Emotion dysregulation explains relations between sleep disturbance and smoking quit-related cognition and behavior

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HIGHLIGHTS

- Emotion dysregulation's association between sleep disturbance and smoking was tested
- Sleep disturbance exerted a significant indirect effect through emotion dysregulation
- Increased sleep disturbance may have detrimental effects on emotion dysregulation

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ABSTRACT

Poor sleep quality and tobacco use are common and co-occurring problems, although the mechanisms underlying the relations between sleep disturbance and smoking are poorly understood. Sleep disturbance lowers odds of smoking cessation success and increases odds of relapse. One reason may be that sleep loss leads to emotion dysregulation, which in turn, leads to reductions in self-efficacy and quit-related problems. To address this gap, the current study examined the explanatory role of emotion dysregulation in the association between sleep disturbance and smoking in terms of (1) self-efficacy for remaining abstinent in relapse situations, (2) the presence of a prior quit attempt greater than 24 h, and (3) the experience of quit-related problems among 128 adults (Mage = 40.2; SD = 11.0; 52.3% female) seeking treatment for smoking cessation. Results suggested that increased levels of sleep disturbance are related to emotion dysregulation which, in turn, may lead to lower levels of self-efficacy for remaining abstinent, more quit-related problems, and being less likely to have had a quit attempt of 24 h or greater. Further, these indirect effects were present above and beyond variance accounted for by theoretically-relevant covariates (e.g., gender and educational attainment), suggesting that they may maintain practical significance. These findings suggest that this malleable emotional risk factor (emotion dysregulation) could serve as a target for intervention among those with poor sleep and tobacco use.

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1. Introduction

Cigarette smoking remains the leading cause of death and disability in the United States (U.S. Department of Health and Human Services [USDHHS], 2014). Between 2005 and 2009, smoking was responsible for over 480,000 premature deaths a year. Although smoking has declined significantly since 1964, large disparities in tobacco use remain across a number of groups (USDHHS, 2014). One such group includes individuals with comorbid health behavior problems and psychiatric symptoms and disorders (Centers for Disease Control and Prevention [CDC], 2013).

One highly common problem among smokers is insomnia. Indeed, high prevalence rates of insomnia (i.e., difficulty initiating or maintaining sleep and/or unsatisfying sleep) and low sleep quality among
smokers (Riedel, Durrence, Lichstein, Taylor, & Bush, 2004; Wetter & Young, 1994) represents both a common problem and a potentially modifiable barrier to smoking cessation. Smokers suffer from insomnia and other sleep problems at higher rates than non-smokers (Riedel et al., 2004; Wetter & Young, 1994), with objective evidence of shorter total sleep time, longer sleep onset latency, and greater time spent awake during the night (Jahne et al., 2012; Soldatos, Kales, Scharf, Bixler, & Kales, 1980; Zhang, Samet, Caffo, & Punjabi, 2006). Indepen-
dently, insomnia is associated with a host of physical and psychological problems, and quality of life among insomniacs is even poorer than among patients with congestive heart failure (Katz & McHorney, 2002). Among those who smoke, there is mounting evidence that the presence of sleep disturbance lowers odds of smoking cessation success and increases odds of relapse (Boutou et al., 2008; Bover, Foulds, Steinberg, Richardson, & Marcella, 2008; Foulds et al., 2006; Hamidovic & de Wit, 2009; Scharf, Dunbar, & Shiffman, 2008). Contextual and mechanistic factors that specifically link sleep and smoking behavior nonetheless remain poorly understood.

Emotion dysregulation is one promisingintegrative, emotion-based construct for bridging gaps in our knowledge and understanding of relations between sleep and smoking behavior. Emotion dysregulation refers to difficulties in both the self-regulation of affective states and self-control over affect-driven behaviors (Mennin, Heinberg, Turk, & Fresco, 2005). Across a range of populations, emotion dysregulation (as a higher-order factor) is related to increased levels of negative affect (Brandt, Zvolensky, & Bonn-Miller, 2013; Vujanovic, Zvolensky, & Bernstein, 2008), more avoidance-oriented coping in response to life stress (Bonn-Miller, Vujanovic, & Zvolensky, 2008), and lower self-efficacy for health behavior (Rellini, Zvolensky, & Rosenfeld, 2012). Emotion dysregulation is related to a longer history of smoking and greater attentional bias to smoking cues (Pucilas, Juliano, & Toll, 2010). In addition, experimental research has shown that instructing participants to use maladaptive emotion regulation strategies (e.g., suppression) results in increased cravings, negative affect, and attentional biases towards smoking cues compared to individuals instructed to use more effective strategies (e.g., cognitive reappraisal; Szasz, Szentagotai, & Hofmann, 2012). Emotion dysregulation may also explain the relation between negative affect symptoms and coping-oriented smoking (Short, Raines, Oglesby, Zvolensky, & Schmidt, 2014). One study found emotion dysregulation explained the relation between threat sensitivity and smoking-based cognitive processes (Johnson, Farris, Schmidt, & Zvolensky, 2012). These data collectively point to the potentially important role of emotion dysregulation in a wide array of clinical correlates of smoking behavior.

Within the sleep literature, evidence for the influence of sleep on emotion regulation continues to mount. In general, sleep loss increases the occurrence of negative emotions, reduces the occurrence of positive emotions, and alters the ways in which individuals understand, express, and modify their emotions (Kahn, Sheples, & Sadeh, 2013; Walker & van der Helm, 2009). At a neurobiological level, decreased connectivity between frontal brain regions (e.g., medial prefrontal cortex) and emotion-based structures (e.g., amygdala) following periods of sleep deprivation is suggestive of broad-based problems with regulatory control (Motomura et al., 2013; Yoo, Hu, Gujar, Jolesz, & Walker, 2007). For example, individuals who are sleep deprived are more likely to make inappropriate comments, make irrational social decisions, take greater risks, have difficulty delaying gratification, and disregard potential negative consequences (Christian & Ellis, 2011; Harrison & Horne, 1998; Killgore et al., 2008).

Smokers who experience greater levels of sleep disturbance may be prone to experience greater levels of emotion dysregulation. As a result, in the absence of alternative adaptive regulatory strategies, smoking may be used to manage negative mood states in the short term. However, it may ultimately result in shorter or less successful quit attempts due in part to the experience of more distressing symptoms during periods of abstinence. Within this framework, there would presumably be negative effects on self-efficacy, as an individual’s beliefs about their ability to successfully maintain abstinence would be compromised. A formative next step is therefore to evaluate whether emotion dysregulation explains the association between sleep disturbance and aspects of smoking behavior that are associated with failed smoking cessation attempts. With this background, the current study tested the hypotheses that, among adult, treatment-seeking daily smokers, emotion dysregulation would explain the relation between sleep disturbance and: (1) lower levels of self-efficacy for remaining abstinent, (2) reduced probability of a prior quit attempt longer than 24 h, and (3) more quit-related problems (see Fig. 1).

2. Method

2.1. Participants

Data were collected as part of a larger randomized controlled trial examining the efficacy of two smoking cessation interventions (Smits et al., 2015). Between January 2010 and July 2014, 136 participants were recruited from the Dallas community and attended a baseline visit. Prior to enrollment, participants provided written informed consent and completed screening consisting of questionnaires, a diagnostic interview (using the Structural Clinical Interview for DSM-IV-TR Axis I Diagnoses, Research Version, Non-Patient Edition [SCID]; First, Spitzer, Gibbon, & Williams, 2002), and a medical examination comprising a physical exam, laboratory work, and maximal exercise testing. Eligible participants met the following criteria at prescreen: (1) adult daily smokers (at least 1 year of smoking a minimum of 10 cigarettes per day); (2) elevated anxiety sensitivity (prescreen score of ≥2.0 on the 16-item Anxiety Sensitivity Index; Reiss, Peterson, Gursky, & McNally, 1986); (3) sedentary (moderate-intensity exercise less than twice a week for 30 min or less); and (4) motivated to quit (reporting a motivation of at least 5 on a 10-point scale). A comprehensive list of exclusion criteria and screening procedures is provided in the study protocol (Smits et al., 2012). For the current study, data from 128 individuals (52.3% female; Mage = 35.2 years, SDage = 15.4 ppm; Mheight = 168 cm, SDheight = 8.2 cm; McGib = 18.9 years of age (SD = 5.1)). Moreover, participants endorsed moderate levels of nicotine dependence, as indexed by an average score of 5.3 (SD = 2.0) on the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) as well as biological verification, determined via baseline expired carbon monoxide (CO; M = 15.4 ppm; SD = 8.2). Most participants (88.0%) reported making at least one previous attempt to quit smoking, endorsing an average of 3.8 (SD = 2.8) ‘serious’ lifetime quit attempts. Less than half (39.8%) of the sample met criteria for at least one current psychological disorder per the SCID-NP with an average of 2.0 (SD = 1.1) diagnoses among those with psychopathology. The most common diagnoses were major depressive disorder (11.7%), alcohol use disorder (10.2%), and specific phobia (10.2%); full diagnostic breakdown (current diagnoses) for the sample is presented in Table 1.

2.2. Procedure

The study was performed after approval by the institutional review board at Southern Methodist University. Written informed consent...
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