Psychology of Addictive Behaviors

0893-164X/11/$12.00 DOI: 10.1037/a0024328

Associations Between Posttraumatic Stress Disorder Symptom Clusters and Cigarette Smoking

Jodie B. Greenberg, Katherine J. Ameringer, Michael A. Trujillo, Ping Sun, Steve Sussman, Molly Brightman, Stephanie R. Pitts, and Adam M. Leventhal
University of Southern California Keck School of Medicine

Understanding the relationship between Posttraumatic stress disorder (PTSD) and cigarette smoking has been difficult because of PTSD's symptomatic heterogeneity. This study examined common and unique lifetime cross-sectional relationships between PTSD symptom clusters [Re-experiencing (intrusive thoughts and nightmares about the trauma), Avoidance (avoidance of trauma-associated memories or stimuli), Emotional Numbing (loss of interest, interpersonal detachment, restricted positive affect), and Hyperarousal (irritability, difficulty concentrating, hypervigilance, insomnia)] and three indicators of smoking behavior: (1) smoking status; (2) cigarettes per day; and (3) nicotine dependence. Participants were adult respondents in the National Epidemiologic Survey of Alcohol and Related Conditions with a trauma history (n = 23,635). All four symptom clusters associated with each smoking outcome in single-predictor models (ps $<$. 0001). In multivariate models including all of the symptom clusters as simultaneous predictors, Emotional Numbing was the only cluster to retain a significant association with lifetime smoking over and above the other clusters, demographics, and Axis-I comorbidity (OR = 1.30, p $<$ .01). While Avoidance uniquely associated with smoking status and nicotine dependence in multivariate models, these relations fell below significance after adjusting for demographics and comorbidity. No clusters uniquely associated with cigarettes per day. Hyperarousal uniquely related with nicotine dependence over and above the other clusters, demographics, and Axis-I comorbidity (OR = 1.51, p $<$ .001). These results suggest the following: (a) common variance across PTSD symptom clusters contribute to PTSD's linkage with smoking in the American population; and (b) certain PTSD symptom clusters may uniquely associate with particular indicators of smoking behavior. These findings may clarify the underpinnings of PTSD-smoking comorbidity and inform smoking interventions for trauma-exposed individuals.

Keywords: posttraumatic stress disorder, smoking, comorbidity, nicotine dependence, posttraumatic stress disorder symptom clusters

The association between posttraumatic stress disorder (PTSD) and cigarette smoking has been widely documented (Feldner et al., 2007; Fu et al., 2007). Extant research indicates that PTSD diagnoses and severity estimates are associated with higher rates of smoking (Acierno, Kilpatrick, Resnick, Saunders, & Best, 1996; Beckham et al., 1995; Buckley, Mozley, Bedard, Dewulf, & Greif, 2004; Hapke et al., 2005; Kirby et al., 2008), heavier smoking (Beckham et al., 1997; Cook, Jakupcak, Rosenheck, Fontana, & McFall, 2009), and increased prevalence and severity of nicotine dependence (Hapke et al., 2005; Koenen et al., 2003, 2005; Thorndike, Wernicke, Pearlman, & Haaga, 2006; Weaver & Etzel, 2003).

Jodie B. Greenberg, Katherine J. Ameringer, Michael A. Trujillo, Ping Sun, Steve Sussman, Molly Brightman, Stephanie R. Pitts, and Adam M. Leventhal, University of Southern California Keck School of Medicine.

This research was supported by National Institutes of Health grants DA026831and DA025041.

Correspondence concerning this article should be addressed to Adam M. Leventhal, University of Southern California Keck School of Medicine, 2250 Alcazar Street, CSC 240, Los Angeles, CA 90033. E-mail: adam.leventhal@usc.edu

Most investigations of the PTSD-smoking relationship regard PTSD as a homogenous construct that can be identified by the presence or absence of a PTSD diagnosis or a combined overall symptom severity score. However, PTSD symptoms are phenomenologically heterogeneous and involve a multidimensional constellation of various cognitive, behavioral, and affective manifestations (APA, 1994). Accordingly, PTSD symptomatology may be best characterized as a collection of several clusters that each include smaller sets of related symptoms that tend to co-occur. Indeed, evidence suggests that different PTSD symptom clusters appear to be psychometrically distinct and relate to disparate psychosocial and biological correlates (Asmundson et al., 2000; Kashdan, Elhai, & Frueh, 2006; Simms, Watson, & Doebbeling, 2002; Weiss, 2007). Accordingly, understanding the unique relation of each PTSD symptom cluster to smoking could shed light on the mechanisms linking PTSD and smoking behavior.

One multidimensional model of PTSD symptoms that has received considerable support is a four-factor model, which includes: Re-experiencing (RE; intrusive thoughts and nightmares about the trauma), Avoidance (AV; avoidance of trauma-associated memories or stimuli), Emotional Numbing (EN; loss of interest, interpersonal detachment, overall affective blunting), and Hyperarousal (HA; irritability, difficulty concentrating, hypervigilance, insom-

nia) (Asmundson et al., 2000; Simms et al., 2002). Some studies have demonstrated significant associations between symptom cluster scores and certain indicators of smoking behavior while others have not. Mixed results have been the reported for studies of RE (Baschnagel, Coffey, Schumacher, Drobes, & Saladin, 2008; Beckham et al., 1997; Kirby et al., 2008; Thorndike et al., 2006; Weaver & Etzel, 2003), AV (Baschnagel et al., 2008; Cook, McFall, Calhoun, & Beckham, 2007; Kirby et al., 2008; Weaver & Etzel, 2003), and HA (Baschnagel et al., 2008; Beckham et al., 1997; Thorndike et al., 2006). Fewer investigations have examined EN, but those that have analyzed this cluster have found significant associations with current smoking status (Kirby et al., 2008) and heavy smoking (i.e., 20 + cigarettes per day; Cook et al., 2009).

It is possible that findings have been mixed because past investigations have been performed in restricted samples selected from very different populations (e.g., battered women, male Iraq and Afghanistan veterans, male Vietnam veterans), have used disparate markers of smoking behavior (e.g., smoking status, cigarettes per day, dependence), and have differed in the extent to which the influence of comorbid Axis-I psychiatric and substance use disorders have been controlled. Accordingly, one way to potentially clarify the extent of PTSD-smoking relations is to examine associations between PTSD symptom clusters and multiple indicators of smoking behavior in a nationally representative sample. Furthermore, past studies of the PTSD-smoking link have most commonly analyzed each PTSD symptom cluster in isolation from one another. It therefore remains unclear the extent to which the relationship between PTSD symptomatology and smoking is attributable to shared variability across the symptom clusters or unique variability specific to each particular cluster.

Elucidating relations between PTSD symptom clusters and smoking behavior may have several important clinical and theoretical implications. For instance, self-medication processes (Jacobsen, Southwick, & Kosten, 2001; Khantzian, 1985) may underlie the PTSD-smoking relation, and symptom cluster analysis may help elucidate specific symptoms for which trauma-exposed smokers might self-medicate. For example, if EN is most robustly associated with smoking, it could be hypothesized that individuals with PTSD symptoms might smoke to overcome affective flattening, which would be consistent with research demonstrating that smokers with trait anhedonia (i.e., inability to experience pleasure in response to natural reinforcers) are more likely to affectively respond to positive mood inductions after nicotine administration (Cook, Spring, & McChargue, 2007). By contrast, data indicating that HA is most strongly related to smoking might suggest that individuals with PTSD symptoms may be motivated to smoke to diminish anxiety, irritability, poor concentration, or other symptoms associated with HA, which have been shown to be suppressed by tobacco (Buchhalter, Acosta, Evans, Breland, & Eissenberg, 2005; Morissette, Tull, Gulliver, Kamholz, & Zimering, 2007). Also, chronic nicotine exposure dysregulates the hypothalamic-pituitary-adrenal (HPA) axis and brain's reward system (BRS) (Heim & Nemeroff, 2009). Alteration in these systems are also associated with PTSD (Heim & Nemeroff, 2009), and it is possible that BRS dysfunction may underlie reward processing deficits linked with EN, whereas HPA axis disturbance may contribute to a wider variety of PTSD symptoms. It is therefore possible that smokers may be more likely to experience certain PTSD symptoms after trauma, given that they may have particular preexisting biological disturbances caused by smoking.

Clinically, this research may be informative for working with patients with PTSD symptoms who smoke or are at risk for smoking. If specific links between particular symptom clusters and smoking are identified, it may behoove clinicians to assess individual symptom clusters in trauma-exposed individuals irrespective of their overall PTSD diagnosis to identify patients with a higher likelihood of comorbid smoking. Furthermore, this line of research may provide clues to designing targeted smoking cessation interventions for smokers with PTSD symptoms. That is, the symptom clusters that are most robustly associated with smoking behavior may require the most intensive treatment in integrative smoking cessation programs for smokers with PTSD symptoms.

The present cross-sectional study examined lifetime associations between number of symptoms experienced within four separate PTSD symptom clusters (RE, AV, EN, and HA) and smoking in a nationally representative sample. Of primary interest was the extent to which each symptom cluster uniquely and incrementally associated with three indicators of lifetime smoking behavior: (1) smoking status; (2) cigarettes per day; and (3) *DSM–IV* nicotine dependence. Because both PTSD and smoking are commonly comorbid with Axis-I emotional and substance use disorders (Grant et al., 2009; Grant, Hasin, Chou, Stinson, & Dawson, 2004), we were also interested whether relations between symptom clusters and smoking were significant after controlling for non-PTSD Axis-I comorbidity.

Method

Sample

Participants were respondents in Waves 1 (2001–2002) and 2 (2004–2005) of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). The NESARC was developed to collect data on the prevalence of alcohol and substance use, psychiatric disorders, psychosocial features, and other clinical features in the U.S. adult population. All participants were noninstitutionalized civilians, aged 18 and older, and resided in the U.S. The sample consisted of individuals living in households, group quarters, or military personnel living off-base.

At Wave 1, in-person interviews were conducted with 43,093 participants. Young adults (18–24 years old) were oversampled by a 2.25 to 1 ratio. Hispanics and African Americans were also oversampled, and each group accounted for approximately 20% of the sample. The overall response rate at Wave 1 was 81.0%. At Wave 2, attempts were made to reinterview all 43,093 respondents. Excluding those who were ineligible (e.g., those who were deceased, had been deported, or were mentally or physically impaired), 34,653 individuals—a response rate of 86.7%—completed the Wave 2 interview. The cumulative response rate of Waves 1 and 2 was 70.2%. Both waves of data were weighted to account for oversampling and were adjusted to represent U.S. population demographics using the 2000 Census of Population and Housing. Further details of the sampling, purpose, demographic profile, and data weighting for the NESARC have been published elsewhere (Grant & Kaplan, 2005; Grant, Moore, Shepart, & Kaplan, 2003).

Of the 34,653 total respondents who completed Wave 2 interviews, only individuals who had experienced at least one traumatic event and endorsed either responding with fear, helplessness, or horror or being confronted with death or serious injury to themselves or others at the time of the event were retained for analyses (n = 23,860). Of these individuals, those who did not have complete PTSD data (n = 106; see measures section) and those who did not have complete smoking status data (n = 119) were eliminated, leaving a final sample of 23,635 respondents for smoking status analyses. Subsequently, those who had never smoked (n = 12,798) and those who did not have complete cigarettes per day data (n = 66) were eliminated, leaving a sample of 10,771 respondents for analyses of nicotine dependence and cigarettes per day. Table 1 provides information on demographic and psychiatric characteristics among those eliminated (n = 11,018) and those retained for analyses (n = 23,635) from the original Wave 2 dataset.

Interviewers and Training

Interviewers administered the Alcohol Use Disorder and Associated Disabilities Interview Schedule-IV(AUDADIS-IV; Grant, Dawson, et al., 2003), which is a diagnostic interview tool intended for experienced, nonclinician interviewers. Data were collected via face-to-face interviews with computer assistance.

Interviewers worked for the U.S. Census Bureau and had an average of five years of experience conducting Census and other health-related national surveys. Their training comprised a five-day self-study course and five-day in-class training at a Census Bureau office. NESARC training supervisors also completed the at-home study and a training session directed by National Institute of Alcohol Abuse and Alcoholism (NIAAA) sponsors and Census Field and Demographics Survey Division headquarters staff.

Procedure

The U.S. Census Bureau contacted potential respondents by mail and informed them about the nature of the study. One adult from each household was randomly selected for the interview. After informed consent was obtained, interviewers administered the AUDADIS-IV.

Measures

The AUDADIS-IV includes questions that assess demographic information, substance use patterns, *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM–IV*) criteria for psychiatric and substance use disorder diagnoses (APA, 1994), and other data. Prior analyses have demonstrated adequate psychometric properties for mental disorder diagnoses and other information

Table 1
Demographic and Psychiatric Characteristics Among Those Eliminated and Those Retained for Analysis

	Respondents excluded from analyses (n = 11,018)	Respondents included in analyses (n = 23,635)	P value for contrast between groups
Demographic characteristics			
Female, %	44%	56%	<.0001
Age, *M* (*SD*)	48.1 (0.22)	48.2 (0.13)	<.77
Ethnicity/race	—	—	
White, non-Hispanic	66%	73%	<.0001
Black, non-Hispanic	12%	11%	
American Indian/Alaskan Native, non-Hispanic	2%	2%	
Asian/Native Hawaiian/Other Pacific Islander, non-Hispanic	6%	4%	
Hispanic, any race	15%	10%	
Income, %	—	—	
< $20,000	21%	17%	<.0001
$20,000 – $34,999	20%	18%	
$35,000 – $69,999	32%	33%	
$70,000 +	27%	32%	
Education, %	—	—	
Some high school or less	18%	12%	<.0001
Completed high school or GED	28%	27%	
Some college or 2-year degree	29%	32%	
Completed college or more	25%	28%	
Marital status, %	—	—	
Married or living with someone as if married	62%	65%	<.0001
Divorced or separated	10%	12%	
Widowed	8%	7%	
Never married	20%	16%	
Lifetime psychiatric characteristics, %			
Major depressive episode	13%	26%	<.0001
Anxiety disorder (other than PTSD)	16%	30%	<.0001
Manic episode	3%	4%	<.0001
Any non-nicotine substance use disorder	32%	29%	<.0001

Note. Percentages are weighted to approximate to the 2000 U.S. census. Chi-square tests and linear regression models were used to compare the two groups on categorical and continuous variables, respectively.

yielded from the AUDADIS-IV (Chatterji et al., 1997; Grant, Dawson, et al., 2003; Ruan et al., 2008).

Lifetime smoking status. Lifetime smoking status was evaluated in the entire sample and was coded positive among respondents who reported ever having smoked 100 or more cigarettes at either wave.

Lifetime cigarettes per day. Lifetime cigarettes per day was evaluated among lifetime smokers and was quantified as the highest number of cigarettes respondents reported smoking on a usual day across both waves.

Lifetime nicotine dependence. All lifetime smokers were evaluated for nicotine dependence and classified as dependent if they met lifetime *DSM–IV* criteria at either wave.

Lifetime PTSD. The PTSD module of the AUDADIS-IV was administered only at Wave 2. Participants were queried on whether they personally experienced, or someone very close to them experienced various types of traumatic events. Those who endorsed at least one event and reported responding with fear, helplessness, or horror or being confronted with death or serious injury to themselves or others at the time of the event were further evaluated for individual *DSM–IV* PTSD symptoms. The test–retest reliability of lifetime PTSD diagnoses was adequate ($K = 0.64$), and the internal consistency of patterns of PTSD item endorsement was good (Cronbach's alpha = .84) (Ruan et al., 2008).[1]

Because partial and subthreshold PTSD is often associated with significant functional impairment (Grubaugh et al., 2005; Marshall et al., 2001; Pietrzak, Goldstein, Southwick, & Grant, 2011; Stein, Walker, Hazen, & Forde, 1997), those individuals who experienced a traumatic event and endorsed either fear/helplessness/horror or threat of death/injury, regardless of PTSD diagnosis, were included in analyses. Table 2 displays the list of traumatic events assessed in the interview and the proportion of each traumatic event endorsed among those in the final sample.

PTSD diagnosis. Respondents were considered to have a PTSD diagnosis if they met lifetime *DSM–IV* criteria for PTSD.

PTSD symptom cluster scores. Despite the *DSM–IV*'s structuring of three categories of symptoms (Cluster B: RE; Cluster C: AV and EN; and Cluster D: HA), previous factor analyses of PTSD symptoms have often identified a four-factor model that contains clusters B and D as unique factors but also separates AV and EN into distinct factors (Asmundson et al., 2000; Simms, Watson, & Doebbeling, 2002). Confirmatory factor analysis was used to compare three separate oblique nested models using the symptom-level data yielded from the AUDADIS-IV in this sample: (a) a one-factor model with all symptoms loading to a common factor; (b) a three-factor model with each symptom loading onto a single factor consistent with *DSM–IV* clustering: and (c) a four-factor model with each symptom loading onto one of four dimensions (RE, AV, EN, and HA). Using a χ^2 difference test for the model comparison (Steiger, Shapiro, & Browne, 1985), results showed that a four-factor model, $\chi^2(df = 146) = 11996.5$, demonstrated significantly better fit than both the three-factor, χ^2 ($df = 149$) = 16489.2, and one-factor, χ^2 ($df = 152$) = 24820.8, models ($ps < .0001$). Accordingly, we applied a four-cluster approach to separating the PTSD symptoms.

As in the *DSM–IV*, the AUDADIS-IV yields dichotomous (present vs. absent) values for each symptom. For creating cluster scores, we first considered identifying a threshold for number of symptoms endorsed within each cluster and categorizing participants as above versus below that threshold. However, this approach is limited because there are no well-defined thresholds for EN and AV, and it does not capture variability across the overall continuum of PTSD symptom severity, which is important given that PTSD symptoms may have a dimensional structure (Broman-Fulks et al., 2006). Accordingly, we opted to define a score based on symptom count as used in prior research (Saladin, Brady, Dansky, & Kilpatrick, 1995). Although scores based on count are limited in that the severity of each symptom is not assessed, extant data indicate high concordance between symptom count estimates and traditional overall severity scores (Foa & Tolin, 2000).

The four clusters have different numbers of total symptoms per cluster, which could complicate comparisons of effect sizes across the clusters. In addition, data were unavailable for some symptoms for some respondents [e.g., participant could not recall whether or not (s)he ever experienced a symptom]. Thus, the final cluster scores used in analyses were based on proportion values which took the sum of symptoms endorsed within a cluster and divided it by the total number of symptoms with data present for that cluster. Participants with less than half of symptom data available for one or more clusters were eliminated from analyses because of poor potential reliability of cluster scores ($n = 106$; 0.4%).

Symptoms within each cluster were as follows: RE (AUDADIS-IV 12-8a, #1 intrusive memories of trauma; #2 bad dreams of trauma; #3 reliving trauma; #4 acting out trauma; #5 upset when reminded of trauma; #6 physical reactions when reminded of trauma), of which 0.1% ($n = 29$) had data available for four symptoms, 0.9% ($n = 209$) for five symptoms, and 99% ($n = 23, 397$) for all six symptoms; AV (#7 avoiding thinking of trauma; #8 avoiding conversations about trauma; #9 avoiding reminders of trauma; #10 inability to recall parts of trauma), of which 0.4% ($n = 91$) had symptom data for three symptoms and 99.5% ($n = 23, 543$) had data for all four symptoms; EN (#11 loss of interest in activities usually enjoyed; #12 feeling emotionally distant from others; #13 inability to feel love or affection toward others; #14 not planning for future), of which 0.3% ($n = 92$) had symptom data for three symptoms and 99.7% ($n = 23, 563$) had data for all four symptoms; and HA (#15 insomnia; #16 anger/irritability; #17 trouble concentrating; #18 hypervigilance; #19 easily startled), of which 0.2% ($n = 40$) had data available for three symptoms, 0.7% ($n = 178$) for four symptoms, and 99.1% ($n = 23, 417$) for all five symptoms.

Data Analysis

Preliminary analyses involved calculating descriptive statistics for key variables and testing correlations among the four PTSD symptom clusters and with *DSM–IV* PTSD diagnoses.

For primary analyses, we calculated regression models to test relations of each symptom cluster to the three indicators of tobacco use behavior. Logistic regression was used in models incorporating

[1] Among the entire sample of respondents who completed the Wave 2 interview ($n = 34,653$), 91.3% ($n = 31,650$) experienced at least one traumatic event, 68.9% ($n = 23,860$) experienced at least one traumatic event and reported either responding with fear, helplessness, or horror or being confronted with death or serious injury to themselves or others at the time of the event, and 6.4% ($n = 2,463$) met *DSM–IV* criteria for lifetime PTSD diagnosis.

Table 2
Rates of Traumatic Events Experienced in the Final Sample for Analyses

Traumatic event	Percent experienced
Ever in active military combat	1106 (5.1%)
Ever serve as a peacekeeper/relief worker in war zone/other terrorized area	306 (1.3%)
Ever an unarmed civilian in war/revolution/military coup	540 (2.5%)
Ever a refugee	303 (1.4%)
Ever in a serious/life-threatening accident	4546 (19.8%)
Ever had a serious/life-threatening illness	4799 (20.2%)
Ever in a serious fire, tornado, flood, earthquake, or hurricane	4331 (18.2%)
Ever sexually assaulted, molested, raped, or experienced unwanted sex	2996 (11.3%)
Physically attacked/beaten/injured before age 18 by parent/caretaker	1156 (4.5%)
Seriously neglected before age 18 by parent/caretaker	1028 (3.9%)
Saw serious fights at home before age 18	3219 (12.6%)
Ever physically attacked/beaten/injured by spouse or romantic partner	2292 (7.9%)
Ever physically attacked/beaten/injured by anyone else	2177 (9.3%)
Ever kidnapped or held hostage by a POW	299 (1.1%)
Ever stalked by anyone	1876 (7.0%)
Ever mugged, held up, or threatened with a weapon	3559 (14.5%)
Ever had someone close to you die in a terrorist attack	245 (1.0%)
Ever had someone close to you injured in a terrorist attack	274 (1.2%)
Ever yourself injured in a terrorist attack	18 (0.1%)
Ever had someone close to you directly experience a terrorist attack	1379 (6.0%)
Ever yourself directly experience a terrorist attack	282 (1.1%)
Ever yourself indirectly experience a terrorist attack, like watching on TV	20,368 (87.2%)
Other than terrorist attack, ever see someone badly injured/killed or ever unexpectedly see a dead body	6594 (28.7%)
Other than a terrorist attack, ever have someone close to you die unexpectedly	11,734 (50.0%)
Ever have someone close to you experience any other serious/life threatening illness, accident, or injury	13,180 (57.0%)
Someone close to you ever have any other very stressful/traumatic experience	4159 (17.4%)
Ever yourself have any other stressful/traumatic experience	1611 (6.6%)

Note. Prevalence of respondents who reported experiencing that traumatic event with weighted percentage within the final sample used for analyses (n = 23,635).

lifetime smoking status and nicotine dependence as outcomes. Linear regression was used in models predicting cigarettes per day. Models predicting lifetime smoking used the entire available sample (n = 23,635), whereas models predicting nicotine dependence and cigarettes per day were tested in the subset of lifetime smokers (n = 10,771). Two sets of models were tested for each smoking outcome: (1) an individual model which included only a single symptom cluster score as the sole predictor; and (2) a combined model which included all four symptom cluster scores as simultaneous predictors to examine their unique associations with smoking outcomes after controlling for their covariance. Both PTSD and smoking are associated with demographic characteristics and Axis-I comorbidity (Grant et al., 2009; Grant et al., 2004). Thus, each of the univariate and the combined models were recalculated after adjusting for demographic covariates (sex, marital status, income, age, ethnicity/race, education), lifetime history of any substance use disorder, major depression, anxiety disorder, and mania to determine whether PTSD-smoking associations were accounted for by these factors. For comparative purposes, we calculated unadjusted and adjusted individual models paralleling those described above, which incorporated *DSM–IV* PTSD diagnoses as the primary predictor. To explore whether relations between symptom cluster count scores and smoking behavior varied as a function of PTSD diagnosis, we conducted four additional interaction models for each symptom cluster score. These analyses paralleled the individual adjusted models described above but also included PTSD diagnosis and the interaction between PTSD diagnosis and the respective symptom count as predictors.

To account for the complex sampling methodology of the NESARC, we performed all analyses in SAS using the PROC SURVEY procedures (SAS Institute, 2009), and sampling weights were used to approximate the U.S. population (Grant & Kaplan, 2005). Primary results of logistic and linear regressions are reported as odds ratios (*OR*s) and beta-weights (β), respectively. Significance was set at $p < .01$ (two-tailed) because of the large number of tests performed. This is consistent with previous approaches that used a .01 criterion to decrease the probability of Type I errors associated with multiple comparisons while not severely limiting statistical power (Leventhal, Kahler, Ray, & Zimmerman, 2009; Schmitz et al., 2000).

Results

Descriptive Statistics of PTSD and Smoking Characteristics

In the final sample of trauma-exposed participants retained for smoking status analysis (n = 23,635), the $M \pm SD$ of the proportion scores for each symptom dimension were as follows: RE (0.34 ± 0.37), AV (0.25 ± 0.36), EN (0.13 ± 0.30), and HA

(0.22 ± 0.35). Among these trauma-exposed participants, 2,443 (9.3% ± 0.22*SE*) met criteria for *DSM–IV* PTSD diagnosis and 10,771 (47.1% ± 0.39 *SE*) endorsed smoking 100+ cigarettes in their lifetime.

In the final subset of trauma-exposed lifetime smokers who had complete nicotine dependence and cigarettes per day data (n = 10,771), the $M \pm SD$ of the proportion scores for each symptom cluster were as follows: RE (0.36 ± 0.38), AV (0.28 ± 0.37), EN (0.15 ± 0.33), and HA (0.24 ± 0.36). Of these participants, 1,943 (16.5% ± 0.42 *SE*) were diagnosed with PTSD. The average number of cigarettes smoked per day was 18.16 (SD = 15.54), and 5,524 participants (51.9% ± 0.58 *SE*) met criteria for *DSM–IV* lifetime nicotine dependence.

Correlations Among and Between PTSD Symptom Clusters and Diagnoses

Correlations between each of the four cluster scores and with *DSM–IV* PTSD diagnoses are listed in Table 3. Consistent with the factor analytic findings, there was a moderate but not substantially large degree of intercorrelation among the PTSD symptom cluster scores (rs = .52–.63). The associations between symptom cluster scores and PTSD diagnosis were similar in magnitude (see Table 3).

Relations of PTSD Symptom Clusters and Diagnoses to Smoking Characteristics

Individual models. As illustrated in Table 4, PTSD diagnoses were significantly associated with all three indicators of smoking behavior in both unadjusted and adjusted models. Broken down by cluster, all four symptom cluster scores were significantly associated with each smoking characteristic in individual models incorporating a single symptom cluster as the sole predictor, with effect sizes being smaller in adjusted than unadjusted models (see Table 4).

Combined models. In combined models that simultaneously incorporated all four symptom clusters, RE did not retain any significant associations with any of the smoking outcomes (see Table 4). AV was significantly associated with lifetime smoking and nicotine dependence in unadjusted combined models. However, these associations fell below significance in models that adjusted for demographics and Axis-I comorbidity. AV did not significantly associate with cigarettes per day in combined models. EN was significantly associated with lifetime smoking in combined models with effect sizes that were smaller than corresponding effects in individual models but did not associate with cigarettes per day or nicotine dependence. HA was significantly associated with nicotine dependence in combined models with effects that were smaller than parallel effects in individual models but did not significantly relate with smoking status or cigarettes per day (see Table 4).

Moderation by PTSD diagnosis. PTSD diagnosis did not significantly moderate the relationships between PTSD symptom clusters and smoking outcomes in adjusted individual models.

Table 3
Correlations (r) Among PTSD Symptom Dimensions and PTSD Diagnosis

	1.	2.	3.	4.	5.
1. Re-Experiencing	—	.59	.52	.63	.44
2. Avoidance		—	.54	.54	.46
3. Emotional Numbing			—	.62	.59
4. Hyperarousal				—	.56
5. PTSD diagnosis					—

Note. n = 23,635. Re-experiencing = proportion re-experiencing symptoms endorsed. Avoidance = proportion of avoidance symptoms endorsed. Emotional numbing = proportion of emotional numbing symptoms endorsed. Hyperarousal = proportion of hyperarousal symptoms endorsed. PTSD diagnosis = *DSM-IV* Posttraumatic Stress Disorder diagnosis.
All associations were significant ($p < .0001$).

Discussion

This study examined the individual and unique relations of PTSD symptom clusters to smoking status, nicotine dependence, and cigarettes per day in a representative sample of U.S. adults. Consistent with past research (Acierno et al., 1996; Beckham et al., 1995; Buckley et al., 2004; Hapke et al., 2005; Kirby et al., 2008), lifetime PTSD diagnosis was associated with smoking status, cigarettes per day, and nicotine dependence. These results replicate and extend past findings to a new sample and document that these associations exist beyond overlap with demographic characteristics and other Axis-I disorders. More importantly, examination of individual PTSD symptom clusters revealed important variability in symptomatology that contributed to PTSD's relation to smoking.

In analyses that did not account for overlap between symptom clusters, each cluster was associated with all three smoking variables. Across all four clusters, endorsing all versus no symptoms within a cluster was associated with a 1.6- to 1.9-fold increase in odds of smoking, a 2.6- to 3.1-fold increase in odds of nicotine dependence, and standardized regression weights of 0.4–0.5 in predicting cigarettes per day among smokers (see Table 4). Although the strength of associations was attenuated in models adjusting for demographics and lifetime mood, anxiety, and substance use disorders, they were not eliminated. Exploratory analyses illustrated that the strength of these relations was not significantly moderated by PTSD diagnosis, which suggests that variation both above and below the diagnostic threshold associate with smoking behavior in a similar fashion.

After entering the symptom clusters into combined models that accounted for correlations among symptom clusters, there was marked variability in the pattern of unique associations found between PTSD symptom clusters and particular indicators of smoking behavior. RE did not retain unique associations with any of the smoking variables in combined models. Previous examinations of restricted samples have illustrated univariate associations between RE and smoking characteristics in some samples (Beckham et al., 1997; Thorndike et al., 2006; Weaver & Etzel, 2003) but not others (Baschnagel et al., 2008; Kirby et al., 2008; Thorndike et al., 2006). These findings in a large nationally representative sample indicate that RE is related to smoking status, cigarettes per day, and nicotine dependence, but this relationship may not be unique from its overlap with the other symptom clusters.

Table 4
Associations of PTSD Symptom Clusters and Diagnoses to Smoking Characteristics

	Re-experiencing		Avoidance		Emotional numbing		Hyperarousal		PTSD diagnosis
	Indiv. model[a]	Comb. model[b]	Indiv. model[a]	Comb. model[b]	Indiv. model[a]	Comb. model[b]	Indiv. model[a]	Comb. model[b]	Indiv. model[a]
Unadjusted models[c]									
Smoking status (*OR*)	1.55†	1.12	1.63†	1.24*	1.87†	1.48†	1.56†	1.04	1.48†
Nicotine dependence (*OR*)	2.56†	1.26	2.56†	1.38*	2.92†	1.34	3.05†	1.87†	2.00†
Cig/day (β)	.04†	.01	.04†	.01	.04†	.01	.05†	.01	.04**
Adjusted models[d]									
Smoking status (*OR*)	1.32†	1.12	1.34†	1.14	1.47†	1.30*	1.27†	0.98	1.26**
Nicotine dependence (*OR*)	1.53†	1.11	1.49†	1.11	1.59†	1.08	1.78†	1.51**	1.33**
Cig/day (β)	.04**	.02	.03**	.01	.03*	.01	.04**	.01	.03*

Note. Models predicting smoking status used the entire available sample (n = 23,653). Models predicting nicotine dependence and cig/day used only lifetime smokers (n = 10,771). *OR* = odds ratio. β = β-weights. Lifetime smoking = smoked 100 + cigarettes in lifetime. Nicotine dependence = *DSM-IV* lifetime nicotine dependence diagnosis. Cig/day = average number of cigarettes smoked per day during period of heaviest use. Re-experiencing = proportion re-experiencing symptoms endorsed. Avoidance = proportion of avoidance symptoms endorsed. Emotional numbing = proportion of emotional numbing symptoms endorsed. Hyperarousal = proportion of hyperarousal symptoms endorsed. PTSD diagnosis = *DSM-IV* Posttraumatic Stress Disorder diagnosis.

[a] Individual model includes the respective PTSD variable as the sole predictor. [b] Combined model includes all four symptom clusters as simultaneous predictors. [c] Includes primary predictor(s) only in the model. [d] Includes primary predictors(s) after adjusting for sex, marital status, income, age, ethnicity/race, education, lifetime history of any substance use disorder, major depression, mania, and anxiety disorder.

† $p < .0001$. * $p < .01$. ** $p < .001$.

Combined models also indicated that AV was uniquely associated with increased prevalence of smoking and nicotine dependence, above and beyond its overlap with the other symptom clusters. However, relationships unique to this cluster fell below significance in analyses adjusting for demographic and Axis-I comorbidity. Prior studies examining individual relations between AV and smoking in smaller samples have generally not found significant associations (Cook, McFall et al., 2007; Kirby et al., 2008; Thorndike et al., 2006; Weaver & Etzel, 2003), although there have been exceptions (Baschnagel et al., 2008). Further, prior reports have shown particularly strong linkages of AV with demographics and Axis-I comorbidity (Beckham et al., 1997; Saladin et al., 1995). Taken together, these findings suggest that although AV may associate with certain markers of smoking behavior in the general U.S. population of trauma-exposed adults, PTSD symptomatology specific to the AV cluster may have little predictive value over and above demographics and Axis-I comorbidity.

Past studies have shown that EN symptoms are elevated in current smokers versus nonsmokers (Kirby et al., 2008) and heavy smokers compared with light and nonsmokers (Cook et al., 2009), which is consistent with results from individual models indicating linkages of EN to all three markers of smoking behavior. The EN cluster is characterized by feelings of detachment, blunted affect or numbing of responsiveness, and diminished interest (NTIS, 2009). These features are conceptually and empirically related with symptoms of major depression, such as anhedonia (Kashdan, Elhai, & Frueh, 2006). Cook et al. (2009) demonstrated that common variance associated with both depressive symptoms and EN cancel out individual contributions toward heavy smoking in a sample of Iraq and Afghanistan veterans and hypothesized that motivation to smoke to elevate blunted positive affect may perhaps be a driving a mechanism underlying smoking in both PTSD and depression. While low positive affect does not constitute an actual symptom of PTSD, it is believed to overlap with EN and may play a role in the relation between smoking and PTSD (Cook, McFall et al., 2007). Furthermore, in the present sample, adjusting for a set of covariates that included major depression reduced the strength of associations of EN with all three indicators of smoking behavior, but not to below significance. In addition, EN was uniquely associated with lifetime smoking over and above the other symptom clusters with effect sizes indicating that endorsement of the entire set of EN symptoms was associated with a 48% increase in odds of lifetime smoking after partialing out covariance with demographics, Axis-I comorbidity, and the other symptom clusters. These results suggest that symptom expressions of EN are tightly and uniquely associated with smoking status in the American population, and other factors, including comorbid major depression, may not account for all of this relationship.

While HA was associated with each smoking characteristic in individual models, HA retained a unique association with nicotine dependence, but not smoking status or cigarettes per day, in combined models. Prior findings on HA-smoking associations in restricted samples of crime victims, war veterans, and local community smokers have been mixed (Baschnagel et al., 2008; Beckham et al., 1997; Thorndike et al., 2006). The present analyses of a large nationally representative sample indicate that HA appears to univariately associate with smoking status, cigarettes per day, and dependence in the U.S. population of adults. They also suggest that co-occurrence of HA and the other symptom clusters may account for associations of HA with smoking status and cigarettes per day, and that this cluster has a unique relationship with nicotine dependence. Specifically, results showed that endorsement of the entire set of HA symptoms was associated with a 51% increase in odds of nicotine dependence after adjusting for overlap with demographics, Axis-I comorbidity, and the other symptom clusters.

The current study had limitations. PTSD was assessed only at Wave 2 in the NESARC, and information about the timing of onset of the specific symptom clusters relative to each other was not

collected. Accordingly, a cross-sectional design examining lifetime data was used, and we could not examine order of onset in these analyses. Thus, interpretations about the temporal or causal aspects of the associations demonstrated herein cannot be made from these data. The NESARC collected information only on the presence versus absence of PTSD symptoms. Therefore, symptom clusters scores were based on symptom count estimates as in prior research (Saladin et al., 1995). Although past data suggest a high concordance between symptom count estimates and overall severity scores (Foa & Tolin, 2000), count scores are limited because they do not take into account the severity or frequency of each individual symptom and may therefore not entirely capture the underlying continuum of PTSD symptom severity.

The NESARC's measurement tool (AUDADIS-IV) was selected for epidemiologic purposes and the interviewers were nonclinicians, which could have impacted the accuracy of trauma assessment. Indeed, the rate of trauma endorsed in the overall sample of respondents who took part in the NESARC Wave 2 was relatively high (91% experienced a trauma, and 69% experienced trauma and responded with fear/helplessness/horror or threat of death/injury). Other studies have reported varying degrees of trauma exposure ranging from 56% to upward of 90% (Breslau et al., 1998; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Pietrzak et al., 2011; Stein et al., 1997), and a review by Breslau (2002) indicates that prevalence of trauma exposure varies based on methodology and parameters for PTSD diagnosis. Thus, the assessment strategy for detecting traumatic events in this study may have had limited specificity and could have yielded some false positives. On a similar note, some symptoms could have been inaccurately assessed because interviewers may not have probed as deeply as what would normally be the standard in clinical settings using more thorough diagnostic interviews.

We selected the three indicators of smoking in the present study to capture different facets of smoking behavior as well as to build off of prior PTSD-smoking research that has used these markers. However, there are many other important smoking characteristics (e.g., cessation, chronicity, relapse) and self-report and biochemical indicators of nicotine dependence that provide clinically relevant information about extent of smoking behavior, which were not included in this study and should be addressed in future work. Furthermore, data on lifetime cigarettes per day and lifetime smoking status were collected using a single item for each variable. Not only may participants have inaccurately reported the number of cigarettes smoked per day on a "usual day," but single item measures also have psychometric limitations and future studies should aim to include multi-item measures to evaluate these constructs.

Finally, examination of moderators of PTSD-smoking relations was beyond the scope of this analysis. Nonetheless, given that findings have been mixed across past samples of varying backgrounds and initial evidence that some factors, such as gender, may moderate PTSD-smoking associations (Baschnagel et al., 2008; Thorndike et al., 2006), future analyses should explore candidate moderators of PTSD-smoking associations in population-based samples.

In spite of these limitations, there are several potentially significant theoretical and clinical implications of these findings. Given that EN was uniquely associated with smoking status, we speculate that EN may potentially play a role in the etiology of smoking onset. Perhaps trauma-exposed individuals with higher EN may be motivated to seek out tobacco for emotional stimulation. However, because EN did not exhibit unique associations with cigarettes per day or nicotine dependence over and above the other symptom clusters, EN may play a less important role in the maintenance of persistent smoking behavior or may not be a unique consequence of heavy tobacco use. By contrast, HA was uniquely associated with nicotine dependence. Interestingly, a laboratory study found that smoking reduced experience of HA symptoms after exposure to a trauma cue (Beckham et al., 2007). One interpretation of Beckham et al.'s finding in the context of the current results is that motivation to suppress HA-related symptoms by smoking may be a unique mechanism underlying nicotine dependence in individuals with PTSD symptoms. At the same time, this relation may perhaps indicate that smokers may also be at higher risk for experiencing HA-related symptoms after a traumatic stressor attributable to HPA axis dysregulation caused by dependent patterns of smoking. More generally, these results might also reflect the possibility that smokers are more likely to respond to stressful situations in maladaptive ways that may put them at risk for developing any of the types of PTSD symptoms after a traumatic event.

From a clinical perspective, the current findings suggest that assessment of PTSD symptoms in the context of smoking treatment should go beyond collective symptom severity estimates or PTSD syndrome diagnoses. Rather, these findings point toward the utility of examining PTSD at the symptom cluster level in the context of smoking treatment. For example, assessment of EN symptoms in trauma-exposed individuals may help to identify individuals who may be at risk for initiating smoking. Also, these results raise the possibility that intervention strategies designed to help trauma-exposed individuals cope with EN may reduce risk of smoking. Additionally, these findings suggest that assessment of HA symptoms may be useful for identifying trauma-exposed smokers who may be most likely to have nicotine dependence. Accordingly, trauma-exposed smokers may perhaps benefit from smoking cessation interventions designed to help them cope with HA-related symptoms during a quit attempt.

In sum, this was the first study to explore common and unique relations between PTSD symptom clusters and smoking behavior in a nationally representative sample. We thoroughly controlled for potential confounding demographic and psychiatric influences by adjusting for these variables in analyses. The results yielded several novel findings. Specifically, results from this study suggest the following in the population of U.S. adults: (1) common variance across PTSD symptom clusters contribute to PTSD's linkage with smoking, cigarettes per day, and nicotine dependence; and (2) certain PTSD symptom clusters may uniquely associate with particular indicators of smoking behavior. Specifically, EN and HA symptom clusters of PTSD appear to have unique associations with lifetime smoking and nicotine dependence, respectively, above and beyond their overlap with other forms of PTSD symptomatology, demographic correlates, and comorbid Axis-I conditions. Future work attempting to elucidate the temporal, casual, and etiologic mechanisms linking these particular symptom clusters to smoking may be useful for informing the development of targeted smoking interventions for trauma-exposed individuals.

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Received November 1, 2010
Revision received February 17, 2011
Accepted March 25, 2011 ■