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A prospective examination of the relationships between PTSD, exposure to assaultive violence, and cigarette smoking among a national sample of adolescents

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ABSTRACT

Research demonstrates robust associations among posttraumatic stress disorder (PTSD), exposure to assaultive violence (i.e., sexual assault, physical assault, and witnessed violence), and cigarette smoking among adults and adolescents. Whether exposure to assaultive violence confers risk for cigarette smoking over and above the effects of PTSD and non-assaultive traumatic events (e.g., motor vehicle accidents) is unclear. The current study prospectively measured PTSD, assaultive violence exposure, non-assaultive traumatic event exposure, and cigarette smoking three times over approximately three years among a nationally representative sample of adolescents (N = 3614, age range 12–17 at Wave 1). Results revealed that multiple exposure to assaultive violence at Wave 1 was a consistent and robust prospective predictor of cigarette smoking at Waves 2 and 3. By contrast, PTSD diagnoses and non-assaultive traumatic event exposures at Wave 1 only predicted cigarette smoking at Wave 2, but not at Wave 3. Theoretical and clinical implications are discussed.

1. Introduction

Posttraumatic Stress Disorder (PTSD) is an anxiety disorder diagnosed at least one month following a traumatic event and characterized by intrusive re-experiencing of a traumatic event, avoidance of trauma-related cues, and physiological hyperarousal symptoms (APA, 2000). A growing body of research suggests substantial comorbidity between PTSD and cigarette smoking (Beckham et al., 1997; Feldner et al., 2007; Hapke et al., 2005). For example, cigarette smoking among adults in the United States general population was recently reported to be approximately 18% (Center for Disease Control & Prevention, 2009). Among United States adolescents, 20% reported smoking cigarettes in the past 30 days (Eaton et al., 2008). By contrast, the prevalence of cigarette smoking is at least twice as high among adults and adolescents with PTSD (Acierno et al., 2000; Acierno, Kilpatrick, Resnick, Saunders, & Best, 1996; Beckham et al., 1995; Hapke et al., 2005; Lasser et al., 2000).

While the rates of smoking are clearly elevated among PTSD populations, whether this elevation is due to potentially traumatic event[1] (PTE) exposure independently, PTSD diagnoses independently, or both is not clear. Beyond the effects of PTE exposure independently, PTSD diagnoses might be expected to confer vulnerability to smoking onset due to attempts to avoid or escape PTSD symptoms (e.g., re-experiencing symptoms) through the intoxicating effects of nicotine (i.e., the self-medication hypothesis; Khantzian, 1985). Evidence in support of the unique effect of PTSD diagnoses on cigarette smoking comes from a prospective study showing that PTSD diagnoses predicted later onset of nicotine dependence controlling for traumatic event exposure (Breslau, Davis, & Schultz, 2003). Similarly, a recent study of trauma-exposed daily smokers found that symptoms of PTSD were positively correlated with self-reported smoking as a means to regulate negative affect (Feldner et al., 2007). Basic research demonstrates that laboratory-administered nicotine leads to enhanced attentional control capacity (Evans & Droobs, 2009; Knott et al., in press), suggesting that cigarette smoking may be a potent means of 'medicating' the cognitive deficits (Vasterling et al., 2002; Vasterling, Brailey, Constans, & Sutker, 1998) associated with PTSD diagnoses. These data suggest a unique

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[1] We prefer the term 'potentially traumatic event' over 'traumatic event' because it is difficult to definitively call an event 'traumatic.' Thus, the term 'potentially traumatic event' is a more conservative descriptive term.

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effect of PTSD diagnoses on cigarette smoking; however, relationships between PTSD diagnoses and cigarette smoking have not always been consistent. That is, other studies have failed to show a relationship between PTSD diagnoses and cigarette smoking (Acierno et al., 1996; Acierno et al., 2000; Baschnagel, Coffey, Schumacher, Drobes, & Saladin, 2008).

There is also inconsistent evidence suggesting a relationship between PTE exposure, above and beyond the effect of PTSD, and cigarette smoking. Cross-sectional studies suggest a robust relationship between PTE exposure and cigarette smoking (Acierno et al., 1996; Acierno et al., 2000; Hapke et al., 2005; Kaplan et al., 1998). For example, one study among a nationally representative sample of adolescents found that a history of physical assault was associated with increased risk of cigarette smoking when controlling for PTSD, but PTSD was not associated with cigarette smoking when controlling for physical assault history (Acierno et al., 2000). Breslau et al. (2003) found when using retrospectively reported data that PTSD, but not PTE exposure independently, predicted current nicotine dependence; however, in a 10 year prospective study they found that both PTSD and PTE exposure independently predicted the onset of future nicotine dependence. These inconsistencies could partly be due to the use of cross-sectional versus prospective designs, the measurement of smoking behavior, or age related differences between sample participants.

The effect of cumulative traumatic event exposure on subsequent smoking behavior has not been well-investigated and might explain some of the inconsistencies. Researchers have suggested that PTSD and substance use disorders broadly (e.g., alcohol use disorders, cocaine abuse, etc.) are related due to shared alterations in stress-related neurobiological pathways (Brady & Sinha, 2005). Cumulative exposure to PTEs may be one factor contributing to alterations in these stress-related neurobiological pathways (Kolassa & Elbert, 2007). While PTE exposure is a diagnostic requirement for PTSD, research also shows that *multiple* PTE exposure is associated with increased risk of PTSD (Hedtke et al., 2008; Kolassa, Kolassa, Ertl, Papassotiropoulos, & De Quervain, 2010; Neuner et al., 2004; Steel et al., 2009); that is, risk of PTSD increases as a function of PTE exposure frequency. For example, Neuner et al. (2004) found among a large sample of refugees that risk of probable PTSD diagnoses increased from 28% among individuals exposed to 1–3 PTEs to 100% among individuals exposed to 28 or more PTEs. Basic research suggests that this cumulative effect may be due to long-lasting alterations in neurobiological structures (e.g., amygdala and prefrontal cortex) subsequent to chronic stress exposure (Arnsten, 2009; Kolassa & Elbert, 2007; McEwen, 2004; Vyas, Mitra, Shankaranarayana Rao, & Chattarji, 2002; Vyas, Pillai, & Chattarji, 2004). Similar to the linear relationship between PTE exposure frequency and risk of PTSD, it also may be the case that greater cumulative PTE exposure, regardless of PTSD development, confers greater risk for cigarette smoking. The type of PTE may also be a risk factor, such that assaultive PTEs (e.g., sexual assault, physical assault, witnessed violence; e.g., Hedtke et al., 2008) are associated with higher rates of PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993) and cigarette smoking (Acierno et al., 1996; Acierno et al., 2000) relative to non-assaultive PTEs (e.g., natural disasters). To our knowledge, no prior study to date has accounted for multiple assaultive PTE exposures when prospectively examining the relationships between PTSD and cigarette smoking.

The aim of the present study was to examine the prospective relationships between cumulative exposure to assaultive PTEs, PTSD, and cigarette smoking in a nationally representative sample of adolescents measured at three time points over approximately three years. The design of the current study addresses some of the limitations of the existing body of literature. First, we are aware of only one previous prospective examination of PTSD, PTE exposure, and cigarette smoking to date (Breslau et al., 2003), and further prospective examinations are accordingly important. Second, this study differentiates assaultive from non-assaultive PTEs to test whether assaultive PTEs specifically are a more 'potent' risk factor (Kessler, Chiu, Demler, Merikanga, & Walters, 2005; Resnick et al., 1993). Third, no previous study has examined the effect of multiple PTE exposures, as opposed to a dichotomous 'exposed' versus 'non-exposed' categorization, on relationships between PTSD and cigarette smoking. Fourth, there is a relative paucity of this research among adolescents (Acierno et al., 2000), which is problematic because smoking during adolescence often continues into adulthood (Brook et al., 2008; Moolchan, Ernst, & Henningfield, 2000). Finally, the present study measured smoking in two ways to address both casual smoking and regular smoking, as relationships may be different as a function of the cigarette smoking index. Based on the emerging evidence in this literature (e.g., Neuner et al., 2004), we hypothesize that multiple assaultive PTE exposure will prospectively predict subsequent smoking behavior. Based on prior research and the self-medication hypothesis (Breslau et al., 2003; Khantzian, 1985), we hypothesize that PTSD will also prospectively predict subsequent smoking behavior.

2. Method

2.1. Participants

The National Survey of Adolescents-Replication (NSA-R) is a longitudinal epidemiological study of adolescents ages 12–17 (N = 3614 at Wave 1; 1808 girls) residing in the U.S. The primary aim of this study is to determine the prevalence and correlates of youths' PTE exposure. For detailed descriptions of the sampling and methodological procedures, refer to McCauley et al., 2010 and Wolitzky-Taylor et al., 2008. The NSA-R sample consisted of a national household probability sample as well as an oversample of urban-dwelling youth. Mean age was 14.50 ($SD = 1.71$) at Wave 1. In regard to racial/ethnic makeup of the sample, 69% were Caucasian, 13% were African-American, 10% were Hispanic, 3% were Native American, and 3% were Asian/Pacific Islander.

2.2. Procedure

Participant selection utilized a multi-stage, stratified, random-digit dial procedure within each region of the country. Trained interviewers at a survey research firm with significant experience managing survey studies administered the interview using a computer-assisted telephone interview system. This system aided this process by prompting interviewers with each question consecutively on a computer screen, and supervisors conducted random checks of data entry accuracy and interviewers' adherence to assessment procedures. Although interviews began with parental consent and a brief parent interview consisting primarily of demographic questions, the majority of the interview was conducted with the adolescent. Data collection occurred from 2005 to 2009.

Mean number of months between Wave 1 and Wave 2 assessment was 15.29 ($SD = 4.58$), and mean number of months between Wave 2 and Wave 3 assessment was 14.44 ($SD = 2.67$).

2.3. Attrition

Of the 3614 adolescents measured at wave 1, 2511 (69%) were measured at wave 2. The 1103 uncompleted wave 2 assessments were due to telephone problems such as technical problems or non-working number (29% of uncompleted); inability to reach the participant (i.e., always busy, no answer, parent or adolescent never available) (24%); adolescent refusal during wave 2 recontact (17%); wrong telephone number (12%); ineligibility (9%); or only partial completion of the interview (9%). Of the original 3614 participants, 1653 (46%) were measured at wave 3. The 858 uncompleted wave 3 assessments were due to telephone problems such as technical problems or non-working number (30% of uncompleted); inability to

reach the participant (i.e., always busy, no answer, parent or adolescent never available) (17%); adolescent refusal during wave 3 recontact (45%); wrong telephone number (3%); ineligibility (1%); or only partial completion of the interview (4%).

Adolescents who completed all three waves were significantly less likely to have ever smoked at Wave 1 (21%) relative to non-completers (28%); χ^2 (1) = 23.04, p < .001, and were also significantly less likely to be regular smokers (4.1%) compared to non-completers (8.9%); χ^2 (1) = 32.96, p < .001. Adolescents who completed all three waves reported significantly less assaultive PTEs at Wave 1 (M = 1.16, SD = 1.82) relative to non-completers (M = 1.54, SD = 2.12); F(1, 3613) = 36.58, p < .001, η_p^2 = .009. Adolescents who completed all three waves were not less likely to have PTSD relative to non-completers; χ^2 (1) = .04, p = .84.

2.4. Measures

2.4.1. PTE history

Assaultive PTE history was assessed by behaviorally specific dichotomous questions regarding a series of events: 1) sexual assault (i.e., anal penetration, vaginal penetration, oral sex on the perpetrator, oral sex from the perpetrator, digital penetration, fondling of the adolescent, forced fondling of the perpetrator, or drug or alcohol facilitated sexual assault), 2) physical assault (i.e., attack with a weapon, attacked with a stick, club, or bottle, attacked without a weapon, threatened with a weapon, attacked with fists), 3) abuse from a caregiver (i.e., spanked, slapped, pushed, locked in a closet, thrown against a hard surface, beaten up with fists or kicked, choked, burned on purpose, cut with sharp object, or threatened with a weapon) 4) witnessed community violence (i.e., witnessed shooting, witnessed stabbing, witnessed sexual assault, witnessed robbery, witnessed threatening with a weapon, witnessed hitting or kicking), and 5) witnessed domestic violence (i.e., witnessing pushing or shoving, punching or hitting, choking, hitting with objects, or threatening with weapon). Non-assaultive PTE history (i.e., motor vehicle accidents, other accidents, fires, natural disasters, bitten by dog) was assessed with questions such as "Have you ever been in a serious motor vehicle accident in a car, truck, or motorcycle?" This PTE assessment methodology was adapted from prior successful epidemiological methodologies (Kilpatrick et al., 2000; Kilpatrick et al., 2003). Specific wording of questions and details of this methodology are available in past publications (Kilpatrick et al., 2000; Kilpatrick et al., 2003). Consistent with the recently proposed criteria for DSM-V (http://www.dsm5.org/), a PTE was considered endorsement of any of the events regardless of whether the individual experienced fear, helplessness, or horror. At Wave 1, for any event endorsed, participants were then asked how many times the event had ever happened to them.

2.4.2. PTSD

The PTSD module of the NSA survey (Kilpatrick et al., 2000) and National Women Survey (Resnick et al., 1993) was used to assess current PTSD symptoms. This structured diagnostic interview assessed each DSM-IV symptom with a yes/no response indicating the presence of a symptom during the last 6 months. Symptoms were not anchored to any specific index traumatic event. A diagnosis of PTSD required exposure to a PTE (regardless of fear, helplessness, or horror) of either assaultive or non-assaultive (i.e., motor vehicle accidents, other accidents, fires, natural disasters, bitten by dog) event categories. Diagnoses also required endorsement of functional impairment, which was defined as either school impairment, social impairment, work impairment, or distress. The measure was validated against the PTSD module of the Structured Clinical Interview for the *DSM* (SCID; Spitzer, Williams, & Gibbon, 1987) administered by mental health professionals (Kilpatrick et al., 1998). The inter-rater kappa coefficient was .85 for the diagnosis of PTSD, and comparisons between the PTSD module used here and SCID yielded a kappa coefficient of .77. Internal consistency in the present study was .88.

2.4.3. Cigarette smoking frequency

Cigarette smoking was assessed by asking two questions that were based off of the Youth Risk Behavior Survey (Brener et al., 2004). First, adolescents were asked "Have you ever [Since the last interview, have you] tried cigarette smoking, even one or two puffs?" Adolescents who answered "yes" were then asked "Have you ever [Since the last interview, have you] smoked cigarettes regularly, that is, at least one cigarette every day for 30 days?" Thus, both casual smoking and regular smoking were indexed. These variables will be referred to as 'ever smoke' and 'regular smoker,' respectively.

2.5. Analytic approach

Relationships between PTSD, cumulative assaultive PTEs, and cigarette smoking were assessed with logistic regression. Consistent with prior epidemiological research, data were weighted to match national demographic characteristics (Kilpatrick et al., 2000; Kilpatrick et al., 2003). Analyses were done separately for the 'ever smoke' (no versus yes) and 'regular smoker' (no versus yes) variables at Wave 2 and Wave 3. Demographic variables, Wave 1 PTSD, Wave 1 Assaultive PTE Exposure, and the corresponding Wave 1 smoking variable (e.g., Wave 1 'Ever Smoke' when predicting Wave 3 'Ever Smoke') were entered as predictors in each analysis. The Wave 1 smoking variables were entered as predictors in order to control for baseline levels of smoking. The demographic variables included: gender (male, 50% of sample, was coded = 1; female, 50% of sample, was coded = 2), minority status (Caucasian, 65% of sample, was coded = 1; Minority, 34% of sample, was coded = 2), and age (ages 12–14, 47% of the sample, was coded = 1; ages 15–18, 53% of sample was coded = 2). Wave 1 Assaultive PTE exposure and Wave 1 Non-Assaultive PTE exposure were coded zero exposures = 0 (52% and 55% of sample, respectively), one exposure = 1 (14% and 21% of sample, respectively), and two or more exposures = 2 (35% and 23% of sample, respectively). This coding separates single from multiple exposures and is necessary to test hypotheses regarding the effect of cumulative assaultive and non-assaultive PTE exposure.

3. Results

3.1. Descriptive statistics

3.1.1. Prevalence of PTSD, Assaultive violence, Non-Assaultive PTE exposure, and cigarette smoking

The frequencies of relevant study variables are presented in Table 1 for the total sample and separately for each gender.

3.2. Logistic regressions among full sample

3.2.1. Wave 2 Ever Smoke

As shown in Table 2, Age and Ethnicity were related to Wave 2 Ever Smoke, with older adolescents and Caucasians more likely to have ever smoked at Wave 2. Both Wave 1 PTSD and Wave 1 multiple Assaultive PTE exposure significantly increased odds of Wave 2 Ever Smoking when controlling for baseline smoking behaviors and other predictors. Wave 1 Non-Assaultive PTE exposure was not significantly related to Wave 2 Ever Smoking.

3.2.2. Wave 2 Regular Smoking

As shown in Table 3, older adolescents and Caucasians were more likely to have regularly smoked at Wave 2. Wave 1 multiple Assaultive PTE exposure significantly increased odds of Wave 2 Regular Smoking. Wave 1 multiple Non-Assaultive PTE exposure also significantly

Table 1
Frequencies of relevant study variables.

Variable		Total sample	Boys	Girls	χ^2 of gender difference	p value of difference
Age[a]					.21	.649
	Age 12–14	50%	50%	51%		
	Age 15–17	50%	50%	49%		
Ethnicity[a]					.58	.448
	Caucasian	69%	69%	68%		
	Minority	30%	29%	31%		
PTSD[a]					16.52*	<.001
	Yes	3.9%	3%	5%		
	No	96.1%	97%	95%		
Non-Assaultive PTE exposure[a]					25.55	<.001
	0 Non-Assaultive PTEs	55%	52%	59%		
	1 Non-Assaultive PTEs	21%	21%	21%		
	2 Non-Assaultive PTEs	23%	27%	20%		
Assaultive PTE exposure[a]					2.71	.258
	0 Assaultive PTEs	52%	50%	53%		
	1 Assaultive PTEs	14%	14%	14%		
	2 Assaultive PTEs	35%	36%	33%		
Ever Smoke						
	Wave 1 Ever Smoke	25%	24%	26%	1.74	.188
	Wave 2 Ever Smoke	21%	21%	20%	.49	.484
	Wave 3 Ever Smoke	26%	27%	24%	2.71	.100
Regular Smoker						
	Wave 1 Regular Smoker	7%	6%	7%	.86	.355
	Wave 2 Regular Smoker	8%	8%	9%	.16	.694
	Wave 3 Regular Smoker	11%	11%	11%	.06	.812

Note.
* $= p < .05$. PTE = potentially traumatic event.
[a] Characteristics at Wave 1.

increased odds of Wave 2 Regular Smoking. Wave 1 PTSD did not increase odds of Wave 2 Regular Smoking.

3.2.3. Wave 3 Ever Smoke

As shown in Table 4, Age and Ethnicity were related to Wave 2 Ever Smoke, with older Adolescents and Caucasians more likely to have ever smoked at Wave 2. Both single and multiple Assaultive PTE exposures significantly increased odds of Wave 2 Ever Smoking when controlling for baseline smoking behaviors and other predictors. Neither Wave 1 Non-Assaultive PTE exposure nor Wave 1 PTSD significantly increased odds of Wave 3 Ever Smoking.

3.2.4. Wave 3 Regular Smoking

As shown in Table 5, older Adolescents and Caucasians were more likely to have regularly smoked at Wave 2. Wave 1 multiple Assaultive PTE exposure significantly increased odds of Wave 3 Regular Smoking after controlling for baseline smoking behaviors. Wave 1 PTSD marginally significantly increased odds of Wave 3 Regular Smoking. Wave 1 Non-Assaultive PTE exposure was not significantly related to Wave 3 Regular Smoking.

Table 2
Logistic regression predicting Wave 2 'Ever Smoke'.

Predictor	Wald	OR	95% CI	p value
Age	46.10*			
12–15[a]		–	–	
16–17		2.36	1.84–3.02	<.001
Ethnicity	28.98*			
Caucasian [a]		–	–	
Other		.46	.35–.61	<.001
Gender	2.68			
Male [a]		–	–	
Female		.82	.65–1.04	.102
Wave 1 PTSD	12.43*			
No[a]		–	–	
Yes		2.47	1.49–4.08	<.001
Wave 1 Assault	9.98*			
0[a]		–	–	
1		1.13	.79–1.61	.516
2 or more		1.54	1.17–2.02	.002
Wave 1 Non-Assault	2.65			
0[a]		–	–	
1		.79	.59–1.07	.125
2 or more		.86	.65–1.15	.315
Wave 1 Ever Smoke	338.19*			
No[a]		–	–	
Yes		9.85	7.72–12.58	<.001

Note.
* $= p < .05$.
[a] Reference category.

4. Discussion

Previous research has yielded inconsistent evidence regarding unique effects of PTSD and PTE exposure on cigarette smoking (Acierno et al., 1996; Acierno et al., 2000; Baschnagel et al., 2008; Breslau et al., 2003). The extant research has scarcely employed prospective designs among large and representative samples and has not accounted for the effect of cumulative assaultive PTE exposure. The present study addressed these limitations and investigated the prospective relationships between PTSD, cumulative assaultive and non-assaultive PTE exposure, and cigarette smoking among a nationally representative sample of adolescents. Wave 1 PTSD was only associated with subsequent casual smoking at Wave 2; it was not

Table 3
Logistic regression predicting Wave 2 'Regular Smoker'.

Predictor	Wald	OR	95% CI	p value
Age	29.04*			
12–15[a]		–	–	
16–17		2.99	2.01–4.45	<.001
Ethnicity	7.59*			
Caucasian [a]		–	–	
Other		.54	.35–.84	.006
Gender	.03			
Male [a]		–	–	
Female		1.03	.73–1.46	.873
Wave 1 PTSD	.34			
No [a]		–	–	
Yes		1.23	.61–2.47	.561
Wave 1 Assault	6.90*			
0 [a]		–	–	
1		1.24	.72–2.15	.435
2 or more		1.71	1.14–2.57	.009
Wave 1 Non-Assault	4.20			
0 [a]		–	–	
1		1.22	.78–1.92	.388
2 or more		1.54	1.02–2.32	.041
Wave 1 Regular Smoker	258.59*			
No[a]		–	–	
Yes		36.67	23.64–56.89	<.001

Note.
* $= p < .05$. [a] = reference category.

Table 4
Logistic regression predicting Wave 3 'Ever Smoke'.

Predictor	Wald	OR	95% CI	*p* value
Age	15.47*			
12–15[a]		–	–	
16–17		1.69	1.30–2.20	<.001
Ethnicity	12.73*			
Caucasian [a]		–	–	
Other		.57	.42–.77	<.001
Gender	2.04			
Male [a]		–	–	
Female		.83	.65–1.07	.153
Wave 1 PTSD	1.00			
No[a]		–	–	
Yes		1.36	.75–2.48	.317
Wave 1 Assault	29.67*			
0[a]		–	–	
1		1.65	1.13–2.42	.010
2 or more		2.28	1.69–3.07	<.001
Wave 1 Non-Assault	3.12			
0[a]		–	–	
1		.81	.59–1.12	.206
2 or more		.77	.56–1.07	.122
Wave 1 Ever Smoke	174.98*			
No[a]		–	–	
Yes		6.81	5.12–9.04	<.001

Note.
* = $p<.05$. [a] = reference category.

associated with Wave 3 casual smoking, Wave 2 regular smoking, or Wave 3 regular smoking. By contrast, assaultive PTE exposure, particularly multiple assaultive PTE exposures, was significantly predictive of all future smoking outcomes measured. Non-assaultive PTE exposure was only predictive of subsequent regular smoking at Wave 2 and did not predict any other smoking outcomes. These relationships were found when controlling for age, gender, ethnicity, and baseline smoking behaviors.

The findings suggest a robust relationship between assaultive PTE exposure and cigarette smoking; however, the relationship was most consistently found for *multiple* assaultive PTE exposures and less consistently found with *single* PTE exposure. These findings suggest that assaultive PTE exposure has a cumulative effect on smoking behavior. These results echo other research documenting the cumulative effect of stressful event exposure on phenotypes such as depression (Risch et al., 2009), PTSD (Kolassa et al., 2010; Neuner et al., 2004; Steel et al., 2009), and other forms of substance use (Hedtke et al., 2008). The results also echo other results suggesting that assaultive PTE exposure is a more 'potent' risk factor for psychopathology outcomes relative to non-assaultive PTE exposure (Breslau, Chilcoat, Kessler, & Davis, 1999; Cougle, Resnick, & Kilpatrick, 2009). To date, this is the first demonstration, to our knowledge, that cumulative assaultive PTE exposure also prospectively confers vulnerability towards subsequent smoking behavior. Cumulative assaultive PTE exposure prospectively predicted both casual smoking and regular smoking. Although the measurement of casual and regular smoking in this study did not detail specific behaviors and was limited to one item for each category, the finding still provides some evidence that the effect of cumulative assaultive PTE exposure extends to both casual and regular smoking. The measurement of smoking behavior in this study also precludes strong inferences regarding the effect of assaultive PTE exposure on nicotine dependence. As such, conclusions from the present results must be tempered, but nonetheless these results suggest an important relation that warrants further empirical attention.

Table 5
Logistic regression predicting Wave 3 'Regular Smoker'.

Predictor	Wald	OR	95% CI	*p* value
Age	18.92*			
12–15[a]		–	–	
16–17		2.32	1.59–3.40	<.001
Ethnicity	8.75*			
Caucasian[a]		–	–	
Other		.50	.31–.79	.003
Gender	.10			
Male [a]		–	–	
Female		.95	.66–1.35	.755
Wave 1 PTSD	3.11			
No [a]		–	–	
Yes		1.90	.93–3.87	.078
Wave 1 Assault	12.10*			
0 [a]		–	–	
1		1.46	.85–2.51	.171
2 or more	2.06		1.37–3.10	.001
Wave 1 Non-Assault	1.12			
0[a]		–	–	
1		1.27	.82–1.97	.573
2 or more		1.09	.70–1.69	.291
Wave 1 Regular Smoker	102.14*			
No [a]		–	–	
Yes		22.43	12.27–41.00	<.001

Note.
* = $p<.05$. [a] = reference category.

Future research will be necessary to examine the processes by which cumulative assaultive PTE exposure confers vulnerability for subsequent smoking. Multiple possible mechanisms exist. For example, prior research suggests that the robust comorbidity between substance use disorders and PTSD may be due to shared disruptions in stress-related neurobiological pathways (Brady & Sinha, 2005). Animal models have demonstrated that chronic stress increases dendritic branching in the amygdala and simultaneously decreases dendritic spine density and length in structures of the prefrontal cortex necessary for cognitive, emotional, and behavioral control (Arnsten, 2009; Mitra, Jadhav, McEwen, Vyas, & Chattarji, 2005; Radley et al., 2006; Vyas et al., 2002; Vyas et al., 2004). As such, cumulative assaultive PTE exposure may contribute to future smoking behavior by increasing reactivity towards motivationally salient cues (e.g., threat cues, smoking cues) while also decreasing cognitive, behavioral, and emotional control (e.g., impaired decision making and exaggerated delay discounting). Alternatively, exposure to assaultive PTEs has also been linked with delinquency (McCart et al., 2007; Zinzow et al., 2009); thus, delinquency might mediate the relationship between cumulative assaultive PTE exposure and smoking. Future research will be necessary to replicate the present finding regarding the robust effect of cumulative assaultive PTE exposure and to examine possible pathways by which exposure contributes to subsequent smoking.

Wave 1 PTSD was a robust prospective predictor of only Wave 2 casual smoking. This finding is partially consistent with models suggesting that individuals with PTSD smoke to regulate or avoid negative affect (Feldner et al., 2007; Khantzian, 1985). The finding that PTSD was only associated with Wave 2, but not Wave 3, casual smoking might suggest a mood-dependent transient effect. For example, perhaps only current symptoms of PTSD may motivate smoking, whereas a history of PTSD (i.e., PTSD symptoms in remission) may no longer confer vulnerability for smoking. Given that PTSD tends to remit over time (Goenjian et al., 2005), current PTSD may only be expected to exert a time-limited effect on smoking behavior. While a prior study documented that PTSD predicted nicotine dependence 10 years later (Breslau et al., 2003), that study did not also control for cumulative assaultive PTE exposure, and given the strong overlap between cumulative assaultive PTE exposure and PTSD (Hedtke et al., 2008; Neuner et al., 2004), it cannot be determined whether the long-term effect of PTSD in that study was due to cumulative assaultive PTE exposure or PTSD. It is also important to note that the hypothesis that individuals with PTSD smoke to regulate or avoid negative affect would also predict that

PTSD should increase odds of regular smoking, which was not found in this study at either Wave 2 or Wave 3. Accordingly, the present study yields more consistent support for models suggesting that the comorbidity between PTSD and smoking is due to shared disruptions in stress-related pathways, and less consistent support for the self-medication hypothesis of smoking in PTSD. It is also important to note that the self-medication hypothesis and shared-stress-related pathways hypothesis are not mutually exclusive and it is possible that they operate in tandem. This is illustrated in analyses of the Wave 2 casual smoking variable, where both PTSD and cumulative assaultive PTE exposure predicted increased odds of Wave 2 casual smoking. Similarly, it remains possible that "smoking to cope" motives moderate the relationship between PTSD and smoking, such that among subgroups of individuals who engage in "smoking to cope," PTSD is more strongly predictive of smoking.

There may be clinical implications of the present study. Specifically, findings supporting the significant association between PTE exposure, especially cumulative PTE exposure, and both transient and chronic smoking behavior illuminate the importance of considering alternative mechanisms that may exist alongside exposure to traumatic events. While mere exposure to PTEs has been linked with detrimental consequences for adolescents (Hedtke et al., 2008; Kessler, 2000; Kilpatrick et al., 2000; Kilpatrick et al., 2003), research suggests that these consequences may be worsened when high risk behaviors (i.e., delinquency, substance use, etc.) are present (Dembo & Schmeidler, 2002; Simpson & Miller, 2002). Thus, it is important to reduce risk for high risk behaviors among adolescents that have experienced PTE exposure, in attempt to lessen detrimental consequences throughout development. To address this problem, the field has recently focused on the development and evaluation of integrated treatments that focus on both victimization and potential for high risk behaviors within the same treatment modules, including interventions such as Seeking Safety (Najavits, Gallop, & Weiss, 2006) and Risk Reduction through Family Therapy (RRFT; Danielson et al., in press). For example, RRFT is an intervention designed for adolescents who have been exposed to sexual assault, which aims to reduce risk of high risk behaviors following victimization.

Although integrated interventions have primarily addressed PTE exposure alongside more "serious" high risk behaviors such as delinquency, alcohol abuse, and drug use, findings from the present study suggest that it may also be important to expand the focus, to additionally include transient and chronic smoking behavior among adolescents. Further, the well-established trajectory from cigarette use into alcohol and drug use among adolescents (Lewinsohn, Rohde, & Brown, 1999; Torabi, Bailey, & Majd-Jabbari, 1993) adds weight to the importance of including smoking behaviors within these interventions. Thus, including prevention of both transient and chronic smoking behavior among adolescents following exposure to PTEs, especially multiple PTE exposure, may reduce the likelihood of a developing pathway into further substance use.

Conclusions from the present study must be tempered by the following limitations. First, the assessment of cigarette smoking was limited to two questions that did not inquire about specific smoking behaviors. For example, it cannot be determined exactly how often the individuals who endorsed casual smoking were actually smoking. Future research will be necessary to examine the unique effects of cumulative PTE exposure and PTSD on specific aspects of smoking (e.g., number of days smoking, number of cigarettes smoked, etc.). Second, dependency and withdrawal were not measured, which hinders comparisons to other studies finding a link between PTSD and dependency (Breslau et al., 2003) and withdrawal (Feldner, Vujanovic, Gibson, & Zvolensky, 2008). Third, attrition was not random, and non-completers reported more Wave 1 assaultive PTEs and were more likely to smoke at Wave 1. As such, it remains possible that different results would have been found if more of the non-completers had been located and measured at Waves 2 and 3. These limitations highlight the need for more research on this topic to replicate the current results and address the limitations.

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Contributors

Authors JMC, ABA, and AMB conducted literature searches, statistical analysis, and contributed to the writing of the manuscript.

Conflict of Interest

All authors contributed to and have approved the final manuscript. All authors declare that they have no conflicts of interest.

References

Acierno, R., Kilpatrick, D. G., Resnick, H. S., Saunders, B. E., Arellano, M. D., & Best, C. L. (2000). Assault, PTSD, family substance use, and depression as risk factors for cigarette use in youth: Findings from the national survey of adolescents. *Journal of Traumatic Stress*, *13*, 381–396.

Acierno, R., Kilpatrick, D., Resnick, H. S., Saunders, B. E., & Best, C. L. (1996). Violent assault, posttraumatic stress disorder, and depression: risk factors for cigarette use among adult women. *Behavior Modification*, *20*, 363–384.

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (revised 4th ed.). Washington, DC: Author.

Arnsten, A. F. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience*, *10*, 410–422.

Baschnagel, J. S., Coffey, S. F., Schumacher, J. A., Drobes, D. J., & Saladin, M. E. (2008). Relationship between PTSD symptomotology and nicotine dependence severity in crime victims. *Addictive Behaviors*, *33*, 1441–1447.

Beckham, J. C., Kirby, A. C., Feldman, M., Hertzberg, M., Moore, S., Crawford, A. L., et al. (1997). Prevalence and correlates of heavy smoking in Vietnam veterans with chronic posttraumatic stress disorder. *Addictive Behaviors*, *22*, 637–647.

Beckham, J. C., Roodman, A. A., Shipley, R. H., Hertzberg, M. A., Cunha, G. H., Kudler, H. S., et al. (1995). Smoking in Vietnam combat veterans with posttraumatic stress disorder. *Journal of Traumatic Stress*, *8*, 461–472.

Brady, K. T., & Sinha, R. (2005). Co-occurring mental and substance use disorders: The neurobiological effects of chronic stress. *The American Journal of Psychiatry*, *162*, 1483–1493.

Brener, N. D., Kann, L., Kinchen, S. A., Grunbaum, J. A., Whalen, L., Eaton, D., et al. (2004). Methodology of the youth risk behavior surveillance system. *Morbidity and Mortality Weekly Report*, *53*(RR-12), 1–13.

Breslau, N., Chilcoat, H. D., Kessler, R. C., & Davis, G. C. (1999). Previous exposure to trauma and PTSD effects of subsequent trauma: Results from the Detroit Area Survey of Trauma. *American Journal of Psychiatry*, *156*, 902–907.

Breslau, N., Davis, G. C., & Schultz, L. R. (2003). Posttraumatic stress disorder and the incidence of nicotine, alcohol, and other drug disorders in persons who have experienced trauma. *Archives of General Psychiatry*, *60*, 289–294.

Brook, D. W., Brook, J. S., Zhang, C., Whiteman, M., Cohen, P., & Finch, S. J. (2008). Developmental trajectories of cigarette smoking from adolescence to the early thirties: Personality and behavioral risk factors. *Nicotine & Tobacco Research*, *10*, 1283–1291.

Center for Disease Control and Prevention (2009). State-specific secondhand smoke exposure and current cigarette smoking among adults-United States, 2008. *Morbidity and Mortality Weekly Report*, *58*, 1232–1235.

Cougle, J. R., Resnick, H., & Kilpatrick, D. G. (2009). Does prior exposure to interpersonal violence increase risk of PTSD following subsequent exposure? *Behaviour Research and Therapy*, *47*, 1012–1017.

Danielson, C. K., McCart, M., de Arellano, M. A., Macdonald, A., Silcott, L., & Resnick, H. (in press). Risk reduction for substance use and trauma-related psychopathology in adolescent sexual assault victims: Findings from an open trial. *Child Maltreatment.*

Dembo, R., & Schmeidler, J. (2002). *Family empowerment intervention: An innovative service for high-risk youths and their families.* Binghamton, NY: Haworth Press.

Eaton, D. K., Kann, L., Kinchen, S., Shanklin, S., Ross, J., Hawkins, J., et al. (2008). Youth risk behavior surveillance-United States, 2007. *Morbidity and Mortality Weekly Report*, *57*(SS04), 1–131.

Evans, D. E., & Droobs, D. J. (2009). Nicotine self-medication of cognitive-attentional processing. *Addictive Biology*, *14*, 32–42.

Feldner, M. T., Babson, K. A., Zvolensky, M. J., Vujanovic, A. A., Lewis, S. F., Gibson, L. E., et al. (2007). Posttraumatic stress symptoms and smoking to reduce negative affect: An investigation of trauma-exposed daily smokers. *Addictive Behaviors*, *32*, 214–227.

Feldner, M. T., Vujanovic, A. A., Gibson, L. E., & Zvolensky, M. J. (2008). Posttraumatic stress disorder and anxious and fearful reactivity to bodily arousal: a test of the mediating role of nicotine withdrawal severity among daily smokers in 12-hr nicotine deprivation. *Experimental and Clinical Psychopharmacology*, *16*, 144–155.

Goenjian, A. K., Walling, D., Steinberg, A. M., Karayan, I., Najarian, L. M., & Pynoos, R. (2005). A prospective study of posttraumatic stress and depressive reactions among treated and untreated adolescents 5 years after a catastrophic disaster. *The American Journal of Psychiatry*, *162*, 2302–2308.

Hapke, U., Schumann, A., Rumpf, H., John, U., Konerding, U., & Meyer, C. (2005). Association of smoking and nicotine dependence with trauma and posttraumatic stress disorder in a general population sample. *The Journal of Nervous and Mental Disease*, *193*, 843–846.

Hedtke, K. A., Ruggiero, K. J., Fitzgerald, M. M., Zinzow, H. M., Saunders, B. E., Resnick, H. S., et al. (2008). A longitudinal investigation of interpersonal violence in relation to mental health and substance use. *Journal of Consulting and Clinical Psychology*, *76*, 633–647.

Kaplan, S. J., Pelcovits, D., Salzinger, S., Weiner, M., Mandel, F. S., Lesser, M. L., et al. (1998). Adolescent physical abuse: Risk for adolescent psychiatric disorders. *American Journal of Psychiatry*, *155*, 954–959.

Kessler, R. C. (2000). Posttraumatic stress disorder: The burden to the individual and to society. *The Journal of Clinical Psychiatry*, *61*(supplement 5), 4–12.

Kessler, R. C., Chiu, W. T., Demler, O., Merikanga, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*, 617–627.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, *52*, 1048–1060.

Khantzian, E. J. (1985). The self-medication hypothesis of addictive disorders: focus on heroin and cocaine dependence. *The American Journal of Psychiatry*, *142*, 1259–1264.

Kilpatrick, D. G., Acierno, R., Saunders, B., Resnick, H. S., Best, C. L., & Schnurr, P. P. (2000). Risk factors for adolescent substance abuse and dependence: data from a national sample. *Journal of Consulting and Clinical Psychology*, *68*, 19–30.

Kilpatrick, D. G., Resnick, H. S., Saunders, B. E., & Best, C. L. (1998). Rape, other violence against women, and posttraumatic stress disorder. In B. P. Dohrenwend (Ed.), *Adversity, stress, and psychopathology* (pp. 161–176). New York: Oxford University Press.

Kilpatrick, D. G., Ruggiero, K. J., Acierno, R., Saunders, B. E., Resnick, H. S., & Best, C. L. (2003). Violence and risk of PTSD, major depression, substance abuse/dependence, and comorbidity: Results from the National Survey of Adolescents. *Journal of Consulting and Clinical Psychology*, *71*, 692–700.

Knott, V., Heenan, A., Shah, D., Bolton, K., Fisher, D., & Villeneuve, C. (in press). Electrophysiological evidence of nicotine's distracter-filtering properties in non-smokers. *Journal of Psychopharmacology*.

Kolassa, I. -T., & Elbert, T. (2007). Structural and functional neuroplasticity in relation to traumatic stress. *Current Directions in Psychological Science*, *16*, 321–325.

Kolassa, I. -T., Kolassa, S., Ertl, V., Papassotiropoulos, A., & De Quervain, D. J. (2010). The risk of posttraumatic stress disorder after trauma depends on traumatic load and the catecho-methyltransferase Val(158)Met polymorphism. *Biological Psychiatry*, *67*, 304–308.

Lasser, K., Boyd, J., Woolhandler, S., Himmelstein, D., McCormick, D., & Bor, D. (2000). Smoking and mental illness. *Journal of the American Medical Association*, *284*, 2606–2610.

Lewinsohn, P. M., Rohde, P., & Brown, R. A. (1999). Level of current and past cigarette smoking as predictors of future substance use disorders in young adulthood. *Addiction*, *94*, 913–921.

McCart, M. R., Smith, D. W., Saunders, B. E., Kilpatrick, D. G., Resnick, H., & Ruggiero, K. J. (2007). Do urban adolescents become desensitized to community violence? Data from a national survey. *The American Journal of Orthopsychiatry*, *77*, 434–442.

McCauley, J. L., Danielson, C. K., Amstadter, A. B., Ruggiero, K. J., Resnick, H. S., Hanson, R. F., et al. (2010). The role of traumatic event history in non-medical use of prescription drugs among a nationally representative sample of US adolescents. *Journal of Child Psychology and Psychiatry*, *51*, 84–93.

McEwen, B. S. (2004). Protection and damage from acute and chronic stress: allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. *Annals of the New York Academy of Sciences*, *1032*, 1–7.

Mitra, R., Jadhav, S., McEwen, B. S., Vyas, A., & Chattarji, S. (2005). Stress duration modulates the spatiotemporal patterns of spine formation in the basolateral amygdala. *Proceedings of the National Academy of Sciences of the United States of America*, *102*, 9371–9376.

Moolchan, E. T., Ernst, M., & Henningfield, J. E. (2000). A review of tobacco smoking in adolescents: Treatment implications. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*, 682–693.

Najavits, L. M., Gallop, R. J., & Weiss, R. D. (2006). Seeking safety therapy for adolescent girls with PTSD and substance use disorder: A randomized controlled trial. *The Journal of Behavioral Health Services & Research*, *33*, 453–463.

Neuner, F., Schauer, M., Karunakara, U., Klaschik, C., Robert, C., & Elbert, T. (2004). Psychological trauma and evidence for enhance vulnerability for posttraumatic stress disorder through previous trauma among West Nile refugees. *BMC Psychiatry*, *4*, 34.

Radley, J. J., Rocher, A. B., Miller, M., Janssen, W. G., Liston, C., Hof, P. R., et al. (2006). Repeated stress induces dendritic spine loss in the rat medial prefrontal cortex. *Cerebral Cortex*, *16*, 313–320.

Resnick, H. S., Kilpatrick, D. G., Dansky, B. S., Saunders, B. E., & Best, C. L. (1993). Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. *Journal of Consulting and Clinical Psychology*, *61*, 984–991.

Risch, N., Herrell, R., Lehner, T., Liang, K. -U., Eaves, L., Hoh, J., et al. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: a meta-analysis. *Journal of the American Medical Association*, *301*, 2462–2471.

Spitzer, R. L., Williams, J. B. W., & Gibbon, M. (1987). *Structured clinical interview for DSM-III-R.* New York: New York State Psychiatric Institute, Biometrics Research Department.

Simpson, T. L., & Miller, W. R. (2002). Concomitance between childhood sexual and physical abuse and substance use problems: A review. *Clinical Psychology Review*, *22*, 27–77.

Steel, Z., Chey, T., Silove, D., Marnane, C., Bryant, R. A., & van Ommeren, M. (2009). Association of torture and other potentially traumatic events with mental health outcomes among populations exposed to mass conflict and displacement: A systematic review and meta-analysis. *Journal of the American Medical Association*, *302*, 537–549.

Torabi, M. R., Bailey, W. J., & Majd-Jabbari, M. (1993). Cigarette smoking as a predictor of alcohol and other drug use by children and adolescents: Evidence of the "gateway drug effect. *The Journal of School Health*, *63*, 302–306.

Vasterling, J. J., Brailey, K., Constans, J. L., & Sutker, P. B. (1998). Attention and memory dysfunction in posttraumatic stress disorder. *Neuropsychology*, *12*, 125–133.

Vasterling, J. J., Duke, L. M., Brailey, K., Constans, J. L., Allain, A. N., Jr., & Sutker, P. B. (2002). Attention, learning, and memory performances and intellectual resources in Vietnam veterans: PTSD and no disorder comparisons. *Neuropsychology*, *16*, 5–14.

Vyas, A., Mitra, R., Shankaranarayana Rao, B. S., & Chattarji, S. (2002). Chronic stress induces contrasting patterns of dendritic remodeling in hippocampal and amygdaloid neurons. *The Journal of Neuroscience*, *22*, 6810–6818.

Vyas, A., Pillai, A. G., & Chattarji, S. (2004). Recovery after chronic stress fails to reverse amygdaloid neuronal hypertrophy and enhanced anxiety-like behavior. *Neuroscience*, *128*, 667–673.

Wolitzky-Taylor, K. B., Ruggiero, K. J., Danielson, C. K., Resnick, H. S., Hanson, R. F., Smith, D. W., et al. (2008). Prevalence and correlates of dating violence in a national sample of adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*, 755–762.

Zinzow, H. M., Ruggiero, K. J., Hanson, R. F., Smith, D. W., Saunders, B. E., & Kilpatrick, D. G. (2009). Witnessed community violence and parental violence in relation to substance use and delinquency in a national sample of adolescents. *Journal of Traumatic Stress*, *22*, 525–533.