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Emotion dysregulation explains the relation between insomnia symptoms and negative reinforcement smoking cognitions among daily smokers

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HIGHLIGHTS

- Emotion dysregulation mediated insomnia and NR smoking outcome expectancies.
- Emotion dysregulation mediated insomnia and NR smoking motives.
- Emotion dysregulation mediated insomnia and HC smoking abstinence expectancies.

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ABSTRACT

Introduction: Insomnia co-occurs with smoking. However, mechanisms that may explain their comorbidity are not well known.
Method: The present study tested the hypothesis that insomnia would exert an indirect effect on negative reinforcement smoking processes via emotion dysregulation among 126 adult non-treatment seeking daily smokers (55 females; $M_{age} = 44.1$ years, $SD = 9.72$). Negative reinforcement smoking processes included negative reinforcement smoking outcome expectancies, negative reinforcement smoking motives, and two negative expectancies from brief smoking abstinence (somatic symptoms and harmful consequences).
Results: 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.
Conclusions: 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.

1. Introduction

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 symptoms (Buysse, 2013; Roth, 2007; USDHHS, 2004). 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 physical and psychological 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).

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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 (i.e., difficulty initiating or maintaining sleep persisting for at least 6 months; Riedel, Durrence, Lichstein, Taylor, & Bush, 2004). Furthermore, available data suggest the relation between insomnia and smoking is complex and bi-directional (Wetter & Young, 1994), such that smoking may contribute to insomnia, and insomnia may promote continued smoking. For example, among smokers, tobacco withdrawal 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., 2017). Indeed, smokers who report co-occurring night smoking and significant sleep disturbance are at greater risk for smoking post-quit compared to smokers with neither risk factor (Peters, Fucito, Novosad, Toll, & O'Malley, 2011). Furthermore, existing work has found sleep disturbance may contribute to negative smoking outcomes after a quit attempt independent of sleep disturbances related to tobacco withdrawal (Augustson et al., 2008). Despite the interconnection between insomnia and smoking, surprisingly little empirical work has explored mechanisms underlying their association.

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 (Yoo, Gujar, Hu, Jolesz, & Walker, 2007).

Theoretically, smokers experiencing insomnia symptoms 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 aversive emotional symptoms (Paulus, Bakhshaie 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). Indeed, emotion dysregulation is associated with early smoking lapse (Farris, Zvolensky, & Schmidt, 2016) among treatment-seeking smokers and is a mechanism that links negative emotional states to problematic substance use (e.g., symptoms of dependence; Paulus, Bakhshaie, et al., 2016; Paulus, Jardin, et al., 2016). 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 >24 h among treatment-seeking smokers (Fillo et al., 2016).

Based upon theoretical models of emotion dysregulation and substance use (Leventhal & Zvolensky, 2015) and the findings of Fillo et al. (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 as a global construct indirectly accounts for the relation between insomnia and negative reinforcement smoking processes, such as anticipated negative reinforcement smoking outcome expectancies (Brandon & Baker, 1991), negative reinforcement smoking motives (McCarthy, Curtin, Piper, & Baker, 2010), and two negative expectancies from brief (24-hour) smoking abstinence (somatic symptoms and harmful consequences; Abrams, Zvolensky, Dorman, Gonzalez, & Mayer, 2011). Furthermore, past work has highlighted smoking outcome expectancies and smoking motives as related although distinct constructs (Ikard, Green, & Horn, 1969). Specifically, negative reinforcement smoking outcome expectancies relates to *beliefs* that smoking will help an individual cope with emotional distress (Brandon & Baker, 1991). Conversely, negative reinforcement smoking motives relates to an individual's *motivation* to smoke for reductions in emotional distress (Piper et al., 2004). Thus, work devoted to independently examining these unique negative reinforcement constructs is warranted to better understand the nature of smoking behavior.

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.

Together, the current study tested the hypothesis that insomnia would exert an indirect effect on negative reinforcement smoking processes via emotion dysregulation (see Fig. 1). Specifically, insomnia was expected to be positively associated with emotion dysregulation, which, in turn, would be associated with smoking criterion variables. In the current study, four clinically significant criterion 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.

2. Materials and method

2.1. Participants

Participants were 126 adult daily non-treatment seeking smokers (55 females; $M_{age} = 44.1$ years, $SD = 9.72$). The racial composition of