immediate predictor of smoking (session 1 $HR = 3.63$, session 2 $HR = 1.95$ versus $HR = 1.41$; *z*-score transformed predictors). These results suggest that craving processes may have greater motivational influence on smoking behavior in the moments leading up to smoking opportunities, although methodological differences between these studies (e.g., duration of abstinence, craving items) limit our ability to draw definitive conclusions. Nevertheless, our findings are consistent with theories of

Table 3 Survival analyses on predictors of smoking in the delay to smoking task

Predictor	β (*SE*)	χ^2	*p*	Hazard ratio	95% C.I.
Session 1					
Model 1: craving and negative affect level ($N = 128$)					
Craving	1.41 (0.18)	**63.28**	**< .0001**	**4.10**	2.90–5.81
Negative affect	− 0.05 (0.12)	0.14	.71	0.96	0.75–1.21
Model 2: craving and negative affect level and change ($N = 120$)					
Craving	1.29 (0.19)	**43.82**	**< .0001**	**3.63**	2.48–5.31
Craving change	0.80 (0.11)	**51.33**	**< .0001**	**2.23**	1.79–2.77
Negative affect	− 0.04 (0.13)	0.08	.77	0.96	0.74–1.25
Negative affect change	− 0.09 (0.10)	0.83	.36	0.92	0.76–1.11
Session 2					
Model 3: craving and negative affect level ($N = 122$)					
Craving	0.79 (0.15)	**26.74**	**< .0001**	**2.20**	1.63–2.97
Negative affect	0.21 (0.13)	2.64	.10	1.23	0.96–1.59
Non-abstinent vs. successfully abstinent	0.31 (0.27)	1.34	.25	1.36	0.81–2.30
Failed vs. successfully abstinent	0.82 (0.30)	**7.26**	**.007**	**2.27**	1.25–4.13
Model 4: craving and negative affect level and change ($N = 89$)					
Craving	0.67 (0.19)	**12.90**	**.0003**	**1.95**	1.35–2.81
Craving change	0.55 (0.13)	**17.55**	**< .0001**	**1.73**	1.34–2.24
Negative affect	0.30 (0.18)	3.01	.08	1.36	0.96–1.91
Negative affect change	0.34 (0.12)	**8.04**	**.005**	**1.40**	1.11–1.77
Non-abstinent vs. successfully abstinent	0.20 (0.33)	0.38	.54	1.23	0.64–2.34
Failed vs. successfully abstinent	1.08 (0.38)	**8.14**	**.004**	**2.94**	1.40–6.16

Table 3 displays inferential statistics from discrete-time hazards models examining craving and negative affect as time-varying predictors of smoking. *SE* = standard error; C.I. = confidence interval. All predictors except sex (male = 0) and abstinence condition (1 = non-abstinent, 2 = successfully abstinent, 3 = failed abstinence) were mean centered and standardized to *z* scores. Models 1 and 3 examine craving and negative affect levels separately as predictors of smoking risk at sessions 1 and 2, respectively. Models 2 and 4 examine change in craving and negative affect as incremental predictors of smoking risk when controlling for craving and negative affect levels at sessions 1 and 2, respectively. Models reporting session 2 analyses include the three-level abstinence manipulation with successfully abstinent smokers as the reference group. Because change scores were only possible to calculate for participants who chose not to smoke on trial 1, sample sizes were smaller for these analyses. Sex, household income, baseline CO level, and nicotine dependence were included as covariates in all models. At session 1, time since last cigarette was also included as a covariate. Bolded values indicate statistical significance at $p < .05$.

addiction that purport a relationship between craving and smoking behavior (Drummond 2001). Importantly, this study included contextual factors that are likely important to real-world smoking behavior, including the presence of smoking cues and immediate cigarette availability (Carter and Tiffany 2001; Ferguson and Shiffman 2009).

A novel finding in this study was that greater momentary increases in craving predicted increased smoking risk. Further, the predictive contribution of change in craving emerged even after controlling for overall craving level. To our knowledge, this is the first laboratory study to demonstrate that changes in craving measured across relatively brief intervals (i.e., 5-min increments) are related to decisions to smoke. These effects were found among both non-abstinent and abstinent smokers. These results, which are consistent with EMA (Moore et al. 2014; Shiffman et al. 1997) and laboratory-based studies (Shadel et al. 2011) that have found increases in craving in the days and hours leading up to smoking lapses, have important theoretical and clinical implications. Our findings suggest that momentary increases in craving, even when overall craving levels are relatively low, may confer risk for smoking. This supposition is compatible with theories of addiction that identify craving as a possible, but not necessary, precipitant of drug use (e.g., Baker et al. 2004; Tiffany 1990). One interpretation of these results is that weaker levels of craving may not instigate decisions to smoke (perhaps because the experiences associated with that craving do not rise above the threshold to trigger action), whereas incremental increases in low-level craving still play a role in motivating smoking behavior. This explanation may, in part, account for studies

demonstrating dissociations between craving and drug use (Gass et al. 2014; Wray et al. 2013). More broadly, our findings suggest that comprehensive models of addiction should offer testable hypotheses as to when, how, and under what circumstances craving change is associated with drug use.

This study revealed fairly robust associations between craving, as well as change in craving, and smoking risk. Relationships of this magnitude are quite rare in the literature (Wray et al. 2013), but there are some conditions under which the associations are stronger (Gass et al. 2014; Sayette et al. 2000; Wray et al. 2013), conditions that we were able to replicate in this research. First, we had a highly reliable measure of craving, which is critical to detecting associations between craving and smoking-related outcomes (Sayette et al. 2000). Second, latencies between craving assessments and risk assessments were very short. To the extent that motivation to smoke waxes and wanes as a function of momentary changes in craving, procedures that capture those changes are more likely to generate better predictions of drug use. Third, in the present research, the decision to smoke was likely highly deliberative, that is, cognitively non-automatic (Tiffany 1990), and associations between craving and drug use have been shown to be markedly stronger when the drug use measure reflects the operation of non-automatic cognitive processing (Gass et al. 2014).

Within the present study, level of negative affect predicted increased risk of smoking, but this effect was no longer significant after accounting for craving level. Change in negative affect was uniquely predictive of smoking risk in session 2, but not in session 1. These findings are consistent with some EMA studies; one study demonstrated that surges in craving, but not negative affect, were associated with substance use relapse (Moore et al. 2014), and another study found that increases in negative affect were associated with smoking lapses that were identified as stress-motivated (Shiffman and Waters 2004). At session 1, negative affect levels were generally low, and it is possible that negative affect is less likely to be associated with smoking behavior during relatively short durations (approx. 1 h 15 min) of abstinence. Our sample size did not allow for examinations of interactions between negative affect and abstinence, which might provide insight into the conditions under which momentary changes in negative affect are predictive of smoking behavior. Future studies might consider examining momentary changes in negative affect after an affect or stress manipulation to more thoroughly investigate the proximal influence of negative affect on smoking risk. In addition, the relationship between abstinence, nicotine withdrawal symptoms, and smoking lapse risk could be examined to determine if relationships between negative affect and smoking lapse are mediated by nicotine withdrawal.

Abstinence condition was also predictive of smoking risk in the present study. Specifically, smokers who attempted but failed to achieve overnight abstinence were at higher risk of smoking compared with successfully abstinent and non-abstinent smokers. This finding is consistent with research that demonstrates an initial smoking lapse is predictive of subsequent smoking behavior (Brandon et al. 1990; Shiffman et al. 1996). Although our study was not designed to observe the effects of successful or unsuccessful abstinence on cognition, cognitive theories of substance dependence argue that beliefs regarding abstinence violation may facilitate the transition from lapse to relapse (Curry, Marlatt, and Gordon 1987) and are an important avenue for future research. Interestingly, we found that successfully abstinent smokers were at no greater risk of smoking than non-abstinent smokers. It is possible that the ability to maintain overnight abstinence generated greater self-efficacy among these participants about the ability to refrain from smoking for extended periods of time. Alternatively, monetary rewards for abstinence may have been more incentivizing for these participants.

A central aim of the present research was to assess craving and its relation to decisions to smoke in a more nuanced fashion. The lability of craving has posed a challenge for substance use researchers for quite some time (Drummond 2001; Tiffany and Wray 2012), and understanding its relevance to smoking lapse and relapse will require methodology that can more comprehensively assess its dynamics. Although laboratory studies cannot mimic all aspects of real-world smoking lapse, they offer well-controlled methodology through which to observe proximal predictors of smoking lapse behavior. One major advantage of the smoking lapse paradigm is its ability to model an initial return to smoking after a period of abstinence in a more controlled environment (McKee 2009). This procedure can accommodate evaluations of initial medication efficacy and their mechanisms of action that may ultimately support larger clinical trials (McKee 2009). Though laboratory "lapses" are not identical to real-world smoking lapse, they allow examinations of clinical phenomena that are fleeting and, by their very nature, difficult to assess.

There are some limitations to this study that warrant discussion. Due to the length of the experimental procedure, we did not include an ad-libitum smoking period after participants decided to smoke, which has been included in previous studies using the smoking lapse paradigm. Therefore, our conclusions are limited to predicting an initial decision to smoke and do not extend to smoking behavior thereafter. We also did not assess craving levels after participants chose to smoke during the task, which may provide additional information on the dynamics of craving as smokers transition from a period of abstinence to subsequent smoking. Participants were non-treatment-seeking smokers, and therefore, results may differ in individuals seeking smoking cessation treatment. Participants were also required to indicate that they were confident in their ability to maintain overnight abstinence, which may limit the generalizability of our findings. Additionally,

we found a significant relationship between nicotine dependence and smoking in session 1 that contradicts prior research (Shiffman et al. 1997 though cf. Roche et al., 2014). This effect was not found in session 2. It is possible that the combination of covariates (nicotine dependence, baseline CO level, and time since last cigarette) included in analyses may have generated a spurious relationship at session 1. It is also important to note that the sample consisted of heavy, highly dependent smokers, and therefore, there was little range in nicotine dependence levels across participants.

Our findings suggest several possible directions for future research. It would be useful to assess how the dynamics of craving relate to smoking behavior in confluence with other factors such as alcohol consumption, stress, and longer durations of abstinence (McKee et al. 2006, 2011, 2012). Learning under what conditions changes in craving lead to smoking lapse behavior may help to improve existing interventions. Measurement of other behavioral or psychophysiological indices throughout the delay to smoking task and their relationships with momentary craving dynamics may provide additional insight into how craving change can promote a decision to smoke. Further, levels of craving obtained immediately after smoking should be examined to determine whether momentary changes in craving prior to and following a choice to smoke are associated with one another.

Although we measured craving prior to decisions to smoke, we cannot be certain about the directionality of these effects. It is possible that choosing to smoke, in fact, is responsible for increasing craving levels in anticipation of smoking. Future research should seek to understand the directionality or bi-directionality between craving change and decisions to smoke. Although researchers have demonstrated that monetary incentives can temporarily increase motivation to abstain from smoking in smokers unmotivated to quit (Alessi, Badger, and Higgins 2004), it will be important to examine whether our findings replicate among treatment-seeking smokers. Finally, researchers should examine whether existing pharmacological treatments can alter craving dynamics and whether these changes are related to medication efficacy. Some research suggests that established smoking pharamacotherapies (e.g., combined nicotine replacement) suppress cigarette craving, and that craving suppression measured on the quit day is directly related to treatment outcomes (Bolt, Piper, Theobald, and Baker 2012). Future research should assess whether pharmacological agents can reduce momentary surges in craving to better understand how those agents aid successful abstinence.

Should the results of this study replicate among treatment-seeking smokers, there are several potential clinical implications. Information about the relationship between craving change and smoking lapse behavior may enhance existing smoking cessation interventions. For example, pre-quit assessments of craving dynamics may allow clinicians to predict how individuals will experience craving change during a cessation attempt. This information could be used to identify those who are most prone to react to modest changes in craving by choosing to smoke and help smokers prepare for craving fluctuations that are likely to emerge during an actual quit attempt. Empowering individuals with such knowledge may increase cessation self-efficacy, an important factor in treatment outcomes (Gwaltney, Metrik, Kahler, and Shiffman 2009). Clinicians might also tailor cessation advice to individualized craving experiences, and assist smokers in identifying and targeting increases in craving before they might typically be recognized.

Overall, as demonstrated during a laboratory delay to smoking task, dynamic changes in craving appear to be significant predictors of smoking behavior. Importantly, we found that momentary changes in craving that may motivate smoking behavior in the real-world can be captured in a laboratory environment using the smoking lapse paradigm. This extension of the paradigm offers a feasible means of accessing important facets of craving that predict smoking behavior after a brief period of abstinence. Moving forward, assessments that allow researchers to capture craving experiences in a more nuanced and dynamic fashion will be crucial to expanding our knowledge on how craving and its fluctuations may predict and perhaps contribute to ongoing smoking behavior and smoking lapse.

Acknowledgements The authors would like to acknowledge Dr. Craig Colder for his assistance with data analyses reported in this manuscript.

Funding information This research was supported by the American Psychological Association (Dissertation Research Award), the Bugelski Fellowship (Department of Psychology, University at Buffalo, SUNY), and the Mark Diamond Research Fund (Graduate Student Association, University at Buffalo, SUNY) awarded to co-author LJG.

Compliance with ethical standards

Conflict of interest Courtney A. Motschman, Lisa G. Germeroth, and Stephen T. Tiffany declare that they have no conflicts of interest. This research was approved by the Institutional Review Board (IRB) at the University at Buffalo, State University of New York. All participants provided written informed consent prior to their participation.

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