Trauma and Posttraumatic Stress Symptoms Influence Alcohol and Other Drug Problem Trajectories in the First Year of College

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Abstract:

Objective: College matriculation begins a period of transition into adulthood, one that is marked by new freedoms and responsibilities. This transition also is marked by an escalation in heavy drinking and other drug use as well as a variety of use-related negative consequences. Trauma and symptoms of posttraumatic stress disorder (PTSD) may affect alcohol and drug problems and, thus, may be a point of intervention. Yet, no studies have examined trauma, PTSD, and alcohol and drug problem associations during this developmental period. The present study provides such an examination. Method: Matriculating college students (N = 997) completed surveys in September (Time 1) and at 5 subsequent time points (Time 2–Time 6) over their 1st year of college. With latent growth analysis, trajectories of alcohol- and drug-related consequences were modeled to examine how trauma (No Criterion A Trauma, Criterion A Only, No PTSD Symptoms) and PTSD (partial or full) symptom status predicted these trajectories. Results: Results showed substantial risk for alcohol- and other drug-related negative consequences that is conferred by the presence of PTSD at matriculation. Those with both partial and full PTSD started the year with more alcohol and drug consequences. These individuals showed a steeper decrease in consequences in the 1st semester, which leveled off as the year progressed. Both alcohol and drug consequences remained higher for those in the PTSD group throughout the academic year. Hyperarousal symptoms showed unique effects on substance consequence trajectories. Risk patterns were consistent for both partial and full PTSD symptom presentations. Trajectories did not vary by gender. Conclusions: Interventions that offer support and resources to students entering college with PTSD may help to ameliorate problem substance use and may ultimately facilitate a stronger transition into college and beyond.

Keywords: college | comorbidity | posttraumatic stress disorder | substance use | trauma

Article:

Of the 8 million college students in the United States, as many as 25% meet *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association,

1994) criteria for an alcohol or other substance use disorder (Blanco et al., 2008; Dawson, Grant, Stinson, & Chou, 2004; Wu, Pilowsky, Schlenger, & Hasin, 2007). Many more engage in problem substance use that, though below diagnostic threshold, can lead to hazardous outcomes both acutely (e.g., sexually transmitted infections, interpersonal violence, intoxicated driving, overdose; Caldeira, Arria, O'Grady, Vincent, & Wish, 2008; O'Malley & Johnston, 2002; Rimsza & Moses, 2005) and long-term (e.g., abuse or dependence; Arria, Vincent, & Caldeira, 2009; McCabe, West, & Wechsler, 2007; O'Neill, Parra, & Sher, 2001). Factors that contribute to substance misuse in college can be targeted in preventive interventions. Data now are emerging to suggest that trauma and posttraumatic stress are among these factors.

Trauma exposure among college students is unfortunately common (Marx & Sloan, 2002; Read, Ouimette, White, Colder, & Farrow, 2011; Scarpa et al., 2002). The traumas that college students experience are significant by any standard—including but not limited to sexual assault and other interpersonal violence, natural disasters, military trauma, life-threatening illness, and motor vehicle accidents (Read et al., 2011; Ullman & Filipas, 2005).

Research supports a dimensional model of psychological responses to trauma, with posttraumatic stress disorder (PTSD) falling at the far end of the continuum (Broman-Fulks et al., 2006; Ruscio, Ruscio, & Keane, 2002). A substantial portion of trauma-exposed individuals do not meet full criteria for a PTSD diagnosis but nonetheless experience significant trauma-associated distress (Mendlowicz & Stein, 2000; Mylle & Maes, 2004; Schnurr et al., 2000; Zlotnick, Franklin, & Zimmerman, 2002). Whereas the prevalence of PTSD in college students is estimated to be around 9% (Read et al., 2011; Smyth, Hockemeyer, Heron, Wonderlich, & Pennebaker, 2008), rates of sub-syndromal PTSD are higher (30%–35%; Lauterbach & Vrana, 2001; Smyth et al., 2008).

Theory and data suggest that PTSD and substance misuse may be etiologically linked and, in particular, that trauma and PTSD may pose risk for the development of problem substance use. This literature is reviewed briefly below.

Substance Involvement Following Trauma and Traumatic Stress: Self-Medication

Prominent among theories posited to explain relations among trauma, PTSD, and substance involvement is the “self-medication” hypothesis (Khantzian, 2003). According to this framework, substance use occurs as an effort to manage distressing affect or symptoms associated with a trauma (Jacobsen, Southwick, & Kosten, 2001; Saladin, Brady, Dansky, & Kilpatrick, 1995).

Some empirical evidence supports the self-medication hypothesis with respect to traumatic stress and substance use (McFarlane et al., 2009; Ouimette, Coolhart, Funderburk, & Brown, 2007; Read, Brown, & Kahler, 2004; Stewart, Pihl, Conrod, & Dongier, 1998). Though studies in college populations have not examined self-medication of PTSD symptoms per se, data do show that students cite stress and negative affect as among the primary reasons for substance use (Flynn, 2000; Kassel, Jackson, & Unrod, 2000; O'Hare & Sherrer, 2000), and negative affect has been linked uniquely to substance consequences in this population, independent of consumption (Martens et al., 2008; Read et al., 2004; Simons, Gaher, Oliver, Bush, & Palmer, 2005).

Trauma Effects Versus PTSD Effects

Consistent with a self-medication formulation is the notion that individuals may use substances not so much in response to trauma alone, but as a result of psychological distress that follows the trauma. Data from older adult samples offer some support for this notion (Breslau & Davis, 1992; Epstein, Saunders, Kilpatrick, & Resnick, 1998; Lepore, 1997; Stewart & Conrod, 2003). Yet, research with college samples has focused primarily on substance involvement following trauma exposure (e.g., Goldstein, Flett, & Wekerle, 2010; Klanecky, Harrington, & McChargue, 2008; McCauley, Ruggiero, Resnick, Conoscenti, & Kilpatrick, 2009) and with only a few exceptions (e.g., McDevitt-Murphy, Murphy, Monahan, Flood, & Weathers, 2010; Reed, Anthony, & Breslau, 2007) has not typically examined the relative contribution of trauma exposure versus the contribution of symptoms resulting from such exposure.

Symptom Cluster Associations

According to the *DSM*, PTSD is comprised of three core symptom clusters: re-experiencing, avoidance/numbing, and hyperarousal. Numerous studies have examined whether specific PTSD symptom clusters may be associated uniquely with substance outcomes, and they have yielded disparate findings. The cluster of symptoms characterized by hyperarousal has been posited by some to be most strongly implicated in self-medication processes (see Stewart et al., 1998), yet empirical support for a specific effect of hyperarousal symptoms has been mixed. Though some work supports a unique link between these symptoms and substance outcomes (Shipherd, Stafford, & Tanner, 2005 [illicit drug involvement only]; Stewart, Conrod, Pihl, & Dongier, 1999 [alcohol only]), other studies find either no effect for hyperarousal (Cook, Jakupcak, Rosenheck, Fontana, & McFall, 2009; McDevitt-Murphy et al., 2010; Read et al., 2004; Shipherd et al., 2005 [alcohol use]) or an effect of hyperarousal, but for other symptoms clusters as well (Jakupcak et al., 2010; McFall, Mackay, & Donovan, 1992; Saladin et al., 1995; Stewart et al., 1999 [illicit drugs]; Taft et al., 2007). No studies have tested these effects prospectively in a college sample.

Gender, PTSD, and Substance Involvement

Gender differences in PTSD, substance involvement, and the interaction between the two clinical phenomena have been noted. Women are at greater risk for PTSD following trauma exposure (e.g., Breslau, 2001; Breslau, Davis, Andreski, Peterson, & Schultz, 1997; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Schnurr, Friedman, & Vernardy, 2002), even when controlling for type of trauma (Breslau, 2001). Further, across the lifespan, male gender is a risk factor for substance use and consequences (Lex, 1991; Spear, 2002; Thompson et al., 2009). At least one recent study has found gender differences in the mechanisms that may underlie co-occurring PTSD and alcohol involvement (Bornovalova, Ouimette, Crawford, & Levy, 2009). Together, existing research suggests that the effects of trauma and PTSD symptom status on alcohol and illicit drug trajectories could differ for men and women.

Summary

The first year of college is a time of excitement and possibility, as students take an important step toward independence. It is also a time of instability and transition, hallmark features of which are an increase in autonomy, a decrease in adult supervision, a shift in both quantity and quality of peer relationships (Arnett, 2000, 2005), and, for many, an increase in substance use (Arria et al., 2008). As Sher and Rutledge (2007) noted, the extant literature does not offer much in the way of examination of risk factors for substance misuse over this transitional period. Self-medication theory would suggest that students with PTSD may rely on substances to help manage the many changes and challenges that they face during this transition as more familiar resources are absent or less available. Though an abundant literature has identified a link between PTSD and substance problems in older adult populations, PTSD as a risk factor for substance misuse in college students has been ignored.

The Present Study

The objective of the present study was to provide what is to our knowledge the first examination of the prospective relationship between trauma, posttraumatic stress symptoms, and substance-related consequences in students at the transition into college. We hypothesized that individuals with significant PTSD symptoms would show more problematic substance involvement across the first year of college.

In our investigation, we modeled four discrete categories of trauma exposure and PTSD symptoms in order to contrast the prospective associations with substance outcomes across exposure and symptom levels. These levels were (1) No Criterion A exposure, (2) Criterion A exposure, but no significant PTSD symptoms, (3) Sub-threshold PTSD symptoms (Partial PTSD), and (4) Full PTSD. We hypothesized alcohol and drug consequence trajectories would differ by trauma and PTSD symptom status, with PTSD symptoms conferring greater risk relative to trauma exposure alone.

In more exploratory analysis, we also sought to understand the contribution of trauma and PTSD symptom characteristics to substance consequence trajectories. Thus, we examined whether unique effects of specific PTSD symptom clusters might be observed for alcohol and other drug consequence trajectories in our college sample. Given the mixed nature of extant findings, and the paucity of research in this area with college students, we did not forward a priori hypotheses. We also sought to examine the contribution of trauma severity to substance outcomes. Here again we forwarded no a priori hypotheses.

Finally, as gender differences have been observed both in posttraumatic stress and in substance involvement, we also evaluated whether the effects of posttraumatic stress on alcohol and other drug consequences were moderated by gender. We posited that the PTSD effects on alcohol/drug consequence trajectories would be stronger for women than for men.

Method

Participants

Participants (N = 997; 65% female) were from a longitudinal study that began at college matriculation. Data analyzed in this study included six time points (see Figure 1) over the first year of college. At Time 1 (T1; September), the average age was 18.12 years (SD = 0.45). Seventy-three percent of participants self-identified as Anglo Caucasian (n = 723), 11% as Asian (n = 113), 9% as Black (n = 90), 3% as Hispanic/Latino (n = 33), less than 1% as American Indian/Alaskan (n = 2) or Hawaiian/Pacific Islander (N = 1), and 3% as multi-racial (n = 31). Four participants did not report ethnicity. Eligibility, recruitment, and sample selection are described below.

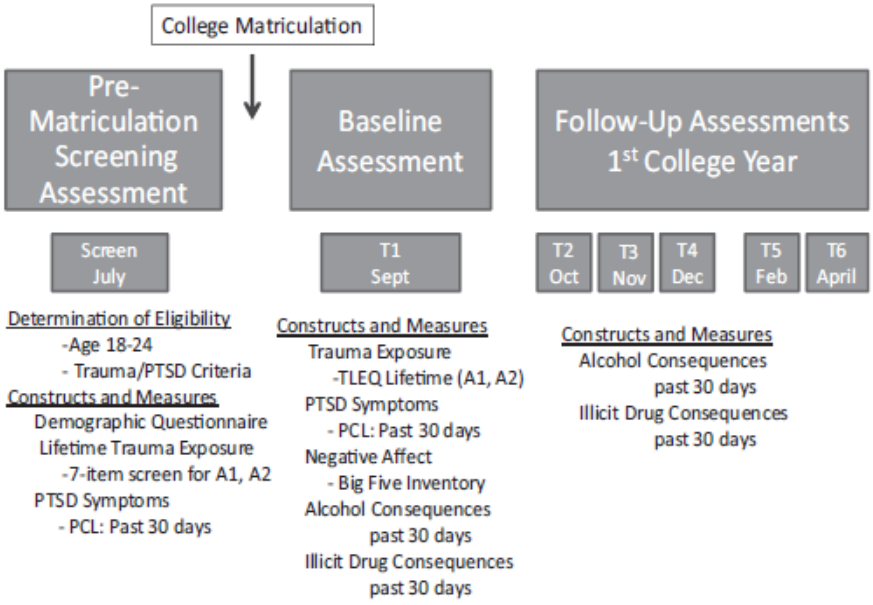

Figure 1. Schedule of recruitment and data collection. T = Time; PTSD = posttraumatic stress disorder; TLEQ = Traumatic Life Events Questionnaire; A1 = Criterion A1; A2 = Criterion A2; PCL = PTSD Checklist–Civilian Version.

Procedure

Initial screening

To obtain the target longitudinal sample, college students at two mid-size public universities in the northeastern (Site A) and southeastern (Site B) parts of the United States screened for trauma and PTSD in the summer prior to matriculation. All students were sent an e-mail with a link to a secure online screening survey. An identical hard-copy packet was sent via postal mail. A return rate of 58% (3,391/5,885) was achieved, comparable to other studies using similar methodologies (e.g., Larimer, Turner, Mallet, & Geisner, 2004; Lewis, Neighbors, Oster-Aaland, Kirkeby, & Larimer, 2007; Neighbors, Geisner, & Lee, 2008). After deletion of cases with insufficient data to determine trauma and PTSD symptom status (see below), the final screening sample consisted of 3,014 students (59% female).

Longitudinal sample selection

To have sufficient representation of students with significant traumatic stress, we invited for participation all those who (1) reported at least one lifetime Criterion A trauma and (2) endorsed at least one symptom from each of the three PTSD symptom clusters. Across sites, 649 participants met these criteria. An additional 585 students who did not meet trauma inclusion criteria were selected randomly for prospective follow-up.

E-mails and a link to the baseline survey (T1) were sent to this selected sample (N = 1,234). At completion of the baseline survey, participants were sent a $20 gift card. Eighty-one percent (N = 997) of those invited completed the baseline survey in September and, thus, constituted the longitudinal sample. This sample was assessed five more times over the year. Across cohorts, the retention rate was 91.3%.

Measures

The present study is focused on a portion of the data from a larger longitudinal study. Measures used in the present study are described below. A schedule of these measures and the time points for which they are analyzed are provided in Figure 1.

Alcohol use

Participants were asked to indicate whether they had consumed any alcohol in the past month. Those participants who indicated that they had consumed alcohol at least once were asked to complete additional questions about their alcohol use. All participants were provided with a standard drink measurement chart to increase accuracy of reporting. Alcohol use was measured with items regarding typical quantity and frequency of alcohol consumption per week in a past 1-month interval (Wood, Read, Palfai, & Stevenson, 2001). Quantity–frequency (QF) indices were created by multiplying item responses. This measure was used for descriptive purposes.

Alcohol-related consequences

Past month consequences associated with alcohol consumption were assessed with the 48-item Young Adult Alcohol Consequences Questionnaire (YAACQ; Read, Kahler, Strong, & Colder, 2006). Items assess a broad array of consequences that range in severity. Response options are rated dichotomously (yes–no), and thus the YAACQ yields a score that ranges from 0 to 48. The YAACQ has strong psychometric properties, including convergent validity and test–retest reliability (Read, Merrill, Kahler, & Strong, 2007). Participants who were not asked to complete these items (i.e., had no past month use) received a consequences score of 0. Cronbach's alpha for the total YAACQ score in this sample was .98.

Illicit substance use

Participants were asked to indicate whether they had used illicit drugs (i.e., cannabis, cocaine, stimulants, inhalants, sedatives or sleeping pills, hallucinogens, or opioids) in the past month (O'Malley & Johnson, 2002). Those participants who answered in the affirmative were asked to indicate the frequency with which they used each drug during the past month for a non-medical

reason. Responses ranged from 0 (*never in the past month*) to 6 (*every day*). This measure was used for descriptive purposes.

Illicit substance-related consequences

Participants who indicated that they had used substances in the past 30 days were asked to respond whether they had experienced a series of 24 substance-related consequences. Items were adapted from the YAACQ and the Brief YAACQ (Kahler, Strong, & Read, 2005), reworded to be relevant to drugs. Items were scored dichotomously and summed to create a total drug consequence score that could range from 0 to 24. Individuals who reported no use received a zero on this measure. Cronbach's alpha for the substance consequences scale in this sample was .98.

Big Five Inventory (BFI; John & Srivastava, 1999)

To isolate the unique influences of PTSD, we controlled for negative affect in all models. We assessed baseline negative affect with the Neuroticism subscale (eight items) of the BFI, a 44-item measure that assesses five personality dimensions: Openness, Conscientiousness, Extraversion, Agreeableness, and Neuroticism. Items consist of short phrases based on adjectives that assess prototypical features of each personality dimension, scored using a 5-point Likert scale. The Neuroticism subscale demonstrated good internal reliability (T1 $\alpha = .84$). Possible scores ranged from 8 to 40.

Trauma exposure

At matriculation, students were screened for Criterion A trauma with items based on the Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000). Seven items assessed exposure to (1) accident/natural disaster/fire; (2) combat; (3) sudden unexpected death of a loved one; (4) life-threatening illness (to self or loved one); (5) physical assault; (6) sexual assault; or (7) other event that was life-threatening, caused serious injury, or caused extreme distress. A follow-up question assessed subjective responses (i.e., fear, helplessness, or horror) to each endorsed event.

In the longitudinal portion of the study, the TLEQ (Kubany et al., 2000; copyright 2004 by Western Psychological Services; format adapted by Jennifer P. Read, University at Buffalo, State University of New York) was used to assess trauma exposure. At Time 1, lifetime exposure was assessed. The TLEQ assesses a range of traumatic experiences consistent with the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM–IV–TR*; American Psychiatric Association, 2000) definition, including the subjective responses that comprise Criterion A2. This measure has demonstrated good psychometric properties and has been used with college students (Kubany et al., 2000). Scores yielded a count of lifetime traumas endorsed that were accompanied by fear, helplessness, or horror (i.e., Criterion A trauma). The number of trauma types that an individual has experienced has been used as an index of trauma severity (Bernat, Ronfeldt, Calhoun, & Arias, 1998; Perkonigg, Kessler, Storz, & Wittchen, 2000) and has been shown to be among the strongest predictors of the development of PTSD. Accordingly,

we used the TLEQ summed score (range = 0–22) to index trauma severity, with total scores representing the number of discrete types of trauma experienced.

Posttraumatic stress symptoms

PTSD was assessed using the PTSD Checklist–Civilian Version (PCL-C; Weathers, Huska, & Keane, 1991; Weathers, Litz, Herman, Huska, & Keane, 1993). This 17-item measure assesses Criteria B (re-experiencing), C (avoidance/numbing), and D (arousal) of the PTSD construct consistent with the *DSM–IV*, and has been shown to correspond strongly to gold-standard interview measures of PTSD (Andrykowski, Cordova, Studts, & Miller, 1998; Lang, Laffaye, Satz, Dresselhaus, & Stein, 2003). Participants rated on a 5-point scale how much they had been bothered by each symptom in the past month. These idiographic traumas were programmed into the PCL-C instructions, and participants were instructed to think about the particular traumatic event(s) that they endorsed while filling out the PCL-C (e.g., "You indicated that you have been in a fire and have experienced the sudden death of a loved one. For the next questions we would like you to think specifically about your response to that event…"). For those who did not report lifetime Criterion A exposure, the computerized PCL-C prompted respondents to think about how they respond in general to stressful events that they have experienced.

Following Blanchard, Jones-Alexander, Buckley, and Forneris (1996), empirically derived severity threshold cut-scores were created for all items on the PCL. This involved dichotomously scoring each item as either "1" or "0" based on the severity rating that the participant assigns on the 5-point Likert-type scale. Items rated as 3 or 4 (depending on the item; see Blanchard et al., 1996) or higher are scored as a "1." All other ratings are scored as a "0." Using this approach, each of the 17 items was dichotomously scored to reflect whether that symptom was "present" or "absent." Thus, the possible range of scores on this measure is from 0 to 17. This scoring produces a clinically meaningful symptom count. Symptoms from Criteria B, C, and D were summed to create a symptom count in each cluster. Possible subscale symptom scores ranged from 0 to 5 for re-experiencing (B), 0 to 7 for avoidance/numbing (C), and 0 to 5 for arousal (D).

Data Analytic Plan

After data cleaning, our first step was to examine frequencies of trauma, PTSD, and substance use variables. We also examined bivariate associations among model variables. Following this, latent growth models (LGMs) were used to test our primary aims because they provide an analytic framework to describe sample average trajectories as well as individual differences in growth and predictors of individual differences in growth (Curran & Muthén, 1999). Our central question was whether baseline trauma exposure and PTSD symptom status (Trauma and PTSD Group) predicted change in alcohol and drug consequences across the first year of college. Accordingly, we tested a series of unconditional growth models to determine the shape of growth in alcohol and drug consequences, and then we tested conditional growth models with trauma and PTSD status as predictors of growth factors. To examine whether both alcohol and drug consequence trajectories differed for men and women, we estimated multiple group models and used nested chi-square tests to examine the equivalence of parameters across gender.

To examine differences in trajectories across trauma and PTSD symptom categories, we created three dummy coded variables based on four orthogonal groups (i.e., participants could only be included as a member of one PTSD symptom category). The groups were (1) No Criterion A trauma, no significant PTSD symptoms (No Criterion A); (2) Criterion A exposure, but no significant PTSD symptoms (Criterion A Only); (3) Criterion A exposure and at least one symptom in each symptom cluster (Partial PTSD); and (4) Criterion A exposure and full PTSD symptoms—1 re-experiencing, 3 avoidance, and 2 hyperarousal—met (Full PTSD). Full PTSD was the reference group, and thus the dummy coded variables contrasted each of the remaining groups to the Full PTSD group. These dummy coded variables were created to allow us to examine the relative contribution of trauma exposure versus posttraumatic stress symptoms to alcohol and drug consequences. Site also was a covariate. In secondary analyses, we also examined the prospective influence of specific symptom clusters (re-experiencing, avoidance/numbing, hyperarousal) and of trauma severity on consequences trajectories. To control for the possible contribution of general negative affect, symptoms of which might overlap with and thus confound the influence of PTSD symptoms, all models included baseline trait negative affect, measured by the BFI.

Growth models were estimated in Mplus Version 6.1 (Muthén & Muthén, 2010). The observed alcohol and drug consequence variables were skewed (values from 1.8 to 4.5), and thus robust maximum likelihood estimation was used to correct fit indices and standard errors for the effects of non-normality. Nested model tests were performed using robust maximum likelihood chi-square difference tests (Muthén & Muthén, 2010). Separate models were estimated for alcohol and drug consequences. In addition to the model chi-square, we report the comparative fit index (CFI), Tucker–Lewis index (TLI), and root-mean-square error of approximation (RMSEA). According to Hu and Bentler (1999), CFI > .95, TLI > .95, and RMSEA < .05 indicate a well-fitting model.

Management of missing data

All participants had complete data on outcome variables at Time 1. Eighty-one percent (n = 810) of the sample completed all six time points; 90% (n = 900) completed at least five time points. Three percent (n = 23) completed two or fewer time points. At the item level, rates of missed/skipped items (including cases with a missing time point) were 9.9% (alcohol) and 9.2% (drug) consequences, respectively, over all six assessments.

To examine the potential influence of missing data, two dummy variables were created to indicate whether a participant had missing data on alcohol and drug consequence variables at any of the five longitudinal time points. We examined whether these dummy variables were related to model variables or to trauma group. For alcohol and drug consequences, missing data were related to neuroticism, site, and trauma group. Missing data on alcohol consequences were not related to T1 alcohol consequences, but presence of any missing data on substance consequences was related to T1 substance consequences. Missing data were unrelated to sex for both outcomes. Missing data did not differ across trauma/PTSD symptom groups for alcohol consequences, $F(3, 993) = 0.22$, $p > .05$, or drug consequences, $F(3, 993) = 2.3$, $p > .05$. To gauge the influence of missing data, we calculated effect sizes for each association. Cohen's d (continuous variables) ranged from .16 to .35, and Craemer's phi (dichotomous) coefficients ranged from .05 to .18—all

small effects (Cohen, 1988). Still, to minimize even a small impact, we used full-information robust maximum likelihood estimation rather than listwise deletion in our analyses.

Results

Rates of Trauma, PTSD, and Alcohol and Drug Involvement

Seventy-four percent of the sample (n = 735) endorsed at least one Criterion A trauma. Among those, the average number of traumas (trauma severity) was 3.09 (SD = 2.19). Trauma types were diverse and included, but were not limited to, interpersonal traumas (e.g., unwanted sexual contact; 13%), physical assault by partner (7%), natural disasters (11%), sudden or unexpected death of someone close (65%), life-threatening illness or injury of other (47%) or self (5%), and motor vehicle accidents (11%). The average number of PTSD symptoms among those with a Criterion A trauma was 4.60 (SD = 4.06). Fifteen percent (n = 152) of participants met criteria for full PTSD (Criterion A trauma exposure and 1 B, 3 C, 2 D symptoms). An additional 15% (N = 151) met criteria for partial PTSD (trauma exposure and one PTSD symptom in each cluster).

At baseline, participants typically consumed 6.42 (SD = 9.70) alcoholic drinks per week in the past month, drinking on an average of 1.43 occasions (SD = 1.48) weekly. The average number of alcohol consequences in the month prior to baseline assessment was 5.34 (7.41). Thirty-nine percent (n = 390) of our participants did not report any alcohol consequences at baseline and thus had a score of zero for this measure.

Across the six time points, rates of ever having used illicit substances were 30% (n = 299) for cannabis, 7.1% (n = 71) for amphetamines, 5.6% (n = 56) for cocaine, 2.2% (n = 22) for inhalants, 9.1% (n = 91) for sedatives, 4.4% (n = 44) for hallucinogens, and 6.2% (n = 62) for opiates. Among those reporting drug use, the modal number of drugs used at any given time point was one. Seventy-nine percent (n = 974) reported no drug consequences.

Group Differences on Demographics, TLEQ, PCL, and Substance-Use-Related Consequences

As shown in Table 1, there were no group differences on age and ethnicity; however, there were gender differences on trauma and PTSD status. Group differences were also observed on neuroticism. These findings suggest that it is important to include gender and neuroticism when testing group differences in subsequent analysis. As expected, there were group differences on the TLEQ and PCL.

ANCOVAs were used to test trauma group differences on alcohol and drug-related consequences in the fall (averaged across the four fall assessments) and spring (averaged across the two spring assessments) semesters. Gender, neuroticism, and site were included so that we could test the unique effects of trauma group. As shown in Table 1, there were group differences in average number of alcohol and drug consequences in both semesters. Post hoc comparisons using the Dunnett–Hsu adjustment for multiple comparisons with the Full PTSD group as the comparison group suggested that those with Full PTSD at matriculation reported more alcohol-related consequences in both the fall and spring semesters compared to the No Criterion A and the

Criterion A Only groups (*p*s < .05). However, no reliable differences were observed between the Full and Partial PTSD groups in either the fall or spring semester (*p*s > .50). Post hoc comparisons showed a similar pattern for drug-related consequences. Those with Full PTSD at matriculation reported higher levels of drug-related consequences in the fall semester compared to the No Criterion A and the Criterion A Only groups (*p*s < .05). This Full PTSD group also reported higher levels of drug-related consequences compared to the No Criterion A group ($p < .05$) and the Criterion A Only group ($p < .07$) in the spring semester, albeit the latter comparison fell short of conventional criteria for statistical significance. Differences between the Full and Partial PTSD groups on drug-related consequences were not statistically reliable in the fall or spring semester (*p*s > .45).

Table 1. Comparison of Trauma and PTSD Group Status on Demographics and Self-Report Measures

	No Criterion A (*n* = 263)		Criterion A Only (*n* = 431)		Partial PTSD (*n* = 151)		Full PTSD (*n* = 152)				
Variable	***n***	**%**	***n***	**%**	***n***	**%**	***n***	**%**	χ^2	***df***	***p***
Sex											
Female	114	43.3	303	70.3	117	77.5	115	75.7	77.59	(3)	.00
Ethnicity											
Caucasian	204	77.6	308	71.5	107	70.9	104	68.4	4.57	(3)	.21
Other	59	22.4	122	28.3	42	27.8	47	30.9			
	M	***SD***	***M***	***SD***	***M***	***SD***	***M***	***SD***	***F***	***dfs***	***p***
Age	18.13	0.39	18.11	0.48	18.05	0.24	18.17	0.57	2.052	(3,993)	.105
No. of Criterion A events (TLEQ)	0	0	2.46	1.68	3.54	2.18	4.44	2.69	255.25	(3,993)	.000
PTSD symptom count (from PCL with Blanchard cutoffs)	1.22	2.64	1.89	1.81	6.22	1.88	10.68	2.57	791.49	(3,993)	.000
Neuroticism (BFI)	2.64	0.77	3.07	0.80	3.32	0.86	3.54	0.75	48.36	(3,993)	.000
Alcohol problems fall semester[a]	2.71	4.57	3.65	5.61	5.22	6.10	5.95	8.04	9.99	(3,991)	.000
Alcohol problems spring semester[b]	2.80	5.50	3.37	6.05	5.64	8.22	5.34	8.18	7.31	(3,192)	.000
Drug problems fall semsester[a]	0.48	1.82	0.59	1.65	1.17	2.69	1.38	2.80	8.59	(3,990)	.000
Drug problems spring semester[b]	0.36	1.68	0.51	1.92	1.27	2.94	0.95	2.50	7.00	(3,906)	.000

Note. PTSD = posttraumatic stress disorder; TLEQ = Traumatic Life Events Questionnaire; PCL = PTSD Checklist–Civilian Version; BFI = Big Five Inventory.

[a] Average number of problems reported across the four fall semester assessments. [b] Average number of problems reported across the two spring semester assessments.

Trauma and PTSD Symptom Effects on Alcohol and Drug Consequence Trajectories

In a preliminary step, the shape of growth was tested for each construct. Growth models included intercept and slope factors. The factor loadings for the slope factors specified polynomial trends (linear, quadratic, and cubic), and the intercept was defined as the first (T1) assessment. We started with a model that specified an intercept and linear growth factor, and then we added quadratic and cubic growth factors sequentially. For alcohol consequences the quadratic model was superior to the linear model, $\Delta\chi^2(4) = 40.84$, $p < .05$. Adding a cubic growth factor resulted in non-convergence, suggesting over-fitting of the model. Accordingly, a model with a fixed cubic factor was estimated, and this model was superior to the quadratic model, $\Delta\chi^2(1) = 108.60$, $p < .05$. The final model with random intercept, linear, and quadratic growth factors and a fixed cubic growth factor fit the data well, $\chi^2(11) = 41.89$, $p < .01$, CFI = .98, TLI = .98, RMSEA = .05. All growth factor means and variances were significantly different from zero ($p <$

.05), and the model accounted for between 78% and 89% of the variability in the observed alcohol consequence variables. Similar results were found for drug consequences. The quadratic model was superior to the linear model, $\Delta\chi^2(4) = 23.96$, $p < .05$. Adding a cubic growth factor resulted in non-convergence. A model with a fixed cubic growth factor was superior to the quadratic model, $\Delta\chi^2(1) = 22.31$, $p < .05$. The final model with random intercept, linear, and quadratic growth factors, and a fixed cubic growth factor fit the data well, $\chi^2(11) = 17.90$, $p > .05$, CFI = .98, TLI = .97, RMSEA = .02. All growth factor means and variances were significantly different from zero ($p < .05$), and the model accounted for between 71% and 90% of the observed variability in the drug consequence variables.

Trauma and PTSD effects on alcohol consequence trajectories

We then added the dummy coded PTSD group variables and control variables to estimate conditional growth models. Growth factors were regressed on control variables, and models with and without paths from the dummy coded PTSD variables were compared to provide an omnibus test of the effect of PTSD on growth in alcohol consequences. The nested chi-square test suggested a significant improvement in model fit when paths from the PTSD variables to the growth factors were added, $\Delta\chi^2(9) = 45.76$, $p < .05$. The conditional model with paths from the PTSD variables fit the data well, $\chi^2(28) = 71.25$, $p < .01$, CFI = .98, TLI = .97, RMSEA = .04, for alcohol consequences, and the increment in R^2 for the intercept, linear, and quadratic growth factors attributable to the PTSD paths was .05, .04, and .04, respectively. In addition to a significant intercept difference for site (northeastern site reporting higher levels of consequences; $p < .01$), we also observed significant baseline intercept effects for the trauma and PTSD contrasts. While there was no intercept difference between the Partial and Full PTSD groups, there were intercept differences between No Criterion A and Full PTSD groups and between Criterion A Only and Full PTSD groups (ps < .05). The nature of these intercept effects was such that students who reported either no Criterion A or Criterion A only (i.e., trauma but no PTSD symptoms) reported fewer alcohol consequences at college matriculation (T1) than those in the Full PTSD group. Differences in both linear and quadratic trends also were observed. The linear trend was more negative for the Full PTSD group compared to Criterion A Only group ($p < .05$) and was marginally more negative compared to the No Criterion A group ($p < .10$), such that the Full PTSD group showed a steeper rate of decline in alcohol consequences at the beginning of the study. The quadratic trend was more positive for the Full PTSD group than for the No Criterion A group or the Criterion A Only group, suggesting a slight upward trend in alcohol consequences as the academic year ended for those with PTSD. A marginally significant difference was found between the Criterion A Only and Full PTSD groups on the quadratic trend ($p < .10$), suggesting that the rate of decline in alcohol consequences at the beginning of the study was slower to level off for the Full PTSD group. The model implied trajectories for each group are plotted in Figure 2. The Full and Partial PTSD groups had very similar trajectories, starting college at the highest levels of consequences and then showing a sharp decline during the fall semester. The No Criterion A and Criterion A Only groups started the college year with fewer alcohol consequences and showed more modest declines during the fall semester.

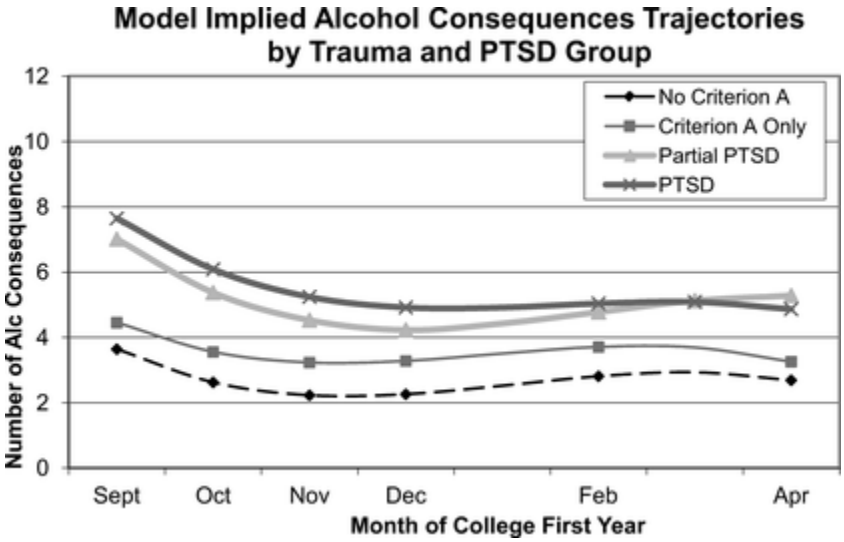

Figure 2. First year college alcohol (Alc) consequences trajectories by trauma and posttraumatic stress disorder (PTSD) symptom status. Full PTSD was the contrast for these comparisons. All models controlled for negative emotionality.

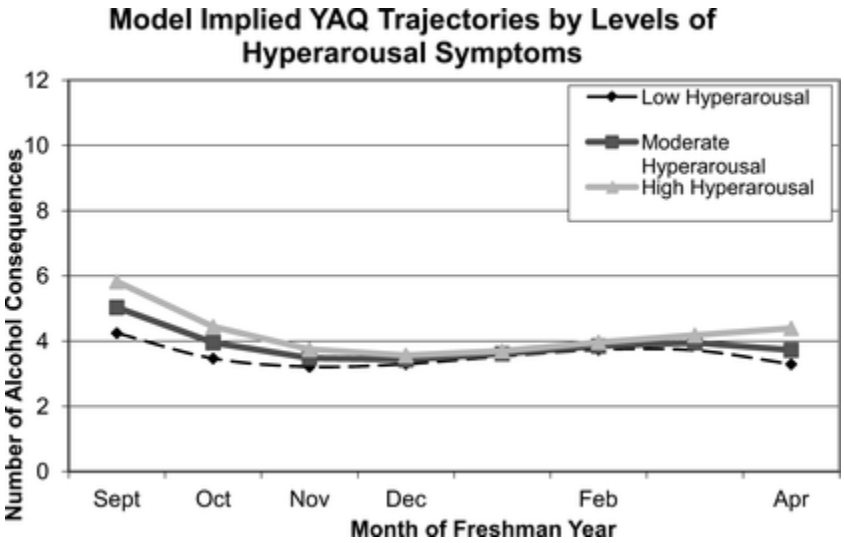

Figure 3. First year college alcohol consequences trajectories by level of Cluster C (hyperarousal) symptoms. All models controlled for negative emotionality. YAQ = Young Adult Alcohol Consequences Questionnaire.

Exploratory analysis of PTSD symptom cluster effects on alcohol consequence trajectories

Next, the dummy coded PTSD predictor variables were removed from the model and were replaced with the PTSD symptom cluster (re-experiencing, avoidance/numbing, hyperarousal) variables. This model conditioned on PTSD symptom clusters fit the data well, $\chi^2(31) = 71.08$, $p < .01$, CFI = .98, TLI = .98, RMSEA = .04, and demonstrated that hyperarousal, but not re-experiencing or numbing, symptoms were associated with the growth factors. Hyperarousal symptoms predicted the intercept and linear and quadratic trends (all *p*s < .05). As shown in Figure 3, high levels of hyperarousal symptoms were associated with high levels of negative

drinking consequences in September and with a relatively rapid decline in consequences during the fall semester. However, negative drinking consequences showed a slight rise at the end of the spring semester for students with high levels of hyperarousal symptoms.

Trauma and PTSD effects on other drug consequence trajectories

Comparison of conditional growth models for drug consequences with and without paths from the dummy coded PTSD group variables suggested a significant improvement in model fit when these paths were included, $\Delta\chi^2(9) = 31.46$, $p < .05$. The conditional model with paths from the PTSD variables fit the data well, $\chi^2(28) = 40.98$, $p > .05$, CFI = .99, TLI = .98, RMSEA = .02, and increment in R^2 for the intercept, linear, and quadratic growth factors attributable to the PTSD paths was .03, .01, and .01, respectively. As with the alcohol consequence model, significant baseline intercept effects were observed for the contrast between No Criterion A and Full PTSD and between Criterion A Only and Full PTSD (ps < .05) groups. The nature of these intercept effects was that students with either no Criterion A trauma or Criterion A trauma only reported fewer drug consequences at college matriculation (T1) than those in the Full PTSD group. There was no intercept difference between the Partial and Full PTSD groups. Differences in linear growth were observed between the No Criterion A and Full PTSD groups ($p < .05$), such that the decline in drug consequences was steeper for the Full PTSD group at the beginning of the academic year. No differences between groups were observed for the quadratic trend. The model implied trajectories for each group are plotted in Figure 4 and suggest trends similar to those observed for alcohol consequences, albeit smaller differences. Again the Full and Partial PTSD groups had similar trajectories, starting at the highest levels of consequences and showing a sharp decline in the fall semester. The No Criterion A and Criterion A Only groups started college with fewer consequences and showed more modest declines.

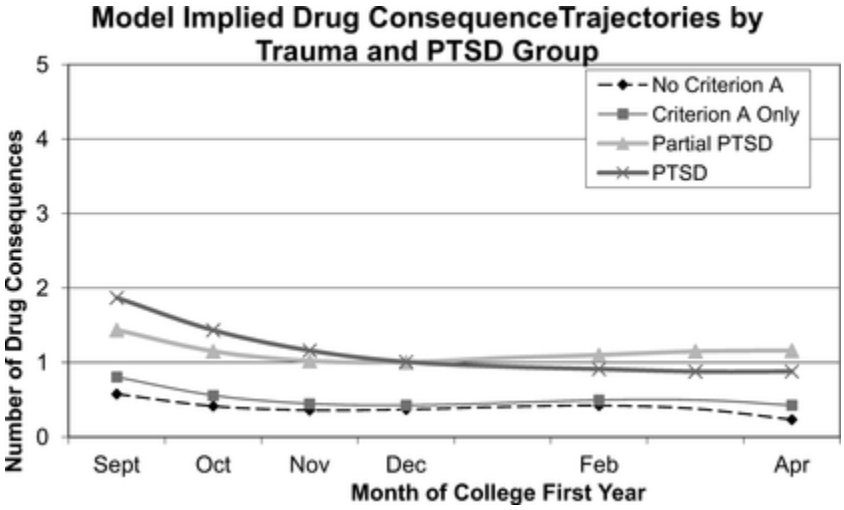

Figure 4. First year college drug consequences trajectories by trauma and posttraumatic stress disorder (PTSD) symptom status. Full PTSD was the contrast for these comparisons. All models controlled for negative emotionality.

Exploratory analysis of PTSD symptom cluster effects on drug consequence trajectories

As we did for alcohol use consequences, the trauma group dummy coded predictor variables were removed from the model and were replaced with the PTSD symptom cluster variables. Results suggested that the substance consequences growth model conditioned on PTSD symptom clusters fit the data well, $\chi^2(31) = 40.95$, $p < .11$, CFI = .99, TLI = .98, RMSEA = .02. None of the symptom clusters, however, reliably predicted the growth factors (*p*s > .20).

Gender differences

Next, we examined potential gender differences in our models. Multiple group models were estimated, and nested chi-square tests were used to test the equivalence of parameters across gender. Two sets of parameters were constrained to be equal across gender, including paths from the PTSD group variables and the means/intercepts of the latent growth factors, in separate nested tests. For both alcohol, $\Delta\chi^2(9) = 10.78$, $\Delta\chi^2(4) = 1.75$, *p*s > .05, and drug, $\Delta\chi^2(9) = 4.07$, $\Delta\chi^2(4) = 10.78$, *p*s > .05, consequences, the nested models suggested no gender differences in causal paths from the PTSD group variables or the means/intercepts of the growth factors. Also, no gender differences were observed when paths from the PTSD symptom clusters were constrained for alcohol, $\Delta\chi^2(9) = 9.07$, $p > .05$, and drug, $\Delta\chi^2(9) = 5.67$, $p > .05$, consequences.

Trauma severity

In a final set of exploratory analyses, we considered the influence of trauma severity on substance consequence outcomes, using number of discrete trauma types derived from the TLEQ. To do this, we conducted our analyses (1) conditioning the alcohol and drug consequence trajectories on trauma severity and then (2) including both PTSD status and trauma severity simultaneously in our models. In the first of these models, we found trauma severity to be significantly associated with both alcohol and drug intercept factors, but we observed no effects for trauma severity on either linear or quadratic slopes. Thus, our main analyses showed that PTSD predicted slopes, but our secondary analysis showed that trauma severity did not. To further explore the effects of trauma severity, we examined the conditional growth trajectories at 1 *SD* above the sample mean, a level equivalent to average levels of trauma severity in our PTSD group. For alcohol consequences, this trajectory suggested that high levels of trauma were associated with 6.5 consequences at the first assessment (one fewer consequence than that predicted for the PTSD group: 7.6; see Figure 2) and with 4.9 consequences at the last assessment (equivalent to that predicted for the PTSD group: also 4.9; see Figure 2). For drug consequences, the model implied trajectory suggested that high levels of trauma were associated with 1.4 consequences at the first assessment (0.5 fewer than that predicted for the PTSD group; see Figure 4), and with 0.9 consequences at the last assessment (equivalent to that predicted for the PTSD group: also 0.9; see Figure 4). This general pattern suggests that PTSD status conferred higher risk at matriculation than did high levels of trauma severity, and this likely accounts for the observation that PTSD, but not trauma severity, predicted declines in consequences.

In the second set of models, in which alcohol and drug consequence trajectories were conditioned on both the dummy coded PTSD variables and trauma severity, we observed trauma severity and PTSD group both to be significantly associated with alcohol and drug consequences intercept factors (*p*s < .05). Not surprisingly, with both of these highly correlated predictors in

the model, there were no significant effects on either the linear or quadratic slope, suggesting that the shared variability in trauma severity and PTSD symptoms left little remaining unique variability for the prediction of these trajectories.[1]

Discussion

In this study, we provide what is to our knowledge the first examination of the prospective relationship between trauma and PTSD and substance involvement in young adults during their transition into college. Findings showed substantial risk for alcohol and other drug consequences that appears to be conferred by the presence of PTSD symptoms at matriculation. This association occurred above and beyond the influence of general trait negative emotionality, suggesting a unique effect of PTSD symptoms.

Our latent growth models show that risk for problem substance use is greatest for those with PTSD (and perhaps also for those with partial PTSD) at the beginning of the academic year. It can be noted from the figures that those in the Full PTSD group started college with almost twice as many consequences as those in the non-symptom groups—a clinically as well as statistically significant difference. Contrary to our expectations, this risk decreased over the course of the college year relative to those with only trauma exposure or no exposure at all. This likely is a function of the fact that those in these PTSD symptom groups began at higher consequence levels and thus had more room for diminution of consequences. Still, despite the declines that we observed, our analyses of covariance (ANCOVAs) showed that differences between those with significant PTSD symptoms and those without persisted across the academic year.

The first months of college are a time of particular risk for students regarding alcohol and other drug involvement (Schulenberg & Maggs, 2002; Sher & Rutledge, 2007; White, Labouvie, & Papadaratsakis, 2005). Though our data and others show that at least some of these consequences resolve naturalistically with time, these early consequences still can have significant acute and long-term implications for the individuals experiencing them. Findings here suggest that trauma exposure and PTSD in particular heighten risk for alcohol and other drug consequences during this period of vulnerability. Moreover, though our models suggest that consequences diminish at a faster rate during the fall semester for those with PTSD symptoms than for those without PTSD, they continue to remain significantly higher over the academic year. This points to a unique effect of PTSD over the most important year of transition into college.

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[1] We also ran our conditional growth models with PTSD operationalized as a continuous variable (a sum of PTSD symptoms). Results for the alcohol consequence model suggested that high levels of PTSD symptoms were positively associated with the intercept ($p < .05$), were negatively associated with the linear trend ($p < .05$), and were marginally positively associated with the quadratic trend ($p < .10$). The nature of these effects mirrors what we found in our main analysis. That is, more severe forms of PTSD (e.g., Full PTSD or a high number of symptoms) are associated with high levels of negative alcohol consequences at the beginning of the study that decline steeply during the course of the first semester. Results for the drug consequence model were also consistent with our main analysis, such that high levels of PTSD symptoms were associated with high initial levels of drug consequences ($p < .05$), and PTSD symptoms were not associated with the slope factors. When the trauma severity (number of traumas) variable was added to these conditional models, the effects of the continuous the PTSD symptom variable on the slope factors were not statistically reliable for both alcohol and drug consequences. This again mirrors our main analysis, suggesting that considering both trauma severity and PTSD symptoms simultaneously leaves little unique variance in the prediction of trajectories.

In our models, Full PTSD was the contrast variable with which other trauma and PTSD groups were compared. Interestingly, though there were consistent differences in consequences between the non-trauma exposed and the Criterion A trauma only groups and the Full PTSD group in drug and alcohol consequence trajectories, there were no differences between Partial and Full PTSD groups. This suggests that the impact of sub-threshold PTSD symptoms on substance consequence outcomes is not substantially different from the impact of full PTSD. These findings offer further support for the clinical significance of PTSD syndromes that fall below a categorical threshold but that nonetheless impact functioning (Mendlowicz & Stein, 2000; Schützwohl & Maercker, 1999; Stein, Walker, Hazen, & Forde, 1997; Zlotnick et al., 2002).

Findings from this study also shed light on another question that has been raised in the PTSD-substance comorbidity literature—that is, whether problem substance use emerges in response to exposure to a trauma event itself or as a result of continued psychological symptoms that follow the event (Epstein et al., 1998; Read et al., 2004; Stewart, 1996; Stewart & Conrod, 2003). We observed the strongest effects for PTSD symptoms—both partial and full PTSD—rather than Criterion A trauma exposure alone. Though to a lesser extent, it also appeared that trauma severity (rather than just exposure) also influenced substance consequence outcomes. These findings corroborate what is to our knowledge the only other study to test the distinct effects of trauma versus PTSD prospectively in a young adult sample (Reed et al., 2007). These investigators found that posttraumatic stress symptoms, and not simply trauma exposure, were associated with risk for substance misuse 1 year later.

We found the hyperarousal cluster to show unique prediction of alcohol consequence trajectories but no evidence of symptom cluster effects for drug trajectories. Though as noted, the research literature has yielded disparate outcomes, findings from the present study are consistent with some previous work (McFall et al., 1992; Shipherd et al., 2005; Stewart et al., 1998; Taft et al., 2007) showing hyperarousal symptoms to be significantly associated with alcohol consequences in particular. Perhaps the perceptual, physical, affective, and cognitive symptoms that comprise the hyperarousal cluster may, when combined with the disinhibiting effects of alcohol, render students particularly vulnerable to externalizing and other problem behaviors (e.g., physical fights, sexual aggression, other risk behaviors).

Limitations and Future Directions

This study had a number of strengths, including a large sample drawn from two universities in different regions of the United States, a high retention rate, prospective design, and frequent assessments over the first college year. Still there were some limitations and also several directions for future investigation. These are discussed below.

In the present study, we sought to understand the prospective effects of trauma and PTSD at college matriculation on alcohol and other drug involvement over the first year of college. We found support for the self-medication hypothesis, which suggests that individuals may use substances to ameliorate PTSD symptoms (Jacobsen et al., 2001; Khantzian, 2003; Saladin et al., 1995). The specific way in which college students may self-medicate is worth noting. Alcohol and drug use in college tends to occur in a fairly consistent pattern that is best represented by weekend use (Colder et al., 2006; Tremblay et al., 2010). Given these typical patterns in college

samples, what is most likely is that to the extent that these students are self-medicating, this is occurring within the context of a strongly routinized substance use pattern. As such, rather than turning to alcohol or drugs every time a PTSD symptom is experienced, students with PTSD instead may be more likely to mismanage their consumption or themselves when drinking and drug use occasions occur. This is consistent with models of psychological functioning that conceptualize stress responses as a function of depletion of cognitive and other coping resources, which may then result in problem behavior in specific circumstances or environments (Bauer & Baumeister, 2011; Muraven & Baumeister, 2000).

Our findings in support of a self-medication process do not exclude other possible directions of association. For example, those who use substances heavily may be at greater risk for accidents, violence, or other types of trauma that may then result in greater posttraumatic stress symptoms (Chilcoat & Breslau, 1998). Heavy substance use also may exacerbate PTSD symptoms (Saladin et al., 1995). Such high-risk pathways were not tested here. We also did not examine the influence of additional traumas or worsening PTSD symptoms over the course of the year. The possibilities for future investigations of association are numerous and point to exciting opportunities for further examination.

In this study, we sought to examine trauma, PTSD, and substance consequences at the time of college matriculation. Conducting over 1,000 clinical interviews with newly incoming students in their first month of college was not feasible. Thus, we employed an online assessment. Our measures were chosen for their strong psychometric properties (Peirce, Burke, Stoller, Neufeld, & Brooner, 2009; Ruggiero, Del Ben, Scotti, & Rabalais, 2003), and prior work supports concurrent validity of the web-based trauma and PTSD measures used here with structured interviews (Read, Farrow, Jaanimagi, & Ouimette, 2009). Still, diagnostic interview remains the gold-standard assessment of clinical syndromes. Replication of these associations with interview data will build on the present findings.

Further, though the PCL has been used in many college samples (e.g., Adkins, Weathers, McDevitt-Murphy, & Daniels, 2008; Hoyt & Yeater, 2010; Read et al., 2011) and has been shown to correlate strongly with interview assessment of PTSD in college students (Adkins et al., 2008; Read et al., 2009; Ruggiero et al., 2003), the cut-scores themselves have not been widely used in college samples. As such, it is possible that the scoring cutoffs recommended by Blanchard et al. (1996) may actually under-estimate PTSD in a sample where the base rates of PTSD are lower.

We did not find gender to moderate the effects of trauma and PTSD on substance consequence trajectories. Other factors may affect these processes and should be tested in future research. For example, socio-environmental influences such as peer group affiliation (heavy drinking peers, sorority/fraternity involvement) and perceptions about normative substance use behavior exert a strong influence on the adoption and maintenance of substance behaviors in college (Borsari & Carey, 2001; Neighbors, Lee, Lewis, Fossos, & Larimer, 2007), and social support has been shown to be relevant to posttrauma adaptation (Charuvastra & Cloitre, 2008; Keane, Marshall, & Taft, 2006). As such, socio-environmental factors may be important moderators to test in future research. Other factors that could be relevant to PTSD–substance misuse include but are not limited to variables such as coping, re-victimization status, or alcohol and drug expectancies.

Future research also will add to the present work by examining a range of psychological distress beyond just posttraumatic distress. Though in our models we controlled for negative emotionality, we cannot from our data know how specific expressions of mood or anxiety (e.g., depression, generalized anxiety, phobias) may affect problem substance use, or how these may interact with PTSD to influence substance trajectories.

This study captures associations among trauma, PTSD, and substance use during a salient passage of young adulthood—the transition into college. Studies of these relations during other periods of vulnerability are needed. Schulenberg and Maggs (2002) have noted that substance use rates tend to increase during periods of transition. Though the transition into college has been identified as one distinct period of risk for escalation in substance use (see Del Boca, Darkes, Greenbaum, & Goldman, 2004), scant attention has been paid to substance use at other potentially important periods of change. Accordingly, an interesting direction for future research will be to examine whether the risk associated with PTSD symptoms persists beyond the first year of college and, perhaps most importantly, whether an effect is again observed during another important developmental transition—the transition out of college and into mature adulthood.

The present study has intervention implications. Risk for problem alcohol and other drug involvement was associated with baseline trauma and PTSD status. This risk was greatest at the point of matriculation, an identified period of vulnerability (Schulenberg & Maggs, 2002; Sher & Rutledge, 2007). Further, despite sharp declines over the first semester, greater alcohol and other drug consequences were observed for those with PTSD symptoms over the academic year. Accordingly, early identification of significant PTSD symptoms may deter problem substance use during the critical developmental transition into college and could offset risk that persists over the first college year. This could include something as simple as screening and outreach for individuals who present to university counseling centers with PTSD symptoms, or as elaborate as developing integrated treatments for PTSD–substance abuse for college students. Several PTSD–substance abuse interventions have been developed (Back, Dansky, Carroll, Foa, & Brady, 2001; Donovan, Padin-Rivera, & Kowaliw, 2001; Najavits, 2002; Triffleman, Carroll, & Kellogg, 1999). Yet, none of these have been tested in a college population (Borsari, Read, & Campbell, 2008).

According to Curran (2000), “There is a great need to link theoretical and statistical models in applied research… this is especially evident in developmental studies of substance use” (p. 3). Here, we present such a link. Our findings suggest that students experiencing PTSD symptoms as they matriculate into college are at greater risk for alcohol and other drug consequences during this time. Interventions offering support and resources to these students may ameliorate this risk and may ultimately facilitate a better transition into college and beyond.

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