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Individual differences in emotion dysregulation and trajectory of withdrawal symptoms during a quit attempt among treatment-seeking smokers

Andrew H. Rogers[a], Jafar Bakhshaie[a], Lorra Garey[a], Thomas M. Piasecki[b], Matthew W. Gallagher[a,c], Norman B. Schmidt[d], Michael J. Zvolensky[a,e,*]

[a] Department of Psychology, University of Houston, Houston, TX, USA
[b] Department of Psychological Sciences, University of Missouri, Columbia, MO, USA
[c] Texas Institute for Measurement, Evaluation, and Statistics, University of Houston, Houston, TX, USA
[d] Department of Psychology, Florida State University, Tallahassee, FL, USA
[e] Department of Behavioral Sciences, University of Texas MD Anderson Cancer Center, Houston, TX, USA

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ABSTRACT

Objective: Cigarette smoking remains the leading preventable cause of death in the United States, and withdrawal symptoms are central to the maintenance of tobacco use. Previous research suggests that individual differences in the propensity to experience negative affect may be related to more severe withdrawal symptoms. However, little research has examined how individual differences in the ability to regulate affect (emotion dysregulation) may impact withdrawal symptoms over time.

Method: Therefore, the current study examined the effects of emotion dysregulation on change in tobacco withdrawal symptoms over 12 weeks following a cigarette quit attempt among 188 (M_{age} = 38.52, SD = 14.00, 46.8% male) treatment seeking smokers.

Results: Results from the study indicated greater emotion dysregulation was associated with greater quit day withdrawal symptoms as well as with as slower decline in withdrawal symptoms over the 12-week period (B = −0.001, SE = 0.001, p = .046).

Conclusion: The current study offers novel evidence into the role of emotion dysregulation in relation to withdrawal symptoms during a quit attempt. Assessing and reducing heightened emotion dysregulation prior to a quit attempt may be a potentially important therapeutic tactic for helping smokers achieve greater success in managing tobacco withdrawal.

Public health significance statement: This study emphasizes the ways in which emotional dysregulation may affect tobacco withdrawal symptoms. This study can be utilized to further target smoking cessation programs for those attempting to quit smoking.

1. Introduction

Cigarette smoking remains the leading preventable cause of death in the United States (U.S. Department of Health and Human Services, 2014). Tobacco withdrawal, conceptualized as the physiological and psychological consequences that occur following cigarette deprivation (Ashare et al., 2016; Benowitz, 2010; Hughes, Higgins, & Hatsukami, 1990), has been implicated as a central construct in the maintenance of tobacco use and identified as a robust risk factor for relapse among smokers attempting to quit (Aguirre, Madrid, & Leventhal, 2015; Hughes, 1992; West, Hajek, & Belcher, 1989). Indeed, a large body of work has sought to clarify the role of withdrawal symptoms in relapse (e.g. Hughes, 1992; Piper, 2015). For example, researchers have sought to explicate withdrawal symptoms across specific periods of time following tobacco deprivation (Pomerleau, Mehringer, Marks, Downey, & Pomerleau, 2000). This work has found that there is a gradual emergence of withdrawal symptoms following a quit attempt, with early signs occurring rapidly (e.g., minutes after the last cigarette smoked; Hendricks, Ditre, Drobes, & Brandon, 2006; Jarvik et al., 2000; Schuh & Stitzer, 1995). Other studies have indicated that the trajectory of tobacco withdrawal is associated with relapse (e.g., steeper increase related to increased odds of relapse; Piasecki, Jorenby, Smith, Fiore, &

* Corresponding author. Dept. of Psychology, 3695 Cullen Blvd., Room 126, University of Houston Houston, TX, 77204, USA.
E-mail address: mjzvolen@central.uh.edu (M.J. Zvolensky).

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Baker, 2003). Tobacco withdrawal has also been identified as a key factor for clinically significant distress among individuals who stop smoking, suggesting the need to understand factors associated with withdrawal (Hughes, 2006). Additionally, across studies, the negative affect component of tobacco withdrawal, in particular, has frequently been rated as the most problematic and directly related to poorer cessation outcomes (Marshall, Johnson, Bergman, Gibson, & Zvolensky, 2009; Piasecki et al., 2000; Piasecki, Kenford, Smith, Fiore, & Baker, 1997).

There is a large body of literature that suggests individual differences in the propensity to experience negative affect states is associated with more severe tobacco withdrawal (Piasecki et al., 1997). More recent research has extended this to suggest that these negative affect states are specifically associated with negative affect tobacco withdrawal symptoms (anger, anxiety), and these symptoms are critical in assessing risk of lapse and relapse (Piper et al., 2011). For example, greater depressive symptoms are significantly related to emotionally-laden tobacco withdrawal symptoms (e.g., depressed mood; Breslau, Kilbey, & Andreski, 1992; Pomerleau et al., 2000) and elevated anxiety-related symptoms and syndromes (e.g., history of panic attacks) are often associated with hyperarousal withdrawal symptoms (e.g., anxiety, irritability; Breslau et al., 1992; Pomerleau et al., 2000; Zvolensky, Lejuez, Kahler, & Brown, 2004). Such clinical research is consistent with experimental tests that document state-like differences in negative affect are related to more severe tobacco withdrawal (Morrell, Cohen, & al'Absi, 2008).

Although available research has indicated that individual differences in negative affect is central to understanding negative affect-related tobacco withdrawal (and ultimately lapse and relapse), scholarly work has increasingly suggested that the underlying *mechanisms* for experiencing negative affect across emotional symptoms and disorders may be a set of (smaller) transdiagnostic vulnerability factors (Leventhal & Zvolensky, 2015; Sauer-Zavala et al., 2012; Zvolensky & Leventhal, 2016). These transdiagnostic vulnerabilities could play a key explanatory role in shaping emotional experiences by enhancing or diminishing a normative response to emotion stimuli and states (Leventhal & Zvolensky, 2015). For example, anxiety sensitivity (fear of anxious arousal) is thought to amplify the perception of physical sensations, leading to withdrawal symptoms to be more distressing and experienced more intensely (Leventhal & Zvolensky, 2015). As such, these processes reinforce the intensity and frequency of future emotional states and exert their influence on emotional symptoms and disorders via their interaction with emotion stimuli, ultimately resulting in a distorted perspective of future emotional experience. For example, when faced with a frustrating experience (e.g., trying to quit smoking), individuals who have a diminished capacity to tolerate frustration should theoretically be more apt to engage in "quick fix" behaviors, such as smoking, that mitigate acute experiential distress, but contribute to low quit success and overall difficulty quitting (Zvolensky & Hogan, 2013).

Several studies have examined transdiagnostic factors in terms of tobacco withdrawal. There is evidence that neuroticism (the tendency to experience negative emotions; Matthews, Deary, & Whiteman, 2003), anxiety sensitivity (fear of anxiety; McNally, 2002), anhedonia (diminished pleasure in response to naturally rewarding stimuli; American Psychiatric Association, 2013), mindful attention (attention and awareness of what is occurring in the present moment; Brown & Ryan, 2003), and distress intolerance (perceived or objective capacity to tolerate distress; Leyro, Zvolensky, & Bernstein, 2010) are related to more severe withdrawal symptoms (Bakhshaie et al., 2018; Langdon et al., 2013; Paulus, Langdon, Wetter, & Zvolensky, 2017). Furthermore, the available literature, although limited in scope, suggests that reducing these transdiagnostic constructs before a quit attempt can produce a faster decrease in withdrawal symptoms following a quit attempt (Bakhshaie et al., 2016; Bakhshaie, Zvolensky, Langdon, Leventhal, & Schmidt, 2017).

Although past work on transdiagnostic factors and tobacco withdrawal is theoretically promising and clinically important (Leventhal & Zvolensky, 2015), there has been little research focused on emotion dysregulation. Emotion dysregulation, defined as dysfunction in one of six domains (emotional awareness, emotional acceptance, emotional clarity, ability to engage in goal-directed behavior, inhibiting impulsive behaviors, flexible use of emotion regulation strategies, and willingness to experience negative emotions; Gratz, 2007), has been implicated in a wide array of psychiatric disorders, including mood, personality, anxiety, eating, and substance use disorders (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Hayes, Wilson, Gifford, Follette, & Strosahl, 1996; Mennin, 2004).

In terms of smoking, research suggests that emotion dysregulation is related to negative affect smoking motives and expectancies (Rogers et al., 2018), greater attentional bias to smoking cues (Fucito, Juliano, & Toll, 2010), increased craving to smoke (Szasz, Szentagotai, & Hofmann, 2012), and decreased quit success (Farris, Zvolensky, & Schmidt, 2016). Other work has found that emotion dysregulation serves as one explanatory factor for the relation between emotional vulnerability factors (e.g., anxiety sensitivity) and expectancies for smoking to relieve negative affect, smoking to reduce negative affect, and barriers to smoking cessation (Johnson et al., 2008; Johnson & McLeish, 2016). These studies suggest emotion dysregulation may be important individual difference factor for better understanding smoking maintenance and relapse because difficulty regulating affect would theoretically increase the likelihood of experiential distress (e.g., withdrawal, negative affect) and subsequently lead to smoking to reduce such distress (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004).

However, research has yet to examine whether emotion dysregulation may be related to tobacco withdrawal following a quit attempt. This gap limits efforts to directly link emotion dysregulation to tobacco withdrawal. Indeed, this question is clinically important, as identifying individual difference factors that prospectively influence tobacco withdrawal severity following a quit attempt may help identify those at increased risk of relapse and thus inform more personalized treatment efforts to promote sustained abstinence. Smokers with greater emotion dysregulation may experience greater withdrawal following a quit attempt because they are unable to regulate their response to these symptoms and therefore the symptoms are perceived as more intense and interfering. For example, following a quit attempt, smokers with greater emotion dysregulation may evince higher quit day withdrawal symptoms because they lack the cognitive flexibility or skillset to respond adaptively to the symptoms. Subsequently, the compounded effect of withdrawal and difficulty managing the symptoms may promote greater withdrawal symptoms on quit day and interfere with one's ability to decrease (or regulate) withdrawal symptoms over time. Indeed, this slower decline may be related to the continuing difficulties regulating affect throughout the post-quit period, as research has found that emotional dysregulation is related to ongoing challenges in regulating emotional states (Ehring & Quack, 2010; Putnam & Silk, 2005).

Overall, the current study sought to examine individual differences in emotion dysregulation in terms of the trajectory of change in withdrawal symptoms during the first three months following a quit attempt among treatment-seeking daily smokers. It was hypothesized that more difficulty regulating emotions would predict greater quit-day withdrawal symptoms, and that these smokers would evince a slower decline in withdrawal throughout the follow-up period (i.e., lower deceleration rate).

2. Method

2.1. Participants

Participants (n = 188; M_{age} = 38.52, SD = 14.00; 46.8% male) were treatment-seeking daily smokers enrolled in a clinical trial evaluating the efficacy of a transdiagnostic smoking cessation treatment,

focused on anxiety sensitivity, relative to a standard smoking cessation treatment (Schmidt, Raines, Allan, & Zvolensky, 2016; Zvolensky et al., 2018). Eligibility criteria for the trial included: (1) 18–65 years of age; (2) being a daily smoker for at least 1 year; (3) currently smoking a minimum of 8 cigarettes per day; and (4) motivation to quit smoking. Exclusion criteria for the trial included: (1) current use of pharmacotherapy for smoking cessation (except the nicotine patch, which was provided by the study); (2) limited mental competency and inability to provide informed, voluntary, written consent; (3) endorsement of current or past psychotic-spectrum symptoms; (4) current suicidality or homicidal ideation that pose an immediate danger; (5) history of significant medical condition that would limit study participation; and (6) planning to relocate within the next 6 months.

2.2. Procedure

Data for the present study were collected during a multi-site randomized controlled clinical trial examining the efficacy of two smoking cessation interventions described in detail elsewhere (Schmidt et al., 2016; Zvolensky et al., 2018). Interested persons responding to community-based advertisements (e.g., flyers, newspaper ads, radio announcements) contacted the research team and were provided with a detailed description of the study via phone. Participants were then screened for initial eligibility, and if eligible, scheduled for a baseline appointment. At the baseline appointment, participants provided written informed consent, were interviewed using the SCID-I/NP, and completed a computerized self-report assessment battery as well as biochemical verification of smoking status to evaluate eligibility criteria.

Participants deemed eligible for the larger trial were randomly assigned to active or control treatment. The two treatment conditions included: a 4-session cognitive-behavioral smoking cessation program with an added anxiety sensitivity reduction component (active; i.e., Panic-Smoking Program), or a standard cognitive-behavioral smoking cessation program (control). Both treatments took place over four, 90-min sessions occurring once per week. The Panic-Smoking Program (active) integrates interoceptive exposure, cognitive restructuring, and psychoeducation exercises developed for panic prevention and treatment programs with standard smoking cessation counseling. The Standard Cessation Program (control) includes only the smoking-related components of the Panic-Smoking Program as well as a review of general health information not specific to anxiety or smoking (to equilibrate contact time across the two conditions). Both treatment groups received nicotine replacement therapy via the transdermal nicotine patch that was initiated at treatment Session 4 (quit-day). Participants were offered the nicotine replacement therapy for up to 12 weeks post-quit. Therapy sessions were supervised by principal investigators (MJZ and NBS) and checked for treatment fidelity by independent reviewers. Participants completed post-treatment assessment at 1-, 2-, 4-, and 12-weeks post quit. Participants were compensated $12.50 for completing the baseline visit, an additional $25 if they completed all treatment sessions, and $15 for each follow-up assessment they completed. We included all participants regardless of quit status during the post quit period because past work suggests excluding those who do not quit can create a potentially under-representative sample (Hughes, 2007). The study protocol was approved by the Institutional Review Boards at the University of Vermont and Florida State University (clinicaltrials.gov # NCT01753141).

2.3. Measures

Smoking History Questionnaire (SHQ; R. A. Brown, Lejuez, Kahler, & Strong, 2002).The SHQ is a self-report questionnaire used to assess smoking history and pattern (e.g. smoking rate, age of onset of initiation). It has been successfully used in previous studies as a measure of smoking history (Zvolensky et al., 2004). The present study used the following variables from the SHQ to characterize the sample: average number of cigarettes smoked per day, age of onset of first cigarette, and age at onset of regular (daily) cigarette smoking. The SHQ was administered at baseline.

Fagerström Test for Cigarette Dependence (FTCD; Fagerström, 2012; Heatherton, Kozlowski, Frecker, & Fagerström, 1991). The FTCD is a well-established 6-item scale designed to assess gradations in tobacco dependence. The measure exhibits good internal consistency reliability, high degree of test-retest reliability (Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994), and positive relations with key smoking variables (Heatherton et al., 1991; Payne, Smith, McCracken, McSherry, & Antony, 1994). The FTCD total score was used as a covariate to account for variations in cigarette dependence. Although the internal consistency for the FTCD was low in the present sample (Cronbach's $\alpha = 0.54$), the alpha was similar to that reported in other work (Bakhshaie et al., 2017; Fillo et al., 2016; Korte, Capron, Zvolensky, & Schmidt, 2013).

Timeline Follow-Back (TLFB). TLFB was used to assess number of cigarettes smoked per day for the 30 days prior to each follow up assessment. Past work suggests that TLFB is a reliable indicator of past substance use, including cigarette use (Robinson, Sobell, Sobell, & Leo, 2014), and average cigarettes smoked 30 days prior to each follow up visit was used as a control variable in the repeated measures analyses to account for current smoking status during the follow up period.

Nicotine Replacement Therapy Monitoring Form. A researcher-generated form was employed to measure use of nicotine patch during the cessation period. This measure has been employed in past work to monitor nicotine replacement usage (Zvolensky et al., 2017). Participants were coded 0 for no NRT use and 1 for any NRT use during the first two weeks following cessation.

Difficulties with Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The DERS is a 36-item self-report measure assessing the degree to which participants have difficulty regulating negative emotional states, rated on a 5-point Likert scale ranging from 1 (*Almost never*) to 5 (*Almost always*). The DERS yields a total score as well as six subscale scores. The total score was used as the primary predictor variable because presently there is a highly limited basis upon which to hypothesize specific lower-order factors of the construct influence smoking and inconsistent evident of the lower-order factor structure (Bardeen, Fergus, & Orcutt, 2012; Fowler et al., 2014). The DERS total score showed good internal consistency (Cronbach's $\alpha = 0.94$) and has been successfully used in past smoking research (Rogers et al., 2018; Zvolensky et al., 2017).

Minnesota Nicotine Withdrawal Scale (MNWS; Hughes & Hatsukami, 1998). The MNWS was used to assess the experience of tobacco withdrawal symptoms over the last 24 h. Utilizing a 5-point Likert-type scale (0 = *none* to 4 = *severe*), respondents rated the degree to which symptoms (including craving, irritability/frustration or anger, anxiety, difficulty concentrating, restlessness, increased appetite, depressed or sad mood, and insomnia) were experienced. The MNWS has shown good validity and reliability in previous studies (Allen, Hatsukami, Christianson, & Nelson, 1996; Hughes, Gust, Skoog, Keenan, & Fenwick, 1991). Internal consistency for MNWS was good in the present sample (Cronbach's $\alpha = 0.81$, 0.87, 0.87, 0.86, 0.89, and 0.88 for pre quit (session 3), quit-day, week 1, week 2, week 4, and week 12 post-quit, respectively).

2.4. Analytic strategy

The present study examined the effects of baseline DERS total score as a predictor of withdrawal symptoms at quit day and change in post-quit withdrawal symptoms over time. Tobacco withdrawal symptoms were modeled using data collected at quit-day and over the post-quit follow-up assessments (1-, 2-, 4-, and 12-weeks post-quit). Preliminary examination of the mean growth in withdrawal over time for all participants yielded curvature, indicating a quadric relationship, and

therefore, a quadratic relationship with time is implied (Biesanz, Deeb-Sossa, Papadakis, Bollen, & Curran, 2004).

Four growth curve models were used to examine post-quit change in withdrawal symptoms from quit-day to post-quit assessments. Data were analyzed using multilevel linear modeling (MLM) in PROC MIXED procedure in SAS 9.4 software (SAS, Inc., Cary, NC). The estimation of missing data in the models was based on restricted maximum-likelihood estimation. The models included two levels, where repeated assessments across time (level-1; variables included time and average cigarettes smoked per day since last assessment) were nested within participants (level-2; variables included baseline DERS, gender, baseline FTCD total score, treatment condition, pre-quit (session 3) withdrawal symptoms, and use of nicotine replacement therapy. To allow for the time irregularity in the assessments, a time-structured predictor was included. Thus, the time values corresponded with the actual time-spaces (in weeks) between each follow-up assessment (Singer & Willett, 2003).

A fully unconditional random intercepts model (Model 1) was used to estimate the intraclass correlation (ICC) and design effect to ensure that the growth curve models were the best data analytic approach in the context of the current study. Second, an unconditional random slopes growth model (Model 2) including examined quit-day levels (intercept), linear change in withdrawal symptoms, and quadratic change in withdrawal symptoms (slopes) were used to examine the fit of the quadratic model. Third, a conditional random slopes model (model 3) with covariates only, including time-varying effects (average cigarettes smoked per day since last assessment) was conducted to predict quit-day levels (intercept) and change in withdrawal symptoms (slopes) as a function of known individual difference variables. Finally, a fully conditional random slopes model with all variables, including DERS total score (Model 4), was used to examine whether DERS total significantly predicted growth over and above the effects of statistical controls. Included in Model 4 was a time by DERS total score interaction to examine differences in trajectories of withdrawal symptoms over time. Covariates included have been used in past smoking research: cigarette dependence (Bakhshaie et al., 2017), gender (Leventhal et al., 2007), treatment condition (Hooten et al., 2014; Schmidt et al., 2016), use of nicotine replacement therapy (Silagy, Lancaster, Stead, Mant, & Fowler, 2004), pre-quit withdrawal symptoms (Shiffman, West, Gilbert, & SRNT Work Group on the Assessment of Craving and Withdrawal in Clinical Trials, 2004), and average number of cigarettes smoked since last assessment (Shiffman et al., 2004).

Following a significant interaction of the highest order product, the interaction was probed such that slopes for the mean DERS total score, +1 SD DERS total score, and −1 SD DERS total score.

In all models, continuous variables were grand mean centered, and time was centered at quit day. First-order autoregressive covariance matrices was used to account for repeated assessments closer in time to being more highly correlated (Gibbons et al., 1993). A random intercept and slope were modeled to allow for intraindividual variability in quit-day withdrawal and post-quit change in withdrawal symptoms. Although the study was concerned with the fixed effects included in the models, the variance and covariance of the random effects were modeled to account for the variability of the observed effects across individuals. The unstructured covariance matrix was used for random effects to allow variance and covariance estimations to be greater than zero (Raudenbush & Bryk, 2001). Satterthwaite approximation was used to calculate the degrees of freedom (Satterthwaite, 1946).

Estimates of effect size were reported as change in R^2 between the covariates only model (model 3) predicting withdrawal symptoms, and the fully conditional model (model 4), including DERS total score and all covariates (Selya, Rose, Dierker, Hedeker, & Mermelstein, 2012). Therefore, the reported R^2 change represents the unique variance in withdrawal symptoms accounted for by the DERS total score. However, due to variance in the outcome coming from both random and fixed effects, a formal significance test of the change in R^2 between models does not provide interpretable results (Nakagawa & Schielzeth, 2013).

3. Results

3.1. Descriptive statistics

Five-hundred twenty-nine participants entered the study and were randomized to treatment. The final sample for the current study included 188 participants who attended and completed the quit day assessment and had at least one value on each of the variables, including covariates. The number of participants that provided withdrawal data on each of the follow up visits is as follows: quit day – n = 188, week 1 – n = 188, week 2 – n = 159, week 4 – n = 140, week 12 – n = 99. Examination of baseline differences between those retained in the current analyses and those excluded indicated that those removed from analysis had significantly higher baseline DERS total scores (p = .01) as well as had a higher proportion of male smokers (p = .02). In terms of smoking behavior, participants reported smoking an average of 15.35 (SD = 7.35) cigarettes per day, reported being a daily smoker for an average of 20.19 (SD = 13.91) years, and reported initiating regular daily smoking at an age of 17.86 (SD = 3.92) years old. The average FTCD score of the sample was 4.97, indicating moderate levels of nicotine dependence (Heatherton et al., 1991). See Table 1 for full demographic characteristics and Table 2 for bivariate relations among variables.

3.2. Growth curve models

Preliminary Analysis. Results from model 1 yielded the ICC (0.34) and the design effect (2.36) for the model. These results indicate that 34% of the observed variance in withdrawal symptoms over time was due to within person differences and about 66% of the observed variance was due to between person differences. The magnitude of the ICC and the design effect support the use of multilevel modeling with the

Table 1
Descriptive statistics.

Demographic Variables	*Mean (SD) or % (n)*
Gender (Male)	46.8%
Age	38.52 (14.00)
Ethnicity	
Caucasian	87.8%
Black (non-Hispanic)	6.4%
Hispanic	3.7%
Other	2.1%
Other Variables	
Baseline DERS total	72.66 (20.38)
Use of Nicotine Replacement Therapy	76.6%
Baseline FTCD Total	4.97 (2.20)
Pre-quit (session 3) Withdrawal	13.77 (4.17)
Condition (active)	56.3%
Average Cigarettes Smoked Since Last Assessment	
Week 1	1.77 (3.97)
Week 2	2.24 (4.59)
Week 4	5.01 (7.31)
Week 12	0.55 (1.68)
Withdrawal	
Quit Day Withdrawal	14.01 (4.60)
Week 1 Withdrawal	14.99 (4.71)
Week 2 Withdrawal	14.53 (4.46)
Week 4 Withdrawal	14.19 (4.78)
Week 12 Withdrawal	13.71 (4.56)

Note: DERS total – Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004); FTCD total – Fagestrom Test for Cigarette Dependence (Fagerström, 2012); Use of Nicotine Replacement Therapy – any use of NRT during the first 2 weeks of the cessation attempt; Average Cigarettes Smoking since Last Assessment – time-varying effect of smoking status during follow up.

Table 2
Bivariate correlations among variables.

	1.	2.	3.	4.	5.	6.	7.
1. Average Cigarettes	–						
2. Use of NRT	−0.003	–					
3. FTCD Total	0.027	.239**	–				
4. Pre-quit Withdrawal	−0.018	0.060	0.025	–			
5. Treatment Condition	−0.016	.130**	0.022	−0.030	–		
6. Gender	0.015	−0.002	0.053	0.016	−0.047	–	
7. DERS	0.030	.076*	−0.041	.354**	-.137**	0.026	–
8. Withdrawal	−0.027	.141**	.133**	.599**	−0.032	0.051	.322**

Note: * indicates significance at the $p < .05$ level, and ** indicates significance at the $p < .01$ level.

Table 3
Solutions for fixed effects in Model 2 (Unconditional Quadratic Model) and Model 3 and 4 (Conditional models with covariates only and with all variables, respectively).

Effect	Estimate	*SE*	*df*	*t value*	*p value*
Model 2					
Intercept	.16	.26	365	.59	.56
Time	.13	.08	747	1.57	.12
Time^2	-.02	.01	722	−2.40	.02
Model 3					
Intercept	-.47	1.06	216	-.44	.66
Time	-.27	.15	243	−1.80	.07
Time^2	.01	.01	295	1.24	.21
Average Cigarettes	-.01	.005	202	−2.25	.03
Use of NRT	1.43	.55	193	2.58	.01
FTCD Total	.14	.10	196	1.47	.14
Pre-quit (session 3) withdrawal	.63	.05	187	12.43	< .001
Condition	-.07	.43	188	-.16	.88
Gender	.09	.43	187	.21	.83
Model 4					
Intercept	-.51	.99	188	-.51	.61
Time	-.25	.16	151	−1.59	.11
Time^2	.01	.01	148	1.02	.31
Average Cigarettes	-.01	.005	183	−2.24	.03
Use of NRT	1.27	.52	165	2.47	.01
FTCD Total	.13	.09	194	1.42	.16
Pre-quit (session 3) withdrawal	.59	.05	184	11.46	< .001
Condition	.06	.41	169	.16	.88
Gender	.06	.40	177	.14	.89
DERS Total	.01	.02	67	.84	.40
Time*DERS Total	.01	.01	126	1.93	.06
Time^2*DERS Total	-.001	.001	112	−2.19	.03

Note: DERS (Baseline difficulties in emotion regulation: Gratz & Roemer, 2004), FTCD (Baseline nicotine dependence; Fagerström, 2012); NRT: Use of nicotine replacement therapy. Condition: treatment condition (active or control); Average Cigarettes: Average cigarettes smoked per day since last assessment; Pre-quit withdrawal: last measurement of withdrawal before scheduled quit day.

data, as there was significant variability at both the between and within individual level.

Fixed Effects. Table 3 presents the fixed effect coefficients for models 2–4. For model 2, time showed a significant quadratic relationship with withdrawal symptoms ($B = -0.02$, $SE = 0.01$, $p = .02$). Results indicated that withdrawal increased and then decreased with time following a scheduled quit attempt (see Fig. 1 and Table 3).

For model 3 with covariates only, average cigarettes smoked since last assessment ($B = -0.01$, $SE = 0.004$, $p = .03$) and pre quit (session 3) withdrawal ($B = 0.63$, $SE = 0.05$, $p < .001$) were significantly associated with withdrawal. Additionally, examination of the random effects indicated there was significant variance in the intercepts ($p < .001$) as well as in the autoregressive structure of the repeated assessments ($p = .01$).

For the fully conditional model (model 4), DERS total score was not a significant unique predictor of withdrawal, suggesting that higher

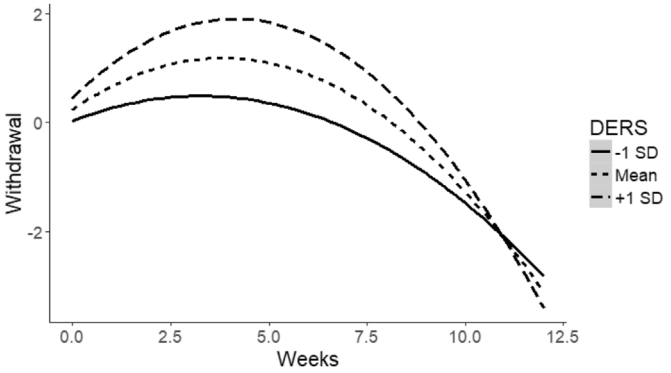

Fig. 1. DERS total baseline score by time^2 interaction from quit week (week 0) to 12 weeks post quit. Lines represent those at mean (short dash) levels of DERS, +1 SD (long dash) levels of DERS, and −1 SD (solid) levels of DERS. Random intercept analyses indicate that those with highest levels of baseline DERS exhibit the highest quit week withdrawal symptoms, followed by the largest increase and decrease in withdrawal symptoms, compared to those with lower baseline DERS scores.

DERS baseline score was not associated with greater withdrawal symptoms ($B = 0.01$, $SE = 0.02$, $p < .40$). However, for the interactive effects of model 4, there was a significant interaction with DERS and time^2 ($B = -0.001$, $SE = 0.0005$, $p = .03$), suggesting differential impact of DERS baseline score on withdrawal symptoms over time (see Fig. 1). Specifically, those with high DERS (+1 *SD* above the mean) evinced the highest withdrawal at quit day (intercept), as well as the greatest increase (slope) and subsequent decrease, leaving them at the highest withdrawal symptoms at week 12, compared to those with mean and low (1 *SD* below the mean) baseline DERS scores.

3.3. Variance explained

The unexplained residual variance of withdrawal symptoms reduced from 19.3% in model 1–15.4% in a model with exclusively covariates predicting withdrawal. Adding DERS total score to the model reduced the unexplained residual variance in withdrawal symptoms to 6.2%. Examining the change in R^2 between the covariates only model, and the fully conditional model, DERS total score explained an additional 40% of the explained variance in withdrawal symptoms.

4. Discussion

The current study examined the relationship of individual differences in emotion dysregulation prior to a cigarette quit attempt with the trajectory of change in tobacco withdrawal symptoms following a scheduled cigarette quit attempt. As hypothesized, individual differences in baseline levels of emotion dysregulation were significantly associated with quit day withdrawal symptoms. Specifically, smokers with elevated baseline emotion dysregulation evinced the greater quit day withdrawal symptoms. These data suggest that emotion

dysregulation is related to more intense withdrawal on quit day, presumably as a result of lacking effective strategies to manage negative affect (Piper & Curtin, 2006). Results also indicated that, following the scheduled quit attempt, baseline emotion dysregulation was associated with slope of change in withdrawal symptoms. Specifically, relative to smokers with lower levels of emotion dysregulation, smokers with greater difficulty regulating negative affect demonstrated a slower overall deceleration in slope, with the largest increase followed by a decline in withdrawal symptoms over the 12-week post-quit period. These results are in line with prior research suggesting that emotion dysregulation is related to maladaptive processes and quit patterns (Farris et al., 2016; Rogers et al., 2018), and extend it to withdrawal symptom severity. The observed effects for both tested models were above and beyond the effects of pre-quit withdrawal symptoms, as well as cigarette dependence, average cigarettes smoking since last assessment, gender, use of NRT, and treatment condition on trajectories of tobacco withdrawal.

Examining the variance accounted for by each of the models indicates that about 20% of the variance in withdrawal is accounted for by the intercept and time. These results are noteworthy, as they suggest that the level of withdrawal an individual reports at quit week, regardless of their level of emotion dysregulation, significantly accounts for withdrawal symptoms over time. However, adding emotion dysregulation further reduces the unexplained residual variance, suggesting that emotion dysregulation is also an important construct to understand the trajectory of withdrawal symptoms over time.

Of note, the current study included smokers who were able to successfully abstain from smoking as well as those that had lapsed during their quit attempt. In the current report, we adjusted for time-varying average number of cigarettes smoked since last assessment. Thus, the present findings are generally applicable to withdrawal during a quit attempt by being evident among treatment-seeking smokers regardless of quit status. Although withdrawal may be expected to be most elevated initially for smokers who maintain abstinence, research suggests withdrawal is nonetheless robustly evident among those that have lapsed/relapsed (Shiffman et al., 1997; Shiffman & Waters, 2004) and even among smokers not engaged in a quit attempt (Baker, Brandon, & Chassin, 2004). Future research employing larger samples may be able to model tobacco withdrawal among smokers that maintain their abstinence and those that do not.

The current findings suggest that emotion dysregulation may have potential clinical value in determining trajectory of withdrawal symptoms over time, which may aid in determining prognosis of smoking cessation and inform treatment planning. Theoretically, addressing pre-treatment emotion dysregulation deficits may theoretically lessen the intensity of withdrawal symptoms experienced on quit day and foster a faster decline in withdrawal symptom severity across the quit period. Given the strong association documented between withdrawal symptoms and smoking lapse/relapse (Piasecki et al., 2000), the findings from the current study highlight potential clinical utility in assessments and interventions focused on emotion dysregulation for smokers who enter a cessation program. Specifically, pre-quit screening of emotion dysregulation followed by the delivery of the interventions to improve more adaptive emotion regulation may help decrease the severity of tobacco withdrawal. By extension, improving emotion dysregulation prior to engaging in a quit attempt could reduce the severity of tobacco withdrawal symptoms during a quit attempt by providing individuals strategies to manage withdrawal-related negative affect, and thereby increase the odds of cessation success. Although no treatments have been developed to target emotion dysregulation in the context of smoking specifically per se, there are some cognitive-behavioral treatments with an explicit focus on reducing emotion dysregulation, including Dialectical Behavioral Therapy (Linehan, 1993), Emotion Regulation Therapy (Mennin, 2004), among others (Barlow et al., 2017; Farchione et al., 2012). Future research could seek to adapt and integrate such emotion dysregulation treatments for smokers with elevated pre-quit emotion dysregulation.

There are several study limitations that warrant comment. First, the current sample was comprised of a relatively homogenous group of Caucasian treatment-seeking community smokers with moderate cigarette dependence. Future research should examine the same questions among diverse samples of smokers with low and high levels of cigarette dependence, as well as among those participants regardless of desire to quit to increase the generalizability of the findings. Second, the current study focused exclusively on emotion dysregulation as a transdiagnostic vulnerability factor for more severe withdrawal symptoms. Because other transdiagnostic vulnerability factors related to more severe withdrawal symptoms (e.g., Bakhshaie, Kulesz, et al., 2018; Bakhshaie et al., 2017), future research could construct and test models incorporating multiple transdiagnostic risk factors in mult-risk factor models. Third, the dependent measure used in the current study was a composite “withdrawal severity” index that was intended to capture overall tobacco withdrawal severity. However, while withdrawal is generally associated with increased risk of lapse and relapse (Piasecki et al., 2000), the current measure includes symptoms that may not be as relevant to risk of lapse (e.g., hunger, concentration; McCarthy, Piasecki, Fiore, & Baker, 2006; Piper et al., 2011). Future research could therefore examine the impact of pre-quit emotion dysregulation on the individual components of tobacco withdrawal.

Fourth, due to the study design, participants only completed a measure of emotion dysregulation at baseline and not following treatment. Although the treatment itself was not focused on emotion dysregulation, it is possible that state-like differences in emotion dysregulation were evident, and either this change, or quit-day level of emotion dysregulation, was a better predictor of withdrawal severity than the baseline measure of individual differences in emotion dysregulation. It may therefore be important to assess state-like emotion dysregulation change in treatment, and examine how that change may impact tobacco withdrawal. Additionally, all measures in the current study were self-report measures, suggesting that the findings may be due to shared method variance, and future research should incorporate both self-report as well as objective measures of emotion dysregulation and tobacco withdrawal. Further, the current study did not examine mechanisms that may link emotion dysregulation to tobacco withdrawal, such as poor coping mechanisms, and future research should examine mediators of the relationships examined in the current study. Finally, we did not measure the extent to which smokers continued to make a ‘serious effort’ toward quitting past quit day. Thus, despite being motivated to quit and enrolling in an intensive treatment for smoking, some smokers may have displayed differential levels of ‘quit effort’ and treatment fatigue. It is unclear how the degree of effort placed in remaining abstinent post quit influenced the current findings (e.g., variability in adherence to the treatment protocol). Future research is needed to explore quit effort as well as treatment fatigue in the context of emotion dysregulation and smoking.

Together, the current study offers novel evidence about the role of individual differences in emotion dysregulation in relation to withdrawal symptoms during a quit attempt. The findings indicated that emotion dysregulation is related to the degree of change in post-quit withdrawal symptoms among treatment-seeking smokers. Thus, there may be clinical utility in assessing and targeting emotion dysregulation prior to engaging in a smoking quit attempt to achieve offset the severity of tobacco withdrawal. Future work could usefully explore the utility in tailoring individual treatments to smokers with elevated emotion dysregulation.

Trial registration

NCT01753141.

Protocol

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

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