Published in final edited form as:

Am J Addict. 2014 March ; 23(2): 108–116. doi:10.1111/j.1521-0391.2013.12075.x.

Posttraumatic Stress Symptoms and Alcohol Problems: Self Medication or Trait Vulnerability?

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Abstract

Background and Objectives—Posttraumatic stress symptoms (PTSD) and problem alcohol use (ALC) commonly co-occur, but the nature of this co-occurrence is unclear. Self-medication explanations have been forwarded, yet traits such as tendency toward negative emotionality and behavioral disinhibition also have been implicated. In this study we test three competing models (self-medication, trait vulnerability, combined dual pathway) of PTSD-ALC prospectively in a college sample.

Method—Participants (N=659; 73% female, M age=18) provided data at college matriculation (Time 1) and one year later (Time 2).

Results—Structural equation models showed disinhibition to mediate the path from PTSD symptoms to alcohol problems, supporting a trait vulnerability conceptualization. Findings regarding negative emotionality and self-medication were more mixed. Negative emotionality played a stronger role in cross-sectional than in prospective analyses, suggesting the importance of temporal proximity.

Conclusions and Scientific Significance—Self-regulation skills may be an important focus for clinicians treating PTSD symptoms and alcohol misuse disorders concurrently.

Introduction

Posttraumatic stress symptoms (PTSD) and problem alcohol use (ALC) commonly co-occur in general and clinical populations, and¹⁻³ recently emerging evidence shows a link between posttraumatic stress and heavy drinking in college students as well^{4,5} The delineation of mechanisms that underlie this co-occurrence may shed light on shared diagnostic features and risk pathways.

PTSD Symptoms and Problem Alcohol Involvement: Competing Conceptualizations

Self-Medication—Some suggest that there is a direct causal relationship between PTSD symptoms and ALC. That is, the presence of one directly causes the development of the other. Grounded in the long-standing view that problem drinking is the product of efforts to regulate, ameliorate, or cope with negative emotions,⁴⁻⁶ self-medication is one example of

such a mechanistic process.^{7,8} According to a self-medication conceptualization, PTSD symptoms lead directly to drinking for distress relief.

Trait Vulnerability—Others argue that the PTSD-ALC relationship is an indirect one, best accounted for by shared traits. From this perspective, it is not PTSD symptoms *per se*, but the effect of PTSD symptoms on traits that confers risk for alcohol misuse.^{9-11,12} In contrast to self-medication, the trait vulnerability pathway is an indirect one, whereby the individual temperamentally vulnerable to PTSD will experience an exacerbation of this vulnerability when PTSD symptoms occur, which may in turn place him/her at risk for problem alcohol use.^{12,68}

The structure of PTSD consists of both internalizing and externalizing dimensions.^{13,15, 16} Two traits that reflect these dimensions, Negative Emotionality and Disconstraint, are believed to be important vulnerabilities connecting PTSD symptoms to ALC.^{9,11,14-17} Negative emotionality is the propensity to experience negative affect. One personality characteristic that captures the dimension of negative emotionality is neuroticism. Disconstraint is a tendency toward risk-taking, impulsive action, and attitudes not bound by rules or social expectations. Both Neuroticism and Disconstraint may worsen following traumatic stress, compromising self-regulatory processes and increasing risk for substance abuse.¹⁶

Gender, PTSD Symptoms, and Problem Alcohol Involvement

Some research on the inter-connectedness of PTSD and ALC has identified gender as a potentially important individual-difference variable which may moderate connections between PTSD and ALC.²⁰⁻²² Indeed, Bornovalova et al.,²⁰ showed that temperamental (impulsivity) paths between PTSD-ALC were stronger for women than men. Beyond this one study, however, examinations of gender in PTSD-ALC associations have been scarce.

Tests of Competing Models

Though often examined in the literature,^{3,23-25} self-medication pathways seldom have been tested against alternative models. One exception is a study by Miller and colleagues¹⁸ who tested self-medication and trait vulnerability models of PTSD-ALC in a sample of veterans. These investigators found no evidence of a direct, self-medication pathway from PTSD to Alcohol Use Disorders. They did find evidence for an indirect, mediated path, through negative emotionality and Disconstraint. The authors concluded that associations between PTSD and ALC could best be characterized as occurring as a function of temperamental traits, presumably exacerbated by PTSD. Yet, these data were cross-sectional, precluding examination of whether this mediated pathway could be observed over time.¹⁹

The Present Study

In this study, we sought to examine the relation between PTSD symptoms and alcohol involvement, with an emphasis on the role of trait characteristics in this association. We tested three competing models. The first was Self-Medication, which posits no role of dispositional traits in PTSD-ALC, reflecting only a direct progression from distress to relief-seeking. The second, the Trait Vulnerability model, tested whether the path from PTSD

symptoms to ALC is fully explained by traits. Finally, we tested a Dual Pathway model. This included both direct and indirect paths, and tested the hypothesis that individual differences contribute to PTSD-ALC, but that there are unique, direct associations not explained by individual differences. We tested models concurrently and then prospectively, to examine whether associations replicated over time. Further, we tested these pathways in a college sample – a population seldom studied in regard to PTSD-ALC.

Method

Participants

Participants (N=659; 73% female) were students at two public universities in the north- and south-eastern U.S. At baseline, the average age was 18.10 (SD=.43). Seventy-one percent were Anglo Caucasian (n = 469), 11.2% Asian (n = 74), 10.3% Black (n = 68), 3.0% Hispanic/Latino (n = 20), and 3.2% multi-racial (n = 21).

Procedure

Recruitment procedures for this study are published elsewhere,^{26,27} but are described in brief here.

Eligibility screen—In the summer prior to matriculation, all incoming freshmen ages 18-24 were web-screened for Criterion A trauma and PTSD symptoms.²⁸ Based on this screening, 1,234 students were targeted for longitudinal follow-up. To ensure a strong representation of trauma and posttraumatic stress, all those reporting at least one lifetime trauma and at least one symptom from each of the 3 PTSD symptom clusters (B, C, and D) during screening were invited for follow-up.

Current sample—Data for the present study were collected in September post-matriculation (T1) and one year later (T2). Eighty-one percent of those invited (N = 1,002) completed the Time 1 survey. The retention rate was 93% (N = 934). Only participants who had experienced at least one Criterion A trauma at T1 (N = 735) were included. We used case-wise deletion for participants with any missing data (N = 76, 10%) on variables of interest, resulting in a final sample of 659 (73% female). Those included in our final sample and those excluded due to missing data did not differ on demographic, drinking, or PTSD symptom indices ($ps > .05$).

Measures

Demographics—Demographic characteristics assessed included gender, age, and ethnicity.

Alcohol Use—Participants were provided with a standard drink measurement chart. Following Wood and colleagues,²⁹ we assessed typical quantity and frequency of alcohol consumption over the past month and then created a quantity-frequency index by multiplying responses to these two items. The frequency question inquired about the average drinking frequency in the past month. Response options ranged from 0 (*Never in the past month*) to 6 (*Every day*). The quantity question read: “In the past month, how many drinks

did you *usually* have on any one occasion (Can of beer, glass of wine, wine cooler, drink of liquor)?” Response options ranged from 0 (*Didn’t drink in the past month*) to 10 (*9 or more total*). Participants who reported no lifetime use received a QF score of 0.

Alcohol Abuse and Dependence—At both T1 and T2, participants who reported alcohol use also completed four items assessing symptoms of alcohol abuse and seven items assessing alcohol dependence.²⁸ Items were scored dichotomously, and summed to create separate abuse and dependence symptom counts for each time point. Lifelong non-drinkers received scores of 0.

Vulnerability Traits—The 44-item Big Five Inventory (BFI)³⁰ assesses five personality dimensions: Openness, Conscientiousness, Extraversion, Agreeableness, and Neuroticism. Short phrases with adjectives describing prototypical features of each trait were rated on a 5-point scale (“*disagree strongly*” to “*agree strongly*”).

We used the BFI Neuroticism subscale to represent the construct of negative emotionality in our models. Disconstraint and other similar constructs (e.g., Psychoticism, Disinhibition) have been shown to be related both theoretically³¹⁻³³ and empirically³⁴⁻³⁷ to the Big Five factors of (low) Conscientiousness and (low) Agreeableness. Accordingly, in the present study we modeled a latent Disconstraint factor represented by Agreeableness and Conscientiousness subscale items, following Markon, et al.³⁵ and others.^{38,39} Low levels of both dimensions reflect a tendency toward impulsivity, recklessness, deception, non-cooperation, and manipulation.⁴⁰

Trauma Exposure—Lifetime trauma was assessed at T1 with the 21-item Traumatic Life Events Questionnaire (TLEQ), 41,^{F1} which includes assessment of exposure (DSM-IV Criterion A.1.), and the subjective experience of fear, helplessness, or horror (A.2.). The TLEQ is psychometrically strong, and has been used in a range of populations, including college students.⁴¹

PTSD Symptoms—At T1, past month PTSD symptoms were measured with the 17-item PTSD Checklist-Civilian Version (PCL-C).^{42,43} This assesses Criteria B (re-experiencing), C (avoidance/numbing), and D (arousal) of the PTSD construct. Participants rate each symptom on a 5-point scale (“*never*” to “*almost always*”). The web-survey was programmed to insert the TLEQ Criterion A trauma(s) that had been endorsed in each PCL-C symptom query. Thus, participants were oriented to their own event(s) while completing the PCL-C.

PCL-C items were rated as symptom or non-symptom using Blanchard et al.’s (2006) empirically derived cut-scores.⁴⁴ This involved re-scoring each symptom as either “1” (present) or “0” (absent), depending on the 5-point severity rating endorsed by the participant. Items were summed within B, C, and D clusters for a clinically meaningful symptom count.

^{F1}Format adapted by J. Read, SUNY at Buffalo, for specific, limited research use under license of the publisher, WPS, 12031 Wilshire Boulevard, Los Angeles, California 90025, U.S.A

Data Analytic Plan

Using a two-step approach,⁴⁵ we first tested a measurement model and then we evaluated a series of structural models comprised of these same variables. In structural models, paths were tested from the latent PTSD symptom variable to the latent alcohol problems variable, through disconstraint (latent) and neuroticism (manifest). Models were tested using AMOS 7.0.46,^{F2} We used MPlus Version 5⁴⁷ to obtain the Satorra-Bentler⁴⁷⁻⁴⁹ correction for chi-squares (S-B χ^2), which is robust to non-normality.

For the cross-sectional measurement model, we estimated covariances among the latent PTSD symptom factor which was comprised of manifest re-experiencing, numbing/avoidance, and arousal symptom indicators; the latent ALC factor which was comprised of manifest alcohol abuse, alcohol dependence, and quantity-frequency indicators; a latent Disconstraint higher-order factor comprised of 2 lower-order factors (Agreeableness, Conscientiousness); and the manifest indicator Neuroticism. Agreeableness and Conscientiousness each were comprised of 4 item parcels of the average of 2-3 BFI items each. For the longitudinal measurement model, we estimated covariances among the latent PTSD symptom factor at T1, the latent ALC factor at T1 and T2, a second-order latent T1 Disconstraint factor (comprised of Agreeableness, Conscientiousness), and a T1 manifest indicator, Neuroticism. Error covariances were estimated between T1 and T2.⁴⁵

Because assumptions of multivariate normality were violated (Mardia's coefficient > 21), we used the bias-corrected bootstrap method⁵⁰ to test the proposed meditational paths.⁵¹

To examine gender invariance, differences in the path coefficients of the estimated models were tested in a simultaneous multiple group analysis in AMOS 7.0. We first specified a model with all direct paths freely estimated and then constrained all paths to be equal across gender,⁵² examining model fit differences using a χ^2 difference test.

Results

Descriptive statistics

See Table 1 for descriptive statistics for all model variables. There were no gender or race (white vs non-white) differences on PTSD symptoms. Females had significantly higher scores on the Conscientiousness and Neuroticism factors of Disconstraint. Males had higher T1 alcohol dependence symptoms and T1 and T2 alcohol use, and white students were higher on all alcohol measures (all $ps < .05$). Participants reported an average of 3.02 (SD = 2.11) Criterion A events (range 1-14). Trauma types were diverse. Based on Blanchard et al.'s⁴⁴ scoring, 20.3% ($n = 134$) of T1 participants reported symptoms of full PTSD (1 B, 3 C, 2 D). Another 20.5% ($n = 135$) reported at least one symptom each in cluster.⁶¹ At Time 2 (T2), 17% ($n = 111$) met for an alcohol abuse diagnosis, and 10% ($n = 65$) met for alcohol dependence.

^{F2}Unique indirect effects were tested in Mplus V.5,⁴⁷ as these estimates are not given in AMOS 7.0.

Cross-sectional Results

In the measurement model, all factors loaded significantly on their indicators; the lowest regression weight was .50 (Table 1).⁵⁴ Measurement model fit was good (Table 2).

Next, we examined three competing models, (1) Self-Medication, (2) Trait Vulnerability, and (3) Dual Pathway. We began with a test of the Self-Medication model, whereby PTSD symptoms and ALC are linked with one another only directly. We controlled for Neuroticism and Disconstraint (Fig. 1a), which allowed us to test whether PTSD symptoms were associated with ALC *above and beyond* influences of personality. Model fit was good (Table 2). PTSD symptoms, Neuroticism (negatively), and Disconstraint all were significantly related to ALC ($ps < .05$).

In the second model, we tested the hypothesis that PTSD symptoms are associated with ALC only through traits Disconstraint and Neuroticism, and not directly (Figure 1b). Model fit again was good (Table 2). PTSD symptoms were significantly associated with both Neuroticism and Disconstraint, and Disconstraint was associated with ALC ($ps < .01$). Neuroticism was marginally and negatively related to ALC ($p < .10$). The total indirect effect was significant (95% CI of $\beta = .076 - .262$). However, tests of each component path coefficient suggested that this was driven by the significant unique indirect path via Disconstraint (95% CI of $\beta = .096 - .297$), as the effect via Neuroticism was non-significant (95% CI of $\beta = -.058 - .003$).

Lastly, in the Dual Pathway Model we specified a model that included paths from PTSD symptoms to ALC that were both direct and indirect (via Neuroticism, Disconstraint; see Figure 1c). Model fit was good (Table 2). The direct path from PTSD symptoms to ALC was $p < .10$, and PTSD symptoms were significantly associated with both Neuroticism and Disconstraint, each of which were associated with ALC (all $ps < .05$). The path from Neuroticism to ALC was negative. Importantly, the indirect effect of PTSD symptoms on ALC through personality was significant (95% confidence interval [CI] of $\beta = .020 - .186$), as were specific indirect effects of PTSD symptoms on ALC through Neuroticism (95% CI of $\beta = -.064 - -.004$) and Disconstraint (95% CI of $\beta = .053 - .221$).

We compared the two nested models, Dual Pathway and Trait-Vulnerability ($S-B\chi^2 = 3.38$, $df = 1$, $p > .05$). Though Akaike's criterion test⁵⁵ values were lowest for the Trait Vulnerability Model, this is due to fewer parameters being estimated. As such, though Trait Vulnerability and Dual Pathway models show equivalent model fit, we assert that the Dual Pathway model provides more clinically and theoretically relevant information, capturing multiple ways that diagnostic and trait variables may be connected. As the Self-Medication model was not nested, model comparisons were not possible.

Longitudinal Results

Measurement model fit was excellent. Fit for the Self-Medication model also was excellent (Table 2). However, only the autoregressive ALC path was significant ($p < .01$, Figure 2a). Neither baseline PTSD symptoms nor personality predicted T2 ALC beyond autoregressivity.

Next, we tested the Trait Vulnerability model (Figure 2b). Here we observed acceptable model fit (Table 2), significant paths from PTSD symptoms to both Neuroticism and Disconstraint, and a significant autoregressive path from T1 to T2 ALC ($ps < .01$). However, the total indirect path was non-significant, as were the unique indirect paths through the two mediators.

Lastly, we tested the Dual Pathway model, with both direct and indirect (via traits; Figure 2c) paths from PTSD symptoms to ALC. Autoregressivity (ALC) was controlled. Model fit was acceptable (Table 2). We observed significant direct effects from PTSD symptoms to Neuroticism to Disconstraint ($ps < .01$), and a marginally significant path from Disconstraint to ALC ($p < .10$). The total indirect effect from PTSD symptoms to ALC also was marginally significant (95% CI is .002 - .040). Though neither unique indirect path was significant, this marginal *total* indirect effect likely was driven by a marginally significant *unique* indirect effect via Disconstraint. We conclude this as each of the component unstandardized path coefficients of this indirect effect were at least marginally significant. This was not the case for indirect effects via Neuroticism.^{F3}

Prospective models showed acceptable to good model fit (Table 2). Model fit was not significantly different between the Trait Vulnerability and Dual Pathway models ($S-B\chi^2 = 2.03$, $df = 1$, $p > .05$). Again interpretation of AIC values is limited (see cross-sectional model discussion).

Gender Invariance

Across all cross-sectional and longitudinal models, chi-square difference tests showed no significant differences in constrained versus unconstrained (across gender) models. This suggests that there are not significant differences in the magnitudes of the path coefficients across gender.

Discussion

Our tests of three hypothesized models emphasize the role of individual differences in PTSD-ALC. Our findings consistently highlighted the role of Disconstraint, which across cross-sectional and prospective models was the most strongly implicated mechanism in this co-occurrence. We did observe some evidence for self-medication processes, but only in cross-sectional models. These results are discussed in detail below, and findings are integrated.

Cross-Sectional Associations

Our cross-sectional findings suggest that individual-level traits account for at least some of the link between PTSD symptoms and problem alcohol use.

^{F3}The Mplus program, from which we were able to obtain bias-corrected bootstrap parameter estimates for the unique indirect effects, is not able to provide 90% confidence intervals to be used to examine marginal significance. Thus, we cite Cohen & Cohen,⁵⁶ who suggest that if both component direct paths are significant, then the unique indirect effect likely is significant as well. Here, we apply this rule of thumb to evaluating marginal significance.

In our tests of the Self-Medication Model, we observed direct, concurrent associations between PTSD symptoms and ALC that were independent of personality traits, though the magnitude of these direct effects was relatively small, and did not replicate in prospective models. Importantly, Neuroticism and Disconstraint both contributed significantly to ALC variance, above and beyond associations with PTSD symptoms.

In the cross-sectional Trait Vulnerability model, we again observed evidence for the mediational role of Disconstraint. Interestingly, when direct paths from PTSD symptoms to ALC were not included, direct associations from PTSD symptoms to both Neuroticism and Disconstraint emerged, but only Disconstraint significantly predicted ALC (Neuroticism was marginal).

In the Dual Pathway model, we observed not only significant indirect (via personality) PTSD-ALC associations, but direct associations as well.

We observed a negative association between Neuroticism and ALC in our college sample. In contrast to non-college populations, where negative affect typically is associated with problem drinking, the literature shows drinking in college to be characterized by positive emotions, celebration, and conviviality.^{57,58} Indeed, some data show negative affect to be *protective* against heavy drinking.⁵⁹⁻⁶¹ The identification of this somewhat unique role of negative emotionality speaks to how individual-difference processes may vary across populations.

Prospective Findings

In contrast with our cross-sectional findings, we observed no support for a direct prospective pathway from T1 PTSD symptoms to T2 ALC (Self-Medication Model). This also was the case in the Dual Pathway Model. Several possibilities might explain this. The most obvious of these is that self-medication is not the best characterization of relations between PTSD symptoms and problem alcohol use over time. Thus, it may be that self-medication processes occur only in response to symptoms that are temporally proximal.^{25,62,63}

Another factor to consider is the timing of assessments. A year may be too long a period in which to capture self-medication processes. Though individuals may not drink to medicate PTSD symptoms from a year ago, they may drink to medicate more recent symptoms. Further, by modeling PTSD symptoms at one time point, we could not capture the relation between changes in these symptoms on subsequent alcohol involvement. Increasingly, researchers are using methods such as daily assessment to capture dynamic variation in behavior. In the future, such approaches will lend themselves well to a better understanding of temporal associations between PTSD symptoms and alcohol outcomes. Finally, strong stability in alcohol involvement (standardized coefficients from .69 to .71) may have obscured self-medication paths.

In the prospective test of the Dual Process Model, we found limited support for a mediated pathway from PTSD symptoms to problem alcohol use through Disconstraint. Whereas when only indirect effects were modeled (Trait Vulnerability), the path through Disconstraint appears unsupported, the Dual Pathway Model provides a different

interpretation, revealing a marginally significant path through this trait. This was consistent with our cross-sectional models, and with prior findings,¹⁸ as well as with conceptualizations of alcohol problem subtypes posited by Cloninger et al.⁶⁴ Notably, Neuroticism did not predict – and thus, did not mediate – problem drinking in either prospective model.

Gender Effects

In both cross-sectional and prospective model tests for gender variability, we found no evidence for variability in structural paths, supporting the gender invariance of these associations. It is important to note, however, that though we met guidelines for number of observations per group in multiple group analysis (N=100),⁶⁹ we had relatively more female than male participants. Future studies with equal gender representation may be better equipped to examine gender differences in these associations.

Limitations and Future Directions

This study advances the literature in several ways. First, we tested a theorized, self-medication conceptualization of PTSD-ALC, and contrasted this with two mediated trait-based models, over the span of one year. Associations were tested in a non-treatment seeking sample. Tests of cross-sectional and longitudinal models allowed us to note convergence and divergence in contemporary versus prospective associations. Additionally, though trauma, PTSD, and heavy drinking all have been identified as problems in college populations, research seldom has focused on co-morbidity in this group.

Limitations to this study should be noted. First, we used self-report assessment, which may be subject to limitations that are less typical of interview assessments.

Here we tested models examining the influence of PTSD symptoms and trait variables on drinking over one year. However, we did not test the prospective effects of PTSD symptoms on these traits; PTSD symptoms and trait variables were assessed concurrently. As such, this study provides only a partial test of the putative temporal pathway. Further, trait vulnerability paths could operate in directions other than those modeled here. For example, problem drinking could lead to exacerbation of trait factors that then lead to PTSD symptomatology, or PTSD symptoms and ALC could be reciprocally related.⁶⁵ Other trait mechanisms could also be examined, including specific expressions of internalizing or externalizing traits (e.g., distress, impulse control).⁶⁶⁻⁶⁷

There also may be features of our college sample that limit the generalizability of our findings, or may even have affected some of the observed associations. For example, rates of alcohol abuse and dependence symptoms in this sample were relatively low compared to clinical samples. Stronger associations may be observed in more heavily alcohol involved samples.

Also, individual-difference variables (e.g., coping) may affect the tendency to self-medicate. Such moderators were not tested here, but will be an important target of future investigations.

Summary, Conclusions, and Clinical Implications

Our data shed light on the nature of PTSD-ALC associations. In our concurrent models, we found evidence for direct associations between PTSD symptoms and ALC, even after accounting for trait variance. These cross-sectional findings may offer support for a self-medication process for symptoms that co-occur temporally. Clearly however, the PTSD-ALC relationship is more complex than just these direct associations. Indeed, perhaps the most compelling evidence to emerge from both our cross-sectional and prospective models is that trait factors such as negative emotionality and Disconstraint share variance with PTSD symptoms, and that at least some of this shared variance contributes to problem alcohol use. In particular, our findings regarding Disconstraint highlight the need for a greater focus on this trait. Clinicians treating these disorders concurrently should consider teaching individuals emotion regulation techniques, including approaches like mindfulness or stimulus control, to facilitate the successful management of externalizing tendencies that may contribute to problematic alcohol outcomes.

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. Craig R. Colder, Paige Ouimette, Jackie White, Ashlyn Swartout, and Sherry Farrow for their contributions to this study design and implementation. We also would like to thank the members of the UB Alcohol Research Lab for their many efforts to support data collection for this study, and the participants for sharing their experiences.

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Figure 1.

Cross-sectional Models. a) Self-Medication Model. (b) Trait Vulnerability Model. (c) Dual Pathway Model. Note: Unstandardized betas are followed by standardized betas in parentheses. Solid line represent marginally significant ($p < .10$) direct paths, bold lines represent significant ($p < .05$) direct paths. ** $p < .01$, * $p < .05$, † $p < .10$

In Figure 1a, Self-Medication (Direct only), T1 Alcohol $R^2 = .182$, In Figure 1b, Trait Vulnerability (Indirect only), T1 Alcohol $R^2 = .216$, T1 Neuroticism $R^2 = .127$, T1

Disconstraint $R^2=.175$. In Figure 1c, Dual Pathway (Direct and Indirect), T1 Alcohol $R^2=.187$, T1 Neuroticism $R^2=.127$, T1 Disconstraint $R^2=.130$

Figure 2.

Longitudinal Models. a) Self-Medication Model. (b) Trait Vulnerability Model. (c) Dual Pathway-Direct Model. Note: Unstandardized betas are followed by standardized betas in parentheses. Solid lines represent marginally significant ($p < .10$) direct paths, bold lines represent significant ($p < .05$) direct paths, dashed lines represent non-significant direct paths. ** $p < .01$, * $p < .05$, † $p < .10$

In Figure 2a, Self-Medication (Direct only), T2 Alcohol $R^2 = .510$, In Figure 2b, Trait Vulnerability (Indirect only), T2 Alcohol $R^2 = .498$, T1 Neuroticism $R^2 = .125$, T1

Disconstraint $R^2=.140$, In Figure 2c, Dual Pathway (Direct and Indirect), T2 Alcohol $R^2=.506$, T1 Neuroticism $R^2=.125$, T1, Disconstraint $R^2=.145$

Table 1
Descriptive Statistics, Internal Reliabilities, and Factor Loadings for Model Variables

	Items	M	SD	Min	Max	α	Factor Loading
Traumatic Stress (total, $\alpha = .94$)							
Re-experiencing	5	1.44	1.49	0	5	.90	.72
Avoidance	7	1.71	1.66	0	7	.85	.80
Hyperarousal	5	1.36	1.53	0	5	.87	.80
Disconstraint (all items, $\alpha = .78$)							
Agreeableness	9	3.53	0.62	1.11	4.89	.72	.74
Parcel 1	2	3.14	0.68	1	5		.50
Parcel 2	2	3.61	0.95	1	5		.68
Parcel 3	2	3.25	1.02	1	5		.61
Parcel 4	3	3.93	0.74	1	5		.70
Conscientiousness	9	3.54	0.65	1.56	5	.76	.52
Parcel 1	2	3.53	0.87	1	5		.71
Parcel 2	2	3.87	0.87	1	5		.66
Parcel 3	2	3.43	0.94	1	5		.69
Parcel 4	3	3.42	0.74	1	5		.65
Neuroticism	8	3.19	0.83	1	5	.83	
T1 Problematic Use							
T1 Quant $\times$ Freq	1	7.91	9.60	0	50		.66
T1 Abuse	4	0.24	0.56	0	4	.78	.70
T1 Dep	7	0.79	1.27	0	7	.92	.83
T2 Problematic Use							
T2 Quant $\times$ Freq	1	8.40	10.12	0	60		.69
T2 Abuse	4	0.24	0.59	0	3	.78	.64
T2 Dep	7	0.75	1.30	0	7	.94	.85

Table 2

Model Testing Sequence and Goodness-of-Fit Indices

	χ^2	S-B χ^2	p	df	RMSEA	SRMR	CFI	AIC	CAIC	GFI
Cross-sectional										
Measurement	221.761	206.563	.00	83	.050	.046	.95	295.761	498.918	.96
Self-Medication	221.761	206.563	.00	83	.050	.046	.95	295.761	498.918	.96
Trait Vulnerability	257.707	240.028	.00	85	.056	.054	.94	327.707	519.882	.95
Dual Pathway	253.444	236.547	.00	84	.055	.053	.94	325.444	523.110	.95
Longitudinal										
Measurement	317.923	284.461	.00	121	.050	.047	.95	417.923	692.459	.95
Self-Medication	317.923	284.461	.00	121	.050	.047	.95	417.923	692.459	.95
Trait Vulnerability	378.766	340.234	.00	125	.056	.066	.93	470.766	723.339	.94
Dual Pathway	376.551	338.192	.00	124	.056	.065	.93	470.551	728.615	.94

Note. Multiple criteria were used to evaluate model fit,^{55,56} including a Goodness of Fit Index (GFI) greater than .90 and Comparative Fit Index (CFI) greater than .95,⁵⁷ and Root Mean Square Error of Approximation (RMSEA) less than .06.⁵⁶