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# Reciprocal Relations between Internalizing Symptoms and Frequency of Alcohol Use: Findings from a Longitudinal Study of Mexican-origin Youth

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## Abstract

**Aims—**Alcohol consumption and internalizing symptoms, which often co-occur, pose considerable risk to the developing adolescent and have lasting public health consequences. Previous research has documented concurrent associations between alcohol use and symptoms of anxiety and depression, but the dearth of longitudinal research, particularly for ethnic minority youth, raises questions about the replicability and causal direction of these effects. The goal of the present research was to clarify these issues, and investigate whether different facets of anxiety and depression are uniquely associated with alcohol use in adolescence.

**Method—**The present research examined cross-lagged relations between frequency of alcohol use and internalizing symptoms, using data from a longitudinal study of 674 Mexican-origin youth (50% female) assessed at ages 14 and 16.

**Results—**Alcohol use at age 14 prospectively predicted increases in overall *internalizing symptoms*, and overall *internalizing symptoms* at age 14 prospectively predicted increases in alcohol use. Reciprocal effects were consistently found for the *general distress* and *anxious arousal* facets, but not for *anhedonic depression* and a scale measuring the cognitive aspects of *anxiety*.

**Conclusions—**The findings provide evidence of reciprocal relations between alcohol use and internalizing symptoms, but also highlight the danger of treating all symptoms of anxiety and depression as interchangeable components of a single broad domain. Instead, symptoms common to both anxiety and depressive disorders (e.g., general distress) have the most robust reciprocal relations with alcohol use. Thus, intervention programs aimed at reducing early alcohol use by Mexican-origin youth should target this component of the internalizing domain.

### Keywords

alcohol use; anxiety; depression; adolescence; longitudinal

Adolescence is a dynamic stage of development during which individuals transition into more challenging academic contexts, navigate increasingly complex peer and romantic

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relationships, and take on adult responsibilities, all while experiencing significant neuro-hormonal changes associated with puberty. These changes put adolescents at risk for myriad emotional and behavioral problems, known to markedly escalate during adolescence. Underage alcohol use, in particular, is increasingly common during adolescence (Johnston et al., 2014a), and contributes to risky sexual behavior, delinquency, school drop-out, and substance use disorders (Brown et al., 2009; Newcomb & Bentler, 1988; Trim et al., 2007).

Alcohol use is also highly comorbid with anxiety and depressive disorders (NIDA, 2010). Two theoretical models have been advanced to account for this comorbidity. The *self-medication model* posits that individuals with pre-existing internalizing symptoms use alcohol to cope with their emotional distress and soothe its physiological concomitants (Trim et al., 2007; Wolitzky-Taylor et al., 2012). Support for the prospective effect of internalizing symptoms on alcohol use has been mixed (Fergusson et al., 2009; Hallfors et al., 2005; Lansford et al., 2008; Maslowsky et al., 2014; Wolitzky-Taylor et al., 2012). The *alcohol-induced disruption model* posits that alcohol use leads to internalizing symptoms by: (a) impeding the development of effective coping and self-regulatory skills; (b) inducing neurotoxic effects that impair cognitive functioning, which creates school-related stress; and (c) alcohol use dysregulates reward mechanisms in the brain, causing adolescents to focus on acquiring alcohol rather than engaging in more adaptive activities that reduce risk for depression and anxiety (Brook et al., 2002; King et al., 2004; Trim et al., 2007). Few studies have tested these mediation pathways, and even the prospective effect of alcohol use on internalizing problems in adolescence is not well established, especially in non-clinical samples (Fröjd et al., 2011; Mason et al., 2008).

The present research examined cross-lagged relations between frequency of alcohol use and internalizing symptoms, using data from a longitudinal study of 674 Mexican-origin youth (50% female) assessed at ages 14 and 16. The study extends previous research in several ways. First, whereas most prior studies examined concurrent associations between alcohol use and internalizing symptoms, we used longitudinal data to examine reciprocal prospective relations and thus establish the temporal order of the effects. Second, our measure of internalizing problems assesses symptoms *unique* to anxiety and depression, as well as symptoms *common* to the disorders, allowing us to elucidate which specific aspects of anxiety and depression are more or less relevant to alcohol use. This is important because the effects of depression on alcohol use appear to be more robust than the effects of anxiety (Hallfors et al., 2005; Hussong & Chassin, 1994; Kaplow et al., 2010), but is difficult to draw conclusions from this literature because many measures of depression include some anxiety content and many measures of anxiety include some depressive content. Third, whereas many prior studies have used clinical samples, our community-based sample allows for more generalizable inferences about how alcohol use and anxiety/depression are associated during normative adolescent development. Finally, we focused on a specific ethnic minority group, Mexican-origin individuals, that remains understudied in the context of alcohol involvement and internalizing problems, despite their growing representation in the population (U.S. Census, 2014), their early onset of alcohol use (Johnston et al., 2014b), and their increased vulnerability to psychiatric and substance use disorders the longer they are exposed to American society (Vega et al., 1998; 2000).

Although the developmental pathways characterizing Mexican-origin and European-origin youth sometimes differ, in the present context we expected to find the same pattern of results observed in prior research. This expectation is based on the fact that the mediating mechanisms posited by the *self-medication* and *alcohol-induced disruption* models reflect basic processes that are unlikely to be culture specific; youth from all ethnic groups are likely to derive some self-medicating benefit from alcohol use and, similarly, alcohol use is likely to lead to impaired self-regulatory skills, neurotoxic effects on brain development, and dysregulated reward processes. Thus, we predicted that alcohol use and internalizing symptoms would be reciprocally related over time, such that alcohol use at age 14 would contribute to subsequent increases in anxiety/depressive symptoms from age 14 to 16, and anxiety and depressive symptoms at age 14 would contribute to subsequent increases in alcohol use from age 14 to 16.

Establishing the generalizability of these reciprocal associations to Mexican-origin youth has important implications. Theoretically, it would suggest that the developmental pathways linking these factors are not culture-specific but rather reflect more basic processes. Practically, it would suggest that intervention programs targeting these pathways (e.g., trying to reduce alcohol use by reducing internalizing problems or improving coping skills) are likely to be effective with Mexican-origin youth. In contrast, if our results differ from prior research, this would highlight the possibility of culture-specific developmental pathways, and suggest that factors unique to Mexican-origin youth exacerbate or diminish the link between internalizing symptoms and alcohol use. For example, the hypothesized pathways may be weaker for first-generation (vs. more acculturated) immigrants, given that first-generation youth are less likely to turn to substance use despite experiencing heightened levels of stress relative to latter generation youth (Garcia-Coll & Marks, 2011). To evaluate this possibility, we tested whether generational status moderates the association between internalizing symptoms and alcohol use. Finally, because the association between internalizing symptoms and alcohol use can be influenced by co-occurring externalizing symptoms (Colder et al., 2013), we also examined the effects of internalizing symptoms after controlling for a measure of externalizing problems.

## Method

### Participants and Procedures

The data come from the California Families Project, a longitudinal study of 674 (50% female) Mexican-origin youth from Norther California. The focal child had to be in the 5$^{th}$ grade, of Mexican origin, and living with his or her biological mother; of eligible families, 73% agreed to participate. The present study used data collected when the children were 14 and 16 years old, when the key measures were available. Of the original sample, 90% ($N$=605) were retained in the study at age 14 and 89% ($N$=600) at age 16. To investigate the potential impact of attrition, we compared individuals who did vs. did not participate in the age 14 and 16 assessments on Wave 1 demographic variables. Adolescents who dropped out of the study did not differ significantly from participating youth in gender, generational status, or family income, all $p$s > .05.

### Measures

**Internalizing symptoms—**At ages 14 and 16, participants completed the Mini-Mood and Anxiety Symptom Questionnaire (MASQ; Casillas & Clark, 2001). Based on Clark and Watson's (1991) tripartite model of anxiety and depression, this 26-item scale measures a general distress factor common to both anxiety and depression as well as specific anxiety and depression factors: General Distress (5 items; "How much have you felt discouraged"), Anhedonic Depression (8 items; "How much have you felt like nothing was very enjoyable"), Anxiety (3 items; "How much have you felt keyed up or on edge"), Anxious Arousal (10 items; "Have you had trouble swallowing"). Participants rated how much they "felt or experienced" each symptom "during the past week" using a 5-point scale at age 14 (1="Not at all"; 2="A little"; 3="Somewhat"; 4="A lot"; 5="Extremely") and a 4-point scale at age 16 (1="Not at all"; 2="A little"; 3="Somewhat"; 4="Very much").

**Frequency of alcohol use—**At ages 14 and 16, participants reported how many times in the past 3 months they used or tried beer; wine or wine coolers, and hard liquor ("more than just a few sips"), using a 5-point scale (1="Never"; 2="Less than once per week"; 3="About once per week"; 4="Two or three times per week"; 5="Almost every day or every day"). *Frequency of alcohol use* was computed by taking the mean of the three items ($M_{age14}$=1.12, $SD_{age14}$=.34, Range=1.00–3.67; $M_{age16}$=1.19, $SD_{age16}$=.45, Range=1.00–4.00). 16% of participants at age 14 and 23% at age 16 reported having tried beer, wine, or liquor at least once in the last 3 months. This is comparable to nationally representative data, where 12% of 14–15-year olds, and 25% of 16–17 year olds report having consumed at least 1 drink in the last 30 days (SAMHSA, 2014).

**Generational status—**Participants were categorized as 1$^{st}$ generation if they were born in Mexico (29%) and as 2$^{nd}$+ generation (71%) if they were born in the United States.

**Delinquency—**At age 14, participants completed a 15-item delinquency scale taken from the Add Health Study (e.g., "*In the past 12 months, how often did you steal something worth more than $50?*). Items were rated on a 4-point scale, ranging from 0 (*Never*) to 3 (*5 or more times*). We excluded one item that specifically asked about selling marijuana and drugs.

## Results

All analyses were conducted with Mplus using the robust maximum likelihood estimator (MLR) to account for non-normal distributions of variables and full information maximum likelihood (FIML) to account for missing data. Alcohol use was modeled as an observed variable, and internalizing symptoms and delinquency were modeled as latent variables. Three parcels of randomly selected items were used to define each latent variable: General Distress (λ's=.83–.91; ω reliability=.87 & .91 at ages 14 and 16, respectively), Anhedonic Depression (λ's=.69–.90; ω=.85 & .82), Anxiety (λ's=.58–.75; ω=.73 & .72), Anxious Arousal (λ's=.70–.85; ω=.84 & .81), and Delinquency (λ's=.73–.89; ω=.86). An Internalizing Symptoms factor was created using the MASQ subscales as indicators (λ's=.46–.91; ω=.65 & .73).

We conducted cross-lagged latent variable regression models to test our predictions (Table 1 shows inter-correlations among the internalizing latent variables). The cross-lagged paths indicate the prospective effect of one variable on the other (e.g., effect of alcohol use at age 14 on Anxiety at age 16), after controlling for concurrent relations between the variables (e.g., association between alcohol use and Anxiety at age 14) and stability over time (e.g., effect of alcohol use at age 14 on alcohol use at age 16; effect of Anxiety at age 14 on Anxiety at age 16). Model fit was satisfactory for all models, with CFIs > .96, TLIs > .94, and RMSEAs < .05.

Table 2 shows the path coefficients from the cross-lagged models. The stabilities for internalizing variables (.31 to .58) were comparable to prior research. Alcohol use was less stable, as expected given many youth are beginning to initiate alcohol use at this age. The cross-lagged effects support our hypothesis that more frequent alcohol use leads to increases in internalizing symptoms, and higher levels of internalizing symptoms lead to increases in alcohol use. This pattern held across all MASQ scales, except for alcohol use to Anxiety. The direction and magnitude of the effects were similar after controlling for Delinquency, but the reciprocal effects of Anhedonic Depression and the Anxiety to alcohol use effect became non-significant.

To test for moderator effects, we conducted multiple group analyses with equality constraints. Models with gender constraints on the cross-lagged paths did not fit significantly worse than freely estimated models (all chi-square difference tests n.s.), suggesting that there is no moderation by gender. Models constraining the cross-lagged paths to be equal for 1st and 2nd+ generation youth did not fit worse than freely estimated models, except for Anxious Arousal. Follow-up analyses showed that the alcohol→Anxious Arousal effect was stronger for 1st generation youth, but generational status did not moderate the Anxious Arousal→alcohol path.

## Discussion

Alcohol consumption and internalizing symptoms, which often co-occur, pose considerable risk to the developing adolescent and have lasting public health consequences (Dodge et al., 2009). The dearth of longitudinal research, particularly for ethnic minority youth, has left gaps in our understanding of how these two risk factors are associated across development. Specifically, although concurrent relations and comorbidity have been established, the degree to which these constructs are prospectively related over time, and the causal direction of the effects, remains unclear. Consequently, the literature has yielded mixed support for the theoretical models proposed to account for the link between internalizing problems and alcohol use. The goal of the present research was to clarify these issues, and investigate whether different facets of anxiety and depression are uniquely associated with alcohol use in adolescence.

At the broad level of internalizing problems, reciprocal cross-lagged associations with alcohol use were robust and held for boys and girls, for 1st and 2nd+ generation youth, and after controlling for gender, generational status, and delinquency. Similarly, the General Distress scale, which includes symptoms that are common to Anxiety and Depression, also

showed robust reciprocal relations with alcohol use that generalized across gender and generational status and held after controlling for all three control variables. However, when we delved *within* the internalizing domain, and examined scales that assess symptoms unique to anxiety and depression, we found several interesting differences. First, Anhedonic Depression, which assesses symptoms distinct to depression, had reciprocal associations with alcohol use in all cases except after controlling for delinquency, suggesting that the tendency for alcohol use to lead to Anhedonic Depression, and for Anhedonic Depression to lead to alcohol use, is confounded, at least in part, by the overlap between these constructs and delinquency. Second, the Anxiety scale, which assesses cognitive/experiential symptoms of anxiety, showed no evidence of the alcohol-induced disruption effect (alcohol use→Anxiety), and the self-medication effect (Anxiety→alcohol use) failed to reach significance when we controlled for delinquency, suggesting that this effect is also confounded by overlap with delinquency.

Third, in contrast to Anxiety, the Anxious Arousal scale, which assesses physical symptoms of anxiety, showed consistent reciprocal associations with alcohol use, even after controlling for delinquency. The differential effects of Anxiety vs. Anxious Arousal on alcohol use may reflect the fact that alcohol is a sedative and thus more directly alleviates the physiological symptoms of anxiety. With regard to the *alcohol disruption pathway*, opponent-process theory (Solomon, 1980) suggests that alcohol use may lead to increases in the physiological symptoms of anxiety as the central nervous system reacts to the sedative effects of alcohol by ramping up physiological arousal to bring the body back to homeostasis. However, these interpretations are highly speculative and future research should rule out the possibility that the Anxiety effects were weaker simply because the latent factor was less reliable.

Finally, contrary to expectation, the effect of alcohol use on Anxious Arousal was stronger for $1^{st}$ (vs. $2^{nd}$+) generation immigrants. It is possible that in recent immigrant families, the physical symptoms of anxiety are less likely to be acknowledged by youth and their families and less likely to be treated due to the stigma attached to using mental health services (Chang et al., 2013; Orozco et al., 2013), leaving alcohol as the only available treatment. A related possibility is that Mexican-origin youth who engage in alcohol use are more likely to experience physical anxiety related to acculturative stress because they have difficulty coping with the conflicting demands of fulfilling the Mexican cultural value of familism–a focus on family harmony and prioritizing the family above of one's personal needs, which tends to prevent youth from seeking mental health services–and fitting in with deviant peer groups that tend to be more acculturated and focused on personal gratification and rejection of family values.

Together, these findings highlight the danger of treating all symptoms of anxiety and depression as interchangeable components of a broader internalizing domain. Instead, our findings suggest that it is the shared anxiety and depressive content that has the most robust reciprocal relations with alcohol use. This pattern places prior research in a new light. Specifically, perhaps the link between depression and alcohol use is more replicable because commonly used depression measures are more likely to include anxiety symptomatology (and therefore capture the overlapping domain), whereas commonly used anxiety measures are less likely to include depressive symptomatology.

Notwithstanding these important differences among the facets of internalizing problems, the overall pattern of findings supports both the *self-medication* and the *alcohol-induced disruption* models. That is, adolescent alcohol use was both a developmental precursor to and a consequence of internalizing symptoms, especially symptoms common to anxiety and depression, in our large, community-based sample of Mexican-origin youth. These findings extend prior research by illustrating prospective reciprocal effects between normative adolescent drinking patterns and subclinical internalizing symptoms. Although the effect sizes were small, the associations observed in the present study may have substantial and lasting consequences through developmental processes that accumulate across adolescence and young adulthood.

Although further research is needed to clarify the mediating processes, a growing literature, including the present study, clearly establishes important implications for interventions. A harm-reduction intervention model that aims to reduce teenage drinking may significantly reduce not only alcohol use, but also future anxiety/depressive symptoms. Similarly, early identification and treatment of anxiety/depressive symptoms in adolescents, including subclinical groups, may reduce the likelihood of youth turning to alcohol as a means of self-medication. Given the current findings, our study proposes the *self-medication* and *alcohol-induced disruptions models* do not have to be positioned against one another. Rather, adolescents may benefit from interventions that integrate both developmental models.

One limitation of the present study is the two-year time interval. Although relatively long compared to some previous longitudinal studies on the topic, this interval is not sufficient to understand the role of more distal influences, such as early emerging anxiety problems or poor parental monitoring during childhood. A second limitation is the reliance on self-reports. Although youth reports are a well-validated method to assess alcohol use and anxiety/depression symptoms, it would be informative to replicate the findings using peer/parent reports and drug testing based on biological samples. Despite these limitations, the present study provides important insights into the etiology of alcohol use in an understudied ethnic minority group. Specifically, we found reciprocal relations between alcohol use and internalizing symptoms in our sample of Mexican-origin youth, supporting both the *self-medication* and *alcohol-induced disruption* models. Thus, interventions aimed at reducing early alcohol use are likely to decrease risk for internalizing symptoms and, conversely, interventions aimed at reducing early emerging internalizing symptoms are likely to decrease subsequent increases in adolescent alcohol use.

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**Table 1**

Intercorrelations among Internalizing Latent Factor Subscales

| | Age 14 | | | | Age 16 | | | |
|---|---|---|---|---|---|---|---|---|
| | **Anxiety** | **Anxious Arousal** | **Anhedonic Depression** | **General Distress** | **Anxiety** | **Anxious Arousal** | **Anhedonic Depression** | **General Distress** |
| Age 14 | | | | | | | | |
| Anxiety | 1.00 | - | - | - | - | - | - | - |
| Anxious Arousal | .33 | 1.00 | - | - | - | - | - | - |
| Anhedonic Depression | .14 | .20 | 1.00 | - | - | - | - | - |
| General Distress | .45 | .50 | .41 | 1.00 | - | - | - | - |
| Age 16 | | | | | | | | |
| Anxiety | .31 | .25 | .16 | .25 | 1.00 | - | - | - |
| Anxious Arousal | .30 | .40 | .20 | .39 | .45 | 1.00 | - | - |
| Anhedonic Depression | .14 | .26 | .43 | .34 | .27 | .37 | 1.00 | - |
| General Distress | .27 | .34 | .29 | .48 | .47 | .57 | .67 | 1.00 |

*Note.* $N = 620$. All correlations are significant at $p < .05$.

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## Table 2

Cross-Lagged Relations between Frequency of Alcohol Use and Internalizing Symptoms

| | Cross-lagged effects | | Stability effects | |
|---|---|---|---|---|
| **Internalizing symptoms** | **Alcohol→ Internalizing** | **Internalizing→ Alcohol** | **Alcohol use** | **Internalizing symptoms** |
| MASQ Total scale | .15* (.14*) | .12* (.12*) | .23* | .58* |
| General Distress | .09* (.08*) | .16* (.14*) | .21* | .48* |
| Anhedonic Depression | .05* (.04) | .08* (.08) | .23* | .43* |
| Anxiety | .03 (.02) | .09* (.06) | .23* | .31* |
| Anxious Arousal | .15* (.10*) | .07* (.05*) | .24* | .38* |

*Note*. *N* = 620. Values in the table are standardized estimates of structural coefficients in bivariate models controlling for gender and generational status. Values in parentheses are structural coefficients after controlling for gender, generational status, and delinquency. Alcohol = frequency of alcohol use; Internalizing = internalizing symptoms; MASQ = Mini-Mood and Anxiety Symptom Questionnaire.

* $p < .05$.