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Smoking status and exercise in relation to PTSD symptoms: A test among trauma-exposed adults

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ABSTRACT

The present investigation examined the interactive effect of cigarette smoking status (i.e., regular smoking vs. non-smoking) and weekly exercise (i.e., weekly metabolic equivalent) in terms of posttraumatic stress (PTSD) symptom severity among a community sample of trauma-exposed adults. Participants included 86 trauma-exposed adults (58.1% female; M_{age} = 24.3). Approximately 59.7% of participants reported regular (≥10 cigarettes per day) daily smoking over the past year. The interactive effect of smoking status by weekly exercise was significantly associated with hyperarousal and avoidance symptom cluster severity ($p \leq .05$). These effects were evident above and beyond number of trauma types and gender, as well as the respective main effects of smoking status and weekly exercise. Follow-up tests indicated support for the moderating role of exercise on the association between smoking and PTSD symptoms, such that the highest levels of PTSD symptoms were observed among regular smokers reporting low weekly exercise levels. Theoretical and clinical implications of the findings are discussed.

The relations among tobacco use, traumatic event exposure, and posttraumatic stress disorder (PTSD[1]) symptoms have been well-established (Feldner, Babson, & Zvolensky, 2007; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). For example, across populations, trauma-exposed individuals are more likely to be current smokers (e.g., Acierno, Kilpatrick, Resnick, & Saunders, 1996), smoke at higher rates (Beckham et al., 1995), and evidence greater levels of nicotine dependence (McClernon et al., 2005), as compared to individuals without clinical *or* subclinical PTSD. Likewise, higher smoking rates and greater levels of nicotine dependence are related to an increased risk for PTSD symptoms among trauma-exposed persons (e.g., Beckham, Feldman, Kirby, Hertzberg, & Moore, 1997). Although smoking and PTSD symptoms commonly co-occur and appear to influence one another, there has been little work focused on factors that may moderate these relations. Elucidating potential protective factors with respect to smoking-PTSD relations has important clinical implications for prevention and intervention efforts.

One such possible moderator of smoking-PTSD associations is exercise. There is a large and diverse empirical literature demonstrating the positive effects of exercise among the general population and many chronic medical populations (e.g., chronic fatigue syndrome, chronic obstructive pulmonary disease, cardiovascular disease, cancer) in regard to improving physical symptoms of fatigue, pain, and dyspnea (e.g., Mostert & Kesselring, 2002). Other work has found that increased exercise can decrease depression and anxiety symptoms, as well as improve positive affect among the general population and many chronic medical populations (Emery, Schein, Hauck, & MacIntyre, 1998; Ströhle et al., 2007). Indeed, population-based studies and well-controlled clinical trials have provided consistent evidence that exercise improves well-being, decreases depression, anxiety, and hostility, as well as offers greater feelings of social connectedness (Otto & Smits, 2011). For example, in controlled clinical trials, programmed exercise can provide depression relief that rivals that provided by antidepressant medication (Stathopoulou, Powers, Berry, Smits, & Otto, 2006).

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[1] For purposes of brevity, the term "PTSD" in this article should be interpreted as referring to all levels of posttraumatic stress (clinical and subclinical). In the present sample, "PTSD" refers to subclinical symptoms.

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In regard to smoking, there is growing scientific literature suggesting that exercise may influence several aspects of tobacco use. For instance, among smokers in the general population, exercise has significant salutary effects on craving and withdrawal symptoms (Ussher, Taylor, & Faulkner, 2012); and offers promising effect sizes for short-term abstinence (Williams et al., 2010), although some mixed findings have been reported (Ussher, West, McEwen, Taylor, & Steptoe, 2003).

Notably, there is limited research on exercise among trauma-exposed populations. Available data suggest there are low rates of exercise among PTSD populations (e.g., de Assis et al., 2008; Zen, Whooley, Zhao, & Cohen, 2012). Furthermore, exercise has been found to mediate the association between PTSD and physical and functional health in a trauma-exposed sample of undergraduate students, with PTSD – Hyperarousal symptoms emerging with particularly salient inverse relations with exercise (Rutter, Weatherill, Krill, Orazem, & Taft, 2011). Furthermore, several small-scale, uncontrolled intervention studies (n's = 9–15), based on trauma-exposed adult (Manger & Motta, 2005; Otter & Currie, 2004) and adolescent (Diaz & Motta, 2008; Newman & Motta, 2007) samples reporting varying levels of PTSD symptoms, have documented significant reductions in PTSD symptoms following aerobic exercise interventions. Although the literature is scant and most studies are methodologically limited (e.g., small sample sizes, lack of control groups), extant work suggests a possible clinically significant role for exercise in terms of both smoking and PTSD and other negative mood symptoms.

Several explanations for the anxiolytic effects of exercise have been proposed (Stathopoulou et al., 2006), a few of which may be particularly relevant for understanding how exercise might influence the smoking-PTSD association. First, it is possible that engagement in exercise modifies traditional emotional action tendencies (e.g., approach vs. avoidance), which in turn serves to attenuate both smoking behavior and PTSD symptoms (Foa, Hembree, & Rothbaum, 2007; Zvolensky & Bernstein, 2005). More specifically, exercise may serve as interoceptive exposure to trauma- and smoking-relevant bodily sensations (e.g., rapid heart palpitations, shortness of breath) in trauma-exposed smokers with PTSD symptoms, which over time, may serve to decrease PTSD symptoms (Smits et al., 2008) and relieve the addictive properties of tobacco (Zvolensky et al., 2008). Second, exercise also appears to reduce reactivity to stress (Puterman et al., 2010, 2011), broadly, and thus may confer protective effects in terms of the development or expression of post-traumatic psychopathology through a stress-buffering pathway. For example, with respect to anxiety-related outcomes, a number of studies have now shown that exercise reduces fear reactivity to established laboratory stressors (e.g., inhalation of carbon dioxide-enriched air; Smits, Meuret, Zvolensky, Rosenfeld, & Seidel, 2009; cholecystokinin tetrapeptide challenge; Ströhle et al., 2005). Third, given the commonality of sleep disturbances to tobacco use (e.g., Okun, Levine, Houck, Perkins, & Marcus, 2011) and PTSD (American Psychiatric Association [APA], 2000; see review by Lamarche & De Konick, 2007), exercise may exert concurrent effects on smoking and PTSD symptoms by improving sleep (Driver & Taylor, 2000). Finally, regular exercise may intensify individuals' present-centered attention and awareness and increase levels of distress tolerance (i.e., ability to withstand negative emotional or physical states; Simons & Gaher, 2005), which may serve to ameliorate both PTSD symptoms and smoking behavior over time (e.g., Brown, Lejuez, Kahler, Strong, & Zvolensky, 2005; Vujanovic, Bernstein, & Litz, 2011).

Despite the emerging empirical literature in this domain, and the theoretical relevance of exercise for the smoking-PTSD co-occurrence, few studies have addressed this topic directly. Furthermore, no published studies to date have examined the interplay of smoking behavior and exercise among trauma-exposed smokers. Thus, the current investigation is the first empirical study of the relations of smoking status and exercise with regard to PTSD symptom severity and PTSD symptom cluster severity. It was hypothesized that, among trauma-exposed persons, the interactive effect of regular smoking status (i.e., $>=10$ cigarettes per day; cf., non-smoking status) and low weekly exercise levels would yield the highest levels of PTSD symptoms, generally, and PTSD – Hyperarousal symptoms, specifically (e.g., Rutter et al., 2011). Weekly exercise was expected to moderate the association between smoking status and PTSD symptoms, such that the highest levels of PTSD symptoms were expected among regular smokers reporting low levels of exercise. These effects were expected above and beyond the variance contributed by theoretically-relevant covariates (i.e., gender, number of trauma exposure types) and the main effects of smoking status and exercise output. Participants were screened out for current (past month) *DSM-IV-TR* (APA, 2000) Axis I psychopathology so as to ascertain that any observed effects were not due to co-occurring Axis I disorders (e.g., Marshall et al., 2008).

1. Method

1.1. Participants

Participants (n = 86, 58.1% female) reported a mean age of 24.3 years (SD = 10.54). The ethnic/racial distribution of the study sample reflected that of the local recruitment environment (State of Vermont Department of Health, 2010): 89.5% (n = 77) identified as White/Caucasian; 4.7% (n = 4) as Black/African-American; 1.2% (n = 1) as Hispanic/Latino; 1.2% (n = 1) as Asian; and 3.5% (n = 3) did not specify race/ethnicity. The majority of the sample was never married (90.7%) and had completed at least high school (61.6%) or some college (25.6%) education.

Participants were selected from a large pooled database that consisted of data from adults ages 18 to 65, who participated in one of three studies on emotional vulnerability. Inclusion criteria for the current study were: (1) exposure to a *DSM-IV-TR* PTSD Criterion A traumatic life event (APA, 2000); (2) available PTSD symptom data; (3) PTSD symptom severity score $\geq$ 1 on the Posttraumatic Diagnostic Scale (PDS; Foa, 1995); and (4) available physical exercise self-report data. Exclusionary criteria were comprised of: (a) limited mental competency and/or the inability to provide informed, written consent; (b) current suicidal/homicidal ideation; (c) current Axis I psychopathology[2]; (d) self-reported smoking as between 1 and 9 cigarettes per day over the past year; (e) current or past serious medical illness (e.g., chronic cardiopulmonary illness, seizure disorder); and (f) pregnancy. Participants were excluded for medical illness and pregnancy due to the experimental nature of a portion of the study protocol not presented here. Of the 86 subjects, 9 were excluded for infrequent tobacco use (cigarettes per day/past year $\leq$ 9), thus 77 participants were included in the final analyses.

2. Measures

2.1. Structured clinical interview for DSM-IV disorders/non-patient version (SCID-I/NP; First, Spitzer, Gibbon, & Williams, 1995)

The SCID-I/NP is a structured-clinical interview used to assess for Axis I psychiatric diagnoses. The SCID-I/NP has been found to have good reliability (Zanarini et al., 2000) and excellent validity

[2] The same pattern of results was found when including individuals with Axis I psychiatric disorders.

(Basco et al., 2000). The SCID-I/NP was employed in the current study to rule out current Axis I psychopathology, as per the exclusionary criteria noted above.

2.2. Posttraumatic Diagnostic Scale (PDS; Foa, 1995)

The PDS is a 49-item self-report instrument designed to assess trauma exposure and the presence of *DSM-IV-TR* PTSD symptoms (APA, 2000). Respondents report if they have experienced any of 12 traumatic events (e.g., "non-sexual assault by a family member or someone you know," "serious accident, fire, or explosion"), including an "other" category, and then indicate which event was most disturbing. The PDS assesses Criterion A trauma exposure as well as the frequency (0 = "not at all" or "only one time" to 3 = "five or more times a week/almost always") of 17 past-month PTSD symptoms for the most disturbing event endorsed. The items are summed to assess PTSD symptom severity (range from 0 to 51) and symptom cluster severity. The PDS has evidenced generally excellent psychometric properties (Foa, Cashman, Jaycox, & Perry, 1997), including high internal consistency (alpha = .92) and high test-retest reliability (kappa = .74). The PDS was found to have 82% agreement with the SCID-I with regard to PTSD diagnosis (Foa et al., 1997) and good convergent validity (alphas = .73–.79) with related measures of depression and anxiety (Foa, 1995). In the current study, internal consistency was good: PDS-Total (α = .92), PDS-Re-experiencing (α = .81), PDS-Avoidance (α = .83), and PDS-Hyperarousal (α = .83).

2.3. Exercise Habits Questionnaire-Revised (EHQ-R; Zvolensky, 2008)

The EHQ-R is a self-report checklist that is used to obtain information about engagement in 29 different types of exercise (e.g., running, swimming, hiking, yoga, resistance training), and the frequency (i.e., number) and length of each session (i.e., time spent; less than 20 min; 20–29 min; 30–39 min; 40–49 min; 50 min or more). Based on previous methodology (e.g., Tart et al., 2010), information from the EHQ-R was used in combination with the compendium of physical activities (Ainsworth et al., 2000) to compute metabolic equivalent (MET) minutes. There are three interrelated steps used in this computation. First, MET is computed (the ratio of working metabolic rate working to resting rate, a value that is assigned based on the type of exercise; see Ainsworth et al., 2000). Next, minutes spent per session of exercise are totaled using the midpoint of the time range (e.g., 20–29 min equaled 24.5 min) and for "50 min or more," 50 min was used. Then, time spent per session is multiplied by its associated MET value to calculate "MET minutes" per activity. These score are then multiplied by the frequency of the respective activity in the past week. Finally, these scores for each of the 29 activities are summed.

2.4. Smoking History Questionnaire (SHQ; Brown, Lejuez, Kahler, & Strong, 2002)

The SHQ is a self-report questionnaire that is used to gather descriptive information about cigarette use. In the current study, the SHQ was used to determine daily smoking rate and smoking history. Smoking status was defined dichotomously, such that regular smoking was defined as 10 or more cigarettes per day over the past year. The cut-off of 10 cigarettes per day was used because it is the established definition of regular smoking (Agrawal et al., 2011).

2.5. Procedure

Participants were recruited from the community via flyer and newspaper advertisements for participation in three separate studies on "emotional distress." Flyers were posted across various well-traveled community spaces (e.g., shopping malls, libraries, supermarkets, coffee shops). The procedures for the three studies were identical in regard to the advertisement, recruitment, completion of study measures, and compensation level. Interested persons contacting the research team were scheduled for a diagnostic evaluation to determine eligibility. Upon arrival to the study center, participants first provided verbal and written consent and were informed of the voluntary nature of the study. Then, participants completed a structured clinical interview and a battery of self-report measures. Participants were compensated $25 for their efforts for that portion of the study. The study protocol involved an experimental procedure, which is not relevant to the current investigation. All study procedures were approved by the site Institutional Review Board.

2.6. Data analytic plan

Analyses were conducted in PASW Statistics 18.0 (SPSS Inc., Chicago, IL, USA). First, zero-order correlations among predictor and criterion variables were examined. Second, a series of four hierarchical multiple regression analyses was performed to examine smoking status and exercise output in relation to self-reported PTSD symptoms. Criterion variables included (a) PTSD – Total symptom severity (PDS-Total score), (b) PTSD – Re-experiencing symptom severity (PDS-Re-experiencing), (c) PTSD – Avoidance symptom severity (PDS-Avoidance), and (d) PTSD – Hyperarousal symptom severity (PDS-Hyperarousal). At step one of each of the models, gender and number of traumatic event types were entered as covariates. At step two, smoking status (coded dichotomously: non-smoker; regular smoker) and exercise output (total MET minutes of aerobic activity during the past week, mean centered) were entered simultaneously. Finally, at step three, the interaction term for smoking status by MET output was entered. All tests were two-tailed with an alpha level set at .05.

The forms of any significant interactions were subsequently examined both graphically (Cohen & Cohen, 1983) and statistically (Holmbeck, 2002). First, we examined the forms of these interactions by inserting specific values for each predictor variable (smoking status dummy coded as 0 = non-smoker and 1 = regular smoker [i.e., ≥10 cigarettes/day]; high and low exercise output defined as .5 standard deviation (*SD*) above and below the mean, respectively) into the regression equation associated with the described analysis (Cohen & Cohen, 1983; p. 323, 419). Second, follow-up probing analyses were conducted to examine the significance of the simple slopes and interactions to test for moderation (see Holmbeck, 2002 for a detailed example).

3. Results

3.1. Participant characteristics

With respect to trauma history, participants reported experiencing 2.1 (*SD* = 1.24) different types of traumatic life events, as per responses on the PDS (Foa, 1995). The sample reported experiencing all types of traumatic events listed in the PDS with the exception of torture, including: serious accident, fire, or explosion (54.5%), non-sexual assault by a stranger (22.1%), non-sexual assault by a family member or someone known (19.5%), life-threatening illness (18.2%), sexual contact when younger than 18 years with someone five or more years older (16.9%), sexual assault by a family member or someone known (14.3%), natural disaster (14.3%), imprisonment (5.2%), sexual assault by a stranger (3.9%), and military combat or a war zone (1.3%). Approximately 37.7% of the sample endorsed the "other" trauma type category (e.g., sudden

unexpected death of a friend or family member). Participants endorsed relatively low PTSD symptom severity levels (PDS-Total score: $M = 9.7$, $SD = 9.03$). PTSD symptom cluster severity scores were as follows: Re-experiencing ($M = 2.6$, $SD = 2.56$), Avoidance ($M = 4.1$, $SD = 4.38$), and Hyperarousal ($M = 3.1$, $SD = 3.27$).

In regard to smoking status, 59.7% reported cigarette use (40.3% were non-smokers), averaging 14.0 ($SD = 6.82$) cigarettes per day and 8.4 years of use ($SD = 7.34$). In regard to exercise, 70.1% of participants met the standard of at least 500 MET minutes per week (US Department of Health and Human Services, 2008). Overall, the sample averaged 1454.9 MET minutes ($SD = 155.46$) per week.

3.2. Zero-order correlations

Please see Table 1 for a summary of zero-order correlations among all studied variables. Gender was significantly, negatively correlated only with weekly exercise ($r = -.23$, $p < .05$). Number of trauma types was significantly, positively associated only with smoking status ($r = .27$, $p < .05$). Weekly exercise (i.e., MET minutes) was significantly, negatively associated only with PTSD – Hyperarousal symptom severity ($r = -.31$, $p < .01$).

3.3. Hierarchical multiple regression analyses

Results of the hierarchical regression analyses are presented in Table 2. With respect to PTSD – Total symptom severity, the model was not significant. Similarly, with regard to PTSD – Re-experiencing symptoms, the model was not significant.

In terms of PTSD – Avoidance symptom severity, step one of the model was not significant. At step two of the model, the main effects of smoking status and exercise output were not significant. Step three of the model contributed 4.9% of unique variance, and the interactive effect of smoking status by exercise was significant ($\beta = -.33$, $p = .05$). Please see Fig. 1 for a graphical depiction of this effect. Follow-up probing analyses of the simple slopes and interactions supported the significant moderating role of weekly exercise in the association between smoking status and PTSD – Avoidance. Specifically, PTSD – Avoidance symptoms were highest among regular smokers reporting low weekly exercise ($t = 1.95$, $\beta = .39$, $p = .05$), however this was not statically significant when exercise was high ($t = 1.69$, $\beta = .22$, $p = .09$).

Finally, with respect to PTSD – Hyperarousal symptom severity, step one was not significant. At step two of the model, 9.3% of variance was uniquely predicted by the main effects ($p = .02$); however only weekly exercise emerged as a significant main effect ($p = .03$). Step three of the model contributed 5.0% of unique variance ($p < .01$), and the interactive effect of smoking status by exercise output was significant ($\beta = -.38$, $p = .04$). Please see Fig. 1 for a graphical representation of this effect. Follow-up probing analyses of the simple slopes and interactions supported the significant moderating role of weekly exercise in the association between smoking status and PTSD – Hyperarousal. Specifically, regular smokers with low weekly exercise levels demonstrated the highest levels of PTSD – Hyperarousal symptoms ($t = 2.01$, $\beta = .39$, $p = .04$), whereas regular smokers reporting high exercise levels exhibited the lowest level of PTSD – Hyperarousal symptoms, though this effect was not significant ($p = .07$).

4. Discussion

The present investigation was the first to empirically document the interplay of smoking and exercise with regard to PTSD symptoms in the context of a trauma-exposed sample. Inconsistent with hypothesis, the interactive effect of smoking status and exercise was not significantly associated with overall PTSD symptom severity ($p = .07$). However, as predicted, the interactive effect was significantly associated with Hyperarousal and Avoidance symptom cluster severity, specifically. Indeed, the highest levels of PTSD symptoms were observed among regular smokers reporting low weekly exercise output. This finding might suggest that the synergistic effect of regular smoking and low exercise levels may increase vulnerability for the expression of PTSD symptoms in the context of trauma exposure, but no definitive conclusions regarding directionality of the observed associations can be determined due to study design limitations. Such findings are consistent with past work documenting inverse relations between exercise and PTSD; namely, lower levels of exercise tend to be correlated with higher PTSD symptoms and vice versa (e.g., Zen et al., 2012). These results also extend this work by elucidating the additional vulnerability of smoking – particularly smoking 10 or more cigarettes per day. Nevertheless, it should be noted that, due to the cross-sectional design of this study, directionality of observed associations cannot be determined. Therefore, it also is possible that higher levels of PTSD symptoms predispose individuals to higher levels of smoking and lower exercise (e.g., de Assis et al., 2008). More research is needed to determine the directionality of these observations, and moreover, to determine whether decreasing smoking and increasing levels of exercise may serve as a protective factor for PTSD among trauma-exposed adults.

Interestingly, trauma-exposed individuals reporting low exercise levels, as compared to those reporting high exercise levels – regardless of smoking status – manifested the highest levels of PTSD – Hyperarousal symptoms. This finding is consistent with past work (Rutter et al., 2011), documenting especially robust associations between exercise and PTSD – Hyperarousal symptoms. It also may suggest that exercise serves a protective function with regard to hyperarousal symptoms, specifically. That is, trauma-exposed individuals who exercise at higher levels may – via the

Table 1
Zero-order correlations among predictor and criterion variables ($n = 86$).

Variable	1	2	3	4	5	6	7	8	Mean (*SD*) or %
Predictor variables									
1. Gender	–	−.01	−.19	−.23*	.18	.22	.09	.19	59.7%
2. Number of trauma types		–	.27**	.06	.17	.14	.16	.14	2.1 (1.24)
3. Smoking status			–	−.17	.15	.04	.15	.19	53.5%
4. MET output				–	−.22	−.16	−.14	−.31**	1454.9 (1553.46)
Criterion variables									
5. PDS–Total Score					–	.83***	.94***	.86***	9.7 (9.03)
6. PDS–Re-experiencing						–	.69***	.57***	2.6 (2.56)
7. PDS–Avoidance							–	.70***	4.1 (4.38)
8. PDS–Hyperarousal								–	3.1 (3.27)

Note. $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$. Gender dummy coded as 0 = male, 1 = female, percent represented is of females; Smoking status dummy coded as 0 = non-smoker and 1 = regular smoker (i.e., ≥10 cigarettes/day), percent represented is of regular smokers; MET output = Metabolic equivalent computed from the Exercise Habits Questionnaire-Revised (EHQ-R; Zvolensky, 2008); PDS = Posttraumatic Diagnostic Scale (Foa, 1995).

Table 2
The interaction between exercise output and smoking status predicting post-traumatic stress symptomatology ($n = 77$).

Predictor variables	ΔR^2	t	β	sr^2	p
Criterion variable: PDS—Total					
Step 1	.060				.10
Gender		1.58	.18	.18	.12
Number of trauma types		1.51	.17	.17	.13
Step 2	.049				.08
Smoking status		.88	.11	.10	.38
MET output		−1.50	−.18	−.17	.14
Step 3	.041				.04
Smoking × Exercise		−1.84	−.30	−.20	.07
Criterion variable: PDS—Re-experiencing					
Step 1	.068				.07
Gender		1.94	.22	.22	.06
Number of trauma types		1.30	.15	.15	.20
Step 2	.014				.18
Smoking status		.12	.02	.01	.90
MET output		−.99	−.12	−.11	.33
Step 3	.002				.27
Smoking × Exercise		−.40	−.07	−.05	.69
Criterion variable: PDS—Avoidance					
Step 1	.034				.28
Gender		.81	.09	.09	.42
Number of trauma types		1.40	.16	.16	.17
Step 2	.027				.33
Smoking status		.86	.11	.10	.40
MET output		−.91	−.11	−.10	.37
Step 3	.049				.13
Smoking × Exercise		−1.98	−.33	−.22	.05
Criterion variable: PDS—Hyperarousal					
Step 1	.058				.11
Gender		1.74	.20	.20	.09
Number of trauma types		1.27	.14	.14	.21
Step 2	.093				.02
Smoking status		1.19	.14	.13	.24
MET output		−2.17	−.25	−.24	.03
Step 3	.050				<.01
Smoking × Exercise		−2.11	−.38	−.22	.04

Note. β = standardized beta weight. Gender dummy coded as 0 = male, 1 = female. Smoking status dummy coded as 0 = non-smoker and 1 = regular smoker (i.e., ≥10 cigarettes/day). MET output = Metabolic equivalent computed from the Exercise Habits Questionnaire-Revised (EHQ-R; Zvolensky, 2008); PDS = Posttraumatic Diagnostic Scale (Foa, 1995).

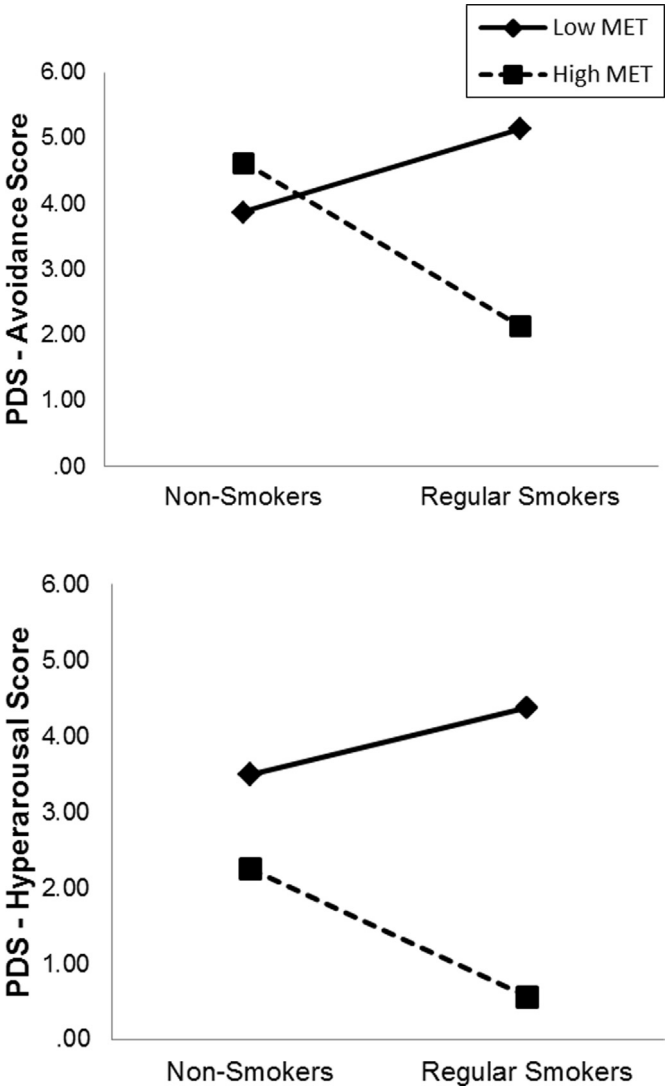

Fig. 1. PTSD — Hyperarousal symptom severity and PTSD — Avoidance symptom severity are represented. High levels of exercise (High MET) refer to .5 standard deviation above the mean for total aerobic output in the entire sample; similarly, low exercise (Low MET) refers to .5 standard deviation below the mean for total aerobic output in the entire sample. Smoking status was coded dichotomously (non—smoker; regular smoker [≥10 cigarettes/day]).

interoceptive exposure effects of exercise (e.g., Smits et al., 2008) — habituate to anxiety-related symptoms, thereby reducing such symptoms with time and consistent exercise. Notably, this effect does not hold for avoidance symptoms, however. This specificity may be due to the non-clinical nature of the sample, or it may suggest a specific association between exercise and hyperarousal symptoms. Future work is necessary to more conclusively delineate these associations.

Notably, no significant effects, at the level of main or interactive effects, were observed with regard to PTSD — Re-experiencing symptoms. While these findings may suggest that smoking and exercise are more robustly associated with PTSD symptoms of Hyperarousal and Avoidance, this effect likely is due to the low levels of Re-experiencing symptoms ($M = 2.6$, $SD = 2.5$) reported by these trauma-exposed adults. It would be important to extend this work among clinical populations with PTSD. Furthermore, the main effects of smoking status and exercise output were not significantly associated with PTSD symptoms. This finding is inconsistent with past work, which has documented significant associations between smoking and PTSD symptoms (e.g., Beckham et al., 1995) as well as exercise and PTSD (Rutter et al., 2011). The nonclinical nature of the sample is likely the factor underlying these results, thus underscoring the importance of future replication and extension of this work with clinical samples to better determine the nature of these associations across samples.

Together, the present study suggests pertinent clinical implications worthy of further empirical exploration. Exercise may be a clinically indicated intervention for trauma-exposed adults experiencing varying levels of PTSD symptoms. Such interventions may help to increase affect tolerance to trauma-relevant interoceptive cues (e.g., heart racing), which in turn may facilitate individuals' willingness to engage in evidence-based treatments for PTSD, such as cognitive processing therapy (Resick & Schnicke, 1993) or prolonged exposure (Foa et al., 2007). Furthermore, such interventions may serve the dual purpose of improving the documented poor physical health functioning of trauma-exposed individuals with subclinical (Rutter et al., 2011) and clinically significant PTSD (de Assis et al., 2008).

There are several limitations worthy of note in the present study. First, the sample was comprised of relatively young, primarily Caucasian, trauma-exposed individuals without current (past month) Axis I psychopathology. Future studies would benefit from including older, more diverse, clinical samples with PTSD and co-occurring disorders. Second, the overall sample endorsed low mean levels of

PTSD symptoms on the PDS ($M = 9.7$, $SD = 9.0$), limiting our ability to readily extrapolate from the present findings to PTSD in clinical samples. It should be noted that participants were comprised of trauma-exposed individuals without current PTSD, which may indicate that (a) they once met criteria for PTSD and/or other Axis I disorders but their symptoms have remitted or (b) they never developed PTSD despite being exposed to trauma. The latter circumstance may indeed indicate that such individuals are especially resilient in the face of adversity and thereby constitute a particular sub-group of the trauma-exposed population. Unfortunately, data regarding the duration of time since trauma exposure were not collected, thus limiting our ability to extrapolate upon the pertinent impact of this variable on outcomes. Third, the participants in this study reported especially high levels of exercise ($M = 1454.9$ MET minutes), considering they constitute a trauma-exposed sample, the majority of whom are smokers; thus, this sample may be unrepresentative of trauma-exposed smokers, generally. This finding may be due to the above average exercise rates often reported for the State of Vermont (Centers for Disease Control, 2012), an artifact of the measurement used (EHQ-R), or a combination of both. Fourth, this study relied exclusively on self-report, which may be subject to report biases. Furthermore, it should be noted that the EHQ-R, the measure of exercise activity utilized in the current study, is a descriptive measure of exercise used in prior research (e.g., Smits, Tart, Rosenfield, & Zvolensky, 2011; Smits et al., 2012). Since it is not extensively studied, it will be important for future studies to extend this work using other exercise instruments. Also, the examination of nicotine dependence (vs. smoking frequency), biochemical verification indices of smoking behavior, as well as interview-based assessments of the frequency and intensity of PTSD symptoms would be pertinent to implement in future work. Finally, given the cross-sectional study design, the direction of the observed associations is not known. As noted previously, it is important to extend this line of work using controlled experimental, longitudinal/prospective, and intervention-based designs to examine potential causal links among these variables.

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