# EMOTION DYSREGULATION EXPLAINS THE RELATION BETWEEN INSOMNIA SYMPTOMS AND NEGATIVE REINFORCEMENT SMOKING COGNITIONS AMONG DAILY SMOKERS

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A Thesis

Presented to

The Faculty of the Department

of Psychology

University of Houston

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In Partial Fulfillment

of the Requirements for the Degree of

Master of Arts

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By

Brooke Y. Kauffman

May, 2017

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## ABSTRACT

Insomnia co-occurs with smoking. However, mechanisms that may explain their comorbidity are not well known. The present study tested the hypothesis that insomnia would exert an indirect effect on negative reinforcement smoking processes via emotion dysregulation among 126 adult daily smokers (55 females; $M_{age}$ = 44.1 years, *SD* = 9.72). Dependent variables included negative reinforcement smoking outcome expectancies, negative reinforcement smoking motives, and two negative expectancies from brief smoking abstinence (somatic symptoms and harmful consequences). Insomnia symptoms yielded a significant indirect effect through emotion dysregulation for negative reinforcement smoking outcome expectancies, negative reinforcement smoking motives, and harmful consequences expectancies from brief smoking abstinence. In contrast to prediction, however, insomnia was not associated with somatic symptom expectancies from brief smoking abstinence through emotion dysregulation. These data may suggest that the indirect effect of emotion dysregulation is more relevant to cognitive-affective negative reinforcement processes rather than somatic states. Overall, the present findings contribute to a growing body of literature linking emotion dysregulation as an explanatory mechanism for insomnia and smoking and uniquely extend such work to an array of clinically significant negative reinforcement smoking processes.

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## Insomnia: Prevalence and Global Impact

Insomnia is characterized as difficulty falling asleep, staying asleep, early morning awakening, or non-restful sleep (Sateia, Doghramji, Hauri, & Morin, 2000). It is one of the most common health behavior problems in the United States (U.S.) with approximately 35% to 50% of the general population experiencing some form of insomnia (Buysse, 2013; Roth, 2007). The disorder poses a large economic burden on society, costing the U.S. an estimated $65 billion dollars in health care costs and lost productivity annually (USDHHS, 2004). Furthermore, insomnia is associated with a number of negative biobehavioral problems, including a variety of physical illnesses (Mellinger, Balter, & Uhlenhuth, 1985), depression (Franzen & Buysse, 2008; Johnson, Roth, & Breslau, 2006; Ohayon, 2002; Ohayon, Caulet, & Lemoine, 1998), stress (Sheikh, Woodward, & Leskin, 2003), anxiety (Johnson et al., 2006; Ohayon, 2002; Papadimitriou & Linkowski, 2005), and a poor quality of life (Bolge, Doan, Kannan, & Baran, 2009; Sateia et al., 2000).

## Insomnia and Smoking Behavior: An Emerging Literature

Insomnia often co-occurs with other problematic health behaviors (e.g., addictive disorders). For example, research has highlighted the substantive comorbidity of insomnia and smoking (Conroy & Arnedt, 2014). One epidemiological study of 769 participants found that 39% of light smokers and 33% of heavy smokers experienced chronic insomnia (Riedel, Durrence, Lichstein, Taylor, & Bush, 2004). Furthermore, available data suggest the relation between insomnia and smoking is complex and bi-directional (Wetter & Young, 1994). Tobacco withdrawal, for instance, is associated with disrupted sleep (Rieder, Kunze, Groman, Kiefer, & Schoberberger, 2001) and desire to smoke during the sleep period (Scharf, Dunbar, & Shiffman, 2008). Conversely, greater levels of insomnia are associated

with an increased risk for smoking lapse and relapse during quit attempts (Augustson et al., 2008; Boutou et al., 2008; Short et al., in press). Despite the interconnection between insomnia and smoking, surprisingly little empirical work has explored mechanisms underlying their association.

**Possible Underlying Mechanism Linking Insomnia and Smoking: Emotion Dysregulation**

There is a need to explicate the processes governing associations between insomnia and smoking to clarify underlying factors that may help to explicate their comorbidity. Emotion dysregulation is one possible factor. Emotion dysregulation reflects difficulties engaging a set of abilities wherein one can observe, understand, evaluate, and differentiate one's emotions and subsequently access strategies to regulate emotions and control behavioral responses (Gratz & Roemer, 2004; Tull & Aldao, 2015). Some initial work has begun to document relations between insomnia and emotion dysregulation among non-smokers. For instance, poorer sleep quality and shortened sleep duration are both robustly associated with emotion dysregulation (Palmer & Alfano, 2016). Experimental work has found that sleep loss impacts connections between the medial prefrontal cortex and amygdala, showing increases in emotional arousal to coincide with deficits in emotional control and monitoring when sleep is inadequate (Yoo, Gujar, Hu, Jolesz, & Walker, 2007).

Theoretically, smokers with greater emotional dysregulation may respond to internal sensations (e.g., somatic agitation associated with sleep disturbance) in a less adaptive fashion, resulting in greater subjective distress and a corresponding tendency to engage in negative reinforcement smoking behavior to manage such aversive states. For example, lesser ability to observe, understand, evaluate, differentiate, and regulate aversive emotional states would be expected to be strongly related to more intense insomnia and related aversive emotional symptoms (Paulus et al., 2016). As a result of such emotion dysregulation, these individuals may use smoking as a means of regulating negative

emotions (Leventhal & Zvolensky, 2015). This perspective is consistent with observations from addictive behavior research wherein emotion dysregulation is an underlying factor that indirectly explains individual differences in negative emotionality and problematic substance use (Paulus et al., 2016; Paulus et al., in press). To our knowledge, there has been only one test of an emotion dysregulation model for insomnia and smoking (Fillo et al., 2016). In this study, emotion dysregulation explained the association between sleep disturbances and less self-efficacy for remaining abstinent in relapse situations, more quit-related problems during past quit attempts, and less quit attempts greater than 24 hours among treatment-seeking smokers (Fillo et al., 2016).

**Limitations of Previous Research**

Based upon theoretical models of emotion dysregulation and substance use (Leventhal & Zvolensky, 2015) and the findings of Fillo and colleagues (2016), there is a need to broaden our understanding of emotion dysregulation in the context of insomnia and smoking in at least three key ways. First, a central tenet of an emotion dysregulation perspective of insomnia and smoking rests on negative reinforcement processes; specifically, insomnia symptoms may interfere with smokers' capacity to regulate emotional states. As a result, smokers may seek out strategies to modulate distress (i.e., smoke), which may, in turn, maintain smoking behavior. Yet, past work has not explored negative reinforcement constructs in the context of insomnia-emotion dysregulation relations. Thus, a clinically and theoretically-relevant domain to evaluate would be whether emotion dysregulation indirectly accounts for the relation between insomnia and negative reinforcement smoking processes, such as anticipated negative reinforcement properties of smoking (outcome expectancies; Brandon & Baker, 1991), negative reinforcement reasons for smoking (motives; McCarthy, Curtin, Piper, & Baker, 2010), and negative smoking abstinence expectancies (expected consequences from brief (24-hour) smoking abstinence; Abrams, Zvolensky, Dorman, Gonzalez, & Mayer, 2011).

Second, past work has not adjusted for psychopathology in models exploring insomnia-emotion dysregulation processes. Given that psychopathology co-varies with smoking (Ziedonis et al., 2008), insomnia (Papadimitriou & Linkowski, 2005), and emotion dysregulation (Tull & Aldao, 2015), it is necessary to adjust for psychiatric disorders when evaluating the validity of this model. Specifically, for the emotion dysregulation mediational model of insomnia-smoking to have maximal clinical significance, it would have to explain variance that is not simply accounted for by psychopathology.

Finally, since the Fillo et al. (2016) study included a sample of treatment-seeking smokers, there is need to test insomnia-emotion dysregulation relations among non-treatment seeking daily smokers in order to understand the generalizability of these findings. For example, treatment-seeking smokers tend to be a more severe population and may therefore be more apt to represent a biased sample when considered in the larger context of the smoking population (i.e., Berkson bias; Berkson, 1946; Rothman, Greenland, & Lash, 2008). Thus, to better ascertain the generalizability of past insomnia-emotion dysregulation work, tests among non-treatment seeking smokers would be timely and an important next research step.

## Present Study Aims and Hypotheses

Together, the current study tested the hypothesis that insomnia would exert an indirect effect on negative reinforcement smoking processes via emotion dysregulation (see Figure 1). Specifically, insomnia was expected to be positively associated with emotion dysregulation, which, in turn, would be associated with smoking dependent variables. In the current study, four clinically significant dependent variables identified in past work were evaluated: negative reinforcement smoking outcome expectancies, negative reinforcement smoking motives, and two negative expectancies from brief smoking abstinence (somatic symptoms and harmful consequences). It was expected that an effect of insomnia via emotion dysregulation would be evident on all criterion measures over and above variance

accounted for by participant sex, number of cigarettes smoked per day (i.e., smoking rate), and psychopathology.

## METHOD

### Participants

Participants were 126 adult daily smokers (55 females; $M_{age}$ = 44.1 years, *SD* = 9.72). The racial composition of the sample was African American (64.3%), Caucasian (29.4%), Hispanic (4.8%), American Indian/Alaska Native (1.6%), Asian American (0.8%), and 4% 'Other.' On average, participants reported smoking 15.1 cigarettes per day (*SD* = 6.08), initiated smoking at age 16.2 (*SD* = 6.56), and reported smoking for 23.6 years (*SD* = 10.91). The average score on the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) was 4.7 (*SD* = 1.49), indicating low to moderate levels of tobacco dependence. Smoking status was confirmed by carbon monoxide (CO) sample analysis of expired breath sample (*M* = 22.6 ppm; *SD* = 11.43), which is above the 10ppm established cut-off for determining a positive smoking status (Cocores, 1993). Regarding current (past-year) psychopathology, 39.7% (*n* = 50) met criteria for one or more diagnoses (range 1-4 diagnoses), which included: posttraumatic stress disorder (30%), major depressive disorder (22%), specific phobia (20%), alcohol abuse/dependence (14%), panic disorder with/without agoraphobia (10%), dysthymia disorder (10%), cannabis abuse/dependence (10%), opioid abuse/dependence (10%), social anxiety disorder (8%), cocaine abuse/dependence (8%), bipolar I disorder (full remission; 4%), bipolar II disorder (4%), mood disorder NOS (4%), generalized anxiety disorder (4%), eating disorder NOS (4%), obsessive-compulsive disorder (2%), bulimia nervosa (2%), sedative, hypnotic, anxiolytic dependence (2%), and poly-substance dependence (2%).

### Measures

A *Demographics Questionnaire* was used to collect demographic data to describe the sample including participant sex, age, and race. Participant sex was used as a covariate in all analysis.

The *Structured Clinical Interview for DSM-IV Diagnosis of Axis I Disorders Non-Patient Version* (*SCID-NP;* First, Spitzer, Gibbon, & Williams, 2007) was used to evaluate the presence of current (past year) psychological disorders, and are reported for descriptive purposes. Additionally, presence/absence of current (past year) psychopathology coded yes (1) or no (0) was used as a covariate in all analysis. The interviews were administered by highly-trained post-baccalaureate research assistants and supervised by the study principal investigator.

Number of cigarettes smoked per day was assessed with the *Timeline Follow-back (TLFB),* a calendar-based assessment that assesses the quantity and frequency of cigarette use during the seven days prior to the in-person assessment. The TLFB has demonstrated good reliability and validity (Brown et al., 1998). Number of cigarettes smoked per day was used as a covariate in all analysis.

The *Smoking History Questionnaire (SHQ: Brown, Lejuez, Kahler, & Strong, 2002),* a 30-item self-report measure of descriptive lifetime smoking history, was utilized to describe the sample in terms of smoking rate, age of smoking initiation, and total years of regular smoking.

A *Vitalograph BreathCO* carbon monoxide (CO; Vitalograph Inc, Lenexa, Kansas) monitor was used to measure the amount of carbon monoxide (in parts per million [ppm]) in an expired breath sample, as a biochemical verification of smoking status and is reported for descriptive purposes.

Level of tobacco dependence was assessed with the *Fagerström Test for Nicotine Dependence (FTND; Heatherton et al., 1991),* a 6-item self-report assessment of gradations in cigarette dependence. Scores range from 0 to 10 with higher scores indicating

higher levels of physiological dependence on tobacco. The FTND items have high test-retest reliability (Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994), acceptable levels of internal consistency, and is closely related to key smoking variables (e.g., saliva cotinine; Heatherton et al., 1991). Internal consistency in FTND items in the current sample was low (Cronbach's α = .43), which is not uncommon for this measure (Korte, Capron, Zvolensky, & Schmidt, 2013). The FTND was reported for descriptive purposes.

Insomnia symptoms were assessed using a subscale from the *Inventory of Depression and Anxiety Symptoms (IDAS; Watson et al., 2007).* The IDAS is a 64-item self-report assessment of major depression and related anxiety disorders. Respondents are asked to rate the degree to which they have experienced symptoms within the past two weeks, rated on a 5-point Likert-type scale from 1 (*not at all*) to 5 (*extremely*). The Insomnia subscale (6 items; e.g., "I had trouble falling asleep") items are summed to derive an index of insomnia symptoms, with higher scores indicative of greater insomnia symptoms in the past two weeks. This subscale has strong psychometric properties including, test-retest reliability, internal consistency, and discriminant validity (Watson et al., 2007). Additionally, the insomnia subscale demonstrated convergent validity with the Beck Depression Inventory-II (Beck, Steer, & Brown, 1996), which includes an insomnia subscale (Watson et al., 2007). Internal consistency for the Insomnia subscale in the current sample was excellent (Cronbach's α = .92). The Insomnia subscale was used as a predictor variable.

The *Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004)* is a 36-item self-report measure used to assess the degree to which respondents experience dysregulated emotional states. Specifically, the DERS assesses six subscale facets which can be summed to create a total score: Non-Acceptance of Emotional Responses, Difficulties Engaging in Goal-Directed Behavior, Impulse Control Difficulties, Lack of Emotional Awareness, Access to Emotion Regulation Strategies, and Lack of Emotional Clarity. Items are rated on a 5-pont Likert-type scale from 1 (*almost never*) to 5 (*almost*

*always*). The total score can be derived, with higher values indicating greater emotion dysregulation (possible range = 36-180). The DERS items have strong psychometric properties (Gratz & Roemer, 2004; Whiteside et al., 2007). Internal consistency for the DERS total score was excellent (Cronbach's α = .95). The DERS total score was utilized as the mediator variable.

The *Smoking Consequences Questionnaire (SCQ; Brandon & Baker, 1991)* is a 50-item self-report measure used to assess the expected consequences of smoking a cigarette. Items are rated on a 10-point Likert-type scale ranging from 0 (*completely unlikely*) to 9 (*completely likely*). The SCQ measure and its constituent factors have demonstrated sound psychometric properties (Brandon & Baker, 1991; Buckley et al., 2005; Downey & Kilbey, 1995). In the current study, items from the Negative Reinforcement subscale (e.g., "If I am disappointed in myself, a good smoke can help") were computed (12 items) and used as a criterion variable in the current analyses. Internal consistency for these items was excellent (Cronbach's α = .95).

The *Wisconsin Inventory of Smoking Dependence Motives (WISDM; Piper et al., 2004)* is a 64-item self-report measure designed to assess 13 different theoretically derived motivational domains related to smoking. Items are rated on a 7-point Likert-type scale ranging from 1 (*not true of me at all*) to 7 (*extremely true of me*). In the current study, the Negative Reinforcement subscale, which includes 6 items, was used (e.g., "I reach for cigarettes when I feel irritable") as a criterion variable. The WISDM subscales have demonstrated acceptable levels of internal consistency (Piper et al., 2004). In the current study, internal consistency for the Negative Reinforcement subscale items was excellent (Cronbach's α = .92).

The *Smoking Abstinence Expectancies Questionnaire (SAEQ; Abrams et al., 2011)* is a 28-item self-report measure that assesses consequences to be expected as a result of acute smoking abstinence (i.e., stop smoking cigarettes/using nicotine for one day). Items

are rated in terms of expected likelihood on a scale from 0 (*very unlikely*) to 6 (*very likely*). In the current study, the somatic symptoms (e.g., “My chest would feel tight”; $\alpha$ = .82) and harmful consequences (e.g., “I would feel like I’m going crazy”; $\alpha$ = .86) subscales were used as criterion variables, consistent with past work (Farris, Langdon, DiBello, & Zvolensky, 2015).

**Procedures**

Adult daily smokers were recruited for an experimental study on anxiety and smoking behavior (Farris & Zvolensky, 2016). Community-recruited smokers were invited for a baseline assessment to determine eligibility for the experimental study. Inclusion criteria were as followed: (1) being between 18 to 65 years of age; (2) daily smoking for at least the past year; (3) smoking an average of ≥ 10 cigarettes per day; (4) biochemical verification of smoking status per expired carbon monoxide (CO) sample ≥ 10ppm; (5) smoking first cigarette of day within at least the first 30 minutes of waking (≥ 2 on the FTND item 1; Heatherton et al., 1991); and (6) stability of daily cigarette use (i.e., had not decreased the number of daily cigarette use by more than half in the past 6 months). Participants were excluded from the study based on evidence of: (1) potentially contraindicated medical condition with biological challenge (e.g., coronary heart disease, chronic obstructive pulmonary disease); (2) limited mental competency and/or the inability to give informed, written consent; (3) self-reported pregnancy or current nursing; (4) current psychotropic medication use; (5) current suicidal ideation/intent; (6) current non-nicotine substance use disorder or psychotic spectrum disorder assessed as determined by the SCID-NP (First et al., 2007); (7) current use of any pharmacotherapy or psychotherapy for smoking cessation; and (8) insufficient command of the English language. Additionally, due to computerized nature of the study assessment and procedures, participants were also excluded based on self-reported low computer literacy. However, the current study is a secondary analysis of data from all participants who completed the baseline assessment,

regardless of whether they were deemed eligible for the experimental phase of the study. Participants were compensated $25 for completing the baseline assessment. All participants provided written, informed consent prior to initiation of any study procedures, and this study protocol was approved by the Institutional Review Board where the study took place.

**Data Analytic Strategy**

Data were first checked for the presence of multivariate outliers; no outliers were detected. Next, sample descriptive statistics and zero-order correlations among study variables were examined. Primary analyses included regression-based path models. In all models, covariates included participant sex (coded: 0 = male and 1 = female), number of cigarettes smoked per day (per the TLFB), and presence/absence of current (past year) psychopathology (coded: 0 = no and 1 = yes). The data analytic strategy utilized (Hayes, 2009; Preacher & Hayes, 2004) allows for estimation and significance testing of the total indirect (mediation) effects through bootstrapping. Bootstrapping generates an empirical representation of the sampling distribution of the indirect effect, from which a confidence interval can be generated (Hayes, 2009). Analyses were conducted using PROCESS, a conditional process modeling program that utilizes an ordinary least squares-based path analytical framework to test for both direct and indirect effects (Hayes, 2013). Conservative confidence intervals (99%) were specified to adjust for Type I error rate inflation (Hayes & Preacher, 2014); the CIs for the indirect effects were estimated with bootstrapped analyses (10,000 resamples) as recommended (Hayes, 2009; Preacher & Hayes, 2004; Preacher & Hayes, 2008). Effect sizes ($\kappa^2$) were calculated for each indirect effect per recommendations of Preacher and Kelly (2011). This value can be interpreted in a similar regard as squared correlation coefficients, as outlined by Cohen (1988). To further strengthen the interpretation of results, alternative models were tested by reversing the proposed mediator for each of the three significant models (Preacher & Hayes, 2004);

specifically, emotion dysregulation was the predictor, insomnia was the indirect variable, and all criterion variables remained the same.

## RESULTS

### Bi-variate Relations

Zero-order correlations among all study variables are presented in Table 1. Average IDAS-Insomnia scores in the current study ($M$ = 12.33, $SD$ = 6.28) were comparable to normative scores for community-recruited individuals ($M$ = 12.7, $SD$ = 5.7; Watson et al., 2007). Insomnia symptoms were positively correlated with emotion dysregulation ($r$ = .57) and all criterion variables ($r$'s = .20-.37). Emotion dysregulation was positively correlated with all criterion variables ($r$'s = .36-.50).

### Indirect Effects

Regression results are presented in Table 2. For negative reinforcement smoking outcome expectancies, the total effects model with insomnia symptoms and covariates in the model accounted for significant variance ($R^2$ = .19, $F$[4, 121] = 7.10, $p$ < .001). The full model with emotion dysregulation was significant ($R^2$ = .29, $F$[5, 120] = 9.78, $p$ < .001). In the test of the indirect effect, results indicated that insomnia symptoms were associated with negative reinforcement smoking outcome expectancies, which indirectly occurred through the effect of emotion dysregulation ($a*b$ = 0.08, $SE$ = 0.03, $CI_{99\%}$ = 0.030, 0.169). The size of the indirect effect was medium to large ($\kappa^2$ = .22).

In terms of negative reinforcement smoking motives, the total effect model accounted for significant variance ($R^2$ = .14, $F$[4, 121] = 4.76, $p$ = .001). The model with emotion dysregulation accounted for additional variance for negative reinforcement smoking motives ($R^2$ = .20, $F$[5, 120] = 6.07, $p$ < .001). The test of the indirect effect indicated that insomnia symptoms were associated with negative reinforcement smoking motives indirectly through the effect of emotion dysregulation ($a*b$ = 0.05, $SE$ = 0.02, $CI_{99\%}$ = 0.009, 0.098). The size of the indirect effect was medium to large ($\kappa^2$ = .16).

In regard to somatic symptom expectancies from brief smoking abstinence, the total effects model with insomnia symptoms and all covariates in the model accounted for significant variance ($R^2$ = .21, $F$[4, 121] = 8.15, $p$ < .001). The full model with the addition of emotion dysregulation accounted for additional variance ($R^2$ = .23, $F$[5, 120] = 7.18, $p$ < .001). In the test of the indirect effect through emotion dysregulation, results indicated that insomnia symptoms were not significantly associated with somatic symptom expectancies from brief smoking abstinence ($a*b$ = 0.11, $SE$ = 0.08, $CI_{99\%}$ = -0.070, 0.338). The size of the indirect effect was medium to large ($\kappa^2$ = .11).

The total effects model for harmful consequences expectancies from brief smoking abstinence accounted for significant variance ($R^2$ = .22, $F$[4, 121] = 8.56, $p$ < .001). The full model including emotion dysregulation was significant ($R^2$ = .31, $F$[5, 120] = 10.90, $p$ < .001). In regard to the indirect effect, insomnia symptoms significantly predicted harmful consequences expectancies from brief smoking abstinence indirectly through emotion dysregulation ($a*b$ = 0.29, $SE$ = 0.10, $CI_{99\%}$ = 0.061, 0.588). The size of the indirect effect was medium to large ($\kappa^2$ = .22).

**Specificity Analyses**

In regard to the alternative models, the indirect effects of the alternate models were non-significant for negative reinforcement smoking outcome expectancies ($a*b$ = 0.00, $SE$ = 0.01, $CI_{99\%}$ = -0.015, 0.016), smoking motives ($a*b$ = 0.00, $SE$ = 0.00, $CI_{99\%}$ = -0.012, 0.011), and harmful consequences expectancies from brief smoking abstinence ($a*b$ = 0.01, $SE$ = 0.02, $CI_{99\%}$ = -0.045, 0.062).

## DISCUSSION

Insomnia often co-occurs with smoking (Riedel et al., 2004) and there is a need to explicate the processes governing these associations toward clarifying underlying factors that explicate their comorbidity. The current study expanded upon the literature by examining whether emotion dysregulation explained, in part, the relationship between

insomnia and negative reinforcement smoking outcome expectancies, negative reinforcement smoking motives, and negative expectancies from brief smoking abstinence (somatic symptoms and harmful consequences).

Consistent with prediction, insomnia symptoms yielded a significant indirect effect through emotion dysregulation for negative reinforcement smoking outcome expectancies, negative reinforcement smoking motives, and harmful consequences expectancies from brief smoking abstinence. Notably, the indirect effects were of medium to large size for all dependent variables ($\kappa^2$ = .11-.22). These results are consistent with the hypothesis that insomnia symptoms are related to emotion dysregulation (the 'a' path), which in turn, is related to a variety of negative reinforcement smoking maintenance processes (the 'b' path). Notably, the observed indirect effects were evident after adjusting for the influence of factors known to correlate with the severity of smoking behavior and insomnia, including participant sex, number of cigarettes smoked per day, and psychopathology. The current findings also are in line with past work that found emotion dysregulation mediated the association between insomnia and self-efficacy for remaining abstinent in relapse situations, prior quit-related problems, and presence of a prior quit attempt greater than 24 hours among treatment-seeking smokers (Fillo et al., 2016). Yet, in contrast to prediction, insomnia was not associated with somatic symptom expectancies from brief smoking abstinence through the indirect effect of emotion dysregulation. These data may suggest that the indirect effect of emotion dysregulation is more relevant to cognitive-affective negative reinforcement processes rather than somatic states. For example, emotion dysregulation may be more pertinent to disrupting cognitive (e.g., "Smoking helps me think more clearly") and affective states (e.g., "I need a cigarette now to calm my nerves") than those that are more physiologic in nature (e.g., "Smoking makes me less aroused"). Although cautiously speculative, future work could compare cognitive-affective versus

somatic negative reinforcement models to better gauge the applicability of this perspective to models of insomnia-smoking comorbidity.

As data were collected at one-time point, competing models were run to examine potential model misspecification. Specifically, for each hypothesized model (testing the indirect effect of insomnia via emotion dysregulation), competing models were run wherein emotion dysregulation was the predictor, insomnia was the indirect variable, and all three criterion variables remained the same. The competing models yielded non-significant indirect effects, adding confidence to the hypothesized model testing the indirect association of insomnia via emotion dysregulation. Overall, the regulation of emotions may represent an important intermediate construct linking insomnia to negative reinforcement cognitive-affective based smoking processes. Indeed, the present data suggest insomnia is related to emotion dysregulation, which, in turn, may affect negative reinforcement smoking outcome expectancies, negative reinforcement smoking motives, and negative expectancies from brief smoking abstinence (harmful consequences). Based upon these findings, future longitudinal work is needed to further evaluate this model.

The results from the present investigation may serve to conceptually inform the development of specialized intervention strategies for smokers prone to experiencing insomnia symptoms. Specifically, for smokers with insomnia symptoms, it may be advisable to understand and clinically address emotion dysregulation to enhance psychological flexibility related to maladaptive smoking cognitions (e.g., “I need a cigarette to cope with my (stressful) life”) and facilitate change in smoking behavior. For example, these results suggest that targeting and improving emotion dysregulation may have benefits in terms of addressing smoking behavior. Such an approach would be consistent with intervention work on emotion dysregulation and substance use (e.g., Berking et al., 2011). For example, changes in the use of maladaptive emotion regulation strategies over the course of treatment have been shown to predict changes in anxiety/stress and alcohol-related

psychopathology (Conklin et al., 2015). It is possible that tailoring these treatments to smokers with insomnia would offer a novel and more personalized approach to improve behavioral health and change smoking behavior.

There are several interpretive caveats to the present study. First, given the cross-sectional nature of these data, causal relations cannot be explicated. Tests were based on a theoretical framework supported by extant empirical data, but did not allow for testing of temporal sequencing. Based upon the present results, future prospective studies are necessary to determine the directional effects of these relations. Second, our sample consisted of community-recruited, daily cigarette smokers with moderate levels of nicotine dependence. Future studies may benefit by sampling from lighter and heavier smoking populations to ensure the generalizability of the results to the general smoking population. Third, as the key variables were assessed via self-report, there is the possibility that the observed relations were in part a function of shared method variance. Future research would benefit by employing a multi-method assessment approach to cross-index the nature of the relations observed in the current report. In particular, utilization of an objective measure of sleep such as actigraphy would be more ideal than sole reliance on subjective sleep reports.

Overall, the present study serves as an initial exploration into the association between insomnia, emotion dysregulation, and negative reinforcement smoking processes. There was consistent empirical evidence of indirect associations of insomnia via emotion dysregulation. Accordingly, if replicated, intervention programs for smokers with insomnia may benefit from consideration of emotion regulation therapeutic tactics.

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Table 1. *Descriptive Statistics and Bivariate Correlations between Study Variables* (*N = 126)*

| Variable | Mean/n (SD/%) | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Participant sex[a] | 55 (43.7%) | _ | | | | | | | | | |
| 2. CPD[a] | 14.68 (6.08) | -.00 | _ | | | | | | | | |
| 3. Psychopathology[a] | 50 (39.7%) | .07 | .02 | _ | | | | | | | |
| 4. Tobacco Dependence | 4.67 (1.49) | .05 | .53*** | .07 | _ | | | | | | |
| 5. Insomnia Symptoms[b] | 12.33 (6.28) | -.11 | .12 | .42*** | .15 | _ | | | | | |
| 6. Emotion Dysregulation[c] | 71.37 (23.10) | .09 | .14 | .33*** | .17 | .57*** | _ | | | | |
| 7. Smoking Expectancies[d] | 4.92 (2.44) | .21* | .25** | .22* | .04 | .28*** | .47*** | _ | | | |
| 8. Smoking Motives[d] | 4.40 (1.65) | .18* | .26** | .13 | .14 | .20* | .37*** | .81*** | _ | | |
| 9. Somatic Symptoms Abstinence Expectancies[d] | 7.08 (7.66) | .11 | .13 | .38*** | .14 | .35*** | .36*** | .30** | .30** | _ | |
| 10. Harmful Consequences Abstinence Expectancies[d] | 7.64 (8.66) | .08 | .22* | .34*** | .31 | .37*** | .50*** | .38*** | .37*** | .70*** | _ |

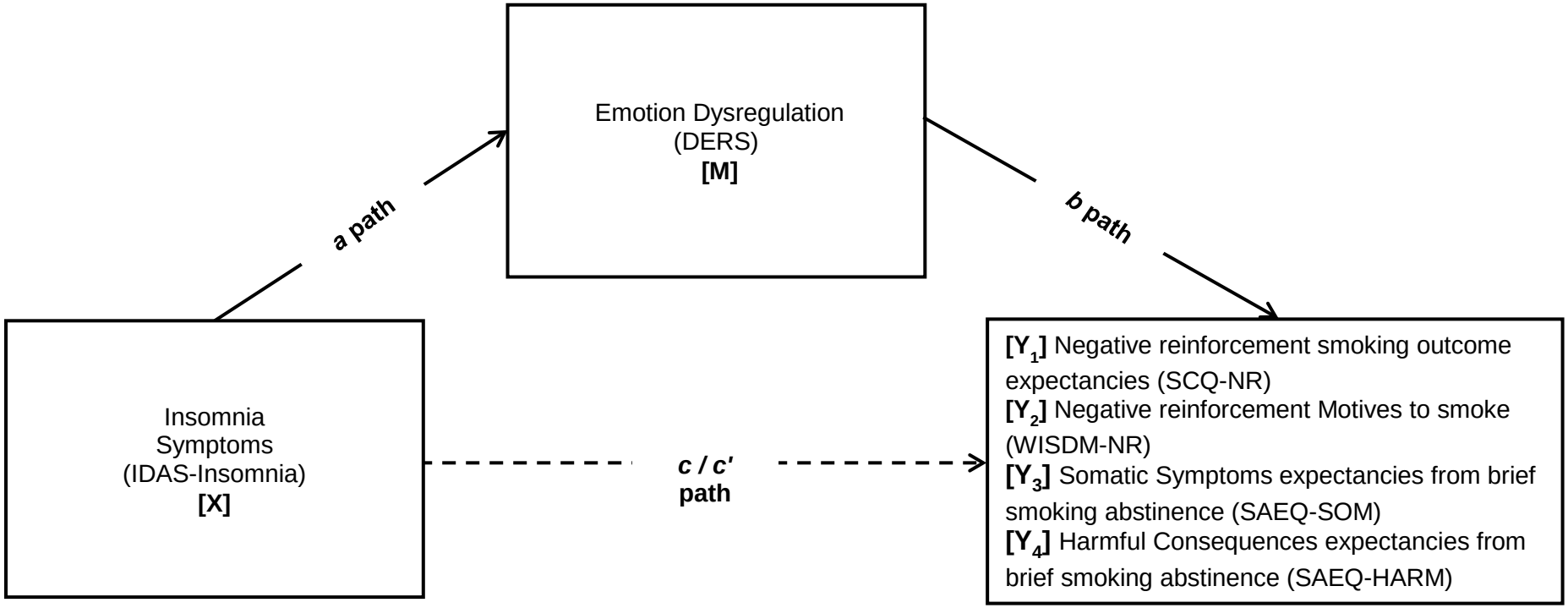
*Note*. ***$p$ < .001, **$p$ < .01, *$p$ < .05. [a]Covariate; [b]Predictor; [c]Mediator; [d]Criterion; Participant sex: % listed as females (Coded: 0 = male and 1 = female); CPD = number of cigarettes smoked per day per the Timeline Follow-back; Psychopathology = Presence/absence of current (past year) psychopathology per the Structured Clinical Interview—Non-Patient Version for DSM-IV (Coded: 0 = no; 1 = yes; First et al., 2007); Tobacco Dependence = Fagerström Test for Nicotine Dependence (Heatherton et al., 1991); Insomnia Symptoms = Inventory of Depression and Anxiety-Insomnia subscale (Watson et al., 2007); Emotion Dysregulation = Difficulties with Emotion Regulation Scale (Gratz & Roemer, 2004); Smoking Expectancies = Smoking Consequences Questionnaire-Negative Reinforcement subscale (Brandon & Baker, 1991); Smoking Motives = Wisconsin Inventory of Smoking Dependence Motives-Negative Reinforcement subscale (Piper et al., 2004); Somatic Symptoms Abstinence Expectancies = Smoking Abstinence Expectancies Questionnaire-Somatic Symptoms subscale (Abrams et al., 2011); Harmful Consequences Abstinence Expectancies = Smoking Abstinence Expectancies Questionnaire-Harmful Consequences subscale (Abrams et al., 2011).

Table 2. *Indirect Effect of Insomnia Symptoms on negative reinforcement smoking outcome expectancies, negative reinforcement smoking motives, and negative expectancies from brief smoking abstinence (somatic symptoms and harmful consequences)*

| Y | Path | $R^2$ | *b* | *SE* | *t* | *p* | CI (l) | CI (u) | $\kappa^2$ |
|---|---|---|---|---|---|---|---|---|---|
| **1** | IDAS-IN → DERS (*a)* | .36 | 2.00 | 0.30 | 6.68 | < .001 | 1.218 | 2.788 | |
| | DERS → SCQ-NR (*b)* | .29 | 0.04 | 0.01 | 4.91 | < .001 | 0.015 | 0.068 | |
| | IDAS-IN → SCQ-NR (*c')* | | 0.01 | 0.04 | 0.12 | .907 | -0.098 | 0.107 | |
| | IDAS-IN → SCQ-NR (*c)* | .19 | 0.09 | 0.04 | 2.47 | .015 | -0.005 | 0.181 | |
| | IDAS-IN → DERS → SCQ-NR (*a*b)* | | 0.08 | 0.03 | | | 0.029 | 0.169 | .22 |
| **2** | DERS → WISDM-NR (*b)* | .20 | 0.02 | 0.01 | 3.15 | .002 | 0.004 | 0.042 | |
| | IDAS-IN → WISDM-NR (*c')* | | 0.00 | 0.03 | 0.01 | .995 | -0.074 | 0.074 | |
| | IDAS-IN → WISDM-NR (*c)* | .14 | 0.05 | 0.03 | 1.85 | .067 | -0.019 | 0.112 | |
| | IDAS-IN → DERS → WISDM-NR (*a*b)* | | 0.05 | 0.02 | | | 0.010 | 0.101 | .16 |
| **3** | DERS → -SAEQ-SOM (*b)* | .23 | 0.06 | 0.03 | 1.67 | .098 | -0.032 | 0.143 | |
| | IDAS-IN → SAEQ-SOM (*c')* | | 0.18 | 0.13 | 1.41 | .161 | -0.155 | 0.517 | |
| | IDAS-IN → SAEQ-SOM (*c)* | .21 | 0.29 | 0.11 | 2.64 | .009 | 0.003 | 0.581 | |
| | IDAS-IN → DERS → SAEQ-SOM (*a*b)* | | 0.11 | 0.08 | | | -0.069 | 0.333 | .11 |
| **4** | DERS → SAEQ-HARM (*b)* | .31 | 0.14 | 0.04 | 4.00 | < .001 | 0.049 | 0.235 | |
| | IDAS-IN → SAEQ-HARM (*c')* | | 0.08 | 0.14 | 0.56 | .579 | -0.283 | 0.435 | |
| | IDAS-IN → SAEQ-HARM (*c)* | .22 | 0.36 | 0.12 | 2.91 | .004 | 0.036 | 0.687 | |
| | IDAS-IN → DERS → SAEQ-HARM (*a*b)* | | 0.29 | 0.10 | | | 0.062 | 0.595 | .22 |

*Note.* Path a is equal in all cases $Y_{1–4}$; therefore, it presented only once to avoid redundancies. N for analyses is 126 cases. The standard error and 99% CI for the indirect effects (a*b) are obtained through bootstrapping with 10,000 re-samples. a path = Effect of X on M; b paths = Effect of M on Yi; c' paths = Direct effect of X on Yi controlling for M; c paths = Total effect of X on Yi. IDAS-IN = Inventory of Depression and Anxiety-Insomnia subscale (Watson et al., 2007); DERS = Difficulties with Emotion Regulation Scale (Gratz & Roemer, 2004); SCQ-NR = Smoking Consequences Questionnaire-Negative Reinforcement subscale (Brandon & Baker, 1991); WISDM-NR = Wisconsin Inventory of Smoking Dependence Motives-Negative Reinforcement subscale (Piper et al., 2004); SAEQ-SOM = Smoking Abstinence Expectancies Questionnaire-Somatic Symptoms subscale (Abrams et al., 2011); SAEQ_HARM = Smoking Abstinence Expectancies Questionnaire-Harmful Consequences subscale (Abrams et al., 2011). Covariates included participant sex, number of cigarettes smoked per day, and psychopathology.

*Figure 1*. Conceptual model




*Note*. a path = Effect of X on M; b paths = Effect of M on $Y_i$; c paths = Total effect of X on $Y_i$; c' paths = Direct effect of X on $Y_i$ controlling for M. Four separate paths were conducted ($Y_{1\text{-}4}$) with the predictor (X). Covariates included participant sex, number of cigarettes smoked per day, and psychopathology.