Published in final edited form as:
Psychol Addict Behav. 2014 September ; 28(3): 828–840. doi:10.1037/a0035805.

Internalizing and externalizing problem behavior and early adolescent substance use: A test of a latent variable interaction and conditional indirect effects

Matthew D. Scalco[a], **Craig R. Colder**[a], **Larry W. Hawk Jr.**[a], **Jennifer P. Read**[a], **William F. Wieczorek**[b], and **Liliana J. Lengua**[c]

[a]University at Buffalo, The State University of New York

[b]Buffalo State College

[c]University of Washington

Abstract

Externalizing problem behavior is a robust predictor of early adolescent substance use (SU); however findings regarding internalizing problems have been mixed, suggesting that there may be important moderators of the relationship between internalizing problems and SU. The present study used a community sample (mean age was 12.1 at the first assessment, 55% female, 83% White) to test a longitudinal latent variable interaction structural equation model to examine whether externalizing problems moderated the relationship between internalizing problems and SU. Peer delinquency was tested as a mediator in the model and prior levels of the mediator and outcome were controlled at each wave to establish temporal precedence. Results suggested that (1) internalizing problems were protective against associating with deviant peers, but only at high levels of externalizing symptomatology, (2) higher levels of peer delinquency were associated with increases in SU, and (3) peer delinquency mediated the effect of the problem behavior interaction on SU. Our findings suggest that the impact of internalizing problems on peer delinquency and SU needs to be considered in the context of externalizing problems. Moreover, developmental models involving internalizing symptoms should consider that internalizing symptoms are generally protective against substance use in early adolescence.

Keywords

adolescence; substance use; internalizing symptoms; externalizing symptoms; peer delinquency

Substance use (SU) and abuse continue to be a major public health concern in the United States (Kessler, Berglund, Demler, Jin, Merikangas, & Walters, 2005). Age of SU initiation is typically between the ages of 13 and 14 (Faden, 2006) and early initiation is associated with high rates of later abuse (Grant & Dawson, 1997) as well as many other adverse outcomes (Grueber, DiClemente, Anderson, & Lodico, 1996; Marmorstein, 2010).

Correspondence regarding this article should be directed to Matthew D. Scalco, Department of Psychology, Park Hall, SUNY at Buffalo, Buffalo, NY 14260-4110., mscalco@buffalo.edu, Phone: 716-645-0216, Fax: 716-645-3801.

Understanding risk and protective factors that influence early SU is important as such research informs the development of effective preventive interventions.

Problem behavior and affiliation with deviant peers figure prominently into developmental models of adolescent SU. Externalizing problems (e.g., rule breaking, aggression) robustly predict adolescent SU and there is evidence that affiliation with delinquent peers mediates this association (e.g., Dodge, Malone, Lansford, Miller, Pettit, & Bates, 2009). The developmental pathways to SU involving internalizing problems (e.g., internal emotional distress, including depression and anxiety) are less well understood in part because the association between internalizing problems and adolescent SU has been equivocal (Colder et al., 2010). One possible explanation for mixed findings is that the association between internalizing problems and adolescent SU may depend on other moderating variables. Given the high rates of co-occurrence of internalizing and externalizing problems in adolescence (Angold, Costello, & Erkanli, 1999) and the robust meditational pathway from externalizing problems to peer delinquency to adolescent SU, externalizing problems are an important context within which to consider the effects of internalizing problems on peer delinquency and SU.

The current study tests a prospective moderated mediational model whereby internalizing and externalizing problems interact to predict peer delinquency, which in turn predicts SU in early adolescence (age = 11–14). We pit two competing models, *the delayed onset hypothesis* and *the dual failure hypothesis*, against each other. Each model posits differing roles (protective, risk) for internalizing symptoms in the context of externalizing symptoms in the prediction of adolescent SU, through their combined influence on affiliation with delinquent peers. One possibility is that aspects of internalizing problems (e.g., fear and social withdrawal) protect youth from selection into delinquent peer networks and thereby reduces the likelihood of early initiation into SU (*the delayed onset hypothesis*, Hussong et al., 2011; Sung, Erkanli, Angold, & Costello, 2004). Alternatively, co-occurring internalizing and externalizing symptoms may place youth at higher risk for selection into delinquent peer networks and thereby increases the likelihood of early initiation into SU (*the dual failure hypothesis*, Capaldi, 1991). We develop these two possibilities in more detail below.

Internalizing Symptoms

Most of the literature conceptualizes internalizing symptoms as a risk factor for adolescent SU and suggests a self-medication mechanism to account for this association whereby SU is motivated to relieve emotional distress (Khantzian, 1997). However, the evidence for a risk pathway involving internalizing symptoms is mixed. Although some research supports an association between high levels of internalizing problems and adolescent SU (King et al., 2004), other research has found protective and null effects of internalizing symptoms on SU, even after controlling for externalizing problems (Colder et al., 2010; Colder, Scalco, Trucco, Read, Lengua, & Wieczorek, in press; Fischer, Najman, Williams, & Clavorino, 2012; Ingoldsby, Kohl, McMahon, & Lengua, 2006; Goodman, 2010; Marmorstein, 2010). Equivocal findings raise the question of whether internalizing problems and the commonly invoked self-medication mechanism is relevant to early adolescent SU (Colder et al., 2010;

Hussong et al., 2011; Sung, et al., 2004). Moreover, findings regarding the relationship between internalizing symptoms and peer delinquency have shown a similar pattern. For instance, findings suggest cross-sectional evidence for internalizing problems as a risk factor for peer delinquency (Wills, Aniette, Gibbons, & Brady, 2007), and longitudinal evidence for protection (Fite et al., 2006) and no association (Moss, Lynch, & Hardy, 2003). Equivocal results in these literatures indicate that it may be important to consider potential moderating variables.

Externalizing Problems as a Moderator of Internalizing Problems

Given that rates of co-occurrence of internalizing and externalizing problems in adolescence are high (Angold et al., 1999; Achenbach & Rescorla, 2001), externalizing problems are an important context in which to study the influence of internalizing problems on peer delinquency and adolescent SU. One possibility is that the effect of internalizing problems on peer delinquency and SU depends on whether they co-occur with externalizing problems. That is, externalizing problems may moderate the effect of internalizing problems on peer delinquency and SU. Testing such moderational effects may help clarify the mixed findings in the literature. There are several ways in which externalizing symptoms could moderate the effect of internalizing symptoms on peer delinquency and SU. The 4 panels in Figure 1 illustrate different possibilities given the present literature and reflect predictions of the theoretical models presented above (dual failure hypothesis and delayed SU onset hypothesis). In general, prior research that has considered both problem behavior domains and co-occurring symptoms tends to support panels C and D, as discussed below.

Miller-Johnson et al. (1997) examined the relationship between co-occurring conduct disorder and depression and adolescent SU using a high-risk community sample. The sample was split into four groups (no psychopathology, pure depression, pure conduct disorder, and co-occurring depression and conduct disorder or co-occurrence group), and then compared on alcohol, marijuana, and tobacco use. The pattern of findings supported a synergistic interaction effect in which internalizing symptoms have a positive association with SU when externalizing problems are high (see Figure 1 panel C). The synergistic effect is compatible with a *dual failure model*, whereby externalizing problems lead to failures in social relationships and academics, which in turn lead to internalizing problems and further disruptions in peer relations and SU (Capaldi, 1991; 1992).

However, evidence for a synergistic effect on peer delinquency and SU should not be overstated. Other studies using similar grouping strategies have found co-occurring internalizing and externalizing problems were comparable to or lower than an externalizing only group on SU (Dishion 2000; Ingoldsby, et al., 2006) and peer delinquency (Ingoldsby et al., 2006; Capaldi, 1992). These studies are consistent with Figure 1 panel D and with the *delayed SU onset hypothesis*, in that they provide evidence that externalizing symptoms in the absence of internalizing problems provide the highest risk for peer delinquency and SU, followed by elevated but intermediate risk when both internalizing and externalizing symptoms are present, and still lower risk associated with high internalizing symptoms that occur in the absence of externalizing symptoms. Finally, the lowest risk is associated with low internalizing and externalizing symptoms.

In considering the protective effects of internalizing symptoms on SU, Hussong et al., (2011) posited that hallmark features of internalizing symptoms such as fearfulness, social withdrawal, and avoidance might safeguard some youth from SU. Indeed, previous research has found that internalizing problems protect youth from selecting into delinquent peer networks, which in turn, decreased the likelihood of adolescent SU (Fite et al., 2006). This is compatible with *the delayed SU onset hypothesis*.

One limitation of much of the prior work on the relationship between co-occuring internalizing and externalizing symptoms and SU was that groups were formed using arbitrary cutoffs of symptom counts. Dichotomizing continuous variables is known to attenuate associations or even create spurious relationships (Cohen, 1983; MacCallum, Zhang, Preacher, & Rucker, 2002). In contrast, dimensional approaches to measuring psychopathology have been shown to be more reliable and valid than categorical approaches (Markon, Chmielewski, & Miller, 2011). In short, sample-to-sample variation in studies that utilize arbitrary cut-offs to form groups may partly explain inconsistent patterns of findings. Colder et al., (in press) used a complex factor analytic approach to examine prospective risk for adolescent substance use associated with “pure” and co-occurring *dimensions* of internalizing and externalizing symptoms. Findings suggested that “pure” externalizing symptoms had the strongest positive relationship with SU, followed by a smaller positive relationship between co-occurring symptoms and SU, while “pure” internalizing symptoms protected youth from SU. These findings are consistent with those of Ingoldsby et al. (2006), Dishion (2000), and the delayed SU onset hypothesis. In these studies, internalizing problems were differentially related to SU depending on whether they co-occurred with externalizing problems suggesting that whether internalizing problems operate as a risk or protective factor depends on levels of externalizing problems.

Another alternative to a grouping strategy or the complex factor model utilized by Colder et al. (in press) is to conceptualize problem behavior on a continuum and test whether externalizing problems moderate the relationship between internalizing problems and adolescent SU. Whether peer delinquency mediates this relationship is a question of interest given (1) the equivocal results in the literature on the relationship between internalizing problems and peer delinquency, (2) the importance of peer delinquency as a proximal influence in developmental models of early adolescent SU, and (3) the delayed SU onset hypothesis would predict that protective effects of internalizing problems on later SU may stem from fear of delinquent peer affiliation or social withdrawal which does not provide the appropriate context for exposure to SU (Hussong et al., 2011).

Summary and the Present Study

There is an implicit assumption made in the adolescent SU literature that internalizing and externalizing problems represent two distinct pathways to SU. However, evidence for an internalizing pathway is equivocal, and little research has explored co-occurring internalizing and externalizing symptoms in developmental pathways to SU. We propose that whether internalizing problems operate as a risk or protective factor may depend on levels of externalizing problems. As discussed above, there is theory and empirical work to support two competing hypotheses, which we pit against each other. The dual failure

hypothesis (Figure 1 Panel C) proposes that internalizing symptoms increases risk for peer delinquency and SU when they co-occur with externalizing symptoms. Alternatively, the delayed SU onset hypothesis (Figure 1 Panel D) predicts that internalizing symptoms decrease risk for peer delinquency and SU when they co-occur with externalizing symptoms. In secondary analyses, we separate internalizing symptoms into depression and anxiety because some researchers have argued that anxiety and depression may operate through different pathways to SU (Hussong & Chassin, 1994) and research suggests stronger evidence for depression as a risk factor above and beyond externalizing problems (Colder et al., 2010).

Hypotheses were tested using a structural equations model (SEM) with latent variable interactions, a 3-wave longitudinal design, and a community sample. This design has several advantages. First, the SEM model allows for more accurate estimation of measurement error than traditional regression models. This is especially relevant for interaction effects, which are notoriously unreliable in the behavioral sciences (Little, Bovaird, & Widaman, 2006). Second, the longitudinal design provides temporal precedence for testing mediation and associations between problem behavior, peer delinquency, and SU in early adolescence. Finally, using moderation to model co-occurring internalizing and externalizing symptoms allows for internalizing symptoms, externalizing symptoms, and their co-occurrence to be modeled with a continuous distribution of symptoms.

Methods

Participants

Participants were part of a three wave longitudinal study of risk and protective factors for adolescent SU. The sample was recruited (April 2007 – February 2009) using Random Digit Dialing (RDD) of listed and unlisted phone numbers for a county (that contains a major city) in the eastern United States. Inclusion criteria were: ages 11 or 12 years old at the time of recruitment, no language or physical disabilities that would preclude understanding or completion of the assessment, and a caregiver willing to participate. The final sample included 387 families (caregiver and adolescent).

The mean age for adolescents at the first assessment was 12.1 (55% female). The second and third assessments occurred at the one-year anniversary of the prior assessment. Participants were Caucasian/non-Hispanic (83%), 9% were Black, 2% were Hispanic, 1% were Asian, and 5% were other (usually of mixed ethnic background). Median family income was $70,000 and public assistance was received by 6% of participants. The majority of parents had completed college or some graduate/professional school (58%). Using the U.S. Census Bureau, the sample compares remarkably well to the general population of the County from which it came across a diverse set of characteristics, including gender, race/ethnicity, income, and receipt of public assistance. Our sample had somewhat more married couple families (76% vs. 65%), fewer female-headed families (21% vs. 28%), and higher levels of education than the county from which it came. The total attrition rate from Wave 1 (W1) to Wave 3 (W3) was 7.5%. Families who did not participate in Wave 2 (W2) or W3 did not differ from families who completed all three assessments on child gender, race, caregiver

marital status, caregiver education, income, or any of the variables used in the current analysis.

Procedure

Families were interviewed in university research offices. Transportation through a local taxi service was offered and provided for families as needed. All study procedures were approved by the University Institutional Review Board (IRB) before the study began. At the start of every interview, consent and assent forms were read aloud and signed by the parent and adolescent, respectively. Each were then brought to separate rooms for the interview. Assessments were typically 2 ½ hours and included questionnaires and laboratory tasks. The present study uses adolescent reported questionnaires that assessed problem behavior, peer delinquency, and SU. All questionnaires were computer administered. For sensitive topics (e.g., SU), the adolescent entered their responses to enhance privacy. Families were compensated $75, $85, and $120 at the first, second, and third assessment, respectively.

Measures

Adolescent Problem Behavior at W1—Problem behavior was assessed using the Youth Self Report (YSR) form of the Achenbach System of Empirical Behavioral Assessment (Achenbach & Rescorla, 2001). The rule breaking and aggressive scales (30 items) were used to measure externalizing problems, and the withdrawn depressed and anxious depressed scales (21 items) were used to measure internalizing problems. The YSR has been used extensively and has been shown to have good reliability and validity (Achenbach & Rescorla, 2001). Cronbach's α was .83 and .86 for the internalizing and externalizing scales, respectively. T-scores in our sample were similar to those reported by Achenbach & Rescorla (2001) in their non-referred sample. For example, 11–18 year old males and females were reported to have an average T-score of approximately 54 for anxious depressed, withdrawn depressed, rule breaking, and aggressive behavior scales, and in the current sample, these T-scores ranged from 51 to 55 for males and females.

Building latent interaction indicators at the item level of the YSR was not feasible given the large number of indicators. Accordingly, we parceled items to create indicators of our internalizing and externalizing factors, and then used these parceled indicators to create indicators of the latent interaction factor. Although there has been debate in the SEM literature about parceling indicators, there is some agreement that when items are unidimensional, parceling can be effective at reducing model complexity by reducing the number of indicators of latent factors (Little, Cunningham, Shahar and Widman, 2002). Factor analytic work on the YSR suggests considerable support for two broad factors representing internalizing and externalizing problems (Achenbach & Rescorla, 2001; Colder et al., 2012; Keiley, Lofthouse, Bates, Bates, Dodge, & Petit, 2003; Lengua, Sadowski, Friedrich, & Fisher, 2001). Accordingly, to create latent internalizing and externalizing factors, items from the aggression and rule breaking scales and items from the anxious depressed and withdrawn depressed scales were each combined into 5 random bundles to create five continuous indicators for each factor.[1]

For secondary analyses, depression and anxiety were separated into different sets of indicators to test for unique effects on peer delinquency and SU. We used the Lengua, Sadowski, Friedrich, & Fisher (2001) factor scheme to distinguish between generalized anxiety and depressive symptoms, and items from their conduct disorder (CD) and oppositional defiant disorder (ODD) scales to represent externalizing problems. In this scoring, 13 items compose the depression subscale, 11 items compose the general anxiety subscale, and 18 items comprise the CD and ODD subscales. Items from each of these subscales were combined into 4 bundles to create continuous indicators for the latent depression, anxiety, and externalizing factors respectively.

Child Substance Use at W1, W2, and W3—Items from the National Youth Survey (NYS) were used to assess lifetime use at W1 and past year alcohol, tobacco, marijuana, and other illegal drug use (Elliott & Huizinga, 1983) at W2 and W3. Lifetime use was assessed with one dichotomous item (no/yes) for each substance. Past year alcohol, marijuana, and tobacco use were assessed with open-ended frequency items. Past year other illegal drug use was assessed with a dichotomous (no/yes) item. In addition, quantity of alcohol use was assessed with an open-ended quantity item that read, “On the days you drink alcohol, how many drinks do you have?” Self-reports of adolescent SU, such as the NYS, have been shown to be valid when the adolescent perceives them to be anonymous and confidential (Winters, Stinchfield, Henly, & Schwartz, 1991).

Given the age of our sample, rates of use were low, especially at the first assessment (see Table 1) where the mean age is 12.1; however rates of use were comparable to other estimates of adolescent SU in the literature. For instance, Sung et al. (2004) reported 4.5% alcohol use, 2.8% tobacco use, and 3.33% for other drug use averaging across 11–13 year olds (see Table 1). Prevalence of alcohol use was higher than other SU, as has been found in large epidemiological studies of adolescent SU, and prevalence rates generally increased over time (Johnston, O’Malley, Bachman, & Schulenberg, 2005). Given the low prevalence rates, dichotomous SU variables were used in our subsequent structural equation modeling analysis with the exception of past year frequency and quantity of alcohol use at W2 and W3. Frequency and quantity of alcohol use at W2 and W3 were used as continuous indicators of latent factors for alcohol use at each wave. The distribution of these variables suggested some extreme observations at W2 and W3. Accordingly, values beyond three standard deviations above the mean were recoded to three standard deviations above the mean to reduce undue influence of a few cases (Tabachnick & Fidell, 2001). One and five cases were recoded for frequency of alcohol use at W2 and W3, respectively, and four and five cases were recoded for quantity of alcohol use at W2 and W3, respectively. Given the low prevalence of tobacco, marijuana, and other illegal SU, these variables were collapsed into dichotomous variables representing lifetime use (no use/use) of drugs other than alcohol

[1]Factor analytic work on the YSR has also suggested a subscale structure within the broad internalizing (withdrawn depressed and anxious depressed) and externalizing (rule breaking and aggression) factors (Achenbach & Rescorla, 2001). An alternative to the parceling strategy we used would be to parcel items within each of these subscales instead of within the broader factors. This strategy is not preferred in our model because it could yield an under-representation of a subscale on the latent interaction factor. Nonetheless, to examine how results might have been affected by our parceling strategy, we reran our models with randomized bundles of items within each subscale (e.g., 3 bundles for the rule-breaking subscale and three bundles for the anxious withdrawn subscale, etc.). Inferences were identical to those reported in the main analysis, suggesting that our results were not heavily influenced by our parceling strategy.

(other SU) at W1, and past year use at W2 and W3 (see bottom of Table 1). Alcohol and the other SU variables were tested in separate models to reduce model complexity.

Peer Delinquency at W1 and W2—Peer delinquency was assessed using 14 items representing delinquent and rule breaking behavior taken from Fergusson, Woodward, and Howard, (1999). The instructions asked the adolescent to "Tell whether or not any of your three close friends have ever done these things," and were keyed as yes (1) or no (0). Sample items are (1) "Sold marijuana or hashish," (2) "Purposefully set fire to a building, a car, or other property, or tried to do so," and (3) "Been in trouble with the police." Cronbach's alpha was .79 at W1 and .83 at W2. Preliminary confirmatory factor analysis was used to test a one factor model for the delinquency items.[2] The one factor model fit the data well at Wave 1 (x^2 (35) = 32.75, p = .57; RMSEA = 0.000; CFI/TLI = 1.00/1.00; WRMR = .60) and Wave 2 (x^2 (54) = 81.63, p = .01; RMSEA = 0.03; CFI/TLI = .98/.97; WRMR = .92), suggesting that the items formed a single dimension. To reduce complexity of our full model, delinquency items were randomly split to form three bundles that would serve as indicators for the peer delinquency latent factor at W1 and W2.

Data Analysis

Hypotheses were tested using structural equation modeling with a latent variable interaction estimated in Mplus version 6.1 (Muthén & Muthén, 1998–2007). Missing data at W2 and W3 were handled using full information maximum likelihood. As such, the full sample (N = 387) was retained for all analyses. Figure 2 contains an example of the full hybrid model that was estimated.[3] The interaction between internalizing and externalizing symptoms was specified using the unconstrained approach (or single mean centered approach; see Marsh Wen, & Hau, 2004) for modeling latent interactions. Although there are several different approaches for testing latent variable interactions (Little et al., 2006; Marsh, et al., 2004), the unconstrained approach has been shown to perform better than many of the other approaches (latent moderated structural equations approach and other "constrained" approaches) to testing latent variable interactions (1) when the first-order factor indicators deviate from normality as did our problem behavior indicators (skew ranged from .73 to 3.28) and (2) when the first order factors are correlated, as were our problem behavior factors (r = .63; Marsh et al., 2004). This approach involves mean centering first-order factor indicators and then creating cross products of the first-order factor indicators, which serve as indicators of the latent interaction factor.

Five of the 25 possible cross product indicators (five internalizing indicators x five externalizing indicators = 25 possible cross product terms) were selected as indicators of the latent interaction factor based on recommendations of Marsh et al., (2004). First, a simple two factor model of internalizing and externalizing symptoms was specified and the bundled first-order factor indicators were rank ordered from the highest to the lowest factor loading and externalizing indicators were paired with internalizing indicators based on these

[2]Given low variability in the peer delinquency items, some items were nearly perfectly correlated, thus precluding a CFA for all 14 items at each wave. The items with low variability were combined based on conceptual similarity into small bundles for the preliminary CFA.

[3]The full correlation matrix of observed variables is available upon request from the first author.

rankings (highest loading bundle from each factor were paired together, second highest loading bundle from each factor were paired together, etc.). Second, the paired bundles were then multiplied to form cross-product indicators of the latent interaction factor.

After determining the indicators for the latent variable interaction, the full hybrid structural equation model for alcohol use was estimated (see Figure 2 for which paths and covariances were estimated between factors). The only difference between the full hybrid model containing alcohol use and other SU (see Figure 4) was that assessments of alcohol use at W1, W2, and W3 were replaced with an observed dichotomous other SU variable at each wave.

Nested chi-square tests were used to determine whether including error covariances between interaction indicators and the first order indicators which formed the interaction indicators improved fit of the hybrid model. Moreover, nested chi-square tests were also used to test whether including auto-correlated error covariances between W1 and W2 peer delinquency factor indicators improved model fit. Modification indices were examined to determine if including any other error covariances between the interaction factor indicators and the first order factor indicators improved model fit. Variances of the internalizing, externalizing, and internalizing x externalizing factors were set to 1 and each of the factor loadings for these factors were estimated to simplify testing the simple slopes if the interaction had a significant effect on any of the outcomes. All other factor variances were freely estimated.

The alcohol model contained all continuous outcomes and was therefore estimated using maximum likelihood robust (MLR), which has been shown to provide more accurate estimates when variables deviate from normality (Finch, West, & MacKinnon, 1997). For the other SU model, the SU outcome variables were dichotomous. As such, the Weighted Least Squares estimator (WLSMV) was used. The Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), and Tucker-Lewis Index (TLI) was used to test model fit for all models, while the Standardized Root Mean Squared Residual (SRMR) was used for the alcohol model and the Weighted Root Mean Squared Residual (WRMR) was used for the other SU model. Because setting specific cut-offs for assessing "good" model fit cannot be generalized across all models (Hu & Bentler, 1999; Marsh, Hau, & Wen, 2004), ranges were used (for RMSEA, .08 is poor, .05 – .07 is acceptable, and < .05 is excellent; for CFI and TLI, < .9 is poor, .9 – .94 is acceptable, and > .95 is excellent; and for SRMR, .09 is poor, .06 – .09 is acceptable, and < .06 is excellent).

Age was included in the full hybrid models as a statistical control variable. Gender was also included as a statistical control variable in initial models, but it did not predict any of the outcomes so it was dropped from the analysis.[4] In both structural models, all continuous variables were mean centered. As such, standardized effects reported in Figure 2 and Figure 4 of the exogenous variables are effects when the moderator is held at mean levels. The

[4]We examined potential gender differences in our model given that some prior research has found problem behavior to predict SU differently for males and females (see Marmorstein, 2010). However, multiple group models testing gender invariance did not converge. Accordingly, we used regression models (for the alcohol and peer delinquency outcomes) and logistic regression models (for the other SU outcomes) with observed variables to test for presence of a 3-way interaction (internalizing x externalizing x gender) predicting peer delinquency and SU. All of the interaction terms including gender were not statistically significant, suggesting that our findings were similar for males and females.

intercepts for all of the factor indicators were set to zero and the intercepts of each of the latent endogenous variables were freely estimated. Estimating the intercept of each latent outcome in this manner was required to test the simple slopes in the case of statistically significant prediction from the latent interaction.

If the latent variable interaction term predicted W2 peer delinquency, W2 SU, or W3 SU then the simple slopes were tested for statistical significance using methods described in Preacher, Curran, and Bauer (2006), which included computing regions of significance for the simple slopes. This method identifies the point on the continuum of the moderator at which the simple slopes become statistically significant, thus avoiding testing simple slopes at arbitrary points (−1 and +1 *SD*). The proposed conditional indirect effect from internalizing symptoms to peer delinquency to SU moderated by externalizing symptoms was tested according to Preacher, Rucker, and Hayes (2007) using Rmediation and distribution of the product estimation (Togfighi & MacKinnon, 2011).

Results

Alcohol Use

Including error covariances between latent interaction indicators and the first order indicators that formed the interaction indicators improved model fit (Δx^2 [10] = 84.09, $p < .01$). A nested test indicated that including auto-correlated errors for the W1 and W2 peer delinquency factors did not improve model fit for the alcohol model (Δx^2 [3] = 4.30, $p = .23$). As such, they were not included. Inspection of modification indices suggested that adding another error covariance between a latent interaction indicator and a first order factor indicator led to a significant improvement in model fit (Δx^2 [1] = 27.86, $p > .01$). The final model with error covariances provided excellent fit to the data, x^2 (297) = 402.77, $p > .01$; RMSEA = 0.03; CFI/TLI = .96/.95; SRMR = .04. Standardized factor loadings ranged from .62 – .76, .69 – .81, .44 – .73, .72 – .81, .74 – .85, .65 – .86, and .60 – .83 for the internalizing, externalizing, internalizing x externalizing, W1 peer delinquency, W2 peer delinquency factors, W2 and W3 alcohol use factors, respectively. All factor variances not set to one were statistically significant as were all factor loadings.

Figure 2 contains path coefficients for the prospective effects of interest for the alcohol model and Table 2 contains the correlation matrix of the latent variables and covariates. W1 externalizing problems prospectively predicted increases in peer delinquency and alcohol use at W2. W1 internalizing problems were prospectively protective against W2 alcohol use and tended to predict low levels of peer delinquency at W2 ($p = .06$). However, the first order effects of problem behavior on W2 peer delinquency were qualified by a significant interaction between the internalizing and externalizing factors predicting W2 peer delinquency. Region of significance analysis revealed that simple slopes of internalizing symptoms became statistically significant at .06 standard deviations above the mean (simple slope = −.02) and at −1.3 *SD* below the mean (simple slope = .06) of externalizing problems. Thus, for W2 peer delinquency internalizing symptoms were protective above the mean of externalizing symptoms, but associated with increased risk at very low levels of externalizing symptoms (see Figure 3 for a graph of the interaction effect at the significant region boundaries). It is notable that although the slope of internalizing symptoms was

positive at low levels of externalizing symptoms, the levels of peer delinquency remain low (below average levels across the continuum of internalizing problems; see Figure 3). Although W1 peer delinquency and W2 alcohol use were associated (see Table 2), W1 peer delinquency did not uniquely predict W2 alcohol use above and beyond other predictors in the model. The internalizing, externalizing, and interaction factors did not reliably predict alcohol use at W3 as a direct effect above and beyond other predictors in the model. However, high levels of W2 peer delinquency prospectively predicted high levels of alcohol use at W3 after controlling for W2 alcohol use.

The general pattern of results suggested a potential conditional indirect effect just above the mean of externalizing symptoms (.06 *SD*) and at low levels of externalizing (−1.3 *SD*). The conditional indirect effect (CIE) from W1 internalizing to W3 alcohol use through W2 peer delinquency was marginally significant above the mean (.06 *SD*) of externalizing problems (CIE = −0.01, CI = −0.02 – .0006) and at very low levels (−1.3 *SD*) of externalizing problems (CIE = 0.01, CI = −.0008 – 0.02). The indirect effect from internalizing problems to alcohol use at W3 through W2 peer delinquency became statistically reliable at .56 *SD* above the mean of externalizing problems (CIE = −0.01, CI = −0.03 – −.000005); however, the CIE below the mean of externalizing problems never reached conventional cut-offs for statistical significance.

Other Substance use

Including error covariances between latent interaction indicators and the first-order indicators that formed the interaction indicators improved model fit (x^2 [10] = 256.59, p > .01). A nested test indicated that including auto-correlated errors for the W1 and W2 peer delinquency factor indicators did not improve model fit for the other SU model (Δx^2 [3] = 5.51, p = .14), and therefore, these covariances were not included in the final model. Modification indices suggested adding three additional error covariances between latent interaction indicators and first order factor indicators, and adding these covariances substantially improved model fit (x^2 [3] = 167.16, p > .01). The final model with error covariances provided excellent fit to the data, x^2 (247) = 445.9, p > .01; RMSEA = 0.05; CFI/TLI = .96/.95; WRMR = .95. Factor loadings for the other SU model were very similar to those in the alcohol model as would be expected because only the SU variables changed in the model. All factor variances not constrained to one were significant as were all factor loadings.

Findings mirrored those for alcohol use. Figure 4 contains the prospective effects of interest for the other SU model and the bottom of Table 2 contains the correlation matrix of the latent variables, covariates, and other SU at each wave. The nature of the internalizing x externalizing latent variable interaction prospectively predicting W2 peer delinquency was the same as that reported in the model for alcohol (the simple slope was statistically reliable above the mean [.09 *SD*] and at low levels [−1.24 *SD*] of externalizing symptoms, p <.05) as would be expected given that little changed in this portion of the structural model. Although W1 peer delinquency and W2 other SU were modestly associated (see Table 2), W1 peer delinquency did not uniquely predict W2 other SU above and beyond other predictors in the model. Mirroring the alcohol model, the general pattern of results suggested a potential

conditional indirect effect above the mean (.09 *SD*) of externalizing and at low levels of externalizing symptoms (−1.24 *SD*). The indirect effect from W1 internalizing to W3 other SU use through W2 peer delinquency was marginally significant above the mean (CIE = −0.05, CI = −0.13 – .0004) of externalizing symptoms and at low levels of externalizing symptoms (CIE = 0.06, CI = −.0005 – 0.14). The indirect effect from internalizing problems to W3 other SU through W2 peer delinquency became statistically reliable at .11 *SD* above the mean of externalizing problems (CIE = −0.05, CI = −0.13 – −.0004) and at −1.27 *SD* below the mean of externalizing problems (CIE = 0.06, CI = .001 – .14).

Secondary Analysis Separating Anxiety and Depression Symptoms

Next anxiety and depression symptoms were disaggregated. Analysis proceeded following the same steps as our main analysis with bundled indicators and latent variable interactions (anxiety symptoms x externalizing behavior and depression symptoms x externalizing behavior). The anxiety symptoms x externalizing interaction and the depression symptoms x externalizing interaction were tested in separate models, but we included a W1 latent depression symptoms factor as a first-order statistical control when testing the anxiety symptoms x externalizing factor, and vice versa. Due to problems with non-convergence in the other SU models (likely due to low rates of other SU at W1 and W2 and high levels of non-normality in the factor indicators of the problem behavior factors at W1 when separating anxiety and depression), we reran this model excluding W1 and W2 other SU and report the results of this model herein. We note that this model does not statistically control for prior levels of other SU, and as such, we cannot fully establish temporal precedence for the second path in our meditational model (W2 Peer delinquency to W3 other SU).

In the model including alcohol use and the depression symptoms x externalizing interaction, including error covariances between latent interaction indicators and the first order indicators that formed the interaction indicators improved model fit (x^2 [8] = 49.31, $p > .01$). The final model with error covariances provided good fit to the data (x^2 [319] = 450.53, $p < .01$; RMSEA = 0.03; CFI/TLI = .95/.94; SRMR = .045) and findings mirrored the main analysis in that the interaction between depression symptoms and externalizing symptoms was a significant predictor of peer delinquency when controlling for anxiety, β = −.26, t = −2.12 $p = .03$. Region of significance analysis for the simple slopes revealed that depression became a statistically significant protective predictor of W2 peer delinquency at .74 *SD* above the mean of externalizing symptoms. However, unlike the main analysis, depression was never positively related to peer delinquency, even at low levels of externalizing symptomatology. Mirroring the main analysis, peer delinquency at W2 was associated with increases in alcohol use at W3 and the CIE from depression symptoms to W3 alcohol use through W2 peer delinquency became significant at 1.19 SD above the mean of externalizing problems (CIE = −0.02, CI = −0.08 – −.00001). For the other SU outcome including the depression symptoms x externalizing interaction, the final model with error covariances provided excellent fit to the data (x^2 [230] = 424.58, $p < .01$; RMSEA = 0.05; CFI/TLI = .96/.95; WRMR = .98) and findings mirrored the alcohol analysis with depression and anxiety in that the interaction between depression symptoms and externalizing symptoms was a significant predictor of peer delinquency when controlling for anxiety, β = −.32, t = −3.09 $p < .01$. Region of significance analysis for the simple slopes

revealed that depression became a statistically significant protective predictor of W2 peer delinquency at −.04 *SD* below the mean of externalizing symptoms. Unlike the main analysis but similar to alcohol models in which depression and anxiety were separated, depression was never positively related to peer delinquency, even at low levels of externalizing symptomatology. Peer delinquency at W2 was associated with increases in alcohol use at W3 and the CIE from depression symptoms to W3 other SU through W2 peer delinquency was significant at −.04 SD below the mean of externalizing problems (CIE = −0.59, CI = −1.27 – −.001).

In the model including alcohol use and the anxiety symptoms x externalizing interaction, a nested test suggested that including error covariance's between indicators of the latent interaction factor and indicators of the first order components improved model fit (x^2 [8] = 62.95, $p > .01$). The final model with error covariances provided good fit to the data (x^2 [319] = 447.94, $p < .01$; RMSEA = 0.03; CFI/TLI = .95/.95; SRMR = .04) and results mirrored the main analysis in that the interaction between anxiety symptoms and externalizing symptoms was a significant predictor of W2 peer delinquency when controlling for depression, β = −.22, t = −2.31 $p = .02$. Region of significance analysis for the simple slopes revealed that anxiety was protective and became a statistically significant predictor of W2 peer delinquency at 1.43 *SD* above the mean of externalizing symptoms. However, unlike the main analysis but mirroring the depression analysis, anxiety was never positively related to peer delinquency, even at low levels of externalizing symptomatology. Moreover, peer delinquency at W2 was associated with increases in alcohol use at W3 and the CIE from anxiety symptoms to W3 alcohol use through W2 peer delinquency became significant at 1.71 SD above the mean of externalizing problems (CIE = −0.02, CI = −0.05 – −.00003). For the other SU outcome including the anxiety symptoms x externalizing interaction, the final model with error covariances provided excellent fit to the data (x^2 [319] = 450.88, $p < .01$; RMSEA = 0.05; CFI/TLI = .96/.95; WRMR = 1.03) and findings mirrored the main analysis in that the interaction between anxiety symptoms and externalizing symptoms was a significant predictor of peer delinquency when controlling for depression, β = −.19, t = −3.65 $p < .01$. Region of significance analysis for the simple slopes revealed that anxiety became a statistically significant protective predictor of W2 peer delinquency at very high levels of externalizing symptoms (2.35 *SD* above the mean) and at −1.52 SD below the mean of externalizing symptoms. Similarly to the main analysis, although the slope of anxiety symptoms was positive at low levels of externalizing symptoms, the levels of peer delinquency remain quite low across the continuum of anxiety problems. As such, the graph of this interaction looked almost identical to Figure 3. Peer delinquency at W2 was associated with increases in alcohol use at W3 and the CIE from anxiety symptoms to W3 other SU through W2 peer delinquency was significant at 2.35 SD above the mean of externalizing problems (CIE = −0.23, CI = −.47 – −.0006) and at −1.52 SD below the mean of externalizing (CIE = −0.23, CI = .0008 – .42).

Discussion

The mixed findings in the literature regarding the association between internalizing problems and adolescent SU suggests that moderational models may be important for clarifying this association. A major contribution of this study was to extend developmental

models of adolescent substance use by testing externalizing problems as a potential moderator of the link between internalizing problems and early adolescent SU. We capitalized on the advantages of a latent variable model and used a longitudinal design to establish temporal precedence and to test the mediating role of peer delinquency. Findings were similar for alcohol use and other SU and supported moderated mediation. In line with the delayed SU initiation hypothesis (Hussong et al., 2011), internalizing problems in the presence of moderately elevated externalizing problems decreased the likelihood of affiliating with delinquent peers, which in turn, prospectively predicted lower levels of alcohol use. Moreover, high levels of internalizing symptoms in the absence of externalizing symptoms were always associated with lower than sample mean levels of peer delinquency and other SU. These findings are discussed below.

Internalizing Problem Behavior and Substance Use

In the context of elevated levels of externalizing problems (at or above the sample mean), high levels of internalizing problems prospectively predicted lower levels of peer delinquency, which in turn, predicted low levels of SU. Given that internalizing problems have been linked to avoidance (Aldao, Nolen-Hoeksema, & Schweizer, 2010) and social problems (Ladd, 2006), these results raise the possibility that the fear and social withdrawal that is characteristic of internalizing problems may protect some youth from selecting into deviant peer networks and subsequently from SU. Other studies have also found that internalizing problems reduce risk for peer delinquency and early adolescent SU (Fite et al., 2006).

Our results also provided evidence for a positive association between internalizing problems and peer delinquency and other SU at very low levels of externalizing problems. At first glance, this may appear like a “risk” effect of internalizing problems, but even though the association was positive, the levels of peer delinquency and SU were very low (much lower than sample averages), suggesting that this effect does not have strong clinical implications. Nonetheless, it is consistent with some prior research supporting prospective positive associations between internalizing problems and SU (e.g., King et al., 2004), albeit with a small effect size. Taken together, our findings along with prior studies suggests that effects of internalizing problems can have varying influences on SU (negative, null, positive), and can be better understood by considering internalizing problems in the context of externalizing problems and peer delinquency.

It is notable that our findings are not consistent with models, such as the dual failure hypothesis, that posit synergistic interactive effects of internalizing and externalizing symptoms on early adolescent SU and peer delinquency (Capaldi et al., 1991; Miller-Johnson et al., 1997). Research examining co-occurring symptoms and adolescent SU that has utilized high-risk clinical samples (Capaldi, 1992; Miller-Johnson et al., 1997) has tended to support a synergistic model (Panel C Figure 1). In contrast, studies utilizing unselected community samples (Colder et al., in press; Ingoldsby et al., 2006) like the present analysis have tended to support highest risk for SU associated with pure externalizing symptoms, with co-occurring symptomatology conferring intermediate risk. Although the number of studies is too small to draw firm conclusions, one possibility is that

internalizing and externalizing problems may operate synergistically for small groups of youth at the extremes of symptomatology, but internalizing symptoms are generally protective at less extreme levels of symptomatology, at least in early adolescence.

Our findings should be placed in the appropriate developmental context. That is, we examined risk and protective pathways to early adolescent SU. High levels of internalizing problems are more consistently linked to SU in later adolescence (Sung et al., 2004) and early adulthood (Hussong et al., 2011), and this suggests that the status of internalizing symptoms as a risk or protective factor may change with age. Perhaps as use becomes more normative (Lee, Mun, White, & Simon, 2010), there is easier access to drugs and alcohol that doesn't depend on affiliation with delinquent peer groups. For instance, internalizing problems like depression and generalized anxiety tend to onset during adolescence and have been linked to maladaptive coping responses to stress (Graber & Sontag, 2009). Such increases in internalizing problems occur at a time of increasing coping demands, which may be attributable to the developmental shifts in autonomy and identity formation (Côté, 2009; Graber & Sontag, 2009). A developmental context in which use is normative combined with motives for self-medication due to increased difficulty in dealing with stress may provide the catalyst for use when internalizing symptoms are high later in adolescence or young adulthood. Another possibility is that substance use in early and middle adolescence influences the development of later internalizing problems, which then promotes self-medication motives for continued SU (i.e. a transactional relationship between these two variables begins later in adolescence). Indeed, previous research has linked adolescent SU to increases in depression later in adolescence and early adulthood (Marmorstein, 2010).

Anxiety and Depression

When we distinguished anxiety and depressive symptoms and examined the effects of one symptom cluster above and beyond the other, findings mirrored our main analysis, albeit with a somewhat weaker protective effect for anxiety than for depressive symptoms at elevated levels of externalizing problems. Our general conclusion is that for most of the population general emotional distress and withdrawal can protect youth from affiliating with delinquent peers, thereby decreasing risk for substance use in the context of high levels of externalizing problems. Previous research has linked general emotional distress in adolescence to rejection (Ladd, 2006) and depression and anxiety have uniquely been linked to avoidance behavior (Aldao et al., 2010; Dickson & MacLeod, 2004). This raises the possibility that in early adolescence (before SU becomes normative) characteristics common between depression and anxiety (rejection and avoidance) may protect youth from selecting into deviant peer networks even when externalizing problems are high. In addition to avoidance, depressive symptoms specifically have been linked to lower approach motivation (Dickson & MacLeod, 2004). This might explain why depression was a stronger protective factor for selecting into a delinquent peer network than anxiety when externalizing problems were above the mean, as youths lower on approach motivation may not seek out such social networks. Future research many benefit from considering this possibility.

The positive association between internalizing symptoms and peer delinquency and other SU that we observed for overall internalizing symptoms was evident for anxiety, but not depression. However, like our main analysis, the levels of peer delinquency and other SU were very low, suggesting that the symptom pattern of elevated anxiety with low levels of externalizing symptoms does not have strong clinical implications for SU, at least in early adolescence.

Limitations

Results from this study should be understood within the context of certain limitations. First, our sample spanned early to middle adolescence and our findings may not generalize to older ages. As stated above, there is evidence that internalizing problems more consistently predict substance use later in adolescence (Sung et al., 2004), and in early adulthood (Hussong et al., 2011) suggesting that different age samples may yield different results. It will be important for long-term longitudinal studies to extend our findings into late adolescence and emerging adulthood to examine if and when the status of internalizing symptoms shifts from a protective to a risk factor.

Second, internalizing and externalizing problems are higher order factors composed of multiple subdomains (e.g. anxiety, depression, rule breaking, aggression, etc.). Although there is little reason to suspect that the subdomains of externalizing problems in the present study would be differentially related to SU, some research has suggested that only certain subdomains of Attention Deficit Hyperactivity Disorder predict adolescent SU (Chang, Lichtenstein, & Larsson, 2012). Moreover, subdomains of internalizing problems not included in our study, such as separation anxiety, have been shown to be protective against SU (Kaplow, Curran, Angold, & Costello, 2001). Co-occurrence between these subdomains of internalizing problems and externalizing problems may be differentially related to risk for substance use. Future research may benefit from extending our work to examine other forms of co-occurring problems.

Third, our measure of peer delinquency was a measure of “perceived” peer delinquency. Previous research has demonstrated that when actual peer reports are obtained, effects on adolescent behavior tend to be smaller (Bauman & Ennett, 1996). While some researchers have argued that the smaller effect reflects an overestimation of the peer to target SU relationship (Bauman & Ennett, 1996), others have argued that the perception of peer behavior is important to the development of adolescent SU (Hill, Emery, Harden, Mendle, & Turkheimer, 2007; Trucco, Colder, & Wieczorek, 2011). Examining the robustness of our conditional indirect effect pathways with other measures of peer delinquency would be a useful direction for future research.

Conclusion

Developmental models of early adolescent substance use typically focus on externalizing behavior problems (Dodge et al., 2011; Zucker, 2006). Our findings support these models in that high levels of externalizing symptoms in the absence of internalizing symptoms (i.e. pure externalizing symptoms) resulted in the highest level of risk for peer delinquency, which, in turn, predicted higher risk for early adolescent SU. There is currently no

developmental model that accounts for the etiological role of internalizing problems in the initiation and escalation of adolescent SU. Our findings suggest that such a developmental model would need to consider both the effect of internalizing problems in the context of co-occurring externalizing problems, and the mediating role of peer delinquency. Considering the co-occurrence with externalizing problems will help clarify the mixed literature on the association between internalizing symptoms and adolescent SU in several ways. First, the general protective effect of internalizing problems that we observed calls into question whether self-medication models of SU are relevant to early adolescent SU in the general population. Second, previous research has typically either pit internalizing and externalizing symptomology against each other in regression models (which partials out co-occurring variance to provide unique associations with peer delinquency and SU; e.g., Fite et al., 2006, King et al., 2004) or ignored one or the other domain of problem behavior when predicting peer delinquency and SU (e.g., Kaplow et al., 2001; McKenzie, Olsson, Jorm, Romaniuk, & Patton, 2010). If the effect of internalizing problems on peer delinquency and SU depends on levels of externalizing symptoms, then one would expect to find positive, negative, and null effects in the literature depending on (1) which forms of problem behavior were measured and statistically controlled in the analysis, and (2) whether co-occurring symptoms are modeled in the analysis. Our findings in the context of equivocal past research indicate that it is crucial for future research to consider internalizing problems in the context of externalizing problems via moderation models.

Acknowledgments

This research was funded by a grant from the National Institute on Drug Abuse (R01 DA020171) awarded to Dr. Craig R. Colder.

References

Achenbach, TM.; Rescorla, LA. Youth Self-Report for ages 11–18. Burlington, VT: ASEBA; 2001.

Aldao A, Nolen-Hoeksema S, Schweizer S. Emotion regulation strategies across psychopathology: A meta-analytic review. Child Psychology Review. 2010; 30:217–237.

Angold A, Costello EJ, Erkanli A. Comorbidity. Journal of Child Psychology and Psychiatry. 1999; 40:57–87. [PubMed: 10102726]

Bauman KE, Ennett ST. On the importance of peer influence for adolescent drug use: Commonly neglected considerations. Addiction. 1996; 91:185–198. [PubMed: 8835276]

Capaldi DM. Co-occurrence of conduct problems and depressive symptoms in early adolescent boys: I. Familial factors and general adjustment at Grade 6. Development and Psychopathology. 1991; 3:277–300.

Capaldi DM. Co-occurrence of conduct problems and depressive symptoms in early adolescent boys: II. A 2-year follow-up at Grade 8. Development and Psychopathology. 1992; 4:125–144.

Chang Z, Litchtenstein P, Larsson H. The effects of childhood ADHD symptoms on early-onset substance use: A swedish twin study. Journal of Abnormal Child Psychology. 2012; 40:425–435. [PubMed: 21947618]

Chassin, L.; Ritter, J. Vulnerability to substance use disorders in childhood and adolescence. In: Ingram, RE.; Price, JM., editors. Vulnerability to Psychopathology: Risk Across the Lifespan. 2. NY: Guildford Press; 2010. p. 107-134.

Colder, CR.; Chassin, L.; Lee, MR.; Villalta, IK. Substance Abuse and Emotion. Washington, DC: American Psychological Association; 2010. Developmental perspectives: Affect and adolescent substance use; p. 109-135.

Colder CR, Scalco MD, Trucco EM, Read JP, Lengua LJ, Wieczorek WF. Prospective associations of internalizing and externalizing problems and their co-occurrence with early adolescent substance use. Journal of Abnormal Child. (in press).

Cote, JE. Identity formation and self-development in adolescence. In: Lerner, RM.; Steinberg, L., editors. Handbook of Adolescent Psychology. 3. Vol. 1. New Jersey: John Wiley & Sons Inc; 2009. p. 266-304.

Dickson JM, MacLeod AK. Anxiety, depression and approach and avoidance goals. Cognition and Emotion. 2004; 18:423–430.

Dishion TJ. Cross-setting consistency in early adolescent psychopathology: Deviant friendships and problem behavior sequelae. Journal of Personality. 2000; 68:1109–1124. [PubMed: 11130734]

Dodge KA, Malone PS, Lansford JE, Miller S, Pettit GS, Bates JE. A dynamic cascade model of the development of substance-use onset. Monographs of the Society for Research in Child Development. 2009:74.

Elliott DS, Huizinga D. Social class and delinquent behavior in a national youth panel. Criminology: an Interdisciplinary Journal. 1983; 21:149–177.

Faden VB. Trends in initiation of alcohol use in the united states 1975 to 2003. Alcoholism: Clinical and Experimental Research. 2006; 30:1011–1022.

Fergusson DM, Woodward LJ, Horwood LJ. Childhood peer relationship problems and young people's involvement with deviant peers in adolescence. Journal of Abnormal Child Psychology. 1999; 27:357–370. [PubMed: 10582837]

Finch JF, West SG, MacKinnon DP. Effects of sample size and nonormality on the estimation of mediated effects in latent variable models. Structural Equation Modeling. 1997; 4:87–107.

Fischer JA, Najman JM, Williams GM, Clavarino AM. Childhood and adolescent psychopathology and subsequent tobacco smoking in young adults: Findings from an Australian birth cohort. Addiction. 2012; 107(9):1669–1676. [PubMed: 22340634]

Fite PJ, Colder CR, O'Connor RM. Childhood behavior problems and peer selection and socialization: Risk for adolescent alcohol use. Addictive Behaviors. 2006; 31:1454– 1459. [PubMed: 16266788]

Galea S, Tracy M. Participation rates in epidemiological studies. Annals of Epidemiology. 2007; 17:643–653. [PubMed: 17553702]

Goodman A. Substance use and common child mental health problems: Examining longitudinal associations in a British sample. Addiction. 2010; 105:1484–1496. [PubMed: 20528814]

Graber, JA.; Sontag, LM. Internalizing problem during adolescence. In: Lerner, RM.; Steinberg, L., editors. Handbook of Adolescent Psychology. 3. Vol. 1. New Jersey: John Wiley & Sons Inc; 2009. p. 642-682.

Grant BF, Dawson DA. Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: Results from the national longitudinal alcohol epidemiologic survey. Journal of Substance Abuse. 1997; 9:103–110. [PubMed: 9494942]

Gruber E, DiClemente RJ, Anderson MM, Lodico M. Early drinking onset and its association with alcohol use and problem behavior in late adolescents. Preventive Medicine. 1996; 25:293–300. [PubMed: 8781007]

Hill J, Emery RE, Harden KP, Mendle J, Turkheimer E. Alcohol use in adolescent twins and affiliation with substance using peers. Journal of Abnormal Child Psychology. 2008; 36:81–94. [PubMed: 17665304]

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, Chassin L. The stress-negative affect model of adolescent alcohol use: disaggregating negative affect. Journal of Studies on Alcohol. 1994; 55:707–718. [PubMed: 7861800]

Hussong AM, Jones DJ, Stein GL, Baucom DH, Boeding S. An internalizing pathway to alcohol use and disorder. Psychology of Addictive Behaviors. 2011; 25:390–404. [PubMed: 21823762]

Ingoldsby EM, Kohl GO, McMahon RJ, Lengua L. Conduct problems, depressive symptomatology and their co-occurring presentation in childhood as predictors of adjustment in early adolescence. Journal of Abnormal Child Psychology. 2006; 34:603–621. [PubMed: 16967336]

Johnston, LD.; O'Malley, PM.; Bachman, JG.; Schulenberg, JE. NIH Publication No. 05-5727. Bethesda, MD: National Institute on Drug Abuse; 2005. Monitoring the future: National survey results on drug use, 1975–2004. Volume I: Secondary school students.

Kaplow JB, Curran PJ, Angold A, Costello EJ. The prospective relation between dimensions of anxiety and the initiation of adolescent alcohol use. Journal of Clinical Child Psychology. 2001; 30:316–326. [PubMed: 11501249]

Kessler RC, Berglund P, Demler O, Jin R, Merikanglas KR, Walters EE. Lifetime prevelance and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. Archive of General Psychiatry. 2005; 62:593–602.

Khantzian EJ. The self-medication hypothesis of substance use disorders: A reconsideration and recent applications. Harvard Review of Psychiatry. 1997; 4:231–244. [PubMed: 9385000]

King KM, Iacono WG, McGue M. Childhood externalizing and internalizing psychopathology in the prediction of early substance use. Addiction. 2004; 99:1548–1559. [PubMed: 15585046]

Ladd G. Peer rejection, aggressive or withdrawn behavior, and psychological maladjustment form ages 5 to 12: An examination of four predictive models. Child Development. 2006; 77:822–846. [PubMed: 16942492]

Lee C, Mun E, White HR, Simon P. Substance use trajectories of black and white young men from adolescence to emerging adulthood: A two-part growth curve analysis. Journal of Ethnicity and Substance Use. 2010; 9:301–319.

Lengua LJ, Sadowski CA, Friedrich WN, Fisher J. Rationally and empirically derived dimensions of children's symptomatology: Expert ratings and confirmatory factor analyses of the CBCL. Journal of Consulting and Clinical Psychology. 2001; 69:683–698. [PubMed: 11550734]

Little TD, Bovaird JA, Widamin KF. On the merits of orthogonalizing powered and product terms: Implications for modeling interactions among latent variables. Structural Equation Modeling. 2006; 13:497–519.

Little TD, Cunningham WA, Shahar G, Widamin KF. To parcel or not to parcel: Exploring the question, weighting the merits. Structural Equation Modeling. 2002; 9:151–173.

MacCallum RC, Zhang S, Preacher KJ, Rucker DD. On the practice of dichotomization of quantitative variables. Psychological Methods. 2002; 7(1):19–40. [PubMed: 11928888]

MacKinnon, DP. Introduction to statistical mediation analysis. Mahwah: Erlbaum; 2008.

Markon KE, Chmielewski M, Miller CJ. The reliability and validity of discrete and continuous measures of psychopathology: A quantitative review. Psychological Bulletin. 2011; 137(5):856–879. [PubMed: 21574681]

Marmorstein NR. Longitudinal associations between depressive symptoms and alcohol problems: The influence of comorbid delinquent behavior. Addictive Behaviors. 2010; 35:564–571. [PubMed: 20171020]

Marsh HW, Hau K, Wen Z. In search of golden rules: comment on hypothesis-testing approaches to setting cutoff values for fit indexes and danger in overgeneralizing Hu and Bentler's (1999) findings. Structural Equation Modeling. 2004; 11:320–341.

Marsh HW, Wen Z, Hau K. Structural Equation Models of Latent Interactions: Evaluation of Alternative Estimation Strategies and Indicator Construction. Psychological Methods. 2004; 9:275–300. [PubMed: 15355150]

McKenzie M, Olsson CA, Jorm AF, Romaniuk H, Patton GC. Association of adolescent symptoms of depression and anxiety with daily smoking and nicotine dependence in young adulthood: Findings from a 10-year longitudinal study. Addiction. 2010; 105(9):1652–1659. [PubMed: 20707783]

Miller-Johnson S, Lochman JE, Coie JD, Terry R, Hyman C. Comorbidity of conduct and depressive problems at sixth grade: Substance use outcomes across adolescence. Journal of Abnormal Child Psychology. 1997; 26:221–232. [PubMed: 9650628]

Moss HB, Lynch KG, Hardie TL. Affiliation with deviant peers among children of substance dependent fathers from pre-adolescence into adolescence: Associations with problem behaviors. Drug and Alcohol Dependence. 2003; 71:117–125. [PubMed: 12927649]

Muthén, LK.; Muthén, BO. Mplus User's Guide. 6. Los Angeles, CA: Muthén and Muthén; 1998–2007.

Preacher KJ, Curran PJ, Bauer DJ. Computational tools for probing interactions in multiple linear regression, multilevel modeling, and latent curve analysis. Journal of Educational and Behavioral Statstics. 2006; 31:437–448.

Preacher KJ, Rucker DD, Hayes AF. Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. Multivariate Behavioral Research. 2007; 42(1):185–227.

Sung M, Erkanli A, Angold A, Costello EJ. Effects of age at first substance use and psychiatric comorbidity on the development of substance use disorders. Drug and Alcohol Dependence. 2004; 75:287–299. [PubMed: 15283950]

Tabachnick, BG.; Fidell, LS. Using Multivariate Statistics. Boston, MA: Pearson Education, Inc; 2007.

Tofighi D, MacKinnon DP. RMediation: An R package for mediation analysis confidence intervals. Behavior Research Methods. 2011; 43:692–700. [PubMed: 21487904]

Trucco EM, Colder CR, Wieczorek WF. Vulnerability to peer influence: A moderated mediation study of early adolescent alcohol use initiation. Addictive Behaviors. 2011; 36:729–736. [PubMed: 21420241]

Wills TA, Aniette MG, Mendoza D, Gibbons D, Brady GH. Self-control, symptomatology, and substance use precursors: Test of a theoretical model in a community sample of 9-year-old children. Psychology of Addictive Behaviors. 2007; 21:205–215. [PubMed: 17563140]

Winters KC, Stinchfield RD, Henly GA, Schwartz RH. Validity of adolescent self-report of alcohol and other drug involvement. The International Journal of the Addictions. 1991; 25:11A, 1379–1395.

Zucker, RA. Alcoholism; a life span perspective on etiology and course. In: Sameroff, AJ., editor. Developmental psychopathology. 2. New York: Kluwer Academic/Plenum Publishers; 2006. p. 569-585.

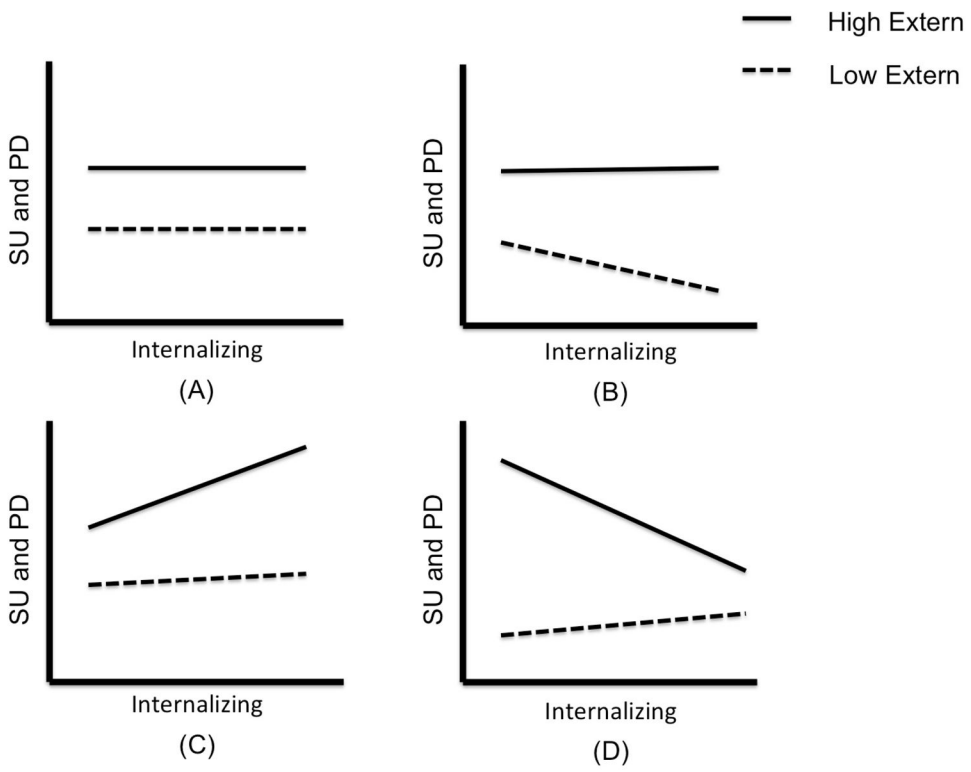

Figure 1.

Hypothetical Internalizing and Externalizing Interaction Patterns.

Note. SU = substance use and PD = peer delinquency. Panel A indicates a non-significant interaction effect, while Panel B indicates a protective effect only in the absence of externalizing symptoms. Panel C represents the dual failure model in that it suggests a synergistic effect of internalizing and externalizing problems on SU and PD. In contrast, Panel D represents the delayed initiation onset hypothesis in that it suggests that internalizing symptoms are protective against SU and PD.

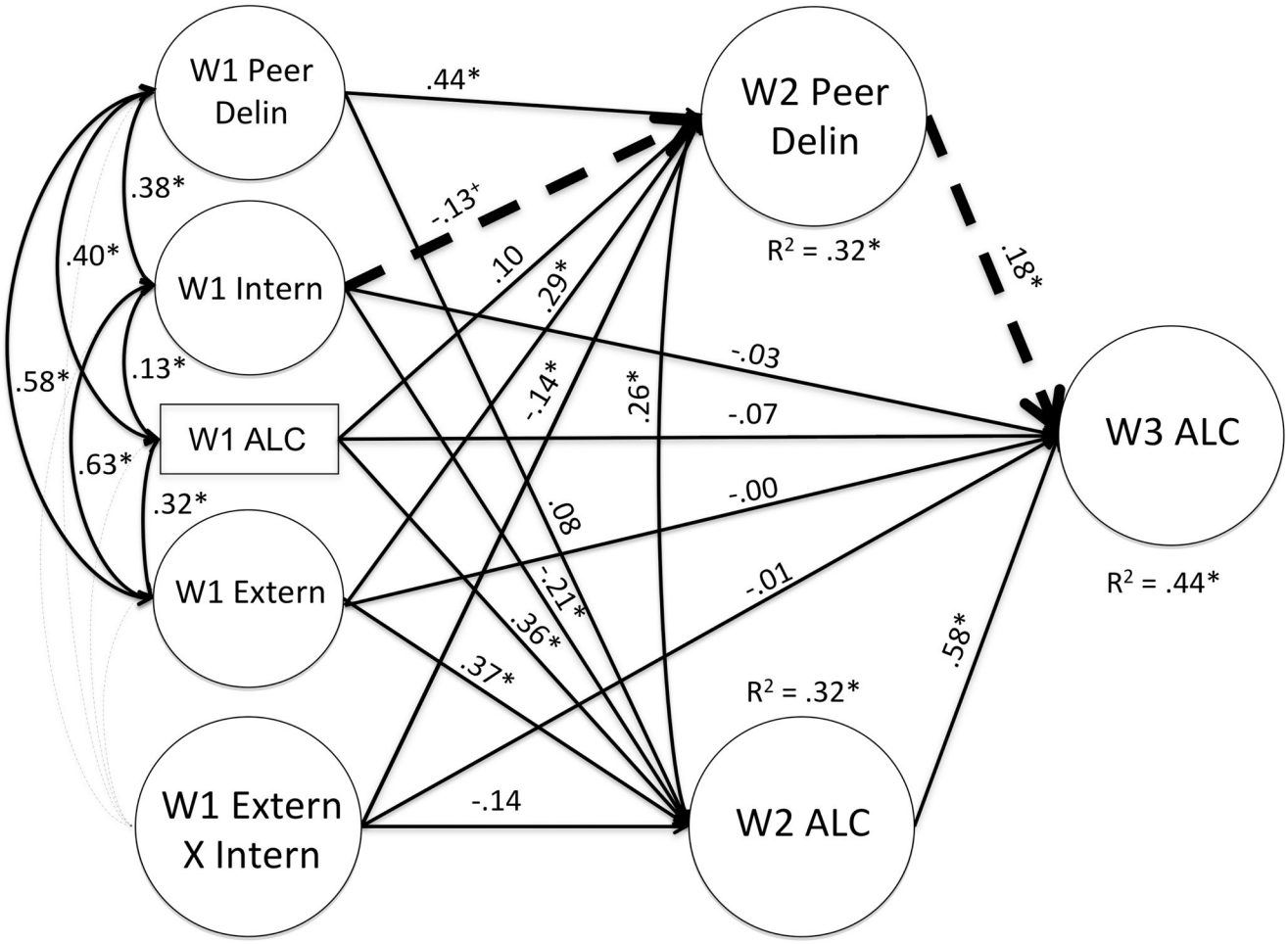

Figure 2.

Estimated Standardized Path Coefficients for the Full Model Predicting Alcohol Use

Note. Age was left out for ease of presentation. * p < .05, + = p < .10. Intern = Internalizing factor, Extern = Externalizing factor, Extern x Intern = Internalizing x Externalizing interaction factor, Peer Delin = peer delinquency, Alc = alcohol, and W = wave. Note that the interaction factor was allowed to correlate with exogenous variables in the model. The pathway that is bolded and has a broken line represents the significant conditional indirect effect from W1 Internalizing problems to W3 Alcohol use through W2 Peer Delinquency and moderated by externalizing problems..

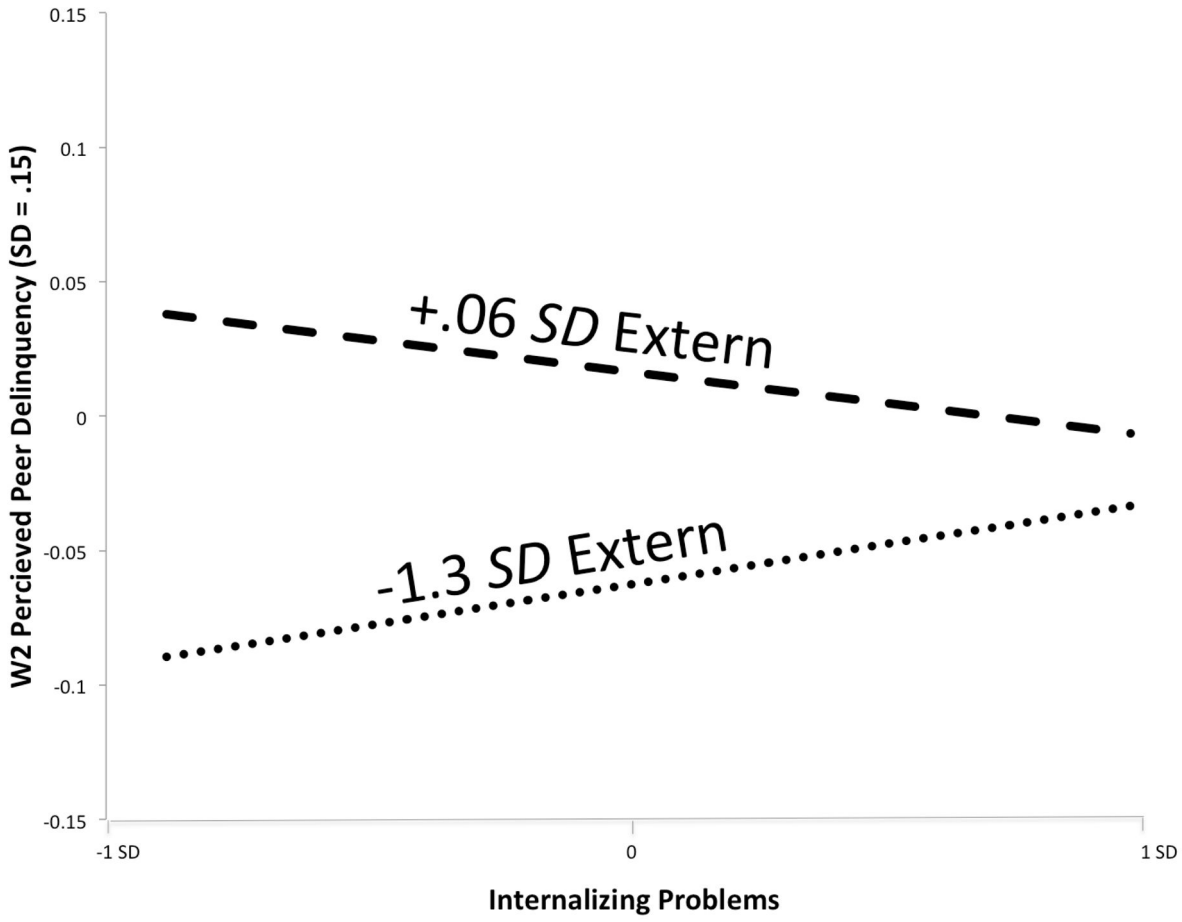

Figure 3.

Region Boundaries for the Effect of Internalizing Problems on Peer Delinquency at Different Levels of Externalizing Symptoms for the Alcohol Model

Note. Extern = externalizing symptoms, W = Wave and *SD* = Standard Deviation. The scales of both internalizing and externalizing symptoms are standardized (mean = 0, *SD* = 1). The .06 *SD* and −1.3 *SD* Extern slopes were significantly different than 0. Slopes between internalizing problems and peer delinquency become steeper, remain negative, and remain significantly different than 0 as externalizing problems increase from .06 *SD*. Likewise, slopes between internalizing problems and peer delinquency become steeper, remain positive, and remain significantly different than 0 as externalizing problems decrease from −1.3 *SD*. A very similar pattern was observed in the Other SU model for the main analysis and for the anxiety x externalizing problems model in which depression and anxiety were separated.

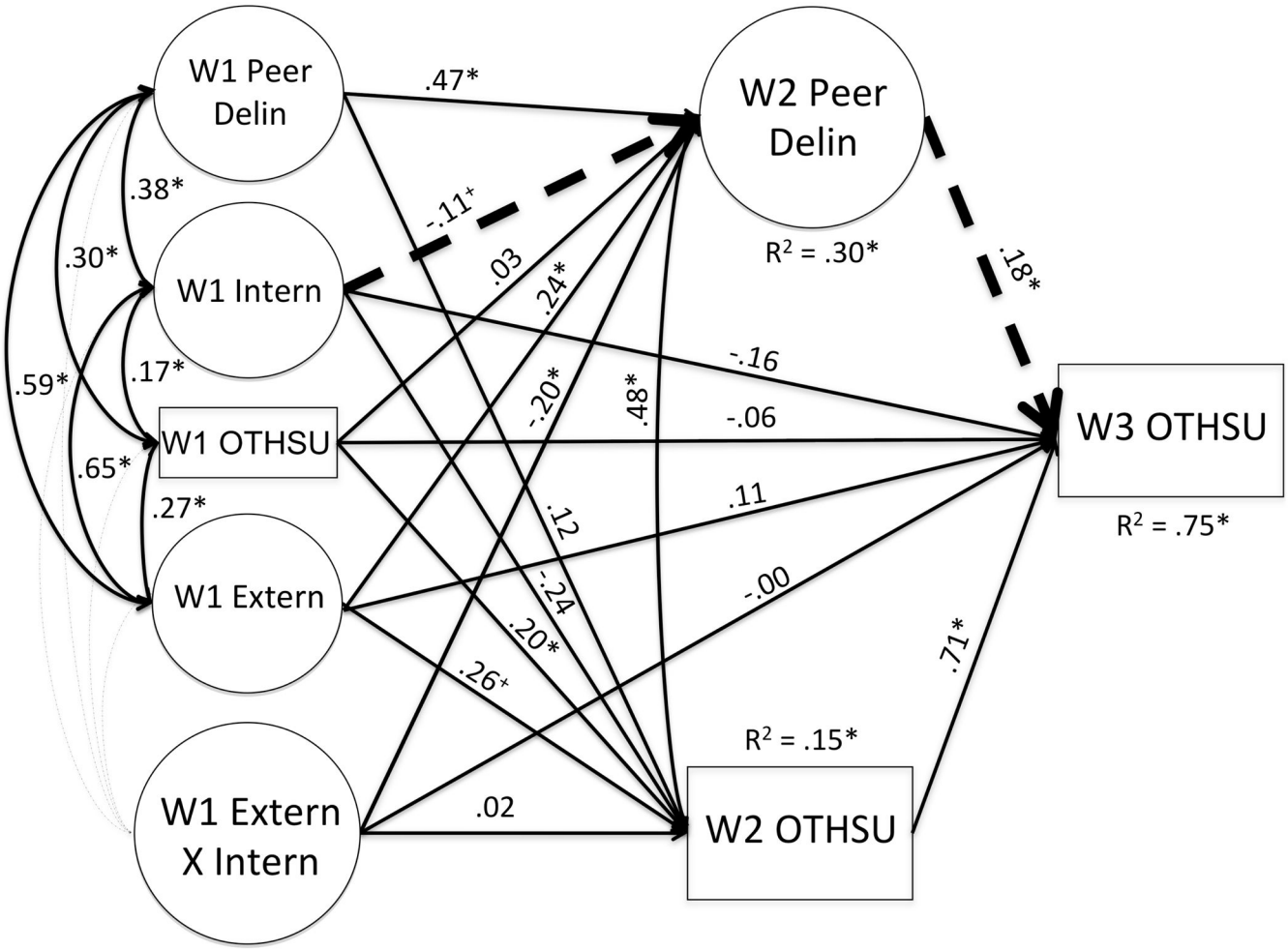

Figure 4.

Estimated Standardized Path Coefficients for the Full Model Predicting Other SU

Note. Age was left out for ease of presentation. * p < .05, + = p < .10. Intern = Internalizing factor, Extern = Externalizing factor, Extern x Intern = Internalizing x Externalizing interaction factor, Peer Delin = peer delinquency, OTHSU = other SU, and W = wave. Note that the interaction factor was allowed to correlate with exogenous variables in the model. Other SU consist of tobacco, marijuana, and illicit drug use. The pathway that is bolded and has a broken line represents the significant conditional indirect effect from W1 Internalizing problems to W3 Other SU through W2 Peer Delinquency and moderated by externalizing problems.

Table 1

Rates of Substance use by Wave

	Wave 1	Wave 2	Wave 3
Alcohol	4.1% (16)	22% (83)	33.3% (121)
Marijuana	0%	1.9% (7)	9.1% (33)
Tobacco	2.3% (9)	3.55% (13)	8.8% (32)
Other Illicit Use	.5% (2)	.5% (2)	1.9% (7)
OTHER SU	2.6% (10)	4.3% (16)	13.2% (38)

Note. OTHER SU is the rate of use when marijuana, tobacco, and illicit use are collapsed into one variable. Percentages at Wave 1 represent lifetime use, while percentages at Waves 2 and 3 represent past year use for each substance. The number in parentheses is the N who reported use. Total sample size = 387. The average age at Wave 1 = 12.1, at Wave 2 = 13.1, and at Wave 3 = 14.1.

Table 2

Correlation Matrix of the Latent Variables for the Main Analysis (Figure 2 and 4)

	1	2	3	4	5	6	7	8	9
1. Extern	1.00								
2. Intern	0.63	1.00							
3. In x Ex	0.58	0.40	1.00						
4. W1 Pdel	0.58	0.38	0.47	1.00					
5. W2 Pdel	0.36	0.14	0.01	0.49	1.00				
6. Age	0.06	0.06	−0.07	0.10	0.15	1.00			
7. W1 Alc	0.32	0.13	0.15	0.40	0.32	0.15	1.00		
8. W2 Alc	0.33	0.06	0.08	0.31	0.44	0.20	0.48	1.00	
9. W3 Alc	0.21	0.02	0.02	0.23	0.42	0.21	0.27	0.64	1.00
							7	8	9
7. W1 Oth SU	0.27	0.18	0.33	0.31	0.15	0.02	1.00		
8. W2 Oth SU	0.31	0.05	0.20	0.22	0.53	0.09	0.27	1.00	
9. W3 Oth SU	0.28	−0.01	0.14	0.28	0.60	0.23	0.21	0.80	1.00

Note. Intern = Internalizing factor, Extern = Externalizing factor, In x Ex = Internalizing x Externalizing interaction factor, Pdel = peer delinquency, Alc = alcohol, oth SU = other substance use, and W = wave