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Authors

Salas, Joanne
Gebauer, Sarah
Gillis, Auston
et al.

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Original Investigation

Increased Smoking Cessation Among Veterans With Large Decreases in Posttraumatic Stress Disorder Severity

Joanne Salas MPH[1,2], Sarah Gebauer MD[1,2], Auston Gillis BA[1], Carissa van den Berk-Clark PhD[1], F. David Schneider MD[3], Paula P. Schnurr PhD[4], Matthew J. Friedman MD, PhD[4], Sonya B. Norman PhD[5], Peter W. Tuerk PhD[6], Beth E. Cohen MD[7], Patrick J. Lustman PhD[8,9], Jeffrey F. Scherrer PhD[1,2]

[1]Department of Family and Community Medicine, Saint Louis University School of Medicine, St. Louis, MO, USA; [2]Harry S. Truman Veterans Administration Medical Center, Research Service, Columbia, MO, USA; [3]Department of Family and Community Medicine, University of Texas Southwestern Medical Center, Dallas, TX, USA; [4]National Center for PTSD and Department of Psychiatry, Geisel School of Medicine at Dartmouth, Hanover, NH, USA; [5]National Center for PTSD and Department of Psychiatry, University of California San Diego, San Diego, CA, USA; [6]Sheila C. Johnson Center for Clinical Services, Department of Human Services, University of Virginia, Charlottesville, VA, USA; [7]Department of Medicine, University of California San Francisco School of Medicine and San Francisco VAMC, San Francisco, CA, USA; [8]Department of Psychiatry, Washington University School of Medicine, St. Louis, MO, USA; [9]The Bell Street Clinic Opioid Addiction Treatment Programs, VA St. Louis Healthcare System, St. Louis, MO, USA

Corresponding Author: Joanne Salas, MPH, Family and Community Medicine, Saint Louis University School of Medicine, 1402 N. Grand Blvd, St. Louis, MO 63104, USA. Telephone: 314-977-8497; Fax: 314-977-5268; E-mail: joanne.salas@health.slu.edu

Abstract

Introduction: Improvement in posttraumatic stress disorder (PTSD) is associated with better health behavior such as better medication adherence and greater use of nutrition and weight loss programs. However, it is not known if reducing PTSD severity is associated with smoking cessation, a poor health behavior common in patients with PTSD.

Aims and Methods: Veterans Health Affairs (VHA) medical record data (2008–2015) were used to identify patients with PTSD diagnosed in specialty care. Clinically meaningful PTSD improvement was defined as ≥20 point PTSD Checklist (PCL) decrease from the first PCL ≥50 and the last available PCL within 12 months and at least 8 weeks later. The association between clinically meaningful PTSD improvement and smoking cessation within 2 years after baseline among 449 smokers was estimated in Cox proportional hazard models. Entropy balancing controlled for confounding.

Results: On average, patients were 39.4 (SD = 12.9) years of age, 86.6% were male and 71.5% were white. We observed clinically meaningful PTSD improvement in 19.8% of participants. Overall, 19.4% quit smoking in year 1 and 16.6% in year 2. More patients with versus without clinically meaningful PTSD improvement stopped smoking ($n = 36$, cumulative incidence = 40.5% vs. 111, cumulative incidence = 30.8%, respectively). After controlling for confounding, patients with versus without clinically meaningful PTSD improvement were more likely to stop smoking within 2 years (hazard ratio = 1.57; 95% confidence interval: 1.04–2.36).

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Conclusions: Patients with clinically meaningful PTSD improvement were significantly more likely to stop smoking. Further research should determine if targeted interventions are needed or whether improvement in PTSD symptoms is sufficient to enable smoking cessation.
Implications: Patients with PTSD are more likely to develop chronic health conditions such as heart disease and diabetes. Poor health behaviors, including smoking, partly explain the risk for chronic disease in this patient population. Our results demonstrate that clinically meaningful PTSD improvement is followed by greater likelihood of smoking cessation. Thus, PTSD treatment may enable healthier behaviors and reduce risk for smoking-related disease.

Introduction

An estimated 14% of adults are current smokers[1]; however, the prevalence of current smoking in individuals with posttraumatic stress disorder (PTSD) is significantly higher than the national average, with estimates ranging from 30% to 40%.[2] A recent meta-analysis observed that adults with PTSD are more likely to be smokers,[3] with evidence that those with trauma and PTSD are over three times more likely to report current smoking than those without a history of trauma.[4] It has been observed that smokers with PTSD have 32%–39% lower odds of smoking cessation compared with smokers without PTSD,[5] despite patients having a desire and intent to quit smoking.[6] During smoking cessation trials, individuals with PTSD are more likely to experience relapse after quit attempts.[7,8]

Smoking may serve as a means of alleviating some PTSD symptoms such as reexperiencing, avoidance, emotional numbing, and hyperarousal.[7,9–12] The "self-medication hypothesis," which states that individuals with PTSD use nicotine or other substances to cope with PTSD symptoms, has been well documented.[13–15] Research has also revealed that increased PTSD severity is associated with increased likelihood of smoking, as well as increased intensity of smoking.[2,16] The relationship does not appear to be bidirectional. The severity of PTSD symptoms significantly predict the amount of cigarette smoking, while cigarette smoking did not predict PTSD symptom severity.[17]

Randomized clinical trials combining PTSD psychotherapy and smoking cessation treatments have demonstrated efficacy. Such integrated care has been associated with nearly a 2.5-fold increase in self-reported prolonged smoking abstinence over the course of 18 months compared with attendance of a smoking cessation clinic alone.[18] A meta-analysis of smoking cessation treatments for individuals with PTSD observed that integrating smoking cessation treatment into PTSD treatment may increase access to smoking cessation counseling, which results in a higher quit rate in individuals receiving integrated treatment.[19] Smoking cessation approaches that take PTSD symptoms into account appear to result in higher quit rates than the traditional adjunctive treatment for smokers with PTSD.

Integrated smoking cessation in PTSD treatment shows promise for smoking cessation, but clinical trials do not include the full range of patients with PTSD and often exclude those too ill to participate, eg, patients with multiple comorbidities or severe mental illness and suicidal ideation. While integrated care may be ideal for patients with PTSD seeking to quit smoking, not all patients have access to such treatment.

This study is designed to determine if clinically meaningful PTSD symptom decrease is associated with greater likelihood of smoking cessation using data from medical records which does not exclude patients ineligible or unwilling to participate in clinical trials. It is also not clear from existing studies if improvement in PTSD alone, independent of smoking cessation treatment, may enable quitting. If true, this would inform the structure of combined treatments, as well as provide additional motivation to those considering initiating PTSD treatment. Given more severe PTSD symptoms are associated with greater cigarette consumption, we hypothesized that patients with large, clinically meaningful PTSD symptom improvement, compared with those without improvement or worsening symptoms, would be more likely to stop smoking in a large cohort of Veterans with PTSD.

Methods

All study procedures were approved by participating institutions as expedited with wavier of informed consent and HIPAA authorization because the investigators did not maintain patient identifiers. Linking chart abstracted data with Veterans Health Affairs (VHA) administrative data was performed by VHA data managers from the VA Informatics and Computing Infrastructure (VINCI).

VHA administrative medical record data were obtained to create variables. The data contain ICD-9 codes, vital signs, type of clinic encounter, pharmacy fills, laboratory results, and health factor data. The PTSD Checklist (PCL) score was used to measure PTSD severity. PCL scores were available in the administrative medical record data and supplemented with chart abstraction for a random sample of 5916 patients aged 18–70 years with a PTSD diagnosis in FY2008 to FY2012. Abt Associates' (www.abtassociates.com) trained medical chart abstractors conducted chart abstraction. Details of chart abstraction have been previously reported.[20–22]

The total observation period was from fiscal year (FY)2008 to FY2015. The eligible cohort was created by first selecting PTSD cases between fiscal (FY)2008 to FY2012. Patients must have had ≥1 PCL score during this time. We then selected the first PCL ≥50 which indicates current PTSD. We created an exposure year to measure change in PCL scores. The exposure year was the 12 months following first PCL ≥50, a value consistent with current PTSD. Change in PCL was measured by taking the difference from the first PCL and the last PCL, which had to occur within the 12-month exposure year and at least 8 weeks after the first PCL. The index date, "baseline," was the end of the exposure year. Eligible patients could enter the cohort anytime between FY2009 to FY2013. Since smoking cessation was defined within the 2 years after baseline, this allowed all eligible patients to have 2 years of follow-up. We have used this method to define an eligible cohort in prior studies.[20–23] We then identified patients who were current smokers in the year prior to index (n = 730), and then limited the sample to persistent smokers who had no indication that they had stopped smoking in the year prior to index (n = 492). Thus, patients who quit and started smoking again in the year prior to index were not eligible. Last, patients had to have a

measure of smoking status from the VHA health factor data in the 2-year follow-up, which resulted in an eligible sample of 449 patients. The sampling approach is illustrated in Figure 1.

Variable Definitions

Detailed variable definitions are shown in Supplementary e-Table 1. Patients with PTSD were identified by the presence of ICD-9 code 309.81 on two separate outpatient visits within a 12-month period or one inpatient stay. This diagnostic algorithm is consistent with an 82% positive predictive value when compared with a PCL score ≥50,[24] and 79.4% agreement with the Structured Clinical Interview for DSM-IV lifetime PTSD diagnosis.[25]

Exposure

Clinically meaningful PTSD improvement was defined by a ≥20 point PCL decrease in the exposure year. Those with <20 point decrease or an increase were classified as not having a clinically meaningful improvement. While 10 points have been suggested as indicating clinically meaningful improvement,[26] we selected an a priori threshold shown to be associated with lower risk for type 2 diabetes,[22] hypertension,[27] and improved health behaviors such as use of weight loss/nutrition programs,[21] use of substance use disorder treatment,[23] and antidepressant medication adherence.[20]

Outcomes

Smoking Cessation

The VHA uses clinical reminder software for various performance measures and all of this information is consolidated into a national health factors database.[28] Current smoking in the year before index was defined by health factor data indicating that a patient is a "current smoker"[28,29] or by an ICD-9 code for nicotine dependence (V15.82, 305.1). We defined smoking cessation during a 2-year follow-up using health factors data values indicating "non-smoker" or "former smoker." The Veterans Aging Cohort Study (VACS) (https://medicine.yale.edu/intmed/vacs/) provides a health factor smoking lookup table on its web site that includes "healthfactortype," a VHA variable in the health factors dataset providing a description of the smoking health factor, and "smokingfactor," a variable generated by VACS to classify each health factor type into current, former, never, or non-smoking.[29] The lookup table was merged with all health factor data pulled for this study. "Healthfactortype" values that were not yet classified by the lookup table available at the time of this study were explored and placed into pertinent "smokingfactor" categories. Example "healthfactortype" values for each category are: (1) Current smoker: "Current Smoker," "Decline smoking cessation meds," "Advise to quit," "Tobacco counsel," "Referred patient to smoking cessation class," "Refused smoking cessation"; (2) Former smoker: "Quit smoking <1YR," "Previous smoker," "Quit smoking," "Quit tobacco," "Tobacco former user"; (3) Never smoker: "Never used tobacco," "Lifetime non-smoker"; and (4) Non-smoking: "Current non-smoker," "Non-tobacco user at this time." Prior studies have shown that the administrative medical record health factor data have moderate (kappa = 0.49)[30] to good agreement (kappa = 0.66)[29] with smoking status obtained via survey.

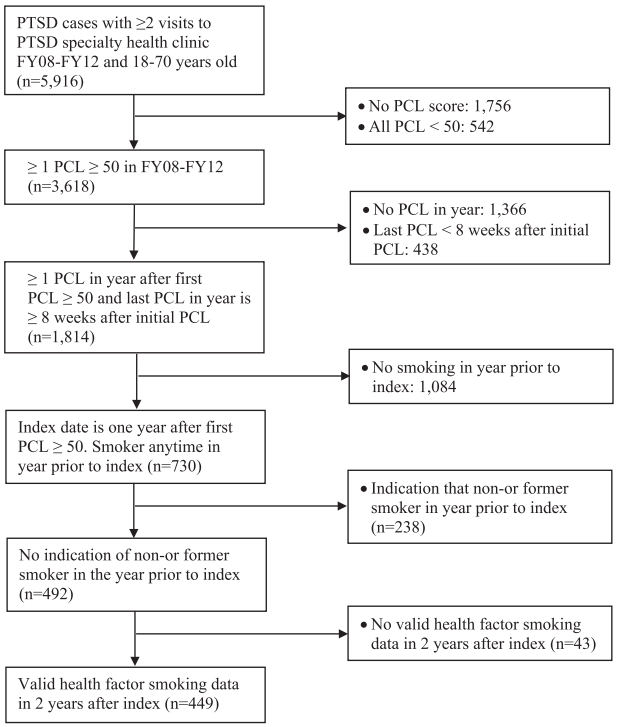

Figure 1. Sampling. PCL = PTSD Checklist; PTSD = posttraumatic stress disorder.

Follow-up Time

Follow-up time was defined as months from index date to either smoking cessation or censoring. Patients without an indication of smoking cessation were censored at last available smoking health factor indicator.

Covariates

We controlled for potential confounding associated with sociodemographic factors, geographic region, health services use, and psychiatric and physical comorbidities. Sociodemographic covariates included age, gender, and race (White, Black, Other). Because clinical practice varies across the United States, we adjusted for region (Northeast, Midwest, South, West). To control for detection bias, we computed the average number of health care encounters per month and used the distribution to categorize high health care use (top 25th percentile) versus not-high. To adjust for seeking mental health care which could be correlated with use of smoking cessation treatment and is associated with PTSD improvement, we included a variable describing the receipt of minimally adequate PTSD psychotherapy. This decision was meant to help isolate the link between clinically meaningful PTSD symptom improvement and smoking cessation.

Psychiatric covariates included severe PTSD defined as an initial PCL score ≥70, diagnoses of depression, any anxiety disorder, and alcohol and drug abuse/dependence. We controlled for asthma and the Charlson–Romano comorbidity index.[31] Detailed variable definitions are shown in Supplementary e-Table 1. All covariates were treated as time invariant and measured from the start of observation (FY08) to index date, except for PTSD psychotherapy duration which was measured during the exposure year.

Analytic Approach

Entropy Balance and Weighting

Primary outcome analyses used entropy balancing and weighting (e-balance) to balance the distribution of confounders between patients with and without a clinically meaningful PCL decrease.[32,33]

This method was chosen over propensity scores and inverse probability of exposure weighting because it does not rely on correctly specifying a propensity score model and better balance between exposure groups can be achieved. This method reweights the PCL decrease <20 group by deriving weights so that specified covariate moments like the mean and standard deviation can match the PCL decrease ≥20 group. Balance was evaluated using the standardized mean difference percent (SMD% = 100 × SMD), where well balanced covariates have an SMD% <10% (SMD <0.1).[34] Stata 16 (StataCorpt, College Station, TX) was used to apply e-balance methods and create weights.

Main Analyses

All main analyses were performed with SAS v9.4 (SAS Institute, Cary, NC) at a two-tailed alpha = 0.05. Bivariate analyses in unweighted data used chi-square tests and independent samples *t* tests to measure the association between confounders and PCL decrease ≥20 versus <20, with SMD% used as an effect size measure. SMD% was also calculated in weighted data to assess whether confounders were balanced or equal between the two groups.

Unweighted and entropy balance weighted Cox proportional hazards models calculated hazard ratios (HRs) and 95% confidence intervals (CIs) for the relationship of meaningful PCL decrease and time to smoking cessation in the 2 years after index. The weighted model was extended to include three separate time-dependent smoking cessation treatment variables that occurred from index to the end of follow-up (see Supplementary e-Table 1): (1) smoking cessation treatment behavioral therapy measured by the presence of clinic stop codes; (2) nicotine replacement therapy measured using fills for nicotine gum, lozenges, patches, or inhalants; and (3) pharmacotherapy measured using fills for varenicline (Chantix) or bupropion (Zyban). This assessed whether increased use of smoking cessation treatment following PCL improvement may be a possible mechanism in the relationship of PCL improvement and smoking cessation. Separately, we used survival models to estimate unadjusted estimates of the association between PCL improvement and use of each type of treatment.

All weighted models used robust, sandwich-type variance estimators to calculate CIs and *p* values.[34] The proportional hazard assumption was tested in all models by including a time-dependent interaction term of PCL decrease and log(follow-up time), where a $p < .05$ for this term indicates violation. In all models, the assumption was met ($p > .70$).

Subgroup Analysis

Because depression and alcohol abuse/dependence are common comorbid conditions among patients with PTSD, we stratified analyses by depression status and separately by alcohol abuse/dependence status. Another subgroup descriptive analysis was conducted to measure whether smoking cessation was successful, which we defined as no evidence of return to smoking in the 12 months after the quit date. For this analysis, we identified 140 eligible patients who quit smoking by 9/30/2014, which allowed for a year to measure return to smoking.

Results

On average, patients were 39.4 (SD = 12.9) years of age, 86.6% were male and 71.5% were white (Table 1). Older age was significantly associated with experiencing a clinically meaningful PTSD improvement. Patients with versus without clinically meaningful improvement had significantly ($p < .0001$) lower average PCL scores at the end of the exposure year (mean = 35.1, SD ± 10.5 vs. mean = 62.2 SD ± 12.1). Alcohol abuse/dependence ($p = .042$) and receipt of minimally adequate PTSD psychotherapy ($p = .004$) were significantly more prevalent among patients with versus without clinically meaningful PTSD improvement.

Table 2 shows that 40.5% of patients with a clinically meaningful PTSD improvement versus 30.8% without stopped smoking in the 2 years after index. The overall incidence rate of smoking cessation in the 2 years after index was 271.4/1000 person-years (PY). The incidence rate of smoking cessation was 250.5/1000PY in patients without clinically meaningful improvement compared with 365.3/1000PY among patients with clinically meaningful PTSD improvement.

In the first year after index, 87 (19.4%) patients had evidence of smoking cessation, and among the remaining 362 patients without cessation in the first year, 60 (16.6%) had evidence of cessation in the second year after index.

As illustrated in Supplementary e-Figure 1, most confounding factors showed imbalance (SMD >10%) between the two groups. However, entropy balancing was very successful in removing differences in the distribution of potential confounding factors between those with and without meaningful PTSD improvement (all weighted SMD% <10%), with most weighted SMDs ≤1.0%.

As shown in Table 2, prior to controlling for confounding, patients with versus without clinically meaningful PTSD improvement were significantly more likely to quit smoking in 2 years (HR = 1.48; 95% CI: 1.02–2.15). After controlling for confounding in weighted data, this association was slightly stronger (HR = 1.57; 95% CI: 1.04–2.36). This association remained largely unchanged (HR = 1.53; 95% CI: 1.01–2.31) after accounting for use of smoking cessation treatment after index. Among patients with clinically significant improvement in PTSD, 11 (12.4%) had smoking cessation behavioral therapy, 22 (24.7%) had nicotine replacement, and 16 (18.0%) had nicotine pharmacotherapy in follow-up (results not shown). Among patients without improvement, 25 (6.9%) had smoking cessation behavioral therapy, 109 (30.3%) had nicotine replacement therapy, and 43 (11.9%) had nicotine pharmacotherapy in follow-up (results not shown). Clinically meaningful PTSD improvement was unrelated to initiation of nicotine replacement therapy in follow-up (crude HR = 0.85; 95% CI: 0.54–1.34), however, there was a trend for meaningful improvement to be related to increased risk of initiation of behavioral therapy (crude HR = 2.01; 95% CI: 0.99–4.09) and pharmacotherapy (crude HR = 1.72; 95% CI: 0.97–3.05). Behavioral therapy (crude HR = 1.32; 95% CI: 0.71–2.45) and nicotine replacement therapy (crude HR = 1.11; 95% CI: 0.75–1.66) were unrelated to likelihood of smoking cessation, however, pharmacotherapy in follow-up was related to a 74% increased likelihood of smoking cessation (crude HR = 1.74; 95% CI: 1.10–2.74).

Results of subgroup analyses by depression and alcohol abuse/dependence status are shown in Tables 3 and 4. As shown in Table 3, we observed patients with a clinically meaningful improvement versus without had a higher incidence of smoking cessation in both those with and without depression. However, the difference in cumulative incidence and incidence rate of smoking cessation between those with versus without a clinically meaningful improvement was much larger in those without depression. Among those without

Table 1. Sample Characteristics Overall and by PCL Decrease for PTSD Cases, Veterans Age 18–70 Years and Smokers (*n* = 449)

Variable, *n* (%) or mean (±SD)	Overall (*n* = 449)	PCL dec <20 (*n* = 360)	PCL dec ≥20 (*n* = 89)	*p*
Age (years), mean (±SD)	39.4 (±12.9)	38.6 (±12.8)	42.5 (±13.1)	.012
Male gender, *n* (%)	389 (86.6)	313 (86.9)	76 (85.4)	.700
Race, *n* (%)				
White	321 (71.5)	254 (70.6)	67 (75.3)	
Black	87 (19.4)	71 (19.7)	16 (18.0)	.598
Other	41 (9.1)	35 (9.7)	6 (6.7)	
Region, *n* (%)				
Northeast	8 (1.8)	7 (1.9)	<5	
Midwest	128 (28.5)	97 (26.9)	31 (34.8)	
South	131 (29.2)	99 (27.5)	32 (36.0)	.050
West	182 (40.5)	157 (43.6)	25 (28.1)	
High primary HCU, *n* (%)	112 (24.9)	96 (26.7)	16 (18.0)	.090
First PCL severe (≥70), *n* (%)	141 (31.4)	109 (30.3)	32 (36.0)	.301
First PCL, mean (±SD)	64.8 (±8.7)	64.3 (±8.8)	66.7 (±8.0)	.019
Last PCL, mean (±SD)	56.8 (±16.0)	62.2 (±12.1)	35.1 (±10.5)	<.0001
Psychiatric comorbidities and treatments[a]				
Depression, *n* (%)	342 (76.2)	273 (75.8)	69 (77.5)	.737
Other anxiety[b], *n* (%)	123 (27.4)	94 (26.1)	29 (32.6)	.220
Sleep disorder, *n* (%)	217 (48.3)	179 (49.7)	38 (42.7)	.235
Alcohol abuse/dependence, *n* (%)	260 (57.9)	200 (55.6)	60 (67.4)	.042
Drug abuse/dependence, *n* (%)	192 (42.8)	146 (40.6)	46 (51.7)	.057
Adequate PTSD psychotherapy, *n* (%)[c]	186 (41.4)	137 (38.1)	49 (55.1)	.004
Physical comorbidities[a]				
Asthma, *n* (%)	38 (8.5)	26 (7.2)	12 (13.5)	.057
Comorbidity index, *n* (%)				
0	309 (68.8)	251 (69.7)	58 (65.2)	
1–2	74 (16.5)	59 (16.4)	15 (16.9)	.598
≥3	66 (14.7)	50 (13.9)	16 (18.0)	

DEC = decrease; FY = fiscal year; HCU = health care utilization; PCL = PTSD Checklist (range: 17–85); PTSD = posttraumatic stress disorder.
[a]Comorbidities occur from start of FY2008 to index date.
[b]Composite of panic disorder, obsessive compulsive disorder, social phobia, generalized anxiety disorder, anxiety not otherwise specified.
[c]Measured only in exposure year. Presence of ≥9 psychotherapy visits in any 15-week period.

Table 2. Quit Smoking Outcome, Descriptive Cumulative Incidence %, Incidence Rate per 1000 Person-Years, and Cox Proportional Hazard Models (*n* = 449)

Group	Total *n*	Quit smoking events	Cumulative incidence %	Incidence rate per 1000PY
Overall	449	147	32.7%	271.4/1000PY
PCL improvement				
PCL decrease <20	360	111	30.8%	250.5/1000PY
PCL decrease ≥20	89	36	40.5%	365.3/1000PY

Cox proportional hazard models[a]

	Crude	Weighted	Weighted + postindex smoking cessation treatment[b]
	HR (95% CI)	HR (95% CI)	HR (95% CI)
PCL decrease ≥20	1.48 (1.02–2.15)	1.57 (1.04–2.36)	1.53 (1.01–2.31)
p	.042	.032	.037

CI = confidence interval; HR = hazard ratio; PCL = PTSD Checklist; PY = person-years.
[a]Proportional hazard assumption, *p* value for all models >.70.
[b]Includes variables behavioral treatment, nicotine replacement, and pharmacotherapy.

alcohol abuse/dependence, a clinically meaningful improvement was not associated with increased smoking cessation. Among those with alcohol abuse/dependence, those with a clinically meaningful improvement had a higher incidence of smoking cessation than patients without a clinically meaningful improvement.

After controlling for confounding, there was no significant difference between patients with and without clinically meaningful improvement in patients with depression and in those without depression. Survival models stratified by alcohol abuse/dependence revealed no association between PTSD improvement and smoking

Table 3. Quit Smoking Outcome, Descriptive Cumulative Incidence %, and Incidence Rate per 1000 Person-Years, by Depression and Alcohol (n = 449)

Group	Total n	Quit smoking events	Cumulative incidence %	Incidence rate per 1000PY
No depression				
PCL decrease <20	87	25	28.7%	238.4/1000PY
PCL decrease ≥20	20	10	50.0%	497.7/1000PY
Depression				
PCL decrease <20	273	86	31.5%	254.2/1000PY
PCL decrease ≥20	69	26	37.7%	331.4/1000PY
No alcohol abuse/dep				
PCL decrease <20	160	58	36.2%	303.0/1000PY
PCL decrease ≥20	29	9	31.0%	292.4/1000PY
Alcohol abuse/dep				
PCL decrease <20	200	53	26.5%	210.5/1000PY
PCL decrease ≥20	60	27	45.0%	398.4/1000PY

PCL = PTSD Checklist; PY = person-years.

Table 4. Results From Cox Proportional Hazards Models Estimating the Association of PCL Decrease and Time to Smoking Cessation, by Depression and Alcohol Abuse/Dependence (n = 449)

	Model 1—crude	Model 2—weighted
	HR (95% CI)	HR (95% CI)
No depression: PCL dec ≥20 vs. <20	2.10 (1.01–4.37)	2.15 (0.92–5.03)
Depression: PCL dec ≥20 vs. <20	1.33 (0.85–2.06)	1.42 (0.89–2.26)
p, depression*PCL dec	p = .291	p = .399
No alcohol abuse/dep: PCL dec ≥20 vs. <20	0.98 (0.48–1.97)	1.00 (0.49–2.03)
Alcohol abuse/dep: PCL dec ≥20 vs. <20	1.92 (1.21–2.06)	1.93 (1.16–3.20)
p, alcohol abuse/dep*PCL dec	p = .115	p = .137

CI = confidence interval; HR = hazard ratio; PCL = PTSD Checklist; PTSD = posttraumatic stress disorder.

cessation among those without alcohol abuse/dependence. Among those with alcohol abuse/dependence, patients who experienced a clinically meaningful improvement, compared with those who did not, had a significantly greater likelihood of smoking cessation (HR = 1.93; 95% CI: 1.16–3.20). For both depression and alcohol abuse/dependence, assessment of effect modification found that neither depression (p = .399) nor alcohol abuse/dependence (p = .137) were significant effect modifiers in the relationship of PTSD improvement and smoking cessation.

Among the 140 patients who quit smoking by 9/30/14, over half (52.9%, n = 74) remained non-smokers in the 12 months after the initial quit date. Rates of sustained smoking cessation did not differ among patients with versus without a clinically meaningful PTSD symptom improvement (52.9% vs. 52.8%).

Discussion

Among patients with PTSD who were current smokers, we found that clinically meaningful decreases in PTSD severity were associated with a 57% greater likelihood of smoking cessation during the 2 years after PTSD improvement. Nearly 53% of those who stopped smoking remained non-smokers for the first 12 months after quitting regardless of their PTSD severity. Thus, our results suggest PTSD improvement is followed by increased smoking cessation, but is not associated with increased probability of remaining a non-smoker for up to 1 year after cessation. This association did not change after accounting for use of postindex smoking cessation treatment. This suggests that PTSD improvement is associated with smoking cessation independent of smoking cessation therapies. The present study adds to the growing evidence that clinically meaningful PTSD improvement compared with less or no improvement is associated with uptake of healthy behaviors such as seeking weight loss counseling,[21] use of SUD treatment,[23] and antidepressant medication adherence.[20]

Subgroup analyses indicated that there were no differences in the association between PTSD improvement and smoking cessation by depression status. Clinically meaningful PTSD improvement was associated with smoking cessation in those with alcohol abuse/dependence but not among patients free of alcohol abuse/dependence, although the difference in HRs between those with versus without alcohol abuse/dependence was not significant. However, subgroup analyses were limited by small cell sizes and further research is needed before drawing strong conclusions about the impact of comorbidity on smoking cessation in patients who do versus do not experience large decreases in PTSD severity. Of note, substance use disorders were more common among those with versus without clinically meaningful PTSD improvement. This could be explained by the slightly higher PTSD severity and the higher prevalence of depression and anxiety disorders in those who had a clinically meaningful PTSD improvement. This group was also more likely to receive an adequate duration of PTSD psychotherapy which may be linked to increased chances that substance use disorders are detected.

Our results are consistent with evidence from a study of National Guard members that found greater PTSD severity was associated with a higher likelihood of current heavy smoking.[35] PTSD severity has also been shown to predict more cigarette consumption at 1-year follow-up.[17] However, we believe this is the first study using data from routine medical care to reveal that PTSD symptom improvement may be followed by increased smoking cessation.

While novel PTSD treatment modalities that integrate smoking cessation therapy have demonstrated efficacy,[18,19] not all persons with PTSD have access to such integrated care. Our study suggests smoking cessation is associated with PTSD symptom improvement alone and does not necessarily depend on cotreatment of PTSD and smoking. Improved self-efficacy following PTSD treatment[36] may be a mechanism that accounts for ability to stop smoking in patients who experience reductions in PTSD severity.

Limitations

Nicotine dependence diagnoses used to identify smokers at baseline may have captured patients using non-smoking nicotine products such as smokeless tobacco and nicotine replacement products. We lacked data on specific PTSD symptoms which vary in their strength of association with smoking and smoking intensity.[16] Persistent smoking in those with PTSD has been attributed to negative reinforcement and emotional numbing.[37] Further research is needed to determine if improvements in specific symptom clusters are followed by increased smoking cessation. Our study is limited to a veteran population and may not generalize to non-veterans. However, we have previously found similar results in veteran and civilian cohorts in studies of opioid use and depression and use of metformin and risk for dementia.[38,39] Although agreement between smoking health factor data and survey data are moderate to good,[29,30] the 2-year quit rate is high and not directly comparable to existing results on 12-month quit rates in veterans with PTSD. However, our first and second year quit rates (19.4% and 16.6%) were similar to 12-month quit rates from a prior study examining targeted smoking cessation outreach to veterans with PTSD.[40] We recognize that a single occurrence of a health factor indicating former or non-smoking does not capture the cyclical nature of quit attempts (ie, multiple successes and failures). Misclassification of smoking cessation should be random with respect to whether a patient improved versus didn't improve PTSD symptoms; thus, the HRs are likely not biased. Last, not all patients from the parent sample had PCL scores and it is possible that those with more severe PTSD or those who remain in treatment longer are more likely to have PCL scores. This could reduce generalizability of our results to all patients with PTSD.

Conclusions

Clinically meaningful decreases in PTSD severity, either through psychotherapy, pharmacotherapy, or spontaneous improvement, are associated with smoking cessation. PTSD is a modifiable risk factor for persistent smoking. Patients with PTSD should be made aware of the opportunity for improved health behaviors following reduction in PTSD severity. Such knowledge may encourage PTSD treatment seeking.

Supplementary Material

A Contributorship Form detailing each author's specific involvement with this content, as well as any supplementary data, are available online at https://academic.oup.com/ntr.

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Declaration of Interests

None declared.

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Data Availability

Interested persons may obtain data following appropriate VA IRB and data access request approvals. The authors may not share data without VA approval.

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