Published in final edited form as:

J Abnorm Psychol. 2013 November ; 122(4): 984–997. doi:10.1037/a0034918.

Reciprocal Associations between PTSD Symptoms and Alcohol Involvement in College: A Three-Year Trait-State-Error Analysis

Jennifer P. Read, Jeffrey D. Wardell, and Craig R. Colder

Department of Psychology, University at Buffalo, State University of New York, Buffalo, NY 14222

Abstract

Bidirectional associations between posttraumatic stress disorder (PTSD) symptoms and alcohol involvement have been theorized, but have not been tested empirically. In this study, we examined these relations at the transition into and over the first three years of college by using an analytic approach (Trait-State-Error Modeling; TSE; Kenny & Zautra, 1995) that allowed us to examine prospective, reciprocal associations among these constructs while accounting for intra-individual stability. Young adults ($N=486$) were recruited at matriculation into college and assessed by web survey in September of the 1st college year (T1) and 11 additional time points over three years. Findings showed evidence of prospective associations from alcohol involvement (both use and problems) to PTSD symptoms over the three year assessment period. We also observed prospective relations from PTSD symptoms to alcohol involvement over time. Patterns of covariation in trait vulnerability for alcohol involvement and PTSD symptoms differed from crossed-lagged associations among state-like variance in these constructs. Results suggest that PTSD symptoms and alcohol involvement each predict the other over the course of college. Findings also highlight the importance of considering both time-varying and stable sources of variation in these associations.

Introduction

For more than 1/3 of individuals ages 18–24 in the U.S., the transition into adulthood co-occurs with transitions into, through, and out of college (U.S. Dept of Education, 2010). During this developmental period these young adults must learn to navigate new freedoms and responsibilities (Arnett, 2005; Bachman, Wadsworth, O'Malley & Johnston, 1997), tasks which may be especially challenging for those coping with trauma and its effects. Trauma and resulting post-traumatic stress (PTSD) symptoms are unfortunately common in young adults attending college (Smyth, Hockemeyer, Heron, Wonderlich, Pennebaker, 2008; Read, Ouimette, Colder, White & Farrow, 2011). Indeed, approximately 2/3 have experienced a traumatic life event, and as many as nine percent report symptoms consistent with a PTSD diagnosis.

Heavy drinking also is widespread on college campuses (Nelson et al., 2009), and is associated with significant negative consequences, both acutely and over the longer term. Evidence suggests that PTSD symptoms and drinking commonly co-occur, and may be mechanistically associated with one another. Yet their co-occurrence seldom has been

studied in college populations. An understanding of these associations may shed light on the nature and course of PTSD symptoms and heavy drinking as they influence one another during the college years.

PTSD-Alcohol: Direction of Association

Numerous theories of association between PTSD symptoms and alcohol involvement have been forwarded. Perhaps primary among these are self-medication models, which posit that alcohol use occurs, in part, as an effort to cope with stress (Khantzian, 2003). From this perspective, PTSD symptoms would be presumed to exert a causal influence on subsequent drinking behavior (Stewart, Pihl, Conrod, & Dongier, 1998) as the individual seeks to alleviate distress resulting from trauma. Indeed, coping motivated drinking has been linked uniquely to problem alcohol outcomes, above and beyond the influence of alcohol use (Carey & Correia, 1997; Cooper, Frone, Russell, & Mudar, 1995; Magid, McLean, & Colder, 2007; Merrill & Read, 2010; Read, Wood, Kahler, Maddock, & Palfai, 2003).

Other directions of association also have been suggested. For example, heavy alcohol involvement can increase the likelihood of exposure to trauma, and experiencing alcohol problems can compromise healthy adaptation to trauma, resulting in greater PTSD symptomatology (Back, Brady, Sonne & Verduin, 2006; Jacobsen, Southwick, & Kosten, 2001). This view, referred to as the Susceptibility Hypothesis, posits that problematic alcohol or other substance use may confer risk for susceptibility to the development or worsening of PTSD symptoms (see Brady, Back, & Coffey, 2004).

Another possibility is that associations between PTSD symptoms and drinking are bidirectional, influencing and being influenced by one another over time. Just as PTSD symptoms may lead an individual to drink to alleviate negative affect, alcohol use and associated consequences also may exacerbate PTSD symptoms, creating a self-perpetuating and reciprocal cycle. This process is consistent with what Bandura described in 1969 as *reciprocal determinism*, whereby two behavior patterns shape one another over time.

Empirical studies of such bi-directionality between alcohol involvement and PTSD symptoms have not, to our knowledge, been conducted in college or any other sample. Related work has examined bidirectional processes in negative affect more broadly as it relates to drinking behaviors, and has offered evidence for a reciprocal process in young adults. Kushner, Sher, & Erickson (1999) found evidence of reciprocal associations between alcohol use and anxiety disorders in their study which followed youths across seven years, beginning in the first year of college. Hussong, Hicks, Levy, and Curran (2001) also observed reciprocal associations between negative affect and alcohol use in their college sample, but found these effects to be specific only to certain types of negative affect (guilt, sadness). Whether reciprocal associations might be observed for distress symptoms specific to PTSD is unknown.

Trait and State Contributions to PTSD-Alcohol Co-Occurrence

Etiological theories that articulate either uni- or bi-directional effects in describing the PTSD-alcohol involvement relationship suggest that functioning in one domain will lead

proximally to changes in the other. Yet the literature on the prospective course of both PTSD symptoms and alcohol involvement offers evidence not just of symptom change, but also of symptom stability (see Bonanno & Mancini, 2012; Del Boca et al., 2004; Chen & Jacobsen, 2012; Greenbaum et al., 2005; Maggs & Schulenberg, 2004; Karlamangla et al., 2006). Prospective studies have not disaggregated stable trait and fluctuating state sources of variability when accounting for PTSD symptom-alcohol involvement associations. This is a significant gap in the literature, as both trait and state aspects of PTSD symptoms and drinking each can contribute uniquely to their co-occurrence.

For example, in terms of trait associations, persistence in PTSD symptoms could lead to vulnerability for problem alcohol involvement, as over time the effects of this distress may tax personal resources. Conversely, continued heavy and problem alcohol involvement will compromise an individual's ability to manage distress, thus perpetuating PTSD symptomatology. In some cases, trait associations could also be suggestive of a shared vulnerability that contributes to the maintenance of each of the two phenomena themselves. In contrast, state associations capture a different type of relation, as they highlight direct associations between symptom episodes that are time varying. For example, an individual's level of PTSD symptomatology and drinking varies across time, and according to self-medication theory, acute onset or exacerbation of PTSD symptoms may lead proximally to increases in drinking in order to cope in response to those symptoms. Conversely, Susceptibility models suggest that an episode of heavy or problematic alcohol use could lead to increased risk for trauma exposure and poorer adaptation in the wake of a traumatic event. Importantly, confounding trait and state sources of co-variation can lead to over- or under-estimation of each, obscuring the complex nature of these connections. Accordingly, modeling these associations over time requires an approach that will delineate trait versus state influences (Cole, 2005). This is especially important for examinations of reciprocal associations, as the examination of unique state associations (distinct from trait) can help to establish temporal associations and temporal precedence, consistent with theories of reciprocity.

The literature examining prospective associations between PTSD symptoms and alcohol outcomes has yielded complex and sometimes discrepant findings (e.g., McFarlane et al., 2009; Miller, Kaloupek, Dillon, & Keane 2006; Read, Brown, & Kahler, 2004; Stewart et al., 1998). No studies to our knowledge have delineated state and trait contributions to these associations. By separating out variance due to stability that is commonly seen in psychological constructs, more proximal associations among acute fluctuations can be observed, and the nature of association between these two constructs can be understood with greater clarity.

Distinguishing Trait and State associations: Trait-State-Error Models

Over the last two decades, models have been developed to capture both stable and changing elements of psychological functioning (e.g., Cole, Martin, & Steiger, 2005; Schmitt & Steyer, 1993; Kenny & Zautra, 2001). Among these are trait-state error models (TSE; Kenny & Zautra, 1995). When applied to prospective data with multiple assessment points, these models parse out variance attributable a general tendency (trait) to experience specific

symptoms or to engage in a particular behavior, as well as occasion-specific variation (state). These models are well suited to the study of reciprocal influences on behavioral constructs over time. This is because in prospective studies with multiple assessments, trait variance (i.e., stable behavioral dispositions) can obscure the ability to detect associations between more acute, state-level symptoms and behaviors (Herzog & Nesselroade, 1987).

Though not specifically modeling the effects of trauma and posttraumatic stress, prior work in adolescent and young adult samples has used trait-state modeling approaches to examine the association between more general psychological distress and alcohol use during important developmental periods (e.g., King et al., 2009; Jackson & Sher, 2003). Findings from this work have been somewhat mixed. For example, King et al. (2009) examined yearly assessments of both state and trait dimensions of family stress to predict latent alcohol trajectories during adolescence (ages 13–17). These authors found that state family stress was directly associated with alcohol use within each time point. These authors also observed modest evidence to suggest that family stress aggregated over time (trait) also contributed to escalations in alcohol use across the adolescent years. The effects of adolescent alcohol use on family stress were not examined, nor were reciprocal associations between these constructs.

Jackson and Sher (2003) tested trait and state associations between psychological distress (measured by the Brief Symptom Inventory) and alcohol involvement in a young adult sample using annual data from a panel study that spanned 11 years. As in the King et al. (2009) study, findings showed that alcohol problems and psychological distress were positively associated when aggregating data across time to represent trait associations. High trait alcohol problems were associated with the tendency to experience high trait levels of distress. Yet, Jackson and Sher found no significant cross-lagged state effects in these models. In this study, covariation between distress and alcohol involvement was due to trait associations and attributed to co-occurring trait vulnerabilities, rather than the direct influence of one construct on the other. Thus, though both studies pointed to trait vulnerability as an important link between psychological distress and alcohol use, evidence regarding direct, time-varying associations was more mixed. The authors of both studies identified long periods between assessment intervals as limitations of their work that may have hampered the detection of state associations. In the present study, we estimated TSE models using multiple assessments over briefer intervals than in prior studies of state-trait associations with alcohol use in order to provide a more fine-grained examination of associations between distress and drinking.

Finally, we recently found evidence to suggest that significant PTSD symptomatology at the beginning of college confers risk for alcohol use and problems over that first college year (Read et al., 2012). Yet PTSD symptoms were modeled only at one time point (matriculation into college), and thus, the current study extends this work with this sample, examining proximal associations between PTSD symptoms and drinking over time over a three year period.

Summary and the Present Study

College is a time of important developmental changes, and also a period of risk for heavy drinking, trauma exposure, and posttraumatic stress symptoms. The literature suggests that PTSD symptoms and alcohol involvement are mechanistically associated with one another, but the nature of these associations is unclear, and no studies have examined bidirectional influences between these two phenomena. Studies distinguishing between state and trait variance will shed light on the nature and direction of the associations between PTSD symptoms and young adult drinking. Though a few studies have used such approaches to examine the relationship between general distress and alcohol use, none have applied such a methodology to PTSD symptoms specifically.

Accordingly, the primary objective of this study was to test covariation between PTSD symptoms and alcohol outcomes over time in a sample of trauma-exposed college students. We sought to distinguish covariation in trait vulnerability for alcohol involvement and PTSD symptoms from crossed-lagged associations among state-like variance in PTSD symptoms and alcohol use and problems. To this end, we tested these associations over the first three years using a trait-state error (TSE) model. As noted by Hussong et al. (2001), these models are well suited to the study of self-medication and other directional models of alcohol involvement, as they allow for the delineation of temporal precedence as well as source of influence. This approach, then, is better suited to examining reciprocal associations among relatively stable constructs than other approaches that do not parse variance into trait and state components (e.g., conventional cross-lagged path models) or emphasize age-related changes or growth one or multiple constructs (e.g., growth modeling). Because alcohol use and problems are distinct constructs that show their own unique trajectories and associations with PTSD and other forms of negative affect (e.g., Cooper et al., 1995; Magid et al. 2007; Read et al., 2003; Read et al., in press), we examined alcohol use and alcohol problems separately to determine whether pathways differed according to type of alcohol involvement. High levels of PTSD symptoms were hypothesized to be associated with high levels of alcohol involvement (use, problems) at both the trait and state level. At the state level, bidirectional cross-lagged associations were expected, such that high levels of PTSD symptoms would be associated with greater subsequent alcohol involvement and vice versa.

Method

Participants and Procedure

Data for the present study are drawn from a larger, longitudinal study of PTSD symptoms and substance use in college. Detailed recruitment procedures for this larger study are described elsewhere (see Read et al., 2012; Read et al., 2011), but here we provide a brief overview (see also Figure 1).

Initial screening survey—Data collection took place at two mid-size public universities in the northeastern (Site 1) and southeastern (Site 2) United States. In the summer prior to beginning college, all incoming first-year students were invited by e-mail and U.S. postal mail to complete a screening survey either online or by paper-and-pencil. This screen was

used to recruit participants for the longitudinal study that is reported on here. Participants received a \$5 gift card for a local retailer upon survey completion. Comparable to other surveys with similar methodologies (e.g., Neighbors, Geisner, & Lee, 2008; Lewis et al., 2007), we achieved a 58% response rate ($N=3391$). Those who responded to the screening survey were relatively representative of the incoming student body, but over-represented women (58% vs. 47% averaged across universities) and Anglo-Caucasian students (72% vs. 62%, averaged across universities). After data cleaning and deletion of cases with missing data on our trauma screening (exposure, PTSD symptoms) variables, the screened sample consisted of 3,014 students.

Longitudinal sample selection—Students were selected for longitudinal follow-up based on trauma exposure and PTSD symptom data from the screening survey. Our goal was to recruit an enriched sample of students with sufficient representation of trauma exposure and PTSD symptoms. Accordingly, we invited for participation all participants who endorsed (1) at least one Criterion A (components A1 and A2, see below) trauma and (2) at least one symptom from each of the three PTSD symptom clusters (i.e., B, C, and D). Across the cohorts, a total of 649 participants met these criteria. Another 585 students who did not meet trauma criteria were selected randomly from the screened sample and invited for longitudinal follow-up. These screening procedures ensured adequate representation of students with trauma exposure and significant PTSD symptoms. No participants were excluded from participation based on their trauma or PTSD symptom history.

E-mails with a link to the Time 1 (Freshman Year September; T1) survey were sent to this selected sample ($N = 1,234$). Participants received a \$20 gift card for completion of the 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. Data from 4 participants were dropped because evidence strongly indicated haphazard responding to survey items. The response rate for participants meeting traumatic stress selection criteria ($n = 527$; 81.2%) was not significantly different than the response for participants who did not meet these criteria ($n = 470$; 80.3%), $\chi^2(1) = 0.70$, $p = .718$. Participants received a \$20 gift card for survey completion. No data were analyzed from this initial screen in the present study.

The present sample—As the objective of this study was to model associations between alcohol involvement and PTSD symptoms over time, we included in our analysis only those individuals who could logically show variability in these outcomes. Accordingly, the present sample included all students ($N=486$) who reported that they had experienced a lifetime traumatic event (PTSD Criterion A, see below) and reported drinking at least once in the past month prior to September of the first college year (T1; see Figure 1). Thus, the sample for the present study was comprised of 486 (73% female) participants. At T1, the average age was 18.11 ($SD=0.48$). Seventy-seven percent self-identified as Non-Hispanic Caucasian ($n = 375$), 8% as Asian ($n = 40$), 7% as Black ($n = 34$), 3% as Hispanic/Latino ($n = 15$), 4% as multi-racial ($n = 17$), and less than 1% ($n = 2$) as other. Three participants did not report ethnicity. Roughly equal numbers of participants reported living on campus ($n = 234$) and off campus ($n = 252$).

Measures

For the current analysis, we used assessments taken 4 times per academic year (September, December, February, and April) for three years (see Figure 1).[†] Thus, a total of 12 waves of data were analyzed. Trauma, traumatic stress symptoms, alcohol use and problems all were assessed at each time point. Surveys were opened and closed (i.e., web accessible to participants) during specific designated assessment periods, thus approximately equating the time between assessments for all participants.

Trauma Exposure—The DSM-IV-TR defines trauma as exposure to a traumatic event (A.1), accompanied by fear, helplessness, or horror (A.2). The Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000©)^{††} was used to assess trauma exposure consistent with this definition. The TLEQ is a 21-item self-report questionnaire that assesses a range of traumatic experiences consistent with the DSM-IV-TR definition, including the subjective responses that comprise Criterion A.2. This measure has demonstrated good psychometric properties, and has been used in a range of populations, including college students (Kubany et al., 2000).

Traumatic Stress Symptoms—Traumatic stress symptoms were assessed at each time point 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-TR, and has been used widely in both patient and non-patient populations, including college students. It has been shown to correspond strongly to gold-standard interview measures of PTSD (Andrykowski, Cordova, Studts, & Miller, 1998; Lang, Laffaye, Satz, Dresselhaus, & Stein, 2003), including the Clinician Administered PTSD Scale (CAPS; Blake et al., 1995). Participants reported according to a 5-point scale how much they have been bothered by each of the 17 PTSD diagnostic symptoms in the past month. A computer-based prompt reminded them of the specific Criterion A trauma(s) they had endorsed on the TLEQ, embedding these Criterion A traumas in the PCL instructions on each web page. Those who did not endorse Criterion A trauma exposure for a given time point completed the PCL with the same instructions, but with reference to how they responded to stressful events more generally in the past 30 days. Responses to the items were summed to create a total PTSD symptom variable reflecting both number and severity of symptoms in the past month. Internal reliability in this sample was very strong (Cronbach's alphas ranged from .92 to .96 across the 12 assessments).

Alcohol use—The alcohol use questions that we used in our quantity-frequency index have been used in multiple investigations of alcohol consumption in college students, and were first used by Sher et al. (1991). Typical weekly frequency and quantity of alcohol use were assessed for the past month at each time point. The frequency question read, "Think of all the times in the past month when you had something to drink. How often have you had some kind of beverage containing alcohol?" Participants responded on a 7-point scale

[†]Data were collected at two additional time points in the first semester (October, November). In order to keep the inter-assessment intervals consistent, these data from these two time points were not included in the analysis.

^{††}Copyright © 2004 by Western Psychological Services. WPS, Format adapted by J. Read, SUNY at Buffalo.

ranging from “never in the past month” to “every day.” The quantity question read, “In the past month, when you were drinking alcohol, how many drinks did you *usually* have on any ONE occasion?” Response options on an 11-point scale ranged from “did not drink in the past month” to “nine or more total drinks.” A standard drink conversion chart was presented to participants before they responded to the quantity item. The frequency and quantity variables were multiplied at each time point to provide a past-month quantity-frequency index. These types of questions show strong associations with interview (timeline follow-back) assessment of alcohol consumption (Grant et al., 1995; Sobell et al., 2003).

Alcohol problems—Problems from alcohol use were assessed with the 48-item Young Adult Alcohol Consequences Questionnaire (YAACQ; Read et al., 2006). This measure assesses a broad range of consequence types that range in severity. Eight domains of alcohol-related problems are assessed: social/interpersonal, academic/occupational, risky behavior, impaired control, poor self-care, diminished self-perception, blackout drinking, and physiological dependence. All domains load on a single higher-order consequence factor. This measure shows good internal and test-re-test reliability, and strong concurrent validity with other alcohol problems measures (Read et al. 2006; Read et al., 2007). Participants provided yes (coded 1) or no (coded 0) responses to items, indicating whether they had experienced that problem in the past month. A total alcohol problems score was created by summing responses to all of the items. Cronbach’s alpha for the YAACQ ranged from .93 to .96 across the 12 time points.

Data Analytic Plan

Data Cleaning, Missing Data, and Descriptive Analysis

We began by examining the means and univariate distributions of the observed PTSD symptoms, alcohol use, and alcohol problems variables. Some of our variables were moderately skewed (skewness range: 0.57 to 2.52) and kurtotic (kurtosis range: 0.18 to 8.08). To accommodate non-normal distributions, we used maximum likelihood estimation with robust estimation to derive scaled model chi-square estimates and corrected standard errors for parameter estimates.

Our longitudinal analysis included 12 repeated measures that were collected over the course of three academic years. Four surveys were administered each year, in September, December, February, and April. Thus assessments were spaced roughly equally (2 months) apart. However, the spacing between the last assessment of the academic year (April) and the first assessment of the next academic year (September) was much larger (about 5 months). All participants who completed the T1 survey continued to be eligible throughout the study and were not excluded for any reason, including dropping out of college. Thus, all missing data were due to participant nonresponse. The proportion of missing data values (total missing data values/total possible data values) was low (12%). Most of the missing data occurred because of occasional missed or incomplete assessments, rather than due to individual items being skipped. Moreover, response rates were high overall. The large majority of participants completed at least 10 of the 12 assessments (79%; $n = 384$). Given the low rate of attrition and low rate of missing values, missing data likely had a limited impact on our findings. Still, we used Full Information Maximum Likelihood Estimation

(FIML) to accommodate missing data, which allowed us to retain participants with missing data in the analyses. FIML assumes data are missing at random, an assumption largely supported by our data. Indeed, the number of missed assessments was not correlated with gender, nonwhite ethnicity, living situation (i.e., on or off campus), nor with baseline PTSD symptoms, alcohol use or alcohol problems (all p s > .05). However, number of missed assessments was correlated with older age ($r = .09, p = .039$).

Analysis of Reciprocal Associations

Our goal was to examine prospective state associations among PTSD symptoms and alcohol outcomes. As noted, given that there also tends to be a great deal of stability in these constructs that could mask prospective state associations, we wanted to apply an analytic technique that allows for the disaggregation of stable, trait variation from state variation. To do this, we used a latent Trait-State-Error modeling approach (TSE; Kenny & Zautra, 1995, 2001), which employs latent variable modeling to parse variance in repeated-measures into 3 components: a stable trait component, a variable state component, and an error component. A bivariate TSE model includes state and trait latent variables for two constructs (here PTSD symptoms and alcohol use/problems) and allows one to test cross-lagged associations among the latent *state* components. Accordingly, these models provide a test of prospective reciprocal associations after accounting for trait-like stability (Kenny & Zautra, 1995).

Consistent with many other studies of college drinkers (Goldstein, Flett, & Wekerle et al., 2010; Martens, Martin, Littlefield, Murphy, & Cimini, 2011; Quinn & Fromme, 2011), within time point correlations between alcohol use and alcohol problems ranged from .50–.70 in this sample, suggesting that, though related to one another, use and problems are distinct constructs. Given this, and in light of a large literature showing that alcohol use and problems demonstrate unique associations with negative affect and related constructs (e.g., Goldstein et al., 2010; Fossos, Kaysen, Neighbors, Lindgren, & Hove, 2011; Merrill & Read, 2010; Read et al., 2003; see also review by Kuntsche, Knibbe, Gmel, & Engels, 2005), we modeled these two indices of alcohol involvement separately. To do so, we specified two separate TSE models using Mplus 6.1 (Muthen & Muthen, 2010); one included PTSD symptoms and alcohol use (quantity-frequency index), and the other included PTSD symptoms and alcohol-related problems. First, we estimated a basic TSE model that did not include cross-lagged paths. This model included the 12 repeated measures of PTSD symptoms and alcohol use/problems as observed variables. We specified these models following the recommendations of Kenny and Zautra (1995). Each observed variable was regressed on two latent variables, including a latent trait and a latent state variable with these factor loadings set to 1.0. Stability coefficients (i.e., autoregressive paths from one variable to the same variable at the next time point) were estimated among latent state variables, and constrained to be equal to one another within each construct.^{†††} Given the unequal spacing of time points within (2–3 months apart) versus across each academic year (5 months apart), and also the fact that these time periods represented a different socio-

^{†††}Equality constraints on path coefficients and factor loadings are required for model identification in TSE models, and are consistent with the model specification guidelines of Kenny and Zautra (1995).

environmental period, separate constraints were placed on within and across academic year stability coefficients.

Error variances (i.e., residual variances for observed variables) and disturbance variances (i.e., residual variances for the endogenous latent variables) were constrained to be equal to one another within each construct. However, in order to accommodate the differences in the stability coefficients spanning across academic year, the disturbance variances for the post-summer state variables (i.e., September years 2 and 3) were estimated to be equal to one another within construct but different from the disturbance variances at the other time points. The covariance among the trait variables was freely estimated. Within-time error covariances between the observed PTSD symptom and alcohol variables were estimated and constrained to be equal across time. The same was done for the within-time disturbance covariances for the latent state variables, except that the September disturbance covariances in years 2 and 3 were estimated to be equal to one another but different from the other time points. The covariance between T1 state variances was estimated but constrained to be a non-linear function of the subsequent state disturbance covariances, stability coefficients, and cross-lagged paths according to recommendations by Kenny and Zautra (1995). This was done to ensure identification of the model. Also in accordance with these recommendations, nonlinear constraints were imposed on the latent state variances at T1 to be a function of the state disturbances and stability coefficients (Kenny & Zautra, 1995). Model fit was considered good if the root-mean square error of approximation (RMSEA) < .06, the comparative fit index (CFI) > .95, and the Tucker-Lewis index (TLI) > .95 (Hu & Bentler, 1999).

The next step in our model estimation was to test whether adding cross-lagged paths improved model fit. Nested model tests were conducted using a program by Crawford and Henry (2003) that calculates scaled chi-square difference tests (see Satorra & Bentler, 2001) to accommodate the chi-square statistic computed from robust maximum likelihood estimation. Because of the longer time interval across academic years and the shift in intervening socio-environmental context (leaving college for the summer) we did not expect cross-lagged associations between state PTSD symptoms and state alcohol involvement across academic years. We did, however, expect to observe meaningful cross-lagged paths within academic year because the closer proximity of the assessments within the same context would make it more likely for state variance in one construct to be associated with subsequent state variance in the other construct. Thus, we tested the addition of the within-year cross-lagged paths and across-year cross-lagged paths in separate nested tests. Cross-lagged paths from PTSD symptoms to alcohol involvement were constrained to be equal to one another over time, as were the paths from alcohol involvement to PTSD symptoms. Based on the results of these nested model tests, the relevant set of cross-lagged paths were retained in our final model.

Results

Descriptive Analysis

The types of traumas reported by students in this sample were diverse, and included sudden/unexpected death of a loved one (n=313, 64%), life-threatening illness or injury to self or

loved one (n=216, 44%), family violence (e.g., child abuse, witnessing partner violence; n=133, 27%), sexual assault (n=104, 21%), unwanted sexual attention (n=87; 18%), and other forms of interpersonal aggression (n=168, 35%). Lifetime exposure was assessed at baseline (T1), and thus these traumas occurred prior to matriculation into college.

Table 1 shows the means, standard deviations, and number of observations for the observed PTSD symptoms, alcohol use, and alcohol problems variables across the 12 assessment points. Mean PTSD symptoms were highest at the first time point, and there was a moderate decline in average symptoms between the first and second time points. Following this, mean PTSD symptoms were fairly steady over the course of the study, showing only a slight decline. This pattern would be expected given that in such enriched samples, there can be decreases in symptoms in the early part of a longitudinal study, which later stabilize (e.g., Nesselrode, Stigler, & Baltes, 1980).

Across the 11 follow-up assessments, the average number of new traumas reported at any given time point ranged from 0.13 (SD=0.46) at T12 to 0.35 (SD=0.75) at T5. The overall average number of new criterion A traumas reported between T2 and T12 was 2.00 (SD = 3.22). Forty percent (n=193) of the sample did not report any additional criterion A traumas after the T1 survey, and 88% (n=430) reported that they experienced 5 or fewer new traumas between T2 and T12.

Consistent with the literature showing the first months of college to be a time of particular risk increases in alcohol use and problems (e.g., Del Boca et al., 2004; Schulenberg & Maggs, 2002; White, Labouvie, & Papadaratsakis, 2005), both mean alcohol use and mean alcohol problems showed a general decline over the three years of college. This decline was steeper for problems. However, these variables had large standard deviations suggesting a great deal of individual variability in alcohol use and problems. Alcohol indicators were re-scaled (divided by a constant) to make their variances comparable to PTSD indicators to foster convergence in Mplus.

Reciprocal Associations: State-Trait-Error Models

PTSD symptoms and alcohol use—The initial Trait-State-Error model without any cross-lagged paths fit the data well, scaled $\chi^2(286) = 404.76$, $p < .001$, CFI = .98, TLI = .98, RMSEA = .03. Adding the within-year cross-lagged paths led to a significant improvement in model fit, scaled $\chi^2(2) = 14.25$, $p < .001$. However, adding the cross-lagged paths that spanned across academic years did not significantly improve model fit, scaled $\chi^2(2) = 1.47$, $p = .480$. Moreover, when these paths were added to the model that included the within-year cross-lagged paths, the across year cross-lags were not statistically significant, as expected (for alcohol use to PTSD symptoms, $B = .04$, $SE = .08$, $p = .639$; for PTSD symptoms to alcohol use, $B = .001$, $SE = .09$, $p = .988$). Thus, we retained only the within-year cross-lagged paths for our final model. This final model fit the data well, scaled $\chi^2(284) = 395.43$, $p < .001$, CFI = .98, TLI = .98, RMSEA = .03.

Figure 2 shows estimates from the final model that included alcohol use. As shown, there was a significant, negative relationship between the PTSD symptom trait variable and the alcohol use trait variable. High levels of stable trait PTSD symptoms were associated with

low stable trait levels of alcohol use. The cross-lagged associations among state variables suggest that high levels of alcohol use prospectively predicted subsequent high levels of PTSD symptoms, and that high levels of PTSD symptoms prospectively predicted high levels of subsequent alcohol use within each academic year. These findings provide evidence for a reciprocal relationship between state fluctuations in PTSD symptoms and alcohol use over the course of 3 academic years. The magnitude of the standardized path coefficients suggests that these were small unique effects above and beyond trait variability and stabilities.

Although not shown in the figure, the within time-point residual covariance between the observed alcohol use and PTSD symptoms variables was significant (covariance = .001, $SE = .001$, $p = .015$, $r = .10$). Also, the within-time point disturbance covariance for the September state factors was significant and negative (covariance = $-.001$, $SE = .001$, $p = .043$, $r = -.27$), whereas the disturbance covariance for the other time points was not significant (covariance = .000, $SE = .000$, $p = .967$, $r = .01$).

In this model, 45% of the total variance in the observed alcohol use variables was accounted for by the trait component, 32% was accounted for by the state component, and 23% was accounted for by the error component. For the observed PTSD variables, 44% of the total variance was accounted for by the trait component, 17% by the state component, and 39% by the error component. Thus, more variance was accounted for by the trait portion of the model for both alcohol use and PTSD symptoms.

PTSD symptoms and alcohol-related problems—The initial model without any cross-lagged paths fit the data well, scaled $\chi^2(286) = 406.40$, $p < .001$, CFI = .97, TLI = .97, RMSEA = .03. Consistent with the alcohol use model, the addition of the within-year cross-lagged paths significantly improved model fit, scaled $\chi^2(2) = 29.74$, $p < .001$, whereas the addition of the across-year cross-lagged paths did not, scaled $\chi^2(2) = 3.49$, $p = .175$. Inclusion of the across year cross-lagged paths (in addition to the within-year cross-lags) suggested that neither across-year paths were statistically significant, as expected (for alcohol consequences to PTSD symptoms, $B = .02$, $SE = .01$, $p = .148$; for PTSD symptoms to alcohol consequences, $B = -.42$, $SE = .30$, $p = .156$). Accordingly, only the within-year cross-lagged paths were retained in the final model. This final model fit the data well, scaled $\chi^2(284) = 399.03$, $p < .001$, CFI = .97, TLI = .97, RMSEA = .03.

Figure 3 shows the final model with the within-year cross-lagged associations. There was no significant association between the trait PTSD symptoms variable and the trait alcohol problems variable. Consistent with the alcohol use model, high levels of alcohol problems prospectively predicted high levels of PTSD symptoms within academic year, although the standardized coefficients suggest that this was a small unique effect above and beyond trait influence and stabilities. The path coefficient from PTSD symptoms to subsequent alcohol problems was not statistically significant. These findings suggest a unidirectional relationship between state fluctuations in alcohol problems and subsequent PTSD symptoms over the course of 3 academic years.

Although not shown in the figure, the within time-point residual covariance between the observed alcohol problems and PTSD symptoms variables was significant (covariance = .004, $SE = .001$, $p = .001$, $r = .10$). Also, the within-time point disturbance covariance for the September state factors was not significant (covariance = $-.001$, $SE = .002$, $p = .778$, $r = -.04$), whereas the disturbance covariance for the other time points was significant (covariance = .002, $SE = .001$, $p = .024$, $r = .35$). In this model, 16% of the total variance in the observed alcohol problems variables was accounted for by the trait component, 56% was accounted for by the state component, and 28% was accounted for by the error component. For the observed PTSD variables, 37% of the total variance was accounted for by the trait component, 22% by the state component, and 40% by error the error component.

Tests of model assumptions and sensitivity analysis—The typical strategy for testing model constraints in structural equation modeling is to start with a fully unconstrained model and then to evaluate constraints on the model in a step-wise fashion using nested tests. In a TSE framework, the unconstrained model is not identified and thus, is not estimable. Given this, we used an alternative, two-stepped strategy to examining the tenability of our model assumptions. First, we tested the equivalence of the trait factor loadings according to the recommendations of Kenny & Zautra (1995). This involved comparing our final model to a model that fixed one factor loading to 1.0, freed the remaining factor loadings, and fixed the proportion of variance in the trait and state components of the model to equal the estimated proportions (Kenny & Zautra, 1995, p. 57). We did this in our bivariate models, but tested the factor loadings for each construct in separate nested tests. Doing this in the model with PTSD symptoms and alcohol problems led to the finding that model fit was not significantly improved by freeing the loadings for the PTSD symptom trait factor, scaled $\chi^2(11) = 11.24$, $p = .423$, nor by freeing the loadings for alcohol consequences trait factor, scaled $\chi^2(11) = 14.76$, $p = .194$. Thus, the data support the equivalence of the trait factor loadings. For the alcohol use model, the data supported the equivalence of the trait factor loadings for the PTSD trait factor (scaled $\chi^2(11) = 17.81$, $p = .086$), but not the alcohol use trait factor (scaled $\chi^2(11) = 32.29$, $p < .001$).

To examine this issue further, we examined modification indices for the alcohol use trait factor loadings in our final model that constrained these loadings. This allowed us to identify specific factor loadings that did not conform to the assumption of the TSE model. We considered modification indices > 6.0 for this evaluation (i.e., p -value approximately .01), given the data driven nature of this approach. The modification indices suggested only one factor loading did not conform to the equality constraint imposed by the TSE model – the constrained alcohol use trait loading corresponding to the T5 observed measure produced a modification index of 9.45 ($p < .01$). In summary, it appears that the trait factor equality constraints were not violated for the alcohol problems model, and that this assumption was reasonable for all but one loading for each trait factor for the alcohol use model.

To test the assumptions of the state portion of the model, we took a “top-down” approach that involved starting with the TSE model, then examining modification indices to test the tenability of each parameter constraint given our data (disturbance and error variances, stabilities, cross-lags, covariances). Again we used a modification index of 6 as a cut off.

Examination of the modification indices for both of our final models (alcohol use and alcohol problems) revealed that only a small proportion of the parameters did not appear to conform to the model constraints. Specifically, of the 111 parameters that were constrained in each of our TSE models, only 14 in the alcohol use model and 14 in the alcohol consequence model had modification indices greater than 6.

Having identified specific parameters that did not conform to the equality assumptions in both the state and trait portions of the model, we proceeded to examine the impact of relaxing these specific constraints on our results. We re-ran our final models allowing the indicated parameters to be freely estimated. Importantly, inspection of the modified models revealed that relaxing these constraints did not have an impact on the substantive findings of the model. That is, in the alcohol use model, the coefficients for the cross-lagged cross paths remained statistically significant ($p < .015$), supporting the conclusion that alcohol use and PTSD symptoms are reciprocally associated at the state level. The coefficients for the two cross-lagged paths that were freely estimated were not statistically significant ($p > .192$). Similarly, in the alcohol problems model, the coefficient for the cross-lagged paths from state alcohol problems to state PTSD symptoms remained statistically significant ($p = .008$) and there was still no significant path from state PTSD symptoms to state alcohol problems ($p = .107$). The freely estimated cross-lagged path from T2 PTSD symptoms to T3 alcohol problems also was not significant ($p = .203$). Furthermore, the direction and significance of the trait covariance estimates in both models were consistent with the original final models. The only difference was that the covariance between the T1 alcohol problems and PTSD state factors was statistically significant in the modified model ($p = .010$), whereas it was nonsignificant in the original model ($p = .136$). This difference does not impact our substantive interpretation of the results. These results provide additional supporting evidence that our TSE models offer a reasonable representation of the data, and that our conclusions are robust to the modest violations.

Discussion

No studies to our knowledge have modeled bidirectional associations between PTSD symptoms and alcohol involvement. In this study, we examined these relations over the first three years of college. We used multiple and relatively frequent assessments, which allowed for the unfolding of bi-directional processes over time, as well as an analytic approach that allowed for delineation of variance due to intra-individual stability in psychological outcomes, and that due to episodic state co-variation in these constructs. As alcohol use and alcohol problems have shown differential associations with other psychosocial constructs, particularly those pertaining to negative affect, we examined PTSD symptom associations with both of these alcohol outcome variables. In our models, we found evidence of prospective associations between alcohol involvement and PTSD symptoms over the three-year assessment period. Importantly, associations varied according to whether they were examined at the trait or the state level. We discuss these findings in detail below.

State Associations

At the state level, we found consistent support for a proximal, direct pathway from alcohol involvement to PTSD symptoms, above and beyond the influence of trait associations. Increased PTSD symptoms were associated proximally with acute increases in subsequent alcohol use. In our alcohol use models, this association was part of a reciprocal relationship between PTSD symptoms and alcohol use, which predicted one another other across the three-year period. These findings are consistent with the concept of reciprocal determinism (Bandura, 1986), and with clinical observations that suggest that PTSD symptoms and substance use are functionally related over time, each helping to maintain the other (Brown, Stout, & Gannon-Rowley, 1998; Morrison et al., in press).

Yet in our alcohol problems models, it was only the unidirectional pathway that was significant; state variability in alcohol problems at each time point was associated with higher levels of PTSD symptoms at the next time point. The robust prediction of PTSD symptoms by both alcohol use and problems is consistent with a “susceptibility” conceptualization of PTSD-alcohol relations which posits that, for both biological and psychosocial reasons, chronic alcohol involvement can contribute to worsening of anxiety symptoms (see Kushner et al., 2000 for a review), and to poor adaptation following trauma exposure (Brady et al., 2004).

A large literature has underscored the importance of differentiating between alcohol use and alcohol problems in college samples (e.g., Cooper et al., 1995; Magid et al. 2007; Read et al., 2003), as these two constructs show differential etiologies, trajectories, and associations with other constructs. This study further highlights the necessity of such differentiation, as we observed different patterns of both state and trait associations depending on whether alcohol use or problems were being modeled.

Our findings have implications for self-medication theory, which suggests that PTSD symptoms drive individuals to drink problematically (Stewart, 1996). Though this theory has dominated the PTSD-alcohol literature, as noted, the evidence for a self-medication pathway has been limited when tested in adolescent and college samples (e.g., Colder & O'Connor, 2005; Colder et al., 2010; Ham et al., 2009; King, Molina, & Chassin, 2009; Read et al., 2012; Read & O'Connor, 2006).

Findings that state-level PTSD predicted later state-level alcohol use suggest that a self-medication process may be occurring in the short-term, and that during periods of distress, increases in drinking and associated problems may follow. Yet state level PTSD symptoms did not prospectively predict alcohol problems. Negative reinforcement drinking (i.e., drinking to alleviate negative affect) typically leads to problem drinking over time, as alcohol involvement progresses (e.g., Breese, Overstreet, & Knapp, 2005; Stasiewicz & Maisto, 1993). This shift toward a negative reinforcement process is thought to characterize a pathological progression toward addiction. One explanation for the differential associations that we observed between PTSD symptoms and alcohol involvement indices is that in this emerging adult sample, problem use may not yet have progressed to a level that reflects such a negative reinforcement process. Specifically, increasing levels of alcohol use may reflect a more temporally proximal response to PTSD symptoms, whereas the influence

on problems resulting from drinking may take longer to reveal itself. This interpretation is consistent with recent data using daily diary methodology that establishes a proximal temporal link between PTSD symptoms and desire to drink (Simpson et al., 2012). Also relevant are data from Murphy et al. (Murphy et al., 2013) showing college students with PTSD symptoms place greater value on alcohol, perhaps suggesting a stronger motivation to drink in these individuals. Replication and further investigation is needed to delineate more clearly the nature of self-medication pathways in college students.

Trait Associations

In contrast to the positive associations between PTSD symptoms and alcohol use observed at the state level, trait PTSD symptoms were negatively associated with trait alcohol use over the 3-year assessment period. We interpret this finding in light of the environmental context in which much drinking in college occurs. Social withdrawal and impaired social functioning are widely observed among individuals with trauma and posttraumatic stress (Frueh et al., 2001; Shea et al., 2010; Trickey et al., 2012). Over time, these continuing symptoms may limit the extent to which college students engage in social activity and, inadvertently, may limit their exposure to drinking occasions. As such, the tendency to continue to experience PTSD symptoms is linked to the tendency to avoid engaging in social activities which may involve alcohol.

Variance accounted for by trait and by state varied across PTSD and alcohol. This and the fact that we observed different associations at the trait and state level point to the advantage of the TSE models and parsing out these two sources of covariation. Further, doing so provided clarity on the nature of the associations between PTSD symptoms and alcohol involvement. First, at the aggregate trait level PTSD symptoms were negatively associated with alcohol involvement, but this does not preclude potential positive occasion specific prospective associations. Indeed, our findings suggest that state or occasion specific events may intensify either PTSD symptoms or alcohol involvement, which in turn, increases levels of the other. These occasion specific associations provide evidence supporting both self-medication (alcohol use) and susceptibility (alcohol problems) hypotheses. In short, different levels of analysis suggest different mechanisms underlying the link between PTSD symptoms and alcohol involvement.

Limitations and Future Directions

This study had a number of strengths, including a large sample from two universities in two regions of the U.S., a prospective design with strong retention, twelve assessments over a three-year period, and an analytic approach that enabled us to examine both the directional nature of PTSD-alcohol associations, and how associations may differ across use and problems. This study also had limitations, which are reviewed below.

It has been noted that trait-level associations may be an indicator of a shared risk factor, such as a genetic vulnerability, personality, or contextual (family, social environment) factors (Jackson & Sher, 2003; King et al., 2008). As there have been no studies that have examined bidirectional associations between PTSD symptoms and alcohol involvement, this was the primary goal of the present study. To do this, we used a design that would enable us

to parse out the contribution of trait variance to the associations of interest. The trait associations and mechanisms which might underlie such an association were not our focus. State-level mediators (e.g., drinking motives, alcohol expectancies) could also serve as intermediary variables in the PTSD-alcohol association. Studies that have examined mediators have found these to account only partially for co-variation in distress and alcohol involvement (e.g., Jackson & Sher, 2003). As such, it does not appear to be the case that indirect, shared risk pathways fully account for associations between distress and drinking. Still, an interesting direction for future research will be tests of shared pathways at both state and trait-level levels.

Another potentially interesting direction for future research lies in the examination of unique PTSD symptom cluster effects (i.e., re-experiencing, avoidance/numbing, hyper-arousal). Numerous studies have examined whether specific PTSD symptom clusters may be associated uniquely with substance outcomes, and have yielded disparate findings (e.g., Jakupcak et al., 2010; McDevitt Murphy et al, 2010; Read et al, 2004; Shipherd et al., 2005; Taft et al., 2007). Examination of associations between specific symptom clusters and drinking outcomes at both state and trait levels may help to clarify discrepancies in the extant literature.

Our findings provide an empirical test of two theorized models of association (self-medication, susceptibility), models that seldom have been tested prospectively in the literature. Yet, it is important to bear in mind that the effect sizes that we observed for the cross-lagged paths, though statistically significant and of potential theoretical relevance, nonetheless were small, and conclusions regarding clinical implications of these findings should be approached with caution.

In this study we sought to address a limitation of previous research by conducting multiple assessments per year, occurring approximately every three months. More – or different - information may come to light with assessments even closer together. Recent years have seen a profusion of assessment approaches that use technology such as the internet or smart phones to assess weekly or even daily symptom and behavior changes. Such approaches would be well suited for micro-examinations of PTSD symptoms and alcohol covariation. Still, such studies cannot reasonably examine longer periods of assessment (i.e., years) and thus would not be able to determine long-term reciprocal processes.

To test the robustness of our models, we used two-stepped strategy which included an examination of the equivalence of the trait factor loadings according to the recommendations of Kenny & Zautra (1995), followed by sensitivity analyses guided by modification indices. Though these analyses suggested that are findings were robust to modest violations of LTSE models, these tests were partially based on post-hoc model modification. Accordingly, some caution is warranted in considering the sensitivity analysis.

In this study, though we modeled PTSD symptoms following new and repeated traumas over the three years, we did not explicitly model the effects of new traumas or repeated victimization. Data show that it is post-traumatic distress, rather than trauma exposure *per se*, that predicts deleterious outcomes, including substance misuse (McDevitt-Murphy et al.,

2010; Saunders, Kilpatrick, & Resnick, 1998; Lepore, 1997; Stewart & Conrod, 2003). As such, we contend that in modeling changes in PTSD symptoms over time, we captured what likely is most essential to understanding the connectedness of the two phenomena.

Though data show that college students report rates of trauma and PTSD symptoms comparable to the general community (see Read, Ouimette, White, Colder, & Farrow, 2011; Smyth et al., 2008), it is important to bear in mind that associations observed here may not necessarily reflect PTSD-alcohol associations for other groups of young adults, or for individuals at other developmental stages. Replication of this work with younger adolescents, older adults, and populations such as veterans or those with alcohol dependence is needed in order to understand how and for whom PTSD symptom-alcohol associations unfold across the lifespan.

Another limitation of the TSE model is that latent factors are formed based on a single observed measure over time. A result of decomposing variance of repeated observed measures is that the latent trait factors also include shared method variance, which can result in biased path coefficients (Cole et al., 2005). An alternative to TSE models that can address such bias are Trait State Occasion models (TSO; see Cole et al., 2005). TSO models require multiple measures of each construct to specify latent variables at each assessment, and variance in the latent variable (rather than an observed variable as in the TSE model) is partitioned into trait and state components. We did not have multiple measures of our constructs, precluding estimation of TSO models, but multi-method studies of PTSD and alcohol involvement could benefit from implementations of these models in future research.

Finally, there are limitations to the types of questions that can be answered with TSE models. For example, though this approach allowed us to examine unique components (trait, state) of relations between PTSD symptoms and drinking outcomes over time, it is not well suited to examine age-related developmental processes. TSE models are predicated on the notion that what is most relevant to what is happening at a given point in time is largely a function of prior events (auto-regressive effects). Although models of reciprocal associations between alcohol and PTSD symptomatology are not developmental (e.g., Stewart & Conrod, 2003), it is possible that age or time might have an impact on these reciprocal associations. This would be an exciting direction for future research, and in this regard, growth modeling and its variants may be particularly useful analytic tools (e.g., Auto-Regressive Latent Trajectory Models, Bollen & Curran, 2004; Latent Difference Score Models, McArdle, 2009).

Summary and Conclusions

Heavy drinking is common on college campuses. It long has been known that such drinking is related to risk for trauma, including sexual assault, interpersonal violence, and serious injury. With this study, we show that alcohol use and associated problems also are linked prospectively and proximally to exacerbation in PTSD symptoms over time, and that PTSD symptoms show a prospective and proximal association with alcohol consumption. As such, both PTSD and heavy drinking are risk factors for one another, each with implications for the other over the course of college.

Acknowledgments

This work was supported by a grant from the National Institute on Drug Abuse (*R01DA018993*) to Dr. Jennifer P. Read.

We would like to thank Drs. Jennifer E. Merrill, Sherry Farrow, Ashlyn Swartout, Jackie White and the members of the UB Alcohol Research Lab for their many efforts to support data collection for this study, and Dr. Patrick Curran for his consultation on data analytic issues. We also would like to thank the participants for sharing their experiences.

References

- Andrykowski MA, Cordova MJ, Studts JL, Miller TW. Posttraumatic stress disorder after treatment for breast cancer: Prevalence of diagnosis and use of the PTSD Checklist–Civilian Version (PCL-C) as a screening instrument. *Journal of Consulting and Clinical Psychology*. 1998; 66:586–590. [PubMed: 9642900]
- Arnett JJ. The developmental context of substance use in emerging adulthood. *Journal of Drug Issues*. 2005; 35(2):235–254.
- Bachman, JG.; Wadsworth, KN.; O'Malley, PM.; Johnston, LD. Smoking, drinking, and drug use in young adulthood: The impacts of new freedoms and new responsibilities. Lawrence Erlbaum Associates, Inc; 1997.
- Back SE, Brady KT, Sonne SC, Verduin ML. Symptom improvement in co-occurring PTSD and alcohol dependence. *The Journal of nervous and mental disease*. 2006; 194:690–696. [PubMed: 16971821]
- Bonanno GA, Mancini AD. Beyond resilience and PTSD: Mapping the heterogeneity of responses to potential trauma. *Psychological Trauma: Theory, Research, Practice, and Policy*. 2012; 4:74–83.
- Brady KT, Back SE, Coffey SF. Substance abuse and posttraumatic stress disorder. *Current Directions in Psychological Science*. 2004; 13:206–209.
- Breese GR, Overstreet DH, Knapp DJ. Conceptual framework for the etiology alcoholism: A 'kindling'/stress hypothesis. *Psychopharmacology*. 2005; 178:367–380. [PubMed: 15765253]
- Brown PJ, Stout RL, Gannon-Rowley J. Substance use disorder-PTSD comorbidity: Patients' perceptions of symptom interplay and treatment issues. *Journal of Substance Abuse Treatment*. 1998; 15:445–448. [PubMed: 9751003]
- Carey KB, Correia CJ. Drinking motives predict alcohol related problems in college students. *Journal of Studies on Alcohol*. 1997; 58:100–105. [PubMed: 8979218]
- Chen P, Jacobson KC. Developmental trajectories of substance use from early adolescence to young adulthood: Gender and racial/ethnic differences. *Journal of Adolescent Health*. 2012; 50:154–163. [PubMed: 22265111]
- Colder CR, O'Connor RM. Attention bias and disinhibited behavior as predictors of alcohol use and enhancement reasons for drinking. *Psychology of Addictive Behaviors*. 2002; 16:325–332. [PubMed: 12503905]
- Colder, CR.; Chassin, L.; Lee, MR.; Villalta, IK. Developmental perspectives: Affect and adolescent substance use. In: Kassel, JD., editor. *Substance Use and Emotion*. American Psychological Association; 2010. p. 109-135.
- Cole DA, Martin NC, Steiger JH. Empirical and conceptual problems with longitudinal trait-state models: Introducing a Trait-State Occasion Model. *Psychological Methods*. 2005; 10:3–20. [PubMed: 15810866]
- Cooper ML, Frone MR, Russell M, Mudar P. Drinking to regulate positive and negative emotions: A motivational model of alcohol use. *Journal of Personality and Social Psychology*. 1995; 69:990–1005. [PubMed: 7473043]
- Del Boca FK, Darkes J, Greenbaum PE, Goldman MS. Up close and personal: Temporal variability in the drinking of individual college students during their first year. *Journal of Consulting and Clinical Psychology*. 2004; 72:155–164. [PubMed: 15065951]

- Desai S, Arias I, Thompson MP, Basile KC. Childhood victimization and subsequent adult revictimization assessed in a nationally representative sample of men and women. *Violence and Victims*. 2002; 17:639–653. [PubMed: 12680680]
- Fossos N, Kaysen D, Neighbors C, Lindgren KP, Hove MC. Coping motives as a mediator of the relationship between sexual coercion and problem drinking in college students. *Addictive Behaviors*. 2011; 36:1001–1007. [PubMed: 21719202]
- Frueh BC, Turner SM, Beidel DC, Cahill SP. Assessment of social function in combat veterans with PTSD. *Aggression and Violent Behavior*. 2001; 6:79–90.
- Goldstein AL, Flett GL, Wekerle C. Child maltreatment, alcohol use and drinking consequences among male and female college students: An examination of drinking motives as mediators. *Addictive Behaviors*. 2010; 35:636–639. [PubMed: 20199849]
- Greenbaum PE, DelBoca FK, Darkes J, Wang CP, Goldman MS. Variation in the Drinking Trajectories of Freshmen College Students. *Journal of Consulting and Clinical Psychology*. 2005; 73:229–238. [PubMed: 15796630]
- Ham L. Positive social alcohol outcome expectancies, social anxiety, and hazardous drinking in college students. *Cognitive Therapy and Research*. 2009; 33:615–623.
- Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*. 1999; 6:1–55.
- Hussong AM, Hicks RE, Levy SA, Curran PJ. Specifying the relations between affect and heavy alcohol use among young adults. *Journal of Abnormal Psychology*. 2001; 110:449–461. [PubMed: 11502088]
- Jackson KM, Sher KJ. Alcohol use disorders and psychological distress: A prospective state-trait analysis. *Journal of Abnormal Psychology*. 2003; 112:99–613.
- Jacobsen LK, Southwick SM, Kosten TR. Substance use disorders in patient with posttraumatic stress disorder: A review of the literature. *The American Journal of Psychiatry*. 2001; 158:1184–1190. [PubMed: 11481147]
- Kenny DA, Zautra A. The trait-state error model for multi-wave data. *Journal of Consulting and Clinical Psychology*. 1995; 63:52–59. [PubMed: 7896990]
- Kenny, DA.; Zautra, A. Trait-state models for longitudinal data. In: Collins, LM.; Sayer, AG., editors. *New Methods for the Analysis of Change*. Washington, DC: American Psychological Association; 2001. p. 243–263.
- Kessler RC. Posttraumatic stress disorder: The burden to the individual and to society. *Journal of Clinical Psychiatry*. 2000; 61:4–12. [PubMed: 10761674]
- King KM, Molina BSG, Chassin L. Prospective relations between growth in drinking and familial stressors across adolescence. *Journal of Abnormal Psychology*. 2009; 118:610–622. [PubMed: 19685957]
- Karlamangla A, Zhou K, Reuben D, Greendale G, Moore A. Longitudinal trajectories of heavy drinking in adults in the United States of America. *Addiction*. 2006; 101:91–99. [PubMed: 16393195]
- Kaysen D, Pantalone DW, Chawla H, Lindgren KP, Clum GA, Lee C, Resick PA. Posttraumatic stress disorder, alcohol use, physical health concerns. *Journal of Behavioral Medicine*. 2008; 31:115–125. [PubMed: 18095150]
- Khantzian EJ. The self-medication hypothesis revisited: The dually diagnosed patient. *Primary Psychiatry*. 2003; 10:47–48. 53–54.
- Kline, RB. *Principles and practices of structural equation modeling*. New York: Guilford Press; 1998.
- Koob GF. Drugs of abuse: anatomy, pharmacology, and function of reward pathways. *Trends in Pharmacological Sciences*. 1992; 13:177–184. [PubMed: 1604710]
- Kubany ES, Haynes SY, Leisen MB, Owens JA, Kaplan AS, Watson SB, Burns K. Development and preliminary validation of a brief broad-spectrum measure of trauma exposure: The Traumatic Life Events Questionnaire. *Psychological Assessment*. 2000; 12:210–224. [PubMed: 10887767]
- Kuntsche R, Knibbe R, Gmel G, Engels R. Why do young people drink? A review of drinking motives. *Clinical Psychology Review*. 2005; 25:841–861. [PubMed: 16095785]

- Kushner MG, Sher KJ, Erikson DJ. Prospective analysis of the relation between DSM-III anxiety disorders and alcohol use disorders. *American Journal of Psychiatry*. 1999; 156:723–732. [PubMed: 10327905]
- Lang AJ, Laffaye C, Satz LE, Dresselhaus TR, Stein MB. Sensitivity and specificity of the PTSD Checklist in detecting PTSD in female veterans in primary care. *Journal of Traumatic Stress*. 2003; 16:257–264. [PubMed: 12816338]
- Lewis MA, Neighbors C, Oster-Aaland L, Kirkeby BS, Larimer ME. Indicated prevention for incoming freshmen: Personalized normative feedback and high-risk drinking. *Addictive Behaviors*. 2007; 32:2495–2508. [PubMed: 17658695]
- Maggs JL, Schulenberg JE. Trajectories of alcohol use during the transition to adulthood. *Alcohol Research and Health*. 2004; 28:195–201.
- Magid V, MacLean MG, Colder CR. Differentiating between sensation seeking and impulsivity through their mediated relations with alcohol use and problems. *Psychology of Addictive Behaviors*. 2007; 32:2046–2061.
- Martens MP, Martin JL, Littlefield AK, Murphy JG, Cimini MD. Changes in protective behavioral strategies and alcohol use among college students. *Drug and Alcohol Dependence*. 2011; 118:504–507. [PubMed: 21612879]
- McFarlane AC, Browne D, Bryant RA, O'Donnell M, Silove D, Creamer M, Horsley K. A longitudinal analysis of alcohol consumption and the risk of posttraumatic symptoms. *Journal of Affective Disorders*. 2009; 118:166–172. [PubMed: 19230982]
- Merrill JE, Read JP. Motivational Pathways to Unique Types of Alcohol Consequences. *Psychology of Addictive Behaviors*. 2010; 24:705–711. [PubMed: 20822194]
- Miller MW, Vogt DS, Mozley SL, Kaloupek DG, Keane TM. PTSD and substance-related problems: The Mediating roles of disconstraint and negative emotionality. *Journal of Abnormal Psychology*. 2006; 115:369–379. [PubMed: 16737401]
- Murphy JG, Yurasek AM, Dennhardt AA, Skidmore JR, McDevitt-Murphy ME, MacKillop J, Martens MP. Symptoms of depression and PTSD are associated with elevated alcohol demand. *Drug and Alcohol Dependence*. 2013; 127:129–136. [PubMed: 22809894]
- Muthen, L.; Muthen, B. *Mplus: The comprehensive modeling program for applied researchers* (Version 6.1). Los Angeles, CA: Muthen & Muthen; 2010.
- Nees F, Tzschoppe J, Patrick CJ, Collstadt-Klein S, Steiner S, Poustka L, Banaschewski T, Barker GJ, Buchel C, Control PJ, Garavan J. Determinants of early alcohol use in healthy adolescents: The differential contribution of neuroimaging and psychological factors. *Neuropsychopharmacology*. 2012; 37:986–995. [PubMed: 22113088]
- Neighbors C, Geisner IM, Lee CM. Perceived marijuana norms and social expectancies among entering college student marijuana users. *Psychology of Addictive Behaviors*. 2008; 22:433–438. [PubMed: 18778137]
- Nelson TF, Xuan Z, Lee, Hang, Weitzman ER, Wechsler H. Persistence of heavy drinking and ensuing consequences at heavy drinking colleges. *Journal of Studies on Alcohol and Drugs*. 2009; 70:726–734. [PubMed: 19737497]
- Nesselroade JR, Stigler SM, Baltes PB. Regression towards the mean and the study of change. *Psychological Bulletin*. 1980; 88:622–637.
- O'Connor RM, Colder CR. Predicting Alcohol Patterns in First-Year College Students Through Motivational Systems and Reasons for Drinking. *Psychology of Addictive Behaviors*. 2005; 19:10–20. [PubMed: 15783273]
- Patton GC, Coffey C, Lynskey MT, Reid S, Hemphill Sh, Carlin JB, Hall W. Trajectories of adolescent alcohol and cannabis use into young adulthood. *Addiction*. 2007; 102:1361–1370.
- Read JP, Brown P, Kahler CW. Substance abuse and posttraumatic stress disorders: symptom interplay and effects on outcome. *Addictive Behaviors*. 2004; 29:2665–2672.
- Read JP, Colder CR, Merrill JE, Ouimette P, White J, Swartout A. Trauma and posttraumatic stress symptoms predict alcohol and other drug consequence trajectories in the first year of college. *Journal of Consulting and Clinical Psychology*. 2012; 80:426–439. [PubMed: 22545739]

- Read JP, Kahler CW, Strong D, Colder CR. Development and preliminary validation of the Young Adult Alcohol Consequences Questionnaire. *Journal of Studies on Alcohol*. 2006; 67:169–177. [PubMed: 16536141]
- Read JP, Merrill JE, Griffin ME, Bachrach R, Kahn S. Post-Traumatic Stress and Alcohol Problems: Self Medication or Trait Vulnerability? *The American Journal on the Addictions*. (in press).
- Read JP, O'Connor RM. High- and low-dose expectancies as mediators of personality dimensions and alcohol involvement. *Journal of Studies on Alcohol*. 2006; 67:1–12.
- Read JP, Ouimette P, White J, Colder CR, Farrow S. Rates of DSM-IV-TR trauma exposure and posttraumatic stress disorder among newly matriculated college students. *Psychological Trauma: Theory, Research, Practice, and Policy*. 2011; 3:148–156.
- Read JP, Wood MD, Kahler CW, Maddock JE, Palfai TP. Examining the role of drinking motives in college student alcohol use and problems. *Psychology of Addictive Behaviors*. 2003; 17:13–23. [PubMed: 12665077]
- Robinson TE, Berridge KC. The neural basis of drug craving: The neural basis of drug craving: an incentive-sensitization theory of addiction. *Brain Research Reviews*. 1993; 18:247–291. [PubMed: 8401595]
- Schmitt MJ, Steyer R. A latent state-trait model (not only) for social desirability. *Personality and Individual Differences*. 1993; 14:519–529.
- Shea MT, Vujanovic AA, Mansfield AK, Sevin E, Liu F. Posttraumatic stress disorder symptoms and functional impairment among OEF and OIF National Guard and Reserve veterans. *Journal of Traumatic Stress*. 2010; 23:100–107. [PubMed: 20127726]
- Schulenberg JE, Maggs JL. A developmental perspective on alcohol use and heavy drinking during adolescence and the transition to young adulthood. *Journal of Studies on Alcohol*. 2002; 114:54–70.
- Simpson TL, Stappenbeck CA, Varra AA, Moore SA, Kaysen D. Symptoms of posttraumatic stress predict craving among alcohol treatment seekers: Results of a daily monitoring study. *Psychology of Addictive Behaviors*. 2012; 26:724–733. [PubMed: 22369221]
- Sher KJ, Walitzer KS, Wood PK, Brenth EE. Characteristics of children of alcoholics: Putative risk factors, substance use and abuse, and psychopathology. *Journal of Abnormal Psychology*. 1991; 100:427–448. [PubMed: 1757657]
- Slutske WS. Alcohol use disorders among US college students and their non-college attending peers. *Archives of General Psychiatry*. 62:321–327. [PubMed: 15753245]
- Smyth JM, Hockemeyer JR, Heron KE, Wonderlich SA, Pennebaker JW. Prevalence, type, disclosure, and severity of adverse life events in college students. *Journal of American College Health*. 2008; 57:69–76. [PubMed: 18682348]
- Stasiewicz PR, Maisto SA. Two-factor avoidance theory: The role of negative affect in the maintenance of substance use and substance use disorder. *Behavior Therapy*. 1993; 24:337–356.
- Messman-Moore TL, Long PJ. Child sexual abuse and its relationship to revictimization in adult women: A review. *Clinical Psychology Review*. 1996; 16:397–420.
- Stewart, SH.; Conrod, PJ. Psychosocial models of functional associations between posttraumatic stress disorder and substance use disorder. In: Ouimette, P.; Brown, PJ., editors. *Trauma and Substance Abuse: Causes, Consequences, and Treatment of Comorbid Disorders*. Washington DC: APA Press; 2003. p. 29-56.
- Stewart SH, Pihl RO, Conrod PJ, Dongier M. Functional associations among trauma, PTSD, and substance related disorders. *Addictive Behaviors*. 1998; 23:797–812. [PubMed: 9801717]
- Trickey D, Siddaway AP, Meiser-Stedman R, Serpell L, Field AP. A meta-analysis of risk factors for post-traumatic stress disorder in children and adolescents. *Clinical Psychology Review*. 2012; 32:122–138. [PubMed: 22245560]
- U.S. Department of Commerce, Census Bureau, Current Population Survey (CPS), October, 1967 through 2009.
- Weathers, FW.; Huska, JA.; Keane, TM. The PTSD Checklist–Civilian Version (PCL-C). 150 South Huntington Avenue, Boston, MA 02130: National Center for PTSD, Boston Veterans Affairs Medical Center; 1991. Available from F. W. Weathers

- Weathers, FW.; Litz, BT.; Herman, DS.; Huska, JA.; Keane, TM. The PTSD Checklist (PCL): Reliability, validity, and diagnostic utility; Paper presented at the annual meeting of the International Society for Traumatic Stress Studies; San Antonio, TX. 1993 Oct.
- White HR, Labouvie EW, Papadaratsakis V. Changes in substance use during the transition to adulthood: A comparison of college students and their non-college age peers. *Journal of Drug Issues*. 2005; 35:281–304.
- White HR, McMorris BJ, Catalano RF, Fleming CB, Haggerty KP, Abbott RD. Increases in alcohol and marijuana use during the transition out of high school into emerging adulthood: The effects of leaving home, going to college, and high school protective factors. *Journal of Studies on Alcohol*. 2006; 67:810–822. [PubMed: 17060997]
- Wood MD, Read JP, Palfai TP, Stevenson JF. Social influence processes and college student drinking: The mediational role of alcohol outcome expectancies. *Journal of Studies on Alcohol*. 2001; 62:32–43. [PubMed: 11271962]

Figure 1.

Recruitment and Data Collection Flowchart. Note. Only those with lifetime trauma exposure and prior alcohol use (N=438) were included in the current sample. This was determined based on the Baseline (T1) assessment.

Figure 2.

Results of the final State-Trait-Error model for alcohol use and PTSD symptoms with cross-lagged associations among time points within each academic year. Unstandardized estimates are shown with standardized estimates in parentheses. Cross-lagged associations spanning across academic years (i.e., T4 to T5 and T8 to T9) were not included in the final models. Errors and disturbances are omitted from the figure for simplicity. ALC = alcohol use, PTS = posttraumatic stress symptoms, t = time point. ** $p < .01$, * $p < .05$.

Figure 3.

Results of the final State-Trait-Error model for alcohol consequences and PTSD symptoms with cross-lagged associations among time points within each academic year.

Unstandardized estimates are shown with standardized estimates in parentheses. Cross-lagged associations spanning across academic years (i.e., T4 to T5 and T8 to T9) were not included in the final models. Errors and disturbances are omitted from the figure for simplicity. ALC = alcohol consequences, PTS = posttraumatic stress symptoms, t = time point. ** $p < .01$, * $p < .05$.

Table 1

Means, standard deviations, and number of observations for alcohol use, alcohol consequences, and PTSD symptoms across the 12 assessments.

		Year 1				Year 2				Year 3			
		Sep (T1)	Dec (T2)	Feb (T3)	Apr (T4)	Sep (T5)	Dec (T6)	Feb (T7)	Apr (T8)	Sep (T9)	Dec (T10)	Feb (T11)	Apr (T12)
Alcohol Use	<i>M</i>	12.01	8.98	11.02	10.67	11.25	9.60	10.12	9.26	10.65	9.72	10.23	10.98
	<i>SD</i>	9.40	8.96	10.14	10.36	10.56	8.94	9.31	8.68	9.67	8.89	9.15	9.94
	<i>n</i>	486	440	418	416	445	431	420	415	444	423	420	416
Alcohol Consequences	<i>M</i>	8.55	5.00	6.41	5.85	6.01	5.01	4.95	4.76	4.88	4.40	4.41	4.76
	<i>SD</i>	8.21	7.44	8.87	8.07	8.44	8.15	7.98	7.67	7.08	7.04	6.93	7.24
	<i>n</i>	469	430	412	406	439	428	417	405	440	416	417	414
PTSD Symptoms	<i>M</i>	36.75	29.27	29.69	29.67	29.97	29.41	28.28	28.01	27.79	28.21	27.26	27.38
	<i>SD</i>	13.19	13.08	13.47	14.53	13.87	13.70	12.79	13.81	13.20	12.95	13.29	12.72
	<i>n</i>	483	433	423	412	440	424	418	412	432	419	411	409