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Original Investigation

Influence of PTSD symptom clusters on smoking status among help-seeking Iraq and Afghanistan veterans

Jessica Cook, Matthew Jakupcak, Robert Rosenheck, Alan Fontana, & Miles McFall

Abstract

Introduction: Despite the strong association between smoking and posttraumatic stress disorder (PTSD), mechanisms influencing smoking in this population remain unclear. Previous smoking research has largely examined PTSD as a homogenous syndrome despite the fact that PTSD is composed of four distinct symptom clusters (i.e., reexperiencing, effortful avoidance, emotional numbing, and hyperarousal). Examination of the relationship between smoking and PTSD symptom clusters may increase understanding of mechanisms influencing comorbidity between smoking and PTSD. The goals of the present study were to (a) examine the influence of overall PTSD symptom severity on likelihood of smoking and smoking heaviness and (b) examine the influence of each PTSD symptom cluster on smoking.

Methods: Participants ($N = 439$) were Operation Iraqi Freedom/Operation Enduring Freedom combat veterans referred to VA mental health services.

Results: Multinomial logistic regression was chosen to accommodate a three-level outcome, in which the likelihood of being a nonsmoker was compared with (a) light smoking (1–9 cigarettes/day), (b) moderate smoking (10–19 cigarettes/day), and (c) heavy smoking (≥20 cigarettes/day). Results showed that veterans with higher levels of overall PTSD symptomatology were more likely to endorse heavy smoking (Wald = 4.56, $p = .03$, odds ratio [*OR*] = 1.65). Veterans endorsing high levels of emotional numbing were also more likely to endorse heavy smoking (Wald = 6.49, $p = .01$, $OR = 1.81$); all other PTSD symptom clusters were unrelated to smoking.

Discussion: The association between emotional numbing and heavy daily smoking suggests that veterans with PTSD may smoke to overcome emotional blunting following trauma exposure.

Introduction

Epidemiological and clinical research demonstrates a strong association between posttraumatic stress disorder (PTSD) and cigarette smoking (for reviews, see Feldner et al., 2007; Fu et al., 2007). In the general population, PTSD is associated with approximately twice the prevalence of smoking (45%) compared with the U.S. adult population at large (23%; Lasser et al., 2000). Moreover, PTSD increases the risk for nicotine dependence fourfold relative to individuals not exposed to trauma (Breslau, Davis, & Schultz, 2003). A strong PTSD–smoking relationship has also been observed in veterans. Beckham et al. (1997) found that 53% of treatment-seeking Vietnam veterans with PTSD reported smoking, and PTSD was strongly associated with smoking heavily. Smokers with PTSD may also experience unique challenges in quitting. Although half of ever-smokers have stopped using tobacco (Fiore, Hatsukami, & Baker, 2002), only 23% of ever-smokers with PTSD have quit (Lasser et al., 2000). In addition, smokers with PTSD and other anxiety disorders have been shown to be at increased risk for early smoking relapse when compared with smokers without Axis I disorders (Zvolensky et al., 2009).

Despite the strong association between smoking and PTSD, mechanisms influencing likelihood of smoking and smoking heaviness in this population remain unclear. Previous research has largely examined associations between PTSD and smoking

Jessica Cook, Ph.D., *Center for Tobacco Research and Intervention, University of Wisconsin School of Medicine and Public Health, Madison, WI*

Matthew Jakupcak, Ph.D., *Mental Illness Research, Education, and Clinical, Center, VA Puget Sound Health Care System, Department of Psychiatry and Behavioral Sciences, University of Washington School of Medicine, Seattle, WA*

Robert Rosenheck, M.D., *Northeast Evaluation Program Evaluation Center, VA National Center for PTSD, Yale University School of Medicine, New Haven, CT*

Alan Fontana, M.D., *Northeast Evaluation Program Evaluation Center, VA National Center for PTSD, Yale University School of Medicine, New Haven, CT*

Miles McFall, Ph.D., *Mental Illness Research, Education, and Clinical, Center, VA Puget Sound Health Care System, Department of Psychiatry and Behavioral Sciences, University of Washington School of Medicine, Seattle, WA*

Corresponding Author:

Jessica Cook, Ph.D., *Center for Tobacco Research and Intervention, University of Wisconsin, 1930 Monroe Street, Suite 200, Madison, WI 53711, USA. Telephone: 608-265-9775; Fax: 608-265-3102; E-mail: jwcook@ctri.medicine.wisc.edu*

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with PTSD conceptualized as a homogenous syndrome. When considering that PTSD is a complex disorder involving different symptom expressions and intermediate phenotypes (King, Leskin, King, & Weathers, 1998), this approach may not convey specific mechanisms underlying comorbidity between smoking and PTSD. Examination of the relationship between underlying dimensions of depression and smoking cessation outcomes has provided preliminary evidence for etiologic processes influencing the depression–smoking relationship (Leventhal, Ramsey, Brown, LaChance, & Kahler, 2008). Using a similar approach, we aim to examine whether unitary PTSD symptom clusters influence smoking status and smoking heaviness among Iraq and Afghanistan combat veterans referred for VA mental health treatment.

Several studies have established four factorially derived PTSD factors consisting of reexperiencing, effortful avoidance, emotional numbing, and hyperarousal (Asmundson et al., 2000; King et al., 1998). The reexperiencing factor consists of symptoms associated with reliving trauma, such as nightmares or intrusive thoughts of trauma. Effortful avoidance is associated with strategic efforts to escape trauma-associated memories (e.g., avoiding talking about trauma). Emotional numbing, believed to be mediated by automatic mechanisms, involves loss of interest, detachment from others, and restricted positive affect (Foa, Zinbarg, & Rothbaum, 1992). Finally, the hyperarousal factor involves a range of agitated states, including feelings of irritability, difficulty concentrating, and hypervigilance (King et al., 1998). Thus, each PTSD symptom cluster represents a distinct, unitary behavioral phenotype, each of which may differentially influence smoking behavior.

A potential association between the effortful avoidance factor and smoking is consistent with previous research suggesting that cigarette smoking is initiated and continued among individuals with PTSD as means of achieving relief from negative affect (Beckham, 1999; Cook, McFall, Calhoun, & Beckham, 2007; Kirby et al., 2008). Individuals with PTSD show a disproportionate negative mood response to nontrauma- and trauma-based stressors (Litz & Gray, 2002; Rauch, van der Kolk, Fisler, & Alpert, 1996). When considering evidence that nicotine reduces negative affect during exposure to stress cues (for a review, see Kassel, Stroud, & Paronis, 2003), individuals with PTSD may smoke to inhibit their hyperresponsivity to environmental stressors. Self-report, ambulatory monitoring, and laboratory studies all provide preliminary evidence that smokers with PTSD self-administer nicotine, in part, to regulate negative affect. For example, smokers with PTSD are more likely to self-report smoking to reduce tension (Beckham et al., 1997) versus other smoking motives (e.g., stimulation, automatic smoking). During ambulatory monitoring, anxiety, stress, and PTSD symptoms predicted *ad libitum* smoking among smokers with PTSD but not among smokers without PTSD (Beckham et al., 2008). Finally, veterans with PTSD who smoked after exposure to laboratory-based trauma and general stress cues reported a reduction in PTSD symptoms (Beckham et al., 2007). If nicotine helps ameliorate PTSD symptoms and related negative mood states, those higher in effortful avoidance symptoms of PTSD may be particularly likely to seek out an avoidance behavior like smoking.

It remains unclear whether alleviation of negative affect is the primary motive influencing smoking among individuals with PTSD. Those with PTSD may also self-administer nicotine to stimulate an underresponsive brain reward system. It has been proposed that PTSD smokers self-administer nicotine to enhance deficient positive affect, which is central to the emotional numbing component of PTSD (Cook, McFall, et al., 2007). In particular, evidence suggests that individuals with PTSD experience attenuated positive mood reactivity to rewarding or pleasurable events (Litz & Gray, 2002). Nicotine administration stimulates brain reward systems (Corrigall & Coen, 1991) and produces mild positive mood enhancement among nondeprived smokers during exposure to a positive psychological stimulus (Cook, Spring, & McChargue, 2007). It is feasible that smokers with PTSD may bolster their ability to experience positive emotions in response to environmental rewards by simultaneously administering nicotine. We posit that nicotine's positive mood enhancing effects may be particularly salient for individuals high in emotional numbing symptoms of PTSD, for whom deficits in positive affect are prominent.

The present study aims to examine the association between overall PTSD symptom severity and smoking status and heaviness in Iraq and Afghanistan combat veterans, followed by examination of the individual contribution to smoking by each of the four factorially derived symptom clusters of PTSD. Given evidence that PTSD may be strongly associated with heavy smoking (Beckham et al., 1997), we examined associations between PTSD symptoms and light, moderate, and heavy smoking (vs. no smoking). Specifically, we hypothesized that higher levels of overall PTSD symptom severity would increase the likelihood of smoking among veterans and that overall PTSD symptom severity would be more strongly related to heavy smoking than to light or moderate levels of smoking. We also hypothesized that higher levels of the effortful avoidance and emotional numbing factors of PTSD would be associated with a greater likelihood of heavier smoking.

Methods

Participants

Subjects. The sample was composed of Iraq and Afghanistan combat veterans ($N = 439$) referred for mental health services at VA Puget Sound Health Care System from 2004 to 2007. The study protocol was approved by the University of Washington Internal Review Board and the Research and Development Committee of VA Puget Sound Health Care System.

The mean age of the sample was 32.35 years ($SD = 9.06$) and most were male (90.2%) and Caucasian (72.4%). The majority (78.8%) reported serving in the Army or the National Guard and 69.5% reported that they were on reserve status when called to duty. The majority of the subjects were not married (55%) and 45% indicated that they were married or remarried. The average years of education were 13.49 ($SD = 2.05$). Approximately half (49.5%) of the veterans referred to mental health services screened positive for PTSD and 29.4% of the sample reported daily smoking. In addition to PTSD, patients screened positive for a range of other mental health problems. From the sample as a whole, 37% screened positive for Major Depressive Disorder, 24.6% for alcohol abuse, and 4% for drug abuse.

Materials

Smoking status. When asked via an intake questionnaire about number of cigarettes smoked daily, veterans responded to one of the following: no smoking, 1–9, 10–19, 20–29, or ≥30 cigarettes. These categories were collapsed into the following: (a) nonsmoker ($n = 301$), (b) 1–9 cigarettes smoked

daily (light smoker, $n = 51$), (c) 10–19 cigarettes smoked daily (moderate smoker, $n = 55$), and (d) ≥20 cigarettes smoked daily (heavy smoker, $n = 23$). Those who reported smoking >30 cigarettes were not retained in a separate category given the small sample size and consequent power limitations.

Posttraumatic stress disorder. The PTSD Checklist Military Version (PCL-M; Weathers, Litz, Herman, Huska, & Keane, 1993) is a 17-item self-report instrument that asks veterans to rate the degree to which they have been bothered by the *DSM-IV* symptoms of PTSD (1 = *not at all*; 5 = *extremely*). The PCL-M total is sum scored, with higher scores indicating greater PTSD symptom severity. The instrument demonstrates strong psychometric properties (Weathers et al., 1993). Screening criteria for PTSD require a PCL-M global score of 50 and the presence of the symptoms endorsed at a moderate or high level across the symptom clusters per the *DSM-IV* algorithm for diagnosing PTSD.

The PCL-M has previously been associated with smoking outcomes (Vasterling et al., 2008) and has been shown to conform to the four-factor structure examined in the present study (Asmundson et al., 2000). In the current sample, each of the four factors demonstrated good internal consistency. The five-item reexperiencing cluster includes symptoms such as "Repeated, disturbing memories, thoughts, or images of a stressful military experience" ($\alpha = .94$). The two-item effortful avoidance cluster includes items such as "Avoiding thinking about or talking about a stressful military experience" ($\alpha = .89$). The five-item emotional numbing cluster includes items such as "Feeling emotionally numb or being unable to have loving feelings for those close to you" ($\alpha = .90$). The five-item hyperarousal cluster includes items such as "Being 'super alert' or watchful or on guard" ($\alpha = .89$). In addition, the overall PCL-M demonstrated a strong internal reliability: $\alpha = .95$.

Depression. The Patient History Questionnaire (PHQ; Spitzer, Kroenke, & Williams, 1999) is a self-report measure based on the clinician-administered Primary Care Evaluation of Mental Disorders (Spitzer et al., 1999) that assesses for a variety of mental disorder symptoms, including symptoms of depression. The PHQ demonstrates high specificity, accurately differentiating depression symptoms from other mental disorder symptoms, such as anxiety (Spitzer et al., 1999). Overall, the PHQ demonstrates validity for assessment of depression in primary care settings (Backenstrass et al., 2006).

Results

Analytic overview

Multinomial logistic regression was chosen to accommodate distribution of the data and a three-level outcome, in which the likelihood of being a nonsmoker was compared with (a) light, (b) moderate, and (c) heavy smoking. Rate of smoking (light, moderate, and heavy smoking) was also collapsed in order to examine whether PTSD symptom severity predicted any smoking (nonsmoker vs. smoker). PTSD and its factors (reexperiencing, effortful avoidance, emotional numbing, and hypervigilance) were examined in single models as continuous variables predicting smoking. Twenty-seven percent of the sample did not meet criteria for PTSD, 20% screened positive for subthreshold PTSD, and 53% screened positive for PTSD. There is a strong evidence that PTSD is best understood as a dimensional construct rather than a discrete disorder (Broman-Fulks et al., 2006), in part, because subthreshold PTSD has been associated with significant functional impairment (Jakupcak et al., 2007; Mylle & Maes, 2004). Dichotomizing the PTSD Checklist into presence versus absence of PTSD results in loss of meaningful variance, particularly among those with subthreshold PTSD. Thus, we utilized the full range of the PTSD Checklist variance to examine the influence of PTSD symptom severity on smoking. Finally, the PTSD Checklist, its subscales, and the Patient History Questionnaire were standardized ($M = 0$, $SD = 1$) to increase interpretability of the observed effects.

Preliminary analyses

Descriptive analyses showed that 33% of veterans who screened positive for PTSD reported daily smoking. Nonsmokers and smokers (light, moderate, heavy) were compared on sociodemographic variables (age, education, and race) using one-way analyses of variance for continuously scaled variables and chi-square tests for dichotomous variables. Only differences in age emerged, $F(1, 419) = 3.58$, $p = .01$; both moderate ($M = 35.4$, $SD = 8.57$) and heavy ($M = 33.91$, $SD = 9.99$) smokers were significantly older than light smokers ($M = 30.00$, $SD = 8.15$). Thus, age was retained as a covariate in all statistical models. In addition, intercorrelations between the PTSD Checklist subfactors were examined. As expected, reexperiencing, effortful avoidance, emotional numbing, and hyperarousal were all positively and significantly correlated (r ranged from .74 to .83, $p < .01$).

Primary analyses

First, binary logistic regression examined the influence of PTSD symptom severity on the likelihood of endorsing any level of smoking (nonsmoker vs. smoker). Results showed that higher PTSD symptom severity was associated with an increased likelihood of endorsing any level of smoking (Wald = 4.69, $p = .03$, odds ratio [*OR*] = 1.27). Next, multinomial logistic regression examined the influence of PTSD symptom severity on smoking heaviness. Nonsmokers were compared with smokers who endorsed light, moderate, and heavy smoking. Multinomial logistic regression, controlling for age, showed that PTSD symptom severity significantly predicted heavy smoking (Wald = 4.56, $p = .03$, $OR = 1.65$) but did not predict light ($p = .60$) or moderate smoking ($p = .08$). For every one point *SD* increase in PTSD symptom severity, the likelihood of endorsing heavy smoking increased 1.65 times.

Next, multinomial logistic regression examined the influence of the severity of the four PTSD factors (reexperiencing, effortful avoidance, emotional numbing, and hyperarousal) on smoking heaviness. Single model analyses showed that PTSD factors were not associated with light or moderate smoking (all p values = *nonsignificant*). However, veterans endorsing higher levels of the emotional numbing factor were more likely to endorse being a heavy smoker versus a nonsmoker (Wald = 6.49, $p = .01$, $OR = 1.81$). In addition, more emotionally numb veterans were more likely to endorse heavy versus light smoking (Wald = 4.23, $p = .04$, $OR = 1.70$); for every one point *SD* increase in emotional numbing, veterans were 1.8 times more likely to endorse heavy smoking than nonsmoking and 1.7 times more likely to endorse heavy smoking than light smoking.

Exploratory analyses

Given the comorbidity between PTSD and depression (Orsillo et al., 1996), we examined whether emotional numbing was a

proxy risk factor for depression (Kraemer, Kazdin, Offord, & Kupfer, 2001). As described by Kraemer et al. (2001), a proxy risk factor is one which predicts an outcome by virtue of its strong correlation with another variable. In this case, the influence of emotional numbing on smoking may be better explained by depression. The emotional numbing component of PTSD and depression was highly correlated ($r = .84$), suggesting that two factors measure similar constructs. First, we tested the individual influence of depression on smoking. Multinomial logistic regression showed that depression predicted heavy smoking (Wald = 8.13, $p = .004$, $OR = 1.90$). When both emotional numbing and depression were entered into the same regression model, neither depression ($p = .16$) nor emotional numbing ($p = .80$) predicted heavy smoking. Results suggest that common variance associated with both depression and emotional numbing cancel out individual contributions toward heavy smoking.

Discussion

Our results showed that veterans reporting higher levels of overall PTSD symptom severity were more likely to report smoking heavily ($OR = 1.65$) when compared with mental health patients with lower levels of PTSD symptoms. When the relationship between PTSD symptom clusters and smoking was examined, the emotional numbing factor predicted heavy smoking ($OR = 1.81$); all other components of PTSD were unrelated to smoking. The observed effects of PTSD symptom severity and emotional numbing and smoking are relatively large when considering the continuous nature of the predictor variables. In other words, the effect size represents the increased likelihood of smoking heavily (≥20 cigarettes) for every one point *SD* increase in PTSD symptom severity or emotional numbing. Overall, results suggest that the relationship between PTSD and heavy smoking may be influenced more by emotional numbing symptoms than other symptom clusters of PTSD.

Our finding that PTSD symptom severity and emotional numbing predicted only heavy smoking is consistent with other research on smoking heaviness in PTSD. Smokers with PTSD smoke more heavily (>25 cigarettes/day) when compared with smokers without PTSD (Beckham et al., 2007). Moreover, Buckley, Susannah, Bedard, and Dewulf (2004) found that smokers with PTSD were heavy smokers, with 73% smoking at least 20 cigarettes/day. Perhaps lower doses of nicotine are not potent enough to regulate PTSD-specific psychobiological vulnerabilities. It has been posited that higher levels of nicotine may also be necessary to mitigate symptoms associated with panic disorder, another anxiety disorder linked with heavy smoking (McCabe et al., 2004). Similar to panic disorder, greater PTSD symptom severity may require more frequent administration of nicotine to offset prolonged and intense PTSD-related affect. Those with lower levels of PTSD may also smoke to regulate mood but may do so with lighter nicotine dosing.

Surprisingly, the effortful avoidance factor of PTSD was not associated with smoking. Effortful avoidance was assessed by items such as "I go out of my way to avoid talking or thinking about my trauma." Effortful avoidance is conceptualized as any strategic attempt to avoid thinking of traumatic events or feeling negative emotions (Foa et al., 1992). Our results suggest that veterans may not explicitly use cigarette smoking to distract themselves from negative thoughts or emotions. Instead, emotional numbing symptoms predicted heavy smoking, a finding consistent with recent data showing that smokers with PTSD endorsed higher emotional numbing than nonsmokers with PTSD (Kirby et al., 2008). Emotional numbing includes symptoms such as restricted positive affect, disinterest in activities, and emotional detachment from others. In contrast to strategic, effortful behaviors associated with the effortful avoidance factor, emotional numbing is believed to be mediated by automatic mechanisms (Foa et al., 1992). Instead of smoking as a conscious strategy aimed at avoiding trauma-related thoughts/emotions, nicotine self-administration may serve to counteract more automatic blunting of emotion following trauma exposure. When considering our finding that smokers high in emotional numbing were more likely to endorse heavy versus light smoking, heavy nicotine dosing may be required to help regulate automatic processes underlying emotional numbing. It may be that over time and with more experience smoking, smokers with PTSD become conditioned to self-administer high doses of nicotine to elevate their blunted affective experience. This is in keeping with literature suggesting that smokers may respond to preconscious stimuli that indicate the need for nicotine in the system (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004).

It is feasible that elevated emotional numbing symptoms in PTSD tend to co-occur with depression. In the present study, emotional numbing and depressive symptoms were highly correlated, raising the possibility that emotional numbing is a proxy risk factor for depression (Kraemer et al., 2001). As such, the influence between emotional numbing and smoking among veterans with PTSD would be explained primarily by depression. However, when both emotional numbing and major depressive disorder (MDD) were placed in the same regression model, neither predicted smoking. It appears that common variance associated with both depression and emotional numbing cancel out individual contributions toward heavy smoking. Similar to PTSD, blunted positive emotions may represent an important mechanism influencing the relationship between depression and smoking.

We postulate that mechanisms maintaining smoking in both depression and PTSD are similar. Deficient positive affect, which is associated with anhedonia and emotional numbing is a core component of both MDD and PTSD (Hasler, Drevets, Manji, & Charney, 2004; Litz & Gray, 2002). It has been hypothesized that smoking may serve to elevate blunted positive emotions that are central to the emotional numbing component of PTSD (Cook et al., 2007) and depression (Leventhal et al., 2008; Spring et al., 2007). Leventhal et al. (2008) found that only anhedonia/low positive affect and not other dimensions of depression predicted cessation-induced withdrawal. In addition, baseline anhedonia/low positive affect decreased the likelihood of maintaining abstinence. Although outcomes in the present study and the work of Leventhal et al. are different (daily smoking vs. smoking cessation outcomes), results suggest that a common behavioral phenotype (i.e., blunted positive emotions) may underlie smoking among different psychiatric disorders. Evidence that nicotine selectively enhances positive mood among anhedonic smokers (Cook et al., 2007) supports the possibility that smoking helps regulate psychopathological positive mood deficits central to both PTSD and depression.

Evidence for the influence of emotional numbing in smoking behavior has treatment implications. Smokers with PTSD and other anxiety disorders have been shown to be at risk for early smoking relapse when compared with smokers without other Axis I disorders (Zvolensky et al., 2009), and efforts to understand mechanisms influencing relapse may improve smoking outcomes. If enhancement of positive emotions proves to be an important mechanism motivating smoking in smokers with PTSD, the greatest challenge in quitting smoking may be helping smokers find behavioral or pharmacological options to activate underresponsive brain reward systems. Behavioral activation (BA) is an efficacious treatment for MDD (Dimidjian et al., 2006). By targeting anhedonia through promoting engagement in meaningful and rewarding activities, BA may help counteract blunted reward functioning in the absence of smoking. In addition, pharmacotherapy treatments such as fluoxetine (Cook et al., 2004) and bupropion (Shiffman et al., 2000) have been shown to elevate positive affect during smoking cessation.

Interpretations are limited by the following. Measurements of PTSD and depression were based on self-report instruments rather than clinician-administered structured interviews. As such, the assessment of these factors may be subject to memory or recall bias as well as shared method variance. In addition, effects associated with treatment-seeking populations have been shown to be exaggerated (Maric et al., 2004), potentially limiting generalizabilty of findings to nontreatment-seeking smokers with PTSD. Confidence in effects observed in the present study may be increased by future use of clinician-administered diagnostic interviews as well as by examining effects among a nontreatment-seeking population. Given that the sample was composed primarily of male veterans, it is also important to examine whether results generalize to women with PTSD.

In addition, the cross-sectional design employed in the present study precludes our ability to identify causal relations between smoking and PTSD, limiting our interpretation to the observation that PTSD and smoking tend to co-occur in this population. Although we speculate that smoking was initiated in the current sample to achieve relief from symptoms of PTSD (e.g., self-medication model), there is also evidence that those who initiate smoking may be more vulnerable to developing PTSD (Koenen et al., 2005). We also were unable to differentiate never smokers from previous smokers, two groups who may have different levels of vulnerability to developing PTSD. Longitudinal research is needed to better elucidate the etiology of the relationship between PTSD and smoking in this population. Finally, participants in this convenience sample were not randomly selected from Iraq and Afghanistan combat veterans, and it cannot be assumed that results will generalize to other veterans.

In sum, PTSD in Iraq and Afghanistan combat veterans increased the likelihood of endorsing heavy smoking. Although Vietnam combat veterans with PTSD report even higher rates of smoking (53%; Beckham et al., 1997) than were reported here (33%), PTSD has been shown to predict late-onset smoking (Koenen et al., 2006). Examining more recently deployed veterans provides a unique opportunity to examine the likelihood of smoking in a population recently exposed to trauma. Perhaps rates of smoking in Iraq and Afghanistan veterans may continue to grow over time as returning veterans attempt to cope with chronic PTSD symptoms. If veterans with PTSD initiate smoking, in part, to overcome emotional numbing, early PTSD interventions may decrease likelihood of initiating smoking by providing alternative mood regulation strategies. For veterans who already smoke, provision of early smoking interventions, particularly with an emphasis on enhancement of positive emotions, may be crucial in effectively treating addiction to nicotine.

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Declaration of Interests

No potential conflicts of interest on the part of any of the authors exist with regard to research reported in this article.

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