Addictive Behaviors 57 (2016) 6–12

Contents lists available at ScienceDirect

Addictive Behaviors

journal homepage: www.elsevier.com/locate/addictbeh

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

ARTICLE INFO

Article history:
Received 18 January 2016
Accepted 22 January 2016
Available online 23 January 2016

Keywords:
Smoking
Sleep
Smoking cessation
Emotion Dysregulation

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 (M_{age} = 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.

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

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http://dx.doi.org/10.1016/j.addbeh.2016.01.013
0306-4603/

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 (Jaehne et al., 2012; Soldatos, Kales, Scharf, Bixler, & Kales, 1980; Zhang, Samet, Caffo, & Punjabi, 2006). Independently, 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 promising integrative, 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, Heimberg, 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, & Rosenfield, 2012). Emotion dysregulation is related to a longer history of smoking and greater attentional bias to smoking cues (Fucito, 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, Sheppes, & Sadeh, 2013; Walker & van der Helm, 2009). At a neurobiological level, decreased connectivity between frontal brain regions (e.g., medial pre frontal 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 ≥20 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; $M_{age} = 40.2$ [$SD = 11.0$]) were available. Pairwise deletion was utilized resulting in 117–125 participants depending on analysis.

Participants had achieved a wide range of education levels; 10.2% had completed graduate school, 3.0% had completed college degrees, 35.2% had completed some college, 17.2% were high school graduates, 3.9% completed some high school, and 1.6% had completed junior high school. Most participants (73.4%) were White, 20.3% were Black/African American, 2.3% were Asian, 3.1% reported “other”, and 0.8% chose not to respond. Additionally, 8.6% of participants identified as Hispanic or Latino. Participants reported smoking an average of 19.4 cigarettes per day ($SD = 9.7$), having smoked their first cigarette at 16.2 years of age ($SD = 4.9$), and beginning to smoke regularly at 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

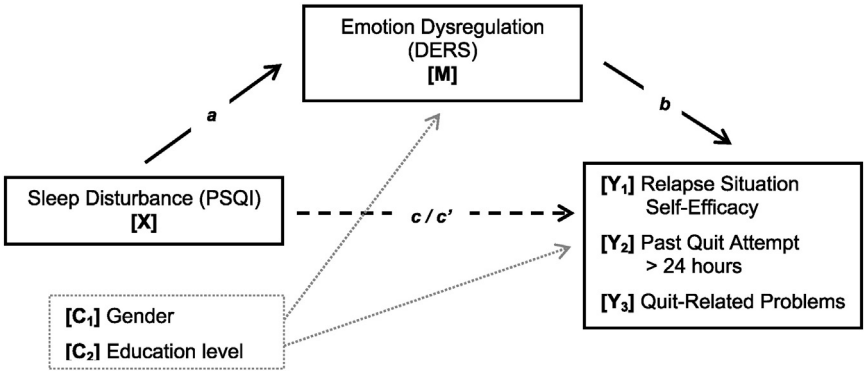

Fig. 1. Conceptual mediation model. Note. a = effect of X on M; b = effect of M on Y_i; c' = direct effect of X on Y_i controlling for M; $a * b$ = indirect effect of X on Y_i through M; c = total effect of X on Y (direct and indirect).

was obtained from each participant at intake. Participants were recruited through the community and physician referrals. Recruitment techniques included post-paid newspaper, radio advertisements, fliers in community-based organizations, and internet-based advertising, including our laboratory website and Craigslist. Participants were compensated $25 for attending the baseline assessment.

Study eligibility was assessed through (1) an initial prescreen (both online and via telephone), and (2) an in-person psychiatric diagnostic screening visit. Eligible participants completed the baseline assessment within 3 weeks of completing their screening visits.

2.3. Measures

2.3.1. Demographics questionnaire

Demographic information collected included gender, age, race, and highest level of education attained.

2.3.2. Structured Clinical Interview—Non-Patient Version for DSM-IV (SCID-I/NP; First et al., 2002)

Diagnostic assessments of past year Axis I disorder were conducted using the SCID-I/NP, which was administered by doctoral level staff or trained research assistants and supervised by licensed clinical psychologists.

Table 1
Current psychopathology among sample.

Diagnosis	Number	Percentage
Major depressive disorder	15	11.7%
Alcohol use disorder	13	10.2%
Specific phobia	13	10.2%
Cannabis use disorder	11	8.6%
Social anxiety disorder	11	8.6%
Stimulant use disorder (cocaine)	7	5.5%
Panic disorder	6	4.7%
Agoraphobia	4	3.1%
Generalized anxiety disorder	4	3.1%
Obsessive–compulsive disorder	3	2.3%
Post-traumatic stress disorder	3	2.3%
Phencyclidine use disorder	2	1.6%
Sedative use disorder	2	1.6%
Binge eating disorder	1	0.8%
Cyclothymic disorder	1	0.8%
Health anxiety	1	0.8%
Substance-induced depressive disorder	1	0.8%
Depressive disorder due to another medical condition	1	0.8%
Persistent depressive disorder/dysthymia	1	0.8%
Stimulant use disorder (amphetamine)	1	0.8%

Note. Diagnoses determined by SCID.

2.3.3. Smoking History Questionnaire (SHQ; Brown, Lejuez, Kahler, & Strong, 2002)

The SHQ is a self-report questionnaire used to assess smoking history (e.g., onset of daily smoking) and pattern (e.g., smoking rate). In the present study, the SHQ was used to describe the sample on smoking history and patterns of use for each individual. Several items were also used as outcome variables indexing (1) the presence of a past quit attempt of at least 24 h and (2) the experience of quit-related problems. Past quit attempt information was obtained via the item, "*Since you started smoking regularly, have you had a quit period of at least 24 hours*?" Participants responded yes (1) or no (0). Additionally, in line with prior work (e.g., Zvolensky, Farris, Leventhal, Ditre, & Schmidt, 2015; Zvolensky, Farris, Schmidt, & Smits, 2014), participants' experience of problems during prior quit attempts was evaluated using 16 items (e.g., "nausea", or "craving for tobacco") that were rated on a scale from 1 (*not at all*) to 5 (*extremely*). Quit-related problems were averaged to create a composite score, with higher scores indicating greater quit-related problems (Cronbach's $\alpha = .91$ in the present sample).

2.3.4. Fagerström Test for Nicotine Dependence (FTND; Fagerström, 1978)

The FTND is a 6-item scale that assesses an individual smoker's "nicotine dependence" (Heatherton et al., 1991). Total scale scores range from 0–10, with higher scores reflecting a greater level of nicotine dependence. The FTND has shown adequate internal consistency ($\alpha = .61$), positive relations with key smoking variables (e.g., saliva cotinine), and high test–retest reliability (Heatherton et al., 1991). In the current sample, the FTND total score internal consistency was low (Cronbach's $\alpha = .46$), which is not uncommon for this measure (see Korte, Capron, Zvolensky, & Schmidt, 2013).

2.3.5. Carbon monoxide

Biochemical verification of smoking status was completed by carbon monoxide (CO) analysis of breath samples. Expired air CO levels were assessed using a CMD/CO Carbon Monoxide Monitor (Model 3110; Spirometrics, Inc., Auburn, ME).

2.3.6. Relapse Situation Self-Efficacy (RSSE; Gwaltney et al., 2001)

Participants' current confidence regarding their ability to resist smoking under a variety of circumstances was measured using the 43-item RSSE. For each item, participants rated their confidence regarding their ability to resist smoking in a particular situation (e.g., "How confident are you that you can resist the temptation to smoke when you are where people are smoking?"). Each item was rated on a 4-point scale ranging from *1* (not at all confident) to *4* (extremely confident). Scores are averaged across all items to create a global score, with higher scores indicating greater relapse situation self-efficacy (Cronbach's $\alpha = .94$ in the present sample). The RSSE has demonstrated acceptable

Table 2
Descriptive statistics and correlations among study variables.

	1	2	3	4	5	6	7	*M* (*SD*) or *n* (%)
1. Gender (% male)	–							61 (47.7)
2. Years of education	−.10	–						2.86 (1.11)
3. PSQI	−.09	.04	–					7.27 (5.46)
4. DERS	.07	−.20*	.21*	–				1.99 (0.58)
5. RSSE	−.04	.05	−.10	−.30**	–			112.38 (32.93)
6. Past quit attempt > 24 h (% yes)	−.03	.02	−.02	−.17	−.16	–		61 (17.4)
7. Quit-related problems	.22*	−.10	.16	.36**	−.23*	−.16	–	37.53 (12.78)

Note. PSQI = Pittsburgh Sleep Quality Index (sleep disturbance component total score). DERS = Difficulties with Emotion Regulation Scale. RSSE = Relapse Situation Self-Efficacy. Gender: −1 = female, 1 = male.
* $p < .05$.
** $p < .01$.

psychometric properties including internal validity and has been shown to predict relapse (Gwaltney et al., 2001).

2.3.7. Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989)

The PSQI is a validated self-report measure of global sleep quality and specific aspects of sleep disturbance. The sleep disturbance component is comprised of 9 items regarding ways in which the respondent may have experienced trouble sleeping during the past month (e.g., "wake up in the middle of the night or early in the morning"). Each item is rated on a 4-point scale ranging from *0* (not during the past month) to *3* (three or more times a week). All items are summed to create a total sleep disturbance score, with higher scores indicating greater sleep disturbance (Cronbach's $\alpha = .78$ in the present sample).[1]

2.3.8. Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004)

The DERS is a 36-item self-report measure that assesses, on a 5-point Likert-type scale from 1 (*almost never*) to 5 (*almost always*), the degree to which respondents experience dysregulated emotional states (e.g., "I experience my emotions as overwhelming and out of control"). Items on the DERS yield a total score as well as six subscale scores: 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. The DERS demonstrates strong psychometric properties, including internal consistency and test–retest reliability, as well as construct and predictive validity (Gratz & Roemer, 2004; Whiteside et al., 2007). In the current study, the average total score was used as the proposed explanatory variable (Cronbach's $\alpha = .93$ in the present sample).

2.4. Data analytic strategy

The explanatory role of emotion dysregulation (DERS), in the relation between sleep disturbance (PSQI) and (1) relapse situation self-efficacy (RSSE), (2) a prior quit attempt longer than 24 h, and (3) problems experienced during past quit attempts was tested using ordinary least squares regression analyses, with bootstrapping to estimate the indirect effect of sleep disturbance on the continuous outcome measures (Hayes, 2009, 2013). Bootstrapping is a nonparametric resampling procedure (Preacher & Hayes, 2008). In all cases, 10,000 bootstrapped samples were used to generate a sampling distribution and a 95% confidence interval for the indirect effect; statistical significance of the indirect effect is determined by the absence of zero from the confidence interval. Logistic regression was used to estimate the indirect effect of sleep disturbance on the binary outcome measure (past quit attempt longer than 24 h). Linearity assumptions were met. Effect sizes (κ^2) were calculated for each indirect effect per recommendations of Preacher and Kelley (2011) as the squared ratio of the obtained indirect effect to the largest possible indirect effect that could have been obtained. This value can be interpreted in a similar manner as squared correlation coefficients, as outlined by Cohen (1988).

Participant gender and highest level of education were included as covariates in all analyses to adjust for the potential effects of these factors on primary dependent measures, as these variables often covary with sleep disturbance and smoking (e.g., Escobedo & Peddicord, 1996; Guindon & Boisclair, 2003).

3. Results

Descriptive data and correlations of the all variables included in the models are presented in Table 2. Sleep disturbance was significantly associated with emotion dysregulation ($r = .21$; 4% shared variance), but not the studied dependent variables. Emotion dysregulation was significantly negatively associated with relapse situation self-efficacy ($r = -.30$) and positively associated with problems experienced during past quit attempts ($r = .36$), but not a past quit attempt longer than 24 h ($r = -.17$).

3.1. Mediation analyses

The total effects model (see Table 3) predicting relapse situation self-efficacy revealed a non-significant effect of sleep disturbance (path c_1). Additionally, once the explanatory variable (emotion dysregulation) was added to the model, the direct effect was not significant (path c_1'). There was a significant indirect effect of sleep disturbance (path $a_1 * b_1$; $b = -0.24$, 95% CI [−0.53, −0.07]), such that sleep disturbance predicted lower relapse situation self-efficacy indirectly through greater emotion dysregulation. The size of the indirect effect was small to medium ($\kappa^2 = .06$). The variance in relapse situation self-efficacy explained by the model ($R^2 = .01$) was largely due to the indirect effect ($R^2 = .009$), accounting for 86% of the total explained variance.

The total effects model predicting a past quit attempt longer than 24 h revealed a non-significant effect of sleep disturbance (path c_2). After accounting for the explanatory variable, the direct effect was not significant (path c_2'). Once again, there was a significant indirect effect of sleep disturbance (path $a_2 * b_2$; $b = -0.02$, 95% CI [−0.07, <−0.01]), wherein sleep disturbance predicted the likelihood of making a quit attempt longer than 24 h indirectly through greater emotion dysregulation. The size of the indirect effect was large ($\kappa^2 = .31$). The variance in past quit attempt longer than 24 h explained by the model ($R^2 = .0005$) was largely due to the indirect effect ($R^2 = .0004$), accounting for 75% of the total explained variance.

The total effects model predicting problems experienced during past quit attempts revealed a significant effect of sleep disturbance (path c_3). Once the explanatory variable was added to the model, the direct effect of sleep disturbance was not significant (path c_3'). However, the indirect effect (path $a_3 * b_3$; $b = 0.15$, 95% CI [0.03, 0.38]) of sleep disturbance via

[1] All reported effects remain significant when analyses are conducted using the scaled (0–3) sleep disturbance component scores (instead total summed score).

Table 3
Bootstrap coefficients and confidence intervals for the unstandardized indirect effect of sleep disturbance on smoking cessation self-efficacy, quitting for more than 24 h, and quit-related problems.

Y	Model	*b*	*SE*	*t/Z*	*p*	Confidence interval		κ^2
						Lower	Upper	
1	PSQI → DERS (a_1)	0.88	.34	2.57	.011	0.20	1.56	
	DERS → RSSE (b_1)	−0.27	.09	−3.14	.002	−0.45	−0.10	
	PSQI → RSSE (c_1)	−0.39	.34	−1.15	.252	−1.07	0.28	
	PSQI → RSSE (c_1')	−0.15	.34	−0.45	.655	−0.82	0.52	
	PSQI → DERS → RSSE ($a_1 * b_1$)	−0.24	.11			−0.53	−0.07	.06
2	PSQI → DERS (a_2)	0.78	.36	2.20	.030	0.08	1.48	
	DERS → Past Quit Attempt > 24 h (b_2)	−0.03	.02	−1.75	.080	−0.06	<0.01	
	PSQI → Past Quit Attempt > 24 h (c_2)	−0.01	.05	−0.28	.780	−0.11	0.08	
	PSQI → Past Quit Attempt > 24 h (c_2')	0.01	.05	0.17	.864	−0.09	0.11	
	PSQI → DERS → Past Quit Attempt > 24 h ($a_2 * b_2$)	−0.02	.02			−0.07	> − 0.01	.31
3	PSQI → DERS (a_3)	0.78	.36	2.20	.030	0.08	1.48	
	DERS → Quit-Related Problems (b_3)	0.20	.06	3.42	<.001	0.08	0.31	
	PSQI → Quit-Related Problems (c_3)	0.48	.23	2.10	.038	0.03	0.93	
	PSQI → Quit-Related Problems (c_3')	0.33	.22	1.46	.146	−0.12	0.77	
	PSQI → DERS → Quit-Related Problems ($a_3 * b_3$)	0.15	.09			0.03	0.38	.06

Note. PSQI = Pittsburgh Sleep Quality Index (sleep disturbance component). DERS = Difficulties with Emotion Regulation Scale. RSSE = Relapse Situation Self-Efficacy. Criterion variables were: RSSE (Y_1; $n = 125$), Past Quit Attempt > 24 h (Y_2; $n = 117$), and Quit-Related Problems (Y_3; $n = 117$).

emotion dysregulation was significant. The size of the indirect effect was small to medium ($\kappa^2 = .06$). The variance in past quit attempt longer than 24 h explained by the model ($R^2 = .03$) was largely due to the indirect effect ($R^2 = .02$), accounting for 68% of the total explained variance.

3.2. Alternative models

We ran two sets of alternative models to provide further insight regarding the direction of the observed relations, as all variables were collected at one time point. In the first set of alternative models, the proposed predictor and mediator variables were reversed. There was no indirect effect of emotion dysregulation on relapse situation self-efficacy through sleep disturbance ($b = -0.01$, 95% CI [−0.07, 0.03]), no indirect effect of emotion dysregulation on quit attempt of 24 h or more through sleep disturbance ($b < 0.01$, 95% CI [−0.01, 0.01]), and no indirect effect of emotion dysregulation on quit-related problems through sleep disturbance ($b = 0.02$, 95% CI [−0.01, 0.06]); all reverse models were rejected.

In the second set of alternative models, the proposed outcome and mediator variables were reversed. There was no indirect effect of sleep disturbance on emotion dysregulation through relapse situation self-efficacy ($b = 0.11$, 95% CI [−0.07, 0.36]) and no indirect effect of sleep disturbance on emotion dysregulation via quit attempt of 24 h or more ($b = 0.12$, 95% CI [−1.17, 0.14]). However, there was a significant indirect effect of sleep disturbance on emotion dysregulation via quit-related problems ($b = 0.23$, 95% CI [0.05, 0.53]).

4. Discussion

The present study examined whether emotion dysregulation explains, in part, the relation between sleep disturbance and self-efficacy for remaining abstinent from smoking during relapse situations, a prior quit attempt longer than 24 h, and problems experienced during past quit attempts. Sleep disturbance exerted a significant indirect effect through emotion dysregulation for all dependent variables. These results are consistent with the hypothesis that increased levels of sleep disturbance may have detrimental effects on emotion dysregulation (the 'a' path) which, in turn, leads 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 (the '*b*' path). These results are also in line with past research that has indicated emotion dysregulation is related to numerous indicators of smoking severity (Gonzalez, Zvolensky, Vujanovic, Leyro, & Marshall, 2008). Namely, they suggest the effect of sleep disturbance on the smoking variables of interest in this study was indirect and dependent upon the degree to which individuals have difficulty regulating their emotional states.[2] Indeed, sleep disturbance did not have a statistically significant direct effect on any of the dependent variables and was not significantly associated with any of the dependent variables at the bi-variate level. Although traditional methods of mediation have considered a correlation between the 'X' and 'Y' variables a prerequisite to the study of mediation (Baron & Kenny, 1986), modern theories of mediation make clear that prerequisite is not necessary (e.g., Hayes, 2013; Kenny, 2013; Rucker, Preacher, Tormala, & Petty, 2011). The indirect effects were significant for all dependent variables studied.

The clinical significance of the current findings is potentially noteworthy for numerous reasons. First, the sizes of the effects were small to medium for relapse situation self-efficacy and quit-related problems and large for a prior quit attempt of 24 h or greater. These effect sizes were estimated with κ^2, which estimates the size of the indirect effect. Further, despite small R^2 for the dependent variables (i.e., the variance in 'Y' accounted for *only* by 'X'), the indirect effects accounted for a larger percentage of this variance (68–86%), relative to direct effects. Thus, the observed effects were empirically meaningful using traditional indicators of effect size. Second, the observed indirect effects were evident after adjusting for the influence of factors known to correlate with the severity of smoking behavior, including gender and educational level (Escobedo & Peddicord, 1996; Guindon & Boisclair, 2003). These data suggest that emotion dysregulation offers unique explanatory value in regard to the studied dependent measures. Given the range of factors modeled in this preliminary study, it is noteworthy that the models' predictive power were consistently observed (Abelson, 1985). Indeed, this is a potentially clinically important finding and it supports a central prediction from the model; namely, 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. Finally, there was generally consistent evidence for the hypothesized direction of the relations. Testing an alternative model with emotion dysregulation as the predictor and sleep disturbance as the

[2] The power to detect the indirect effect (path '$a * b$') can be obtained analytically as the power to detect an effect for the '*a*' path multiplied by the power to detect an effect for the '*b*' path (Fritz & MacKinnon, 2007). In the current study, there was adequate power ($1 - \beta$) of 0.972–0.980 to detect an indirect effect. Furthermore, despite potential concerns about low power to detect an effect in a relatively small sample ($n = 177$–125), significant indirect effects *were present* in all models.

mediator, we found no significant indirect effect in predicting any of the criterion variables. In the second set of alternative models, there was no significant indirect effect for relapse situation self-efficacy or prior quit attempt of more than 24 h, whereas there was an indirect effect for quit-related problems. Together, these alternative models suggest that there are likely bi-directional relations between these variables, particularly for quit-related problems, which showed a significant indirect effect in both the hypothesized model and one of the alternative models. To more fully explore the nature of relations among these variables over time and further probe the practical significance of the models tested, future prospective modeling of the temporal ordering of sleep disturbance and emotion dysregulation in relation to smoking is warranted.

Clinically, the findings from the present investigation may serve to conceptually inform the development of specialized intervention strategies for smokers with sleep disturbance. Specifically, among smokers with sleep disturbance, it may be advisable to understand and clinically address emotion dysregulation in order to enhance psychological flexibility related to smoking, address maladaptive smoking cognitions (e.g., "I need a cigarette to cope"), and facilitate change in smoking behavior. Acceptance-based techniques (e.g., experiential awareness, openness, willingness, mindfulness, cognitive diffusion) have been shown to reliably reduce emotion dysregulation (McCallion & Zvolensky, 2015) and may be useful in this regard. Likewise, based on established direct links between inadequate/poor quality sleep and deficits in emotion regulation (Kahn et al., 2013; Walker & van der Helm, 2009), behavioral interventions targeting smoking might consider incorporating strategies for improving sleep health.

There are a number of interpretive caveats to the present study that warrant further consideration. First, given the cross-sectional nature of these data, it is unknown whether sleep disturbance is causally related to greater emotion dysregulation or to the smoking variables. The present tests were solely based on a theoretical framework and 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, treatment-seeking 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, the sample was largely comprised of a relatively homogenous group of treatment-seeking smokers. To rule out a selection bias and increase the generalizability of these findings, it will be important for future studies to recruit a more ethnically/racially diverse sample of smokers. 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. Fourth, the models tested were unable to account for method-based measurement error. As such, results must be interpreted in light of error associated with mon-method measurement. Finally, assessment of sleep patterns using objective measures (e.g., actigraphy) would provide a more accurate understanding of sleep than subjective accounts. Future research would benefit by employing a multi-method assessment approach to cross-index the nature of the relations observed in the current report.

Overall, the present study serves as an initial investigation into the nature of the associations between sleep disturbance, emotion dysregulation, and smoking. Future work is needed to explore the extent to which emotion dysregulation accounts for relations between sleep disturbance and other smoking processes (e.g., withdrawal, cessation outcome) to further clarify theoretical models of sleep disorders, emotional vulnerability, and smoking.

Protocol

The full trial protocol can be found at pubmed.gov; PMCID: PMC3522063.

Funding

This study was funded by grants from the National Institute on Drug Abuse: R01DA027533 (awarded to Drs. Zvolensky and Smits; MPI) and K01DA035930 (awarded to Dr. Powers; PI).

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