PTSD Symptom Course During the First Year of College

Jennifer P. Read and Rachel L. Bachrach  
University at Buffalo, State University of New York  
Aidan G. C. Wright  
University of Pittsburgh  
Craig R. Colder  
University at Buffalo, State University of New York

Objectives: In this study we examined patterns of transition in posttraumatic stress disorder (PTSD) symptoms over the 1st year of college. We also examined 2 factors that might predict these transitions: trauma exposure and alcohol involvement. Method: Matriculating students (N = 944; 65% female) completed assessments of PTSD, trauma exposure, and alcohol use and consequences multiple times in their freshman year. Three symptom classes (no symptoms, moderate symptoms, and severe symptoms) were identified. Examination of transitions from 1 status to another was conducted with latent transition analysis. Results: These models revealed considerable variability in the course of PTSD symptoms. The most common pattern was resolution, yet a significant portion of students showed other patterns. Symptom worsening was more commonly observed in the 2nd semester. Trauma exposure had a deleterious effect on PTSD symptom change trajectories, as did alcohol involvement, though less consistently so. Conclusions: Interventions that focus on the timing and correlates of symptom progression may benefit college students with posttraumatic distress.

Keywords: PTSD trajectories, college, alcohol, trauma, latent transition analysis

Supplemental materials: http://dx.doi.org/10.1037/tra0000087.supp

Recent data highlight surprisingly high rates of trauma exposure in college students (Read, Ouimette, White, Colder, & Farrow, 2011; Smyth, Hockemeyer, Heron, Wonderlich, & Pennebaker, 2008), with college attendance being a risk factor for particular trauma types such as sexual assault. Moreover, students remain at risk for experiencing new or repeated trauma throughout their college careers (Frazier et al., 2009). A substantial portion of college students with trauma exposure go on to develop symptoms consistent with a posttraumatic stress disorder (PTSD) diagnosis (Lauterbach & Vrana, 2001; Read et al., 2011). The emerging adult years are a period marked by both risk and growth (Arnett, 2000; Wright, Pincus, & Lenzenweger, 2011), and studies of posttraumatic stress during this period may provide important information about the course of posttrauma adaptation in this developmental context. Yet, little is known about the relative stability or change of these symptoms in college students. Further, factors that influence the course of these symptoms—contributing to resolution, maintenance, or exacerbation—have not been examined in college samples.

The Latent Structure of PTSD

In both clinical and community samples, empirical findings have suggested there is substantial individual variability in the response to potentially traumatic events and in the presentation of PTSD symptoms, both cross-sectionally (Breslau, Reissouin, Anthony, & Storr, 2005) and longitudinally (see Bonanno & Mancini, 2012, for a review). Prior research has adopted both variable-centered (e.g., confirmatory factor analysis; Palmieri, Weathers, Difede, & King, 2007; Simms, Watson, & Doebbeling, 2002) and person-centered (e.g., latent class analysis [LCA]; Breslau, Reissouin, Anthony, & Storr, 2005; Forbes, Elhai, Miller, & Creamer, 2010; McDevitt-Murphy et al., 2009) approaches to describe the latent (i.e., unobserved) structure of PTSD symptoms.
The evident clinical significance of subthreshold PTSD syndromes (Pietrzak, Goldstein, Southwick & Grant, 2011; Wolff et al., 2011), combined with a growing literature emphasizing the utility of considering psychological phenomena as existing along a severity spectrum, have drawn attention to the need for alternative ways of capturing the nosology of PTSD and other psychological symptoms (e.g., Beseler, Taylor, Kraemer, & Leeman, 2012; Wright et al., 2013). LCA can be useful for such examination because it can capture a range of symptom profiles, some of which may be more strongly associated with severity of illness and functional impairment than are others (Lanza, Collins, Lemmon, & Schafer, 2007).

Past work using epidemiological samples has supported the utility of using LCA to model the latent structure of individual responses to trauma. For example, Breslau and colleagues (2005) found that trauma-exposed individuals could be differentiated primarily on the basis of the level and to some degree on the configuration of their symptoms, with a three-class solution best capturing the latent structure of PTSD symptom heterogeneity. These three identified classes roughly conform to (a) full PTSD/severe posttraumatic stress response, (b) partial PTSD/moderate posttraumatic stress response, and (c) healthy/negligible posttraumatic stress response.

The Longitudinal Course of Traumatic Stress

To date, longitudinal approaches have typically relied on latent growth curve modeling or latent growth mixture modeling to identify latent groups of individuals who share a similar change trajectory on a given dimension. An alternative approach to understanding individual patterns of change is to extend LCA by studying the transitions between latent statuses of individuals over time in what has been termed latent transition analysis (LTA; Collins & Lanza, 2010). In LTA, class membership at a given time point is referred to as a status reflecting the potentially transient nature of group assignment.

A strength of LTA is the ability to examine moderators of the transitions between statuses across time. Thus, LTA offers psychopathologists the ability to establish not only what predicts an individual’s status at a given time point but characteristics of the individual (i.e., time-invariant predictors) or experiences that the individual encounters (i.e., time-varying predictors) that predict a shift in symptom status. In the present study we focus on the impact of two such factors that are both common on college campuses and have been theoretically and empirically linked to the development of PTSD symptoms over time: trauma exposure and alcohol involvement (e.g., Breslau, Chilcoat, Kessler, & Davis, 1999; Compton, Mager, Spitznagel, & Janca, 1992). Next, we briefly review the literature, which highlights the potential significance of these risk factors for PTSD.

Risk Factors for PTSD Transitions

Trauma Exposure

PTSD is unique among the disorders of the Diagnostic and Statistical Manual of Mental Disorders in that, by definition, it includes an etiological event: trauma exposure. Further, the literature has documented a cascading relationship among trauma exposure and reexposure and PTSD. Prior trauma exposure is among the strongest predictors of reexposure (Green et al., 2000; Marx, Heidt, & Gold, 2005), and multiple trauma exposures are a risk factor for the development of PTSD (Follette, Polusny, Bechtle, & Naugle, 1996; Schumm, Briggs-Phillips, & Hobfoll, 2006), as well as a predictor of PTSD course (Kolassa et al., 2010). Some evidence has suggested that recent trauma exposure may exert a particularly deleterious influence on PTSD outcomes (Horesh, Apter, & Zalsman, 2011). Accordingly, consideration of the longitudinal course of PTSD must take trauma exposure into account, especially the influence of new traumas that may have occurred in the recent past.

Alcohol Involvement

PTSD and heavy alcohol consumption commonly co-occur and have been etiologically linked. Though much of the extant literature has focused on the extent to which PTSD affects later drinking outcomes (“self-medication” models; e.g., McFarlane et al., 2009; Shiperd, Stafford, & Tanner, 2005), some research has suggested that the reverse is also true: Drinking and its consequences may influence both the presence and course of PTSD (e.g., Bisby, Brewin, Leitz, & Curran, 2009; Stewart, Conrod, Pihl, & Dongier, 1999). The high risk hypothesis highlights the potential significance of alcohol involvement for the development of PTSD symptoms. This model asserts that alcohol use and its consequences may confer risk for psychopathology through the physiological and psychosocial impairment that may accompany it (e.g., Bisby et al., 2009; Read, Merrill, Griffin, Bachrach, & Khan, 2014). As such, alcohol involvement may be a marker of psychological vulnerability (e.g., Jessor, 1987). Yet, studies of the influence of alcohol involvement on PTSD symptom trajectories have been few. None have focused on college students specifically.

Objectives

In the present study, we sought to examine patterns of transition in posttraumatic stress symptoms over the first year of college. To accomplish this, we applied LTA to a large sample of first-year college students. We also examined whether trauma and alcohol involvement exerted an influence on symptom transitions. We expected both of these risk variables to be related prospectively to the likelihood of transitioning into higher severity PTSD status as the college year progressed.

There is a large literature highlighting overlap between posttraumatic stress disorder and a general tendency toward negative affect (e.g., Armour et al., 2015; Breslau & Schultz, 2013; Simms et al., 2002). Further, negative affect has been found to account for some of the link between trauma and PTSD (e.g., Breslau & Schultz, 2013) and alcohol involvement and PTSD (e.g., Miller, Vogt, Mozley, Kaloupek, & Keane, 2006). Accordingly, in the present study, to isolate processes unique to PTSD, we controlled for neuroticism in our models. In addition, because sex has been shown to influence both alcohol involvement and posttraumatic stress (Breslau & Anthony, 2007; Substance Abuse and Mental Health Services Administration, Office of Applied Studies, 2008), we also controlled for sex. We also considered other dimensions of personality to provide some external criterion validity for our latent classes.
Method

Participants

Participants were enrolled in an ongoing longitudinal study of PTSD and substance use behavior in college. The sample for the present study consisted of 944 (65.1% female) participants. At Time 1 (T1), the average age was 18.11 (SD = 0.44). Seventy-two percent self-identified as non-Hispanic Caucasian (n = 678), 12% as Asian (n = 112), 9% as Black (n = 84), 3.4% as Hispanic/Latino (n = 32), less than 1% as Hawaiian/Pacific Islander (n = 1), less than 1% as American Indian/Native Alaskan (n = 2), and 3.3% as multiracial (n = 31). Four participants did not report ethnicity.

Procedure

Extensive details of this procedure are published elsewhere (Read et al., 2012; Read, Guinette, White, Corder, & Farrow, 2011). However, they are reviewed briefly below.

Initial recruitment screen. Participants were incoming freshmen at two midsize public universities in the United States. In the summer prior to matriculation, an initial screen was sent out to assess trauma exposure and PTSD symptoms as well as demographic information. After data was cleaned and cases with significant missing data were deleted, the final screening sample consisted of 3,014 students.

Longitudinal sample selection. Next, students were selected from the screening pool for participation in the longitudinal study. To ensure sufficient representation of students with significant traumatic stress, we invited for participation all those who endorsed (1) at least one Criterion A trauma (see the Measures section) and (2) at least one symptom each from PTSD symptom clusters B, C, and D. A total of 649 participants met these criteria. These and another 585 students from the screening sample who did not meet trauma criteria were invited for longitudinal follow-up.

E-mails and a survey link were sent to this selected sample (N = 1,234). Participants received a $20 gift card for completion of this baseline (T1) survey. Eighty-one percent (N = 1,002) of those invited to participate in the study completed the baseline survey in September of their freshmen year. This constituted the final longitudinal sample.

This longitudinal sample was then assessed three more times (October, November, December) in the first semester and twice (February, April) in the second semester. Data from the September (T1), December (T2), and April (T3) assessment points were used in the present investigation. However, data from the October, November, and February surveys (i.e., interim assessment points) were used to estimate both trauma exposure status and level of alcohol involvement in order to retain and model this behavior when predicting symptom transitions. Participants who had data for at least two out of the three assessment points were retained in analyses (N = 944; 94% of the original T1 sample).

Measures

Trauma exposure. The Traumatic Life Events Questionnaire (Kubany et al., 2000) is a 21-item self-report questionnaire that assesses a range of traumatic experiences consistent with the definition according to the Diagnostic and Statistical Manual of Mental Disorders (4th ed., text rev.; DSM–IV–TR; American Psychiatric Association, 2000). This measure has demonstrated good psychometric properties and has been used in a range of populations, including college students (Kubany et al., 2000). T1 trauma exposure was coded as having occurred if the participant endorsed one or more lifetime Criterion A events. Subsequent trauma exposure captured events that were endorsed between T1 and T2, and T2 and T3, respectively. Thus, any trauma exposure between assessment points was treated as a binary variable at T1, T2, and T3 (0 = did not experience a trauma, 1 = experienced trauma[s]).

Traumatic stress symptoms. Traumatic stress symptoms were assessed at T1, T2, and T3 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 (reexperiencing), C (avoidance/numbing), and D (arousal) of the PTSD construct consistent with the DSM–IV–TR. Participants rated on a 5-point scale (never to almost always) how much they had been bothered by each symptom in the past month. PTSD symptoms were queried specifically with regard to participants’ own Criterion A stressors. These traumatic events were listed in the PCL–C instructions on each web page of the survey.

PTSD symptoms from the PCL–C were then recoded dichotomously as 0 (nonsymptom) or 1 (symptom) on the basis of empirically derived cutoff scores from Blanchard, Jones-Alexander, Buckley, and Forneris (1996). With this method, each symptom was recoded from the 5-point Likert-type scale to a dichotomous scale, so that symptoms were identified as either present (1) or absent (0). Items were summed to obtain a total symptom count, with a range from 0 to 17. This method is most consistent with how clinical diagnostic interviews would classify symptom scores and is more rigorous in its classification of PTSD. Internal consistency in this sample was strong across all three waves (α from .89 to .91).

Using dichotomous scoring of individual symptoms also allows for calculating symptom presentations that would be consistent with a diagnosis of partial or full PTSD. Thus, for descriptive purposes, students were categorized as having partial PTSD if they endorsed at least one PTSD symptom in each of the B, C, and D clusters (see Schnurr, Lunney, Sengupta, & Spiro, 2005). Students were also categorized as having full PTSD on the basis of DSM–IV–TR criteria (i.e., at least one B symptom, three C symptoms, and two D symptoms).

Alcohol status. At each assessment point, participants reported on their past-month alcohol use. This included the interim assessment points that are not included in the formal LTA analysis. Those individuals who reported drinking any alcohol between study time points were coded as drinkers. Individuals who reported not drinking were treated as nondrinkers. We chose to dichotomize alcohol use for several reasons. First, alcohol use was highly skewed, with close to half the sample reporting no drinking in the past month. PTSD symptoms were queried specifically with regard to participants’ own Criterion A stressors. These traumatic events were listed in the PCL–C instructions on each web page of the survey.
Alcohol-related consequences. Previous-month consequences from drinking 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 load on a single, higher order consequences factor. These include Interpersonal Consequences (e.g., “I have become very rude, obnoxious or insulting after drinking”), Academic/Occupational Consequences (e.g., “I have neglected my obligations to family, work, or school because of my drinking”), Risky Behavior (e.g., “I have taken foolish risks when I have been drinking”), Impaired Control (e.g., “I often drank more than I originally had planned”), Poor Self-Care (e.g., “I have been less physically active because of drinking”), Diminished Self-Perception (e.g., “I have felt badly about myself because of my drinking”), Blackout Drinking (e.g., “I have awakened the day after drinking and found that I could not remember a part of the evening before”), and Physiological Dependence (e.g., “I have felt anxious, agitated, or restless after stopping or cutting down on drinking”). For a complete listing of all items see Read et al. (2006). The YAACQ has strong psychometric properties, including convergent validity and test–retest reliability (Read, Merrill, Kahler, & Strong, 2007). Like alcohol use, consequences were highly skewed and hence also dichotomized. Thus, for the present study, participants received a score of either 1 if they endorsed experiencing at least one consequence or 0 if they did not endorse any consequences prior to T1 or between assessment points.

Neuroticism. To isolate the unique influences of PTSD, we controlled for trait negative affect proneness in all LTA models. We assessed this construct with the Neuroticism subscale (eight items) of the 44-item Big Five Inventory (BFI; John & Srivastava, 1999). Items consist of short phrases based on adjectives that assess prototypical features of each personality dimension (Openness, Conscientiousness, Extraversion, Agreeableness, and Neuroticism), scored using a 5-point Likert scale. The remainder of the traits assessed by the BFI were used to help establish criterion validity for the latent classes at Time 1. The Neuroticism subscale demonstrated good internal reliability (T1 α = .84).

Data Analytic Plan
To assess the underlying structure of PTSD symptoms, we estimated a series of LCA models on the basis of the dichotomously scored PCL–C items at T1, T2, and T3 using PROC LCA in SAS (Lanza et al., 2007; Lanza, Dziak, Huang, Xu, & Collins, 2011). Model fit was evaluated using the Bayesian Information Criterion (BIC; Schwarz, 1978) along with interpretability of classes. Next, measurement invariance was evaluated, meaning that the equality constraints were imposed on the LCA structure across time points. The fit of this solution was compared to a solution allowing each time point’s parameters to be freely estimated.

The probability of transitioning between latent PTSD classes was evaluated with PROC LTA in SAS (Lanza & Collins, 2008). First, we examined the stability of latent classes between T1 (September) and T2 (December) and then between T2 and T3 (April) without covariates. Second, we examined the effect of our covariates (i.e., trauma exposure, alcohol involvement, neuroticism, sex) on the probability of transitioning. This can be understood as regressing the transition probabilities on the covariates.

Results
Attrition
There was a very low rate of attrition in this sample across the first year of college. Because we included only those individuals who provided at least two waves of data, the full sample consisted of 944 students. An additional 40 participants who did not provide data on covariates were excluded in the LTA. Though PROC LTA uses full information maximum likelihood estimation and can handle missing data in the dependent variables, missing data on the covariates results in listwise deletion. Though attrition was minor, we still compared those retained from those excluded on a number of variables that might be related to attrition. These groups did not differ on sex, ethnicity, age, drinking status, baseline trauma, or total PCL–C symptom count at T1 (all ps > .05). Thus, attrition likely had little effect on the results.

Descriptive Statistics
With regard to classification as drinkers versus nondrinkers, at T1 597 (63.2%) participants reported having at least one drink in the month prior. Among T1 drinkers, participants drank an average of 4.63 drinks per occasion (SD = 2.35). Between T1 and T2, 665 (70.4%) individuals were classified as drinkers (Mdrinks = 3.84, SD = 2.45), and between T2 and T3, 605 (64%) individuals were classified as drinkers (Mdrinks = 4.39, SD = 2.48). In terms of negative alcohol-related consequences, at T1 508 (53.8%) participants reported having experienced one or more past-month consequences (M = 9.19, SD = 7.72). Between T1 and T2, 548 (58.1%) experienced at least one alcohol-related consequence (M = 4.97, SD = 6.37), and between T2 and T3, 492 (52.1%) experienced at least one alcohol-related consequence (M = 5.75 SD = 7.52). Given that the modal response was zero for both use and consequences, and the typical drinks per occasion (i.e., within “binge” range) and consequences endorsed among drinkers were substantial (e.g., nearly 68% of drinkers experienced five or more consequences at T1), our categories of use and consequences represent moderate to high levels of alcohol involvement. Rates of PTSD symptom endorsement (i.e., PCL–C items) at each assessment can be found in Supplementary Table 1 of the online supplemental material.

Latent Class Analysis
As a first step, an LCA model was estimated within each time point using the 17 PCL–C items as observed indicators. At T1, the BIC suggested a four-class solution, but at T2 and T3, a three-class solution was supported (see Table 1). Following close consideration of three- and four-class models, we ultimately retained a three-class solution based on interpretability, replicability across time points, and convergence with prior PTSD symptom-based LCA models in community samples (e.g., Breslau et al., 2005). The three classes captured groupings of increasing severity of PTSD symptoms, and were labeled as (1) severe symptoms, (2) moderate symptoms, and (3) no symptoms. We then tested mea-
surement invariance across assessment waves to establish that the classes have the same interpretation at each time point. Comparison between a freely estimated and a constrained model across time points suggested a considerably lower BIC for the constrained model (BIC difference = 438.39). This supports measurement invariance across time points. Table 2 presents the estimated parameters from the measurement invariant model, including item endorsement probabilities and class membership at each time point.

Comparison of Classes on External Variables

To better understand the class solution, we compared the classes at T1 on a range of demographic, trauma-related, PTSD, and personality criteria (see Supplementary Table 2 in the online supplemental material) using analysis of variance. Classifying individuals on the basis of their most likely class membership was supported by the entropy of the T1 LCA (entropy = .83), with values above .80 supporting this form of group comparison (Clark & Muthén, 2015). Classes did not significantly differ on participant age, Extraversion, or Openness. The classes did differ on sex, with fewer males in the moderate and severe symptoms classes. As can be seen in Supplementary Table 2, across-class differences in traits were observed. Agreeableness and Conscientiousness scores tended to be lower for the more-severe symptoms classes, and Neuroticism scores were greater in the more-symptomatic PTSD classes. These patterns in trait differences across symptom severity were generally consistent with prior meta-analytic results (Kotov, Gamez, Schmidt, & Watson, 2010) and further support the latent class distinctions.

Describing Symptom Transitions: Latent Transition Analysis Without Covariates

The proportion of individuals in each class (or status) at each time point can be found at the bottom of Table 2. The probabilities of transitioning between classes (latent transition probabilities) are presented in Table 3. The bold values on the diagonals in this table represent “stabilities,” or the probability of an individual remaining in that particular class between time points. It is notable that the transition probabilities are much different moving from T1 to T2 compared to moving from T2 to T3. Going from T1 to T2, there were dramatic shifts whereby the symptomatic statuses primarily moved into less-severe statuses, whereas the no symptoms class was highly stable, with only 9% moving into a symptomatic class. Going from T2 to T3, the stabilities were higher in the symptomatic classes. Yet, the no symptoms status was less stable; during this time, 15% of these individuals shifted into a symptomatic status. Similarly, 17% of the moderate symptoms class moved to the severe symptoms class from T2 to T3.

Supplementary Table 3 in the online supplemental material summarizes the 15 most common symptom trajectories. The most common of these trajectories on the basis of the LTA is one of stability and maintaining one’s symptom profile across waves. These rates are followed by rates of individuals decreasing in severity. Yet a sizable portion of individuals got symptomatically worse, transitioning into more-severe classes over time.

Predicting Symptom Transitions: Latent Transition Analysis With Covariates

We next conducted an LTA with covariates. This included our substantive covariates, which we believed might influence PTSD transitions (trauma exposure, alcohol use and consequences) and also our control variables (sex and neuroticism).

The conditional model focused on the effect of covariates on the transition into a given status at a subsequent time point. Thus, for T1, the latent statuses are regressed on the covariates in a multinomial logistic regression, whereas for T2 and T3 it is the transi-
tion probabilities that are regressed on the T1 covariates in the regression. The LTA with covariates are estimated within one model, thus correcting for measurement error.

Specifically, the following variables were included simultaneously as covariates: sex, T1 neuroticism, lifetime (in the case of T1/new/repeated Criterion A event endorsement, whether the individual drank in the past month (in the case of T1) or between assessment points, and whether the individual experienced any consequences associated with drinking. Results of this and all multinomial logistic regressions are presented in odds ratios (OR). An OR of 1 indicates no relationship, greater than 1 indicates a positive relationship, and a value less than 1 indicates a negative relationship. In these analyses, the no symptoms group was used as the reference class. Here, we have conservatively adopted a convention of 1.5, 2.5, and 4.3 (0.67, 0.40, and 0.23 for negative relationships) for small, medium, and large effects, respectively (Haddock, Rindskopf, & Shadish, 1998). On the basis of these general guidelines, we found that T1 symptom class was unrelated to sex (ORs of 0.98 and 1.01 for transition to the moderate [M] and severe [S] classes, respectively), whereas neuroticism had a small to medium effect size (ORs of 1.45 [M] and 2.21 [S]), trauma had a medium to large effect size (ORs of 3.79 [M] and 4.25 [S]), and being a drinker (OR of 1.22 [M] and 1.33 [S]) or having experienced consequences (OR of 1.16 [M] and 1.01 [S]) had very modest effect sizes.

Table 4 reports the ORs for covariate effects on PTSD transitions. When predicting transitions from T1 to T2, status at T1 always served as the reference class (i.e., movement out of one status into the other two). For these models, an OR greater than 1 indicates an increased likelihood of transitioning out of a status, whereas an OR less than 1 indicates a decreased likelihood of transitioning out, or an increased likelihood of maintaining status. We found that for the severe symptoms group, trauma predicted retaining severe status relative to transitioning in to the no symptoms class, but otherwise the covariates had little effect on movement from the severe class. For the moderate symptoms group, trauma again exerted the strongest effects, leading to a 2.21 OR of transitioning into the severe status relative to maintaining status and to some degree lessened the likelihood of transitioning into the no symptoms status. Alcohol consequences also had a very modest effect of predicting a lower likelihood of transitioning to the no symptoms class. Drinking modestly predicted movement from the moderate symptoms class, but this was equivalent in both directions. Surprisingly, in terms of moving out of the no symptoms class, those with trauma and who were drinkers were less likely to move into the severe symptoms group. Trauma and drinker status did however predict movement from the no symptoms into the moderate symptoms class. Last, alcohol consequences predicted moving from the no symptoms to the moderate and severe symptoms classes. Effect sizes were 1.45 (small) and 2.86 (medium), respectively.

From T2 to T3, experiencing a trauma made transition out of the severe symptoms class less likely. Accordingly, trauma is predictive of moving from the moderate to the severe symptoms status and negatively predictive of moving into the no symptoms status. Engaging in drinking between T2 and T3 was also predictive of moving into the severe symptoms class. Experiencing alcohol-related consequences had a very modest effect on moving out of the moderate symptoms class in either direction. Finally, neuroticism and trauma had small and medium effects as predictors of moving from the no symptoms status to both symptomatic statuses, as did consequences to some degree. However, being a drinker made it less likely that one transitioned from the no symptoms class to either symptomatic class.

**Discussion**

In this study, we applied latent class analysis to identify the latent structure of PTSD symptoms in college students. We then used latent transition analysis to understand the course of PTSD symptoms during this year of transition and to explicate factors that may influence this course. Our findings revealed significant variability in PTSD symptom course over the first college year. Exposure to trauma during this period contributed to this variability, exerting an influence on the maintenance and worsening of PTSD symptoms. Alcohol involvement also showed a deleterious influence on PTSD symptom status over time, though less consistently so. Importantly, these associations were observed above and beyond contributions by general negative affectivity, highlighting a process that is unique to posttraumatic stress. We elaborate on these findings next.

### Table 3

**Latent Transition Probability Estimates**

<table>
<thead>
<tr>
<th>Class</th>
<th>Severe symptoms</th>
<th>Moderate symptoms</th>
<th>No symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>December latent status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe symptoms (n = 187)</td>
<td>.37 (69)</td>
<td>.40 (75)</td>
<td>.23 (43)</td>
</tr>
<tr>
<td>Moderate symptoms (n = 388)</td>
<td>.08 (31)</td>
<td>.32 (124)</td>
<td>.61 (237)</td>
</tr>
<tr>
<td>No symptoms (n = 369)</td>
<td>.02 (7)</td>
<td>.07 (26)</td>
<td>.91 (336)</td>
</tr>
<tr>
<td>Estimated n</td>
<td>(107)</td>
<td>(225)</td>
<td>(616)</td>
</tr>
<tr>
<td>September latent status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe symptoms (n = 104)</td>
<td>.63 (66)</td>
<td>.18 (19)</td>
<td>.19 (20)</td>
</tr>
<tr>
<td>Moderate symptoms (n = 225)</td>
<td>.17 (38)</td>
<td>.54 (122)</td>
<td>.29 (65)</td>
</tr>
<tr>
<td>No symptoms (n = 615)</td>
<td>.03 (18)</td>
<td>.12 (74)</td>
<td>.85 (523)</td>
</tr>
<tr>
<td>Estimated n</td>
<td>(122)</td>
<td>(215)</td>
<td>(608)</td>
</tr>
</tbody>
</table>

*Note. N = 944. Data in parentheses are n values. Probability of maintaining latent status along the diagonal is bolded.*
Latent Class Analysis Findings

We identified three classes of PTSD symptoms that appear to reflect a linear pattern of severity. Associations with related constructs support these classifications. The identification of these severity-based symptom classes is consistent with a growing literature that highlights the utility of conceptualizing PTSD symptoms as existing along a spectrum of severity, rather than as a dichotomous classification (disorder/no disorder) based on diagnostic cutoffs (e.g., Broman-Fulks et al., 2006; Ruscio, Ruscio, & Ruscio, 2010). Severity-based classification also offers clinical utility. For example, it is well known that PTSD symptoms that fall short of conventional diagnostic criteria for a PTSD diagnosis can and do result in substantial psychological distress and functional impairment (Mendlowicz & Stein, 2000; Schnurr, Friedman, & Bernardy, 2002). Classifications such as those identified here may direct attention to such symptom presentations that are below diagnostic threshold but which may well be of functional and quality of life significance.

Latent Transition Analysis Findings

We observed significant variability in PTSD symptom change over the first year of college. Encouragingly, the most typical pattern was toward resolution. This is consistent with work showing evidence for a natural recovery process in those with PTSD (Blasco-Ros, Sanchez-Lorente, & Martinez, 2010; Krause, Kaltman, Goodman, & Dutton, 2008). Yet the more severe patterns of PTSD symptoms appeared to be those most firmly entrenched, showing less movement toward amelioration. Indeed, for a non-trivial portion of our sample, there was no resolution of PTSD symptoms at all. For others still, there was evidence of symptom worsening.

The patterns of stability and change that we observed in our latent transition analyses also point to particular periods of risk. In the first semester, between-time stabilities were lower than they were in the second semester, with general trends toward decreasing symptom severity. As the academic year progressed, the no-symptoms class became less stable, with a substantial portion shifting into a symptomatic status. We observed a similar pattern in shift from the moderate symptoms class to the severe symptoms class during this second half of the year. In addition, the symptomatic classes became more stable, with less likelihood of change.

There likely are both sampling and nonsampling reasons for the observed differences in stability patterns across the study. For example, we oversampled individuals who reported a prior trauma and PTSD symptoms at T1. In enriched samples there is often a dropoff in symptoms in the early part of a longitudinal study, which then stabilizes at successive time points (e.g., Nesselroade, Stigler, & Baltes, 1980). This may account for the early patterns of change that we observed. Alternatively, the first set of transitions may be capturing the acclimatization of the participants to college life. If this is the case, then it may be that, once acclimated, a particular subset of students is at risk for emerging into escalation of PTSD severity. This raises the question of what factors may place students at risk for continued or escalating PTSD symptoms, a question that we sought to address in our covariate analyses.

Covariate Effects

Trauma effects. There has been much discussion in the literature regarding the relevance of trauma exposure to ensuing PTSD symptoms (Brewin, Lanius, Novac, Schnyder, & Galea, 2009; Kilpatrick, Resnick, & Acierno, 2009) and, particularly relevant to this study, the role of new or recent traumas in contributing to the onset of PTSD symptoms, or the derailment of PTSD recovery (Blasco-Ros et al., 2010; Horesh et al., 2011). Findings from the present study contribute to the dialogue. We observed a clear and consistent impact of trauma exposure on PTSD class status and on transitions into and out of PTSD symptom classes, with a uniformly negative effect on PTSD symptom outcomes. Prior trauma exposure predicted baseline PTSD symptom class membership, as well as transitions from the no symptoms class to both symptom classes. Consistent with at least one prior study (Blasco-Ros et al., 2010), trauma also predicted either worsening or maintenance of existing symptoms. Importantly, here we show for the first time

Table 4
Odds Ratios Reflecting the Effect of Covariates on Transitions Between Waves

<table>
<thead>
<tr>
<th>Time and latent status</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latent status at Time 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Severe symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>—</td>
<td>0.98</td>
<td>0.97</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>—</td>
<td>0.92</td>
<td>0.96</td>
</tr>
<tr>
<td>Trauma</td>
<td>—</td>
<td>0.82</td>
<td>0.67</td>
</tr>
<tr>
<td>Drinker</td>
<td>—</td>
<td>0.97</td>
<td>1.20</td>
</tr>
<tr>
<td>Consequences</td>
<td>—</td>
<td>1.01</td>
<td>0.96</td>
</tr>
<tr>
<td>2. Moderate symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>0.79</td>
<td>—</td>
<td>0.80</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>1.11</td>
<td>—</td>
<td>1.23</td>
</tr>
<tr>
<td>Trauma</td>
<td>2.21</td>
<td>—</td>
<td>0.78</td>
</tr>
<tr>
<td>Drinker</td>
<td>1.28</td>
<td>—</td>
<td>1.34</td>
</tr>
<tr>
<td>Consequences</td>
<td>0.94</td>
<td>—</td>
<td>0.78</td>
</tr>
<tr>
<td>3. No symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>0.60</td>
<td>0.82</td>
<td>—</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>1.09</td>
<td>0.93</td>
<td>—</td>
</tr>
<tr>
<td>Trauma</td>
<td>0.65</td>
<td>1.45</td>
<td>—</td>
</tr>
<tr>
<td>Drinker</td>
<td>0.68</td>
<td>1.35</td>
<td>—</td>
</tr>
<tr>
<td>Consequences</td>
<td>2.86</td>
<td>1.45</td>
<td>—</td>
</tr>
</tbody>
</table>

Latent status at Time 3

<table>
<thead>
<tr>
<th>Time 2</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Severe symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>—</td>
<td>1.03</td>
<td>0.87</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>—</td>
<td>0.94</td>
<td>0.93</td>
</tr>
<tr>
<td>Trauma</td>
<td>—</td>
<td>0.66</td>
<td>0.73</td>
</tr>
<tr>
<td>Drinker</td>
<td>—</td>
<td>0.82</td>
<td>1.10</td>
</tr>
<tr>
<td>Consequences</td>
<td>—</td>
<td>1.05</td>
<td>0.81</td>
</tr>
<tr>
<td>2. Moderate symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>0.78</td>
<td>—</td>
<td>0.87</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>1.05</td>
<td>—</td>
<td>1.05</td>
</tr>
<tr>
<td>Trauma</td>
<td>1.40</td>
<td>—</td>
<td>0.71</td>
</tr>
<tr>
<td>Drinker</td>
<td>1.51</td>
<td>—</td>
<td>1.12</td>
</tr>
<tr>
<td>Consequences</td>
<td>0.75</td>
<td>—</td>
<td>0.77</td>
</tr>
<tr>
<td>3. No symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>0.61</td>
<td>1.20</td>
<td>—</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>1.45</td>
<td>1.40</td>
<td>—</td>
</tr>
<tr>
<td>Trauma</td>
<td>2.99</td>
<td>2.66</td>
<td>—</td>
</tr>
<tr>
<td>Drinker</td>
<td>0.83</td>
<td>0.76</td>
<td>—</td>
</tr>
<tr>
<td>Consequences</td>
<td>1.45</td>
<td>1.18</td>
<td>—</td>
</tr>
</tbody>
</table>

Note. N = 904. Dashes indicate the reference class.
that trauma may also derail the resolution of PTSD symptoms for those already experiencing them. Trauma decreased the likelihood of transitioning to no symptoms status among those with moderate symptoms. Together, these strong and consistent effects have important implications for intervention. Rates of trauma—particularly sexual trauma—are surprisingly high in college populations (Read et al., 2011; Testa, Hoffman, & Livingston, 2010), especially in the first college year (Fromme, Corbin, & Kruse, 2008). As such, identification and referral of students who have experienced trauma may help to ameliorate subsequent psychological distress.

**Alcohol effects.** At least some prior work has suggested that alcohol involvement can have a deleterious influence on both the presence and course of PTSD (Bisby et al., 2009). Our findings are consistent with this work and suggest that alcohol use and alcohol problems may contribute to worse PTSD outcomes. Drinking status and alcohol consequences both predicted baseline membership in the PTSD symptom classes and predicted the probability of transitioning from one PTSD symptom class to another. In many cases, both alcohol use and consequences predicted transitioning into more-severe PTSD symptom classes. Moreover, alcohol consequences were negatively associated with naturalistic evolution out of PTSD during the first semester.

The findings pertaining to the risk conferred by alcohol use and consequences on PTSD symptoms in the first college semester in particular are worth noting, because it is during this period that students are at greatest risk for heavy drinking and consequences (Sher & Rutledge, 2007; White et al., 2006). Accordingly, the ubiquity of heavy alcohol consumption and related consequences at the transition into college may represent risk not just for the hazardous alcohol outcomes themselves but also for the effects of these outcomes on other psychological distress—in this case, post-traumatic distress.

Yet the effects of alcohol involvement on PTSD transitions were not uniform. We saw this most clearly for those who started the first semester in the moderate symptoms class. Here, both alcohol use and alcohol consequences were associated with transitioning out of this class, with equal likelihood of transitioning to more- or less-severe symptoms status. We posit that as more-severe symptoms patterns tend to be more entrenched, the less-severe symptoms group may be less firmly established and more readily influenced both for better and for worse in the context of alcohol use and related consequences.

Somewhat surprising was our finding that being a drinker was associated with a decreased likelihood of transitioning from a no symptoms class to one of the symptomatic classes. The explanation for this seemingly protective effect of drinker status may lie in the unique nature of drinking in the college environment. That is, drinking in college tends to be celebratory and convivial (e.g., Bachrach, Merrill, Bytschkow, & Read, 2012) and thus is associated with some level of positive social functioning. Thus, it may be that being interpersonally tethered enough to engage in continued social contact serves as a buffer against worsening traumatic stress symptoms. Research that seeks to explicate the unique reasons why students are engaging in alcohol behavior will help shed further light on this association. A particularly fruitful avenue for further investigation may be in the area of drinking motives (Lindgren, Neighbors, Blayney, Mullins, & Kaysen, 2012; Merrill & Read, 2010). For those students motivated to drink for social, celebratory, or mood-enhancing purposes, drinking may be adaptive, associated with social engagement and connecting with others. In contrast, for those drinking to cope with trauma or other psychological distress, the pathway may be a darker one, leading to poorer mental health outcomes.

**Limitations and Future Directions**

This study had limitations that qualify our findings and highlight future research directions.

Our approach to modeling trauma in our analyses enabled us to capture trauma exposure that occurred between assessments. This afforded a fairly comprehensive evaluation of the effects of trauma. Yet, there are features of trauma exposure not modeled here. For example, we assessed whether trauma exposure had occurred since the last assessment point but not when it had occurred. Thus, the effects of the timing of the trauma are unknown. It also is possible that the effects of trauma and alcohol are not only additive but multiplicative. This was not examined here. We also did not examine the unique effects of different types of trauma. For example, some evidence has suggested that the course of PTSD may differ for those with interpersonal violence relative to other forms of trauma (Forbes et al., 2012). A determination of whether specific types of trauma (e.g., interpersonal trauma) or simply trauma exposure more broadly influence PTSD symptom change is an important next step in future research.

Some readers may also question whether the observed effects of the covariates on the latent transitions were truly different across the first and second semesters of the study. In other words, were the processes nonstationary? Though we tried, we were unable to definitively test this question, because PROC LTA does not allow for parameter constraints on the covariate effects. Other software such as Mplus has added this capability, and we tried to estimate our models using this software. However, the complexity of our models and the large number of covariates resulted in problems of estimation. In sum, whether there are in fact differences across semester, and if so what might cause these differences (e.g., maturation, changing social processes), remains an open question to be addressed as statistical software advances.

An objective of this study was to provide what is to our knowledge the first examination of how alcohol involvement may be linked to PTSD symptom changes over time. To do this, we considered the most basic level of differentiation—whether students had been using alcohol and whether they were experiencing consequences of this use. We did this partly for data analytic reasons and partly for conceptual clarity. From our data, it is clear that our dichotomized “drinker” and “consequence” distinctions were associated with a level of alcohol involvement that was not trivial and that in some cases was linked to shifts in PTSD symptoms. However, it remains unknown whether level or degree of involvement makes a difference in these shifts. This is an important question for future research.

Reviews of the literature have identified myriad factors that may moderate the risk for the development and presentation of PTSD symptoms (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003; Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012). Clearly, not all of these factors could be modeled in a single study. With this study, our focus was on two important predictors common on college campuses: trauma expo-
sure and alcohol involvement. Examination of the influence of additional psychosocial variables will continue to build on the present findings. Further, in future work, it will be interesting to extend this investigation to periods beyond the first college year or even as students transition out of college. Also, it makes sense to consider factors such as physical activity, diet, or sleep (Barry & Piazza, 2010; DeMartini & Fucito, 2014; Eisenberg & Fitz, 2014) that occur in the natural environment and that may be linked to symptom change. Of course, formal treatment may also influence the course of PTSD, and this variable was not modeled here.

Last, one of the strongest findings to emerge from our examination of transition patterns was movement toward health and recovery. In considering future directions for investigation, it will be important to consider not only what contributes to symptom worsening but also what factors—either personal or environmental—may increase the likelihood of resilience. As with risk factors, these resilience factors can be targeted in interventions.

Summary and Conclusions

Findings here suggest that though many college students resolve PTSD symptoms on their own over time, a significant portion do not. Moreover, both trauma exposure and alcohol involvement appear to confer risk for symptom worsening. This is an important finding when considered in the context of the first college year, which has been noted for both the high risk for trauma and the ubiquity of alcohol use and consequences. An increased clinical focus on PTSD and factors that may affect its course is warranted for this population.

References


This document is copyrighted by the American Psychological Association or one of its allied publishers. This article is intended solely for the personal use of the individual user and is not to be disseminated broadly.

Received January 27, 2015
Revision received September 9, 2015
Accepted October 2, 2015

PTSD TRANSITIONS IN COLLEGE

11

This document is copyrighted by the American Psychological Association or one of its allied publishers.
This article is intended solely for the personal use of the individual user and is not to be disseminated broadly.