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Panic attack history and smoking topography

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ABSTRACT

Background: Little is known about panic attacks and puffing topography, a behavioral index of the value of smoking reinforcement. This study examined smoking style during the course of smoking of a single cigarette among adult daily smokers with and without a history of panic attacks.
Method: Participants ($n = 124$, $M_{age} = 43.9$, $SD = 9.7$; 44.4% female) were non-treatment seeking daily smokers. Lifetime panic attack history was assessed via diagnostic assessment; 28.2% ($n = 35$) of the sample had a panic attack history. Participants smoked one cigarette during an ad libitum smoking trial. Puff volume, duration, and inter-puff interval were measured using the Clinical Research Support System (CReSS) pocket device.
Results: Regression analyses revealed that panic attack status was not associated with significant differences in average puff volume, duration, or inter-puff interval. Multi-level modeling was used to examine puffing trajectories. Puff-level data revealed that there was a significant quadratic time x panic effect for puff volume and duration. Those with a panic attack history demonstrated relatively sustained levels of both puff volume and duration over time, whereas those without a history of panic attacks demonstrated an increase followed by a decrease in volume and duration over time. These effects were not accounted for by the presence of general psychopathology.
Discussion: Smokers with a panic attack history demonstrate more persistent efforts to self-regulate the delivery of nicotine, and thus may be at risk for continued smoking and dependence. Tailored treatment may be needed to address unique vulnerabilities among this group.

1. Introduction

Panic attacks reflect an abrupt autonomic surge of intense discomfort and extreme fear or impending doom accompanied by a strong flight-or-fight action tendency (American Psychiatric Association, 2013). Panic attacks can occur from calm or anxious states – and regardless of preceding anxious states, the peak intensity of fear or discomfort is reached within minutes. That is, panic attacks are discrete in nature (Craske et al., 2010), and distinct from anxiety or other generalized negative emotional states (Craske, 1991). Psychophysiological data suggest that panic attacks are indeed reflected by abrupt surges of arousal, typically cardiorespiratory activation/instability that reaches a peak within minutes and subsides within minutes (Craske et al., 2010; Meuret and Ritz, 2010). Research suggests that panic attacks may 'mark' risk for multiple forms of psychopathology, including anxiety disorders (Baillie and Rapee, 2005; Reed and Wittchen, 1998), major depressive disorder (Bittner et al., 2004; Bovasso and Eaton, 1999; Hayward et al., 2000), substance use disorders (Baillie and Rapee, 2005), personality disorders (Goodwin et al., 2005), and severe mental illness (Goodwin et al., 2004).

It is estimated that approximately 28% of the general United States population has experienced a panic attack some point in their life, with 23% experiencing panic attacks without ever meeting criteria for panic disorder and/or agoraphobia (Kessler et al.,

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2005). Lifetime panic attacks are associated with an increased odds of smoking (e.g., Breslau et al., 1991; Breslau and Klein, 1999; Goodwin and Hamilton, 2002; Johnson et al., 2000; Pohl et al., 1992). The prevalence of smoking is significantly higher among those with a history of panic attacks relative to the general non-psychiatric population, and smoking cessation rates are significantly lower (Lasser et al., 2000). Indeed, smoking is a risk factor for the onset and maintenance of panic attacks (Bakhshaie et al., 2016; Breslau et al., 2004; McFall et al., 2005). Research also suggests that panic attacks may maintain smoking behavior (Zvolensky et al., 2003b). For example, panic attacks are associated with more severe nicotine withdrawal symptoms during quitting (Marshall et al., 2009), shorter durations of abstinence from smoking (Zvolensky et al., 2004), and overall lower success rates in quitting (Piper et al., 2011).

Negative reinforcement models of smoking (Zvolensky et al., 2003b) and drug addiction more broadly (McCarthy et al., 2010) posit that smokers with a history of panic attacks may be especially likely to rely on cigarettes (nicotine) for temporary amelioration of aversive internal bodily states. Aligned with this theory, empirical data indicate that panic attack history is predictive of *self-reported* coping-oriented smoking motives (Johnson et al., 2013), which in turn, may maintain tobacco dependence (Farris et al., 2014). Such self-report data suggest that smokers with panic attacks may utilize cigarettes to manage negative affect states, which may increase the reinforcing value of nicotine for this subgroup of smokers.

Style of puffing behavior (topography) is used to comprehensively examine factors that maintain regular tobacco use (e.g., nicotine dependence, smoking frequency/quantity), and to understand individual aspects of nicotine regulation (Burling et al., 1985; Frederiksen et al., 1977). Puffing style also provides a *behavioral index* of the value of smoking reinforcement (Perkins et al., 2010). Empirical data convincingly indicate that smokers will change (compensate) how they smoke to maintain stable levels of nicotine (Ashton and Watson, 1970; Kumar et al., 1977; Sutton et al., 1978), including following laboratory-manipulated anxiety/stress paradigms (Farris and Zvolensky, 2016; McKee et al., 2011; Rose et al., 1983). That is, a smoker may not only rely on smoking as an affective-regulatory aid but also tailor *how* he/she smokes (puffs/inhales) as a means to increase the negative reinforcing value of a cigarette. Initial data also indicate that smokers with affective symptoms/psychopathology (e.g., depression, posttraumatic stress disorder) display altered smoking styles relative to those without (e.g., larger puff volume, McClernon et al., 2005; Perkins et al., 2010), although this set of findings has not always been consistently reported (Malpass and Higgs, 2007).

The majority of studies that have examined smoking topography calculate averages of topographic behavior of a smoked cigarette (e.g., Corrigall et al., 2001; Kassel et al., 2007; Perkins et al., 2010). However, puff behavior typically changes during the course of smoking a cigarette (e.g., Collins et al., 2010; Guyatt et al., 1989; Kolonen et al., 1992). Smokers tend to take longer and larger initial puffs, potentially to increase immediate consumption of nicotine (Guyatt et al., 1989). Over the course of a cigarette, the magnitude of puff volume and puff duration decreases and the inter-puff interval increases (Guyatt et al., 1989; Kolonen et al., 1992); varying patterns may reflect individual differences in sensitivity to nicotine/cigarette components, satiation, and persistence in efforts to self-regulate the delivery of nicotine (Guyatt et al., 1989). Thus, the topographic trajectory may provide unique and nuanced information about the reinforcing value of smoking, and may vary as a function in psychological vulnerabilities.

In a prior study, we found that an intense surge in panic/arousal (induced in the laboratory) resulted in *reductions* in average puff volume and duration (Farris and Zvolensky, 2016), possibly a result of the intensity of the acute subjective and cardiorespiratory distress. The current study aimed to extend this line of work examining smoking topography among smokers with and without a history of panic attacks. This approach allows for broader characterization of smoking topography and panic, beyond a context-specific test. Such a test could potentially provide novel and nuanced information about the nature of panic vulnerability on smoking reinforcement and risk for tobacco dependence. We are aware of at least one study among light adolescent smokers that examined trait anxiety symptoms in terms of puff-to-puff changes in topography during the course of a single cigarette (Veilleux et al., 2011). Findings indicated that higher anxiety symptoms were associated with linear increases in puff volume and duration over the course of the cigarette (Veilleux et al., 2011). Moreover, smokers with an 'atypical' pattern of increasing puff volume profile had a more rapid progression to tobacco dependence over the course of two years (Veilleux et al., 2011). Although not directly tested, these data suggest that adolescent smokers with anxiety symptoms may be an at risk group for tobacco dependence based on their smoking topographical profile. This methodological approach has not been extended to panic attack psychopathology specifically.

The current study examined differences in ad libitum smoking topography, a behavioral index of smoking motivation, among non-treatment seeking adult smokers with and without a lifetime history of panic attacks. Smoking topography indices were examined in two ways: (a) averaging puff data across a single cigarette to derive a mean index and (b) utilizing puff-to-puff data to examine variability during a single cigarette. It was hypothesized that (1) Smokers with a history of panic attacks would have larger average puff volumes and duration and shorter inter-puff intervals compared to smokers with no history of panic attacks and (2) Smokers with a history of panic attacks would demonstrate an 'atypical' pattern of smoking over the course of one cigarette – increasing puff volume and duration. We examined whether these associations were unique beyond the effect of other psychopathology, gender, and tobacco dependence.

2. Material and methods

2.1. Participants

Non-treatment seeking adult daily smokers were recruited for an experimental study on anxiety and smoking behavior (Farris and Zvolensky, 2016). Community-recruited smokers who were between 18 and 65 years of age, reported smoking 10 or more cigarettes per day for at least one year, and smoked within the first 30 min of waking in the morning, were invited for a baseline assessment to determine eligibility for the experimental study (described in Farris and Zvolensky, 2016). Participants were excluded from participation during an initial telephone screen if they reported frequent drinking (≥9 standard drinks/week), illicit drug use (≥3 days/week), unstable medical conditions, or current psychotic symptoms. The current study is a secondary analysis of data from participants who completed the baseline assessment ($n = 126$), regardless of whether or not they were deemed eligible for the experimental phase of the study. Two participants were missing smoking topography data due to equipment malfunction, thus were excluded from the analyzed sample. Thus, 124 participants ($M_{age} = 43.9$, $SD = 9.7$; 44.4% female) were included in analyses.

2.2. Procedure

Participants were screened for potential eligibility by telephone, scheduled for an in-person appointment and instructed to bring their usual brand of cigarettes (at least 2 full cigarettes) to the

laboratory. Upon arrival, participants provided a carbon monoxide (CO) analysis of expired breath to verify smoking status, completed a diagnostic clinical interview and a series of self-report assessments. Next, all participants completed an *ad libitum* smoking trial at a standardized point during the baseline assessment (approximately 90 min after arrival to the laboratory). Participants were told they could have a 'smoke break' during which they were oriented to the portable CReSS device and shown how to use it. The experimenter accompanied the participant outdoors, alongside the laboratory, and informed the participant that he/she would have the opportunity to smoke one cigarette using the device. The participant was told to smoke as usual, and was given as much time as desired. Next, then participant returned inside the laboratory for an adaptation period, during which he/she completed approximately 75 min of self-report assessments, although data from this portion of the assessment are not included in the current analyses. After, participants were dismissed if ineligible, or were informed that they had the opportunity to complete the experimental portion of the study (if eligible). All participants were provided $25 compensation for their time and participation in the baseline phase of the study. Study procedures were approved by the Institutional Review Board where the study took place.

2.3. Measures

2.3.1. Panic attack history. The *Structured Clinical Interview for DSM-IV Disorders-Non-Patient Version* (SCID-I/NP; First et al., 2007), a clinician-administered semi-structured diagnostic assessment, was used to assess the presence of past-year Axis-I psychopathology based on the DSM-IV-TR diagnostic guidelines. All participants were administered the panic disorder section of the anxiety disorder module (F.1). Based on the focus of the parent study (Farris and Zvolensky, 2016), *lifetime* presence of recurrent panic attacks was assessed (i.e., ever occurring). Specifically, all participants were asked: *"Have you ever had a panic attack, when you suddenly felt frightened or anxious or suddenly developed a lot of physical symptoms?"* If yes, participants were considered having a lifetime panic attack history if they endorsed having at least two in their lifetime (Y/N), if the symptoms come on 'all of a sudden' [acute onset] (Y/N), and if ≥4 or more symptoms were present during the episodes. DSM-IV criteria (A1/A2) were also assessed (to assess presence of panic disorder) but not required to denote presence of panic attacks (American Psychiatric Association, 2013). This included A1: recurrent *unexpected* panic attacks and A2: At least one attacks is followed by ≥1 month of persistent concern about having additional attacks and/or worry about implications of the attack/consequences). Additionally, the mood episodes, psychotic screening, and somatoform disorders modules were administered to all participants. Substance use, other anxiety disorder, and eating disorder modules were administered only based on positive endorsement of symptoms on the SCID screening module. Diagnostic assessments were conducted by highly-trained post-baccalaureate level research assistants. All research assistants completed a 6-session training conducted by the principal investigator and two additional doctoral-level graduate students, shadowed administration of three assessment cases with a doctoral-level graduate student, completed two live-supervised assessments, and demonstrated diagnostic accuracy on three test cases, prior to being 'signed off' as SCID-trained. In the current study, all diagnostic assessments were audio-recorded and 100% of cases were supervised by the study principal investigator for diagnostic accuracy. A random 20% of recordings were subjected to blinded inter-rater reliability review by a doctoral-level clinical psychology graduate student. No cases of diagnostic disagreement were noted (100% accuracy).

2.3.2. Descriptive smoking indices.

2.3.2.1. Smoking History Questionnaire. The *Smoking History Questionnaire* (SHQ; Brown et al., 2002), a 30-item self-report measure, was used to gather information about smoking history (e.g., age of smoking initiation, smoking rate, years smoking).

2.3.2.2. Fagerström Test for Nicotine Dependence. The Fagerström Test for Cigarette Dependence (FTND; Heatherton et al., 1991), a 6-item self-report measure (possible range 0–10), was used to assess level of tobacco dependence (higher scores reflecting greater dependence). The FTND has adequate internal consistency, positive relations with key smoking variables (e.g., saliva cotinine), and high test-retest reliability (Heatherton et al., 1991; Pomerleau et al., 1994). Respondents were also asked to determine use of their usual cigarette brand, and indicate specific properties of their preference cigarette (e.g., filtered, menthol, etc).

2.3.2.3. Carbon monoxide (CO). A *carbon monoxide* (CO) analysis, using the Vitalograph Breath Co carbon monoxide monitor, was conducted to measure the amount of CO (in parts per million (ppm)) in an expired breath sample, which is an indirect, noninvasive measure of blood carboxyhemoglobin. CO analysis was conducted at baseline, upon arrival to the laboratory.

2.3.3. Smoking topography assessment. The *Clinical Research Support System* (CReSS; Plowshare Technologies, Borgwaldt KC, Inc., Virginia), specifically the portable CReSS pocket device, was used to assess puff topography. The device has a sterilized flow meter mouthpiece that is connected to a pressure transducer, which converts pressure into a digital signal that is sampled at 1000 Hz. CReSS computer software transforms the signal to a flow rate (mL/s), from which puff topography data are computed. The reliability and acceptability of use of the portable CReSS device is well documented (Blank et al., 2009; Perkins et al., 2012), and is recommended over direct observation (Blank et al., 2009). Puff topography data included: *puff volume* (amount of CO in mL of smoke inhaled), *puff duration* (time in seconds inhaled during puff), and *inter-puff interval* (time in seconds between successive puffs).

2.4. Data analytic procedures

Differences between smokers with and without a panic attack history (0 = no panic attack history, 1 = panic attack history) were explored in relation to demographic, smoking, and psychological characteristics. Puff-level smoking topography data were examined for range and outlying variables. Next, raw puff-level smoking topography data were examined for outliers using standard scores, with a criterion of $z = 3.5$ to retain maximum data. A small number of outliers were detected for puff volume (2.4%), puff duration (1.3%) and inter-puff interval (1.0%). The outliers were determined to be legitimate high-magnitude values and were recoded as one unit higher than the next lowest non-outlying value (Tabachnick and Fidell, 2000). The observed range of puffs during ad libitum of the cigarette ranged from 7 to 43, with an average of 21.1 puffs ($SD = 7.74$). Volume, duration, and inter-puff interval were all non-normally distributed and were transformed to correct for this. Specifically, puff volume and duration were square-root transformed, and inter-puff interval was log-transformed and then cubic transformed. These transformations were successful in normalizing the data. Puff-level data were averaged to create mean scores, and linear regression models were conducted to examine group differences by panic attack status in terms of each smoking topography variable. The patterning of results is unchanged when the outliers are removed (widsordized) versus retained (as-is). Given

the outliers affect the underlying assumptions of normality, the windorized variables utilized.

To examine puff-level data, multilevel modeling was used as intraclass correlation (ICC) values for each dependent variable were above the conservative recommended value of 0.1 (range 0.23–0.51). Random intercepts and slopes were included in all models. Huber/White/sandwich robust standard errors were estimated in order to adjust for heteroskedasticity in the error terms, and an unstructured covariance matrix was specified to allow for each covariance to be uniquely estimated (StataCorp LP, 2015). Time was mean centered to prevent multicollinearity when using higher-order polynomials (Aiken and West, 1991). Gender (coded 0 = male, 1 = female) and FTND were entered as model covariates. Significance was determined based on $p < 0.05$.

3. Results

Based on the SCID-I/NP diagnostic assessment, 28.2% ($n = 35$) of the sample had a history of panic attacks. The average age of panic attack onset was 28.9 ($SD = 12.6$) years. Among smokers with a history of panic attacks, 62.9% had a current (past-year) emotional disorder (anxiety/mood) and 20.0% had a past-year substance use disorder; 14.3% met diagnostic criteria for current (past-year) panic disorder. In contrast, the prevalence of current emotional and substance use disorders among smokers without a panic attack history was 18.0% and 2.2%, respectively. See Table 1 for smoking characteristics for smokers with and without panic attacks. Relative to smokers with no panic attack history, smokers with panic attacks were more frequently female, were younger in age when first initiating smoking, but did not differ in terms of smoking rate, tobacco dependence, expired CO, or use of menthol cigarettes.

3.1. Average smoking topography

Female gender was associated with shorter mean puff volume and duration (r's = −0.18-0.20, p's < 0.05), thus was entered as a model covariate. Tobacco dependence (per FTND) was entered as a covariate based on consistency with prior studies (Veilleux et al., 2011), although in these data, higher tobacco dependence was only associated with shorter average inter-puff interval ($r = -0.22$, $p = 0.016$). The presence of past-year psychopathology (coded 0 = no; 1 = yes) was not related to puff volume or duration, however was significantly associated with shorter inter-puff interval ($r = -0.24$, $p = 0.006$). Puff volume was significantly correlated with puff duration ($r = 0.69$, $p < 0.001$), but not inter-puff interval ($r = 0.16$, $p = 0.078$). Puff duration and inter-puff interval were significantly correlated ($r = 0.30$, $p = 0.001$). Additionally, menthol cigarette use was not associated with different puff topography indices (r's = −0.01 − 0.08).

3.1.1. Puff duration. Average puff duration among smokers with and without a panic attack history was 1.7 s ($SD = 0.60$) and 1.8 ($SD = 0.70$), respectively (untransformed means presented). The model predicting average puff duration was non-significant ($F[3120] = 1.78$, $p = 0.154$; total $R^2 = 4.3\%$); only female gender was associated with shorter average puff duration ($b = -0.10$, $se = 0.05$, $t = -2.16$, $p = 0.033$).

3.1.2. Puff volume. Average puff volume among smokers with and without a panic attack history was 64.0 mL ($SD = 15.27$) and 67.1 ($SD = 29.57$), respectively (untransformed means presented). The regression model predicting average puff volume was non-significant ($F[3120] = 1.33$, $p = 0.268$; total $R^2 = 3.2\%$); female gender was associated with smaller average puff volume ($b = -0.53$, $se = 0.27$, $t = -1.99$, $p = 0.049$). Neither tobacco dependence nor panic attack status predicted significant unique variance in average puff volume.

3.1.3. Inter-puff interval. Average puff duration among smokers with and without a panic attack history was 12.2 s ($SD = 5.26$) and 13.2 ($SD = 5.29$), respectively (untransformed means presented). The regression model predicting average inter-puff interval was significant ($F[3120] = 3.05$, $p = 0.031$; total $R^2 = 7.1\%$). The model significance was driven by the significant effect of tobacco dependence ($b = -0.09$, $se = 0.04$, $t = -2.39$, $p = 0.019$); the effect of panic attack status was non-significant.

All regression results were unchanged when the past-year psychopathology was entered in the models as a covariate.

3.2. Puff-Level smoking topography

3.2.1. Puff duration. See Table 2 for complete model results. Visual inspection of the raw puff duration data suggested that a quadratic effect of time was most appropriate for the model, and this was statistically significant (see Fig. 1, top). In addition, there was a significant quadratic time x panic status interaction ($b = 0.0006$, $SE = 0.0002$, $z = 2.43$, $p = 0.015$), but not a main effect of panic status. The form of the interaction indicated that smokers without a history of panic attacks demonstrated an increase followed by a decrease in puff duration over time, whereas those with a panic attack history demonstrated relatively sustained puff durations over time. In terms of covariates, there was a main effect of gender but not FTND. When FTND was removed from the model, the pattern of results was identical.

3.2.2. Puff volume. Visual inspection of the raw puff volume data suggested that a quadratic effect of time was most appropriate for this model, and this was statistically significant (see Fig. 1, middle). There was a significant quadratic time x panic status interaction ($b = 0.0041$, $SE = 0.0016$, $z = 2.56$, $p = 0.011$), but not a main effect of panic status. The form of the interaction indicated that smokers without a history of panic attacks demonstrated an increase followed by a decrease in puff volume over time, whereas those with a panic attack history demonstrated relatively sustained puff volumes over time. In terms of covariates, gender but not FTND was a significant predictor. When FTND was removed from the model, the pattern of results was identical.

3.2.3. Inter-puff interval. Visual inspection of the raw inter-puff interval data suggested that a cubic effect of time was most appropriate for the data, and this was statistically significant (see Fig. 1, bottom). There was non-significant cubic time x panic attack status interaction or a main effect of panic status. There was a main effect of the FTND, but not gender. When Gender was removed from the model, the pattern of results was identical.

All multilevel models results were unchanged when the past-year psychopathology was entered in the analyses as a covariate. Results are not presented here, although are available upon request.

4. Discussion

The current study was the first to explore the nature of smoking topography among smokers with and without a lifetime history of panic attacks. The present results suggest smokers with a history of panic attacks appear to have different smoking topographical profiles compared to smokers without a history of panic attacks. Specifically, smokers with a history of panic attacks demonstrated sustained puff volumes and duration over the course of a cigarette. In contrast, smokers without a history of panic attacks demonstrated a more typical pattern of topography wherein puff volume and duration decreased over the course of smoking (Guyatt et al.,

Table 1
Descriptive characteristics among smokers with and without panic attack history.

Variable	Total Sample (n = 124)	Panic Attack History (n = 35)	No Panic Attack History (n = 89)	X^2 or t
Gender (n, % Female)	55 (44.4%)	22 (62.9%)	33 (37.1%)	6.76**
Race (n, %)				0.99
White	37 (29.8%)	12 (34.3%)	25 (28.1%)	
Black/African-American	79 (63.7%)	20 (57.1%)	59 (66.3%)	
Other	8 (6.5%)	3 (8.6%)	5 (5.6%)	
Age *(M, SD)*	43.9 (9.71)	42.5 (10.49)	44.5 (9.39)	1.07
Age of smoking initiation	16.2 (6.60)	14.0 (3.82)	17.1 (7.26)	2.36*
Age of regular daily smoking	19.4 (6.98)	18.3 (6.93)	19.8 (6.99)	1.09
Years as a smoker	23.5 (10.95)	22.2 (11.99)	24.0 (10.54)	0.79
Avg. cigarettes/day past week	14.7 (6.06)	15.9 (6.43)	14.3 (5.88)	−1.36
Expired CO at arrival	22.6 (11.34)	19.5 (9.84)	23.8 (11.71)	1.92
Tobacco Dependence (FTND)	4.7 (1.49)	4.7 (1.67)	4.7 (1.42)	−0.21
Smoking 5-min waking (n,%yes)	66 (53.2%)	19 (54.3%)	47 (52.8%)	0.02
Menthol Cigarette Use (n,%yes)	80 (64.5%)	21 (60.0%)	59 (66.3%)	0.43
Any Psychopathology (n,%yes)	50 (40.3%)	26 (74.3%)	24 (27.0%)	23.38***
Emotional Disorder (n,%yes)	38 (30.6%)	22 (62.9%)	16 (18.0%)	23.81***
Substance Use Disorder (n,%yes)	9 (7.3%)	7 (20.0%)	2 (2.2%)	11.76**

* $p < 0.05$.
** $p < 0.01$.

Table 2
Results from puff-level analyses.

Variable	Puff Duration (sec.)			Puff Volume (mL)			Inter-Puff Interval (sec.)		
	b	*z*	*p*	*b*	*z*	*p*	*b*	*z*	*p*
Linear Time	−0.0033	−1.39	0.164	0.0077	0.46	0.643	**0.5341**	**5.68**	**<0.0001**
Quadratic Time	**−0.0007**	**−4.33**	**<0.0001**	**−0.0055**	**−4.83**	**<0.0001**	**−0.0571**	**−7.07**	**<0.0001**
Cubic Time	–	–	–	–	–	–	**0.0015**	**4.11**	**<0.0001**
Panic status	−0.0302	−0.60	0.549	0.0599	0.23	0.818	1.5145	−0.68	0.499
Linear Time x Panic	−0.0026	−0.71	0.476	−0.0235	−1.00	0.318	−0.1428	−1.01	0.314
Quadratic Time x Panic	**0.0006**	**2.43**	**0.015**	**0.0041**	**2.56**	**0.011**	0.0002	0.01	0.989
Cubic Time x Panic	–	–	–	–	–	–	0.0004	0.64	0.519
FTND	**−0.0864**	**−2.00**	**0.045**	**−0.5698**	**−2.22**	**0.026**	2.1044	−1.42	0.157
Gender	−0.0020	−0.14	0.890	0.0571	0.69	0.490	**−1.0984**	**−2.00**	**0.045**

Note: significant effects are presented in bold for ease in viewing.

1989). These observed effects were significant after adjusting for participant gender and level of tobacco dependence. Additionally, the patterning of results was unchanged when considering the presence of past-year general psychopathology, suggesting that the observed effects may be unique to panic attack history. We were unable to directly compare individuals with a history of panic attacks to those with other forms of psychopathology groups in terms of smoking topography given the small number of smokers with only panic attacks (with no present psychopathology, n = 9). This is an important area for future examination.

This set of findings is broadly consistent with prior findings among high-anxiety adolescent smokers (Veilleux et al., 2011), who demonstrated linear increases in puff volume and duration while smoking. Additionally, these behavioral data bolster and extend the existing self-report data that document the link between panic attacks and affect-regulatory motivation for smoking (e.g., Farris et al., 2014; Johnson et al., 2013). Although there were no overall average differences in nicotine intake among smokers with and without a history of panic attacks, the differential puff topographical patterns indicate that the trajectory of nicotine intake differs between these groups. Specifically, smokers with no history of panic attacks demonstrated steeper initial curves in nicotine intake, followed by a drop (indexing potential satiation). In contrast, smokers with a history of panic attacks demonstrated more steady and graduate intake in nicotine. When contextualized with the previous findings that acute panic-relevant arousal produces *decreases* in puff volume and duration (Farris and Zvolensky, 2016), the current pattern of puffing may suggest that panic-prone smokers are averse to *rapid rises* in nicotine levels and arousal. Although the mechanism underlying the associations between panic attack history and smoking topography are not clear, the current data provide unique and detailed information about the reinforcing value of smoking (Perkins et al., 2010) and risk for tobacco dependence (Veilleux et al., 2011) among a high-risk group of smokers (i.e., smokers with a history of panic attacks).

Notably, smoking topography did not vary between smokers with and without a history of panic attacks when topographical indices were averaged across the cigarette smoked. This observation is in contrast to the findings observed in prior work that has found that puff volume is larger in smokers with PTSD (McClernon et al., 2005) and depression (Perkins et al., 2010), although another study found no difference in average topography for smokers with and without depression (Malpass and Higgs, 2007). Although averaging topography data is a commonly used analytic approach, the divergent findings across studies suggest that it may be particularly informative to examine puff-level topography data. Indeed, the variability in puff-to-puff behavior is not captured when puff topography data are averaged, which decreases specificity in the measured outcome, and thus, may limit the ability to detect nuanced differences in puffing behavior. Based on the current set of findings, future studies examining smoking topography, especially in the context of psychopathology, may be bolstered by testing topographical trajectories.

Several study limitations warrant comment. First, a single measurement of smoking topography was used in the current study. It is possible that lack of familiarity of smoking with the CReSS device affected smoking behavior, although acceptability of the CReSS pocket device has been documented (Blank et al., 2009). Additionally, all participants smoked outdoors, which was specifically designed to increase ecological validity of smoking behavior.

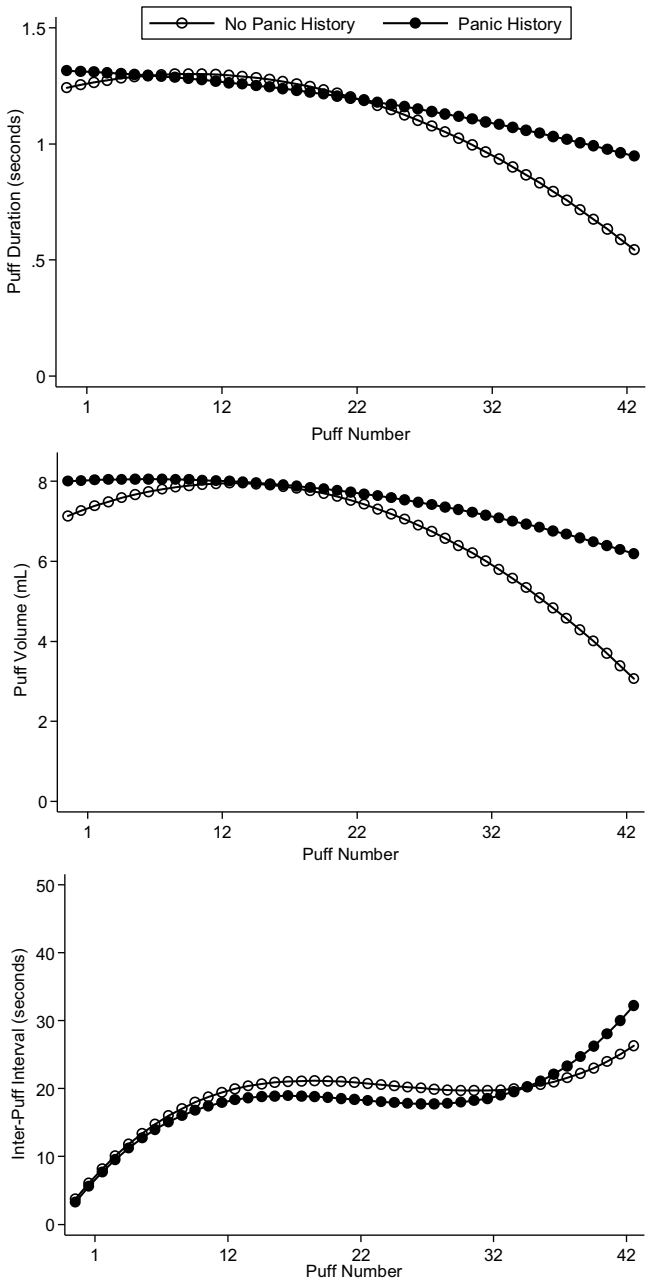

Fig. 1. (top) Quadratic time x panic on puff duration; (middle) Quadratic time x panic on puff volume; (bottom) Cubic time on inter-puff interval.

However, we cannot rule out the possibility that being in a research study/laboratory setting, versus an individual's naturalistic environment, could influence smoking topography (Ossip-Klein et al., 1983). Second, smoking behavior prior to the experimental laboratory visit and timing of study appointments were not standardized (e.g., participants were seen in the morning and afternoon for the experiment). Data were not systematically collected on the time since last cigarette use. These factors could have affected observable puffing behavior if individuals had recently smoked. Expired CO breath samples suggest that smokers likely had smoked within the past 12–24 h, thus they were not arriving at the appointment in a state of nicotine deprivation. Additionally, there was a non-significant difference between expired CO values between smokers with and without panic attack history. Thus, these data may most closely reflect naturalistic smoking behavior. Third, we assessed lifetime history of panic attacks, which is consistent with previous studies (e.g., Farris et al., 2014; Goodwin and Hamilton, 2002; Johnson et al., 2013), although it is also possible that some smokers may have remitted panic attacks (i.e., that are not present in the past-year). In contrast to lifetime panic attacks, the presence of current panic arousal may be differentially related to aspects of smoking topography (Farris and Zvolensky, 2016). Last, the sample was comprised of participants who primarily identified race as Black/African-American (63.7%) and the prevalence of menthol cigarette use was high (64.5%). Thus, generalizability may be limited to the studied sample of smokers.

Overall, the current study offers novel insight into the negative reinforcing value of smoking among individuals with a history of panic attacks. Specifically, the current findings suggest that a lifetime panic attack history may be related to subtle alterations in puffing style that are only detected when examining the *trajectory* of puffing behavior. Future work should examine the concordance between self-reported measures of motives for smoking and actual smoking behavior. Given that persistent puffing behavior demarcates increased risk for tobacco dependence (Veilleux et al., 2011), it may be important to assess and address puffing style among panic-vulnerable smokers. Providing tailored feedback to smokers about *how* they smoke could be meaningfully integrated into tailored panic-smoking cessation intervention (Zvolensky et al., 2003a).

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Contributors

Drs. Farris and Zvolensky developed the study, on which Dr. Goodwin was a consultant. Drs. Farris and Zvolensky managed the literature searches and summaries of previous related work. Drs. Farris and Brown completed statistical analyses. All authors contributed to and have approved the final manuscript.

Conflict of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this paper.

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