Published in final edited form as:

Drug Alcohol Depend. 2013 September 1; 132(0): 149–157. doi:10.1016/j.drugalcdep.2013.01.017.

Alcohol and Tobacco Use Disorder Comorbidity in Young Adults and the Influence of Romantic Partner Environments

Meredith C. Meacham, Jennifer A. Bailey, Karl G. Hill, Marina Epstein, and J. David Hawkins

Social Development Research Group, School of Social Work, University of Washington

Abstract

Background—Although there is considerable evidence that the development of tobacco dependence (TD) and that of alcohol use disorder (AUD) are intertwined, less is known about the comorbid development of these disorders. The present study examines tobacco dependence and alcohol use disorder comorbidity in young adulthood within the context of romantic partner relationships.

Methods—Data were drawn from the Seattle Social Development Project, a contemporary, ethnically diverse, and gender balanced longitudinal panel including 808 participants. A typological person-centered approach was used to assign participants to four outcome groups: no disorder, tobacco dependence (TD) only, alcohol use disorder (AUD) only, and comorbid (both). Multinomial logistic regression was used to determine the association between partner general and substance-specific environments and single or dual alcohol and tobacco use disorder diagnosis in young adulthood (ages 24–33, $n = 628$). Previous heavy alcohol and tobacco use were controlled for, as were dispositional characteristics, gender, ethnicity, adult SES, and adult depression.

Results—Greater partner conflict increased the likelihood of being comorbid compared to having TD only or AUD only. Having a smoking partner increased the likelihood of being comorbid compared to having AUD only, but having a drinking partner did not significantly distinguish being comorbid from having TD only.

Conclusions—Findings demonstrated the utility of a comorbidity-based, person-centered approach and the influence of general and tobacco-specific, but not alcohol-specific, partner environments on comorbid alcohol and tobacco use disorders in young adulthood.

© 2013 Elsevier Ireland Ltd. All rights reserved.

Correspondence concerning this article should be addressed to Meredith Meacham, Social Development Research Group, University of Washington, 9725 3rd Ave. N.E., Suite 401, Seattle, WA 98115; mmeacham@uw.edu; Phone: 206-685-1997; Fax: 206-543-4507.. Respective email addresses of coauthors are: jabailey@uw.edu, khill@uw.edu, marinaep@uw.edu, jdh@uw.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Contributors

Drs. Bailey and Hill designed the study. Ms. Meacham implemented the analyses and was the primary writer. Drs. Bailey, Hill, Epstein, and Hawkins consulted on analyses and contributed to the writing of the manuscript. All authors have contributed to the study and have read and approved the final manuscript.

Conflict of Interest

No conflict declared

Keywords

comorbidity; alcohol use disorder; tobacco dependence; romantic partner; nicotine; partner conflict

1. INTRODUCTION

The health and societal consequences of tobacco dependence and alcohol abuse and dependence are well known. Tobacco smoking and excessive alcohol consumption are both leading contributors to national and global morbidity and mortality (Drobes, 2002; Falk et al., 2006; Mokdad et al., 2004). When used together, heavy alcohol and tobacco use increase the risk for certain cancers and cardiovascular disease (Drobes, 2002; National Institute on Alcohol Abuse and Alcoholism, 1998).

There is considerable evidence that the development of tobacco dependence (TD) and that of alcohol use disorder (AUD) are intertwined (Chen and Kandel, 1995; Jackson et al., 2003; John et al., 2003; Sher et al., 1996). Although some people who become tobacco dependent do not develop alcohol use disorder, and vice versa, overall, drinkers are more likely to smoke and smokers are more likely to drink, and individuals who are dependent on one substance are often dependent on the other (Drobes, 2002; Le Strat et al., 2010). Furthermore, a common set of environmental risk and protective factors influence early initiation of both substances (Hawkins et al., 1992), and early initiation of smoking and drinking has been shown to significantly increase the probability of developing tobacco and alcohol use disorders in adulthood. As individuals transition from adolescence into young adulthood, when tobacco and alcohol use disorders tend to peak (Substance Abuse and Mental Health Services Administration, 2004), a dominant environmental influence is romantic partner relationships (Daley and Hammen, 2002; Meier and Allen, 2009).

The present study examines tobacco dependence and alcohol use disorder comorbidity within the context of romantic partner relationships; specifically, this study examines how the quality of one's relationship with a romantic partner and their partner's substance use at ages 21–24 influence the degree to which individuals develop both TD and AUD vs. only one disorder from ages 24–33.

1.1 Typological approach to comorbidity

Research on alcohol and tobacco use disorders has traditionally approached these disorders and treated users as homogenous groups, yet researchers have suggested that there may be qualitatively different subtypes of individuals with alcohol and tobacco use disorders and that these subtypes may have different etiologies (Babor et al., 1992; Jackson et al., 2000). Despite burgeoning research on the biological mechanisms of addiction, the lack of consistent findings in genetic research on these disorders may be due in part to the heterogeneity of substance use disorders. Conway and colleagues (2006) called for the development of more useful and complex phenotypes that consider, among other things, polysubstance abuse, developmental processes, individual characteristics, and environmental factors.

Typologies provide a person-centered approach to identifying different groups of disorder subtypes or phenotypes. Person-centered approaches identify groups or subtypes of individuals with shared attributes and assume population heterogeneity with respect to how variables influence outcomes (Laursen and Hoff, 2006). The typological approach may be contrasted with variable-centered approaches that seek to develop a model of overall sample behavior and identify processes similar to all members of the group. Such approaches may be less applicable when there are different types of disorders within the sample (Hill et al., 2000; Laursen and Hoff, 2006).

In this study, we build on a typological latent class approach of Jackson et al. (2000), which found a comorbid alcohol and tobacco use disorder class. We describe potential subtypes of alcohol and tobacco use disorders that focus on the comorbid cumulative prevalence of these disorders, apply these groupings to the behavior of young adults after the peak years of substance use initiation and during peak years of substance abuse, and utilize romantic partner environments to examine different etiologies for these subtypes.

1.2. General and specific influences

Geneticists and social scientists have demonstrated that the development of AUD and TD disorders is influenced by a combination of environmental and genetic risk factors (Kreek et al., 2005; Rutter et al., 2006). Furthermore, these factors may be substance specific or may represent a common or shared vulnerability to substance use disorders (Jackson et al., 2009; Tsuang et al., 1998). Environmental predictors of substance use problems have likewise been shown to be general and substance specific, especially in the family context (Catalano and Hawkins, 1996; Hawkins et al., 1992; Bierut et al., 1998).

In adulthood, romantic relationships become increasingly influential, often succeeding that of the family of origin (Bachman et al., 1997, 2002; Catalano and Hawkins, 1996; Daley and Hammen, 2002; Meier and Allen, 2009). The quality of a romantic relationship and partner substance use have both emerged as important factors associated with the escalation of substance use as well as the development of substance use disorders (Catalano and Hawkins, 1996; Fleming et al., 2010; Homish and Leonard, 2005; Homish et al., 2006; Horwitz and White, 1991; Rhule-Louie and McMahon, 2007; Kearns-Bodkin and Leonard, 2005). Thus, partner relationship quality may constitute a general influence on young adult AUD and TD, whereas partner drinking and smoking may constitute substance-specific influences on young adult AUD and TD, respectively.

1.3. Mechanisms of partner behavior similarity

Two primary mechanisms explaining romantic partner similarity in substance use and problem behavior have been hypothesized: assortative mating and behavior contagion (Rhule-Louie and McMahon, 2007). Under the assortative mating mechanism, people select partners with similar characteristics, implying non-random relationship formation. For example, someone who drinks heavily is more likely to spend time with other heavy drinkers and so partner with another heavy drinker. Under the behavior contagion mechanism, individuals' experiences subsequent to relationship formation lead to behavioral similarity, implying a more causal association between partners' behaviors, e.g., someone

who drinks only occasionally would end up drinking more heavily if partnered with a heavy drinker.

Although the literature on antisocial behavior similarity in partners tends to show stronger evidence for assortative mating, studies that control for previous behavior tend to find evidence for behavior contagion (Leonard and Das Eiden, 1999; Leonard and Mudar, 2003; Rhule-Louie and McMahon, 2007). The relative influence of these mechanisms may also vary by behavior type, and so are likely to differ from crime to illicit substance use to licit substance use to substance use disorders (Merline et al., 2008). By including information on previous behavior, the influence of their partner's behavior above the individual's previous behavior can be examined further. The availability of longitudinal data in the present study allows us to control for previous heavy use in adolescence so that any effect of the partner's behavior is more likely to be causal and reflect behavior contagion, rather than a secondary result of similar background or traits selected through assortative mating.

1.4. Other influences on the development of AUD and TD

Dispositional characteristics, demographics, and mental health status are also known to be associated with the development of alcohol and tobacco use disorders. Behavioral disinhibition, an externalizing tendency toward impulsivity and novelty seeking despite negative consequences or societal disapproval, has been associated with early initiation of tobacco and alcohol use, the development of alcohol use disorders, and problem behaviors in general (Hill et al., 2010, 2000; Iacono et al., 1999, 2008; Sher and Trull, 1994). Although findings are less consistent, inhibition and internalizing traits (Leonard and Das Eiden, 1999) such as anxiety and poor reactions to stress have been associated with the development of alcohol and tobacco use disorders as individuals attempt to medicate or minimize these internal states with alcohol or tobacco use (Clark and Sayette, 1993; Hill et al., 2010; Khantzian and Albanese, 2008; Swendsen et al., 2000).

In general, males are more likely to drink in larger quantities and to develop alcohol use disorders than are women (Hasin et al., 2007; Naimi et al., 2003), and individuals with lower socioeconomic status (SES) are more likely to smoke and develop tobacco dependence (Centers for Disease Control and Prevention, 2010). Research on substance use disorders has shown that individuals with major depressive episode are also more likely to use alcohol and tobacco and develop use disorders (Grant and Harford, 1995; Tully et al., 2010). Therefore, the present study considers these other influences when investigating the role of partners.

This work extends previous research by investigating factors that shape the development of alcohol, tobacco, and comorbid substance use disorders during young adulthood. Specifically, the impact of the quality of romantic relationship and romantic partner alcohol and tobacco use are examined over and above enduring person-level characteristics, demographics, and adolescent substance use. Accordingly, this investigation was guided by the following research questions and hypotheses:

1. To what extent do general and substance-specific partner environments increase the likelihood of being comorbid for both substance use disorders?

- a. We expected that general negative partner environment, operationalized as partner conflict, would be associated with increased likelihood of later having both substance use disorders compared to having only one.
 - b. We expected that substance-specific partner environment, operationalized as partner regular use of alcohol or tobacco, would be associated with increased likelihood of later having both substance use disorders compared to having only one.
2. Is there evidence of contagion effects of a partner's behavior, after controlling for adolescent substance use, on comorbidity in later young adulthood? We expected that the effect of partner influences on comorbidity would be attenuated, but would remain significant.
3. Do the effects of partner environments remain after controlling for demographic variables and adult confounders? In line with previous literature in this area, we expected that general baseline risk factors such as male gender, lower SES, and externalizing or internalizing traits, and adulthood risk factors of low income and depression, would be associated with increased likelihood of having both substance use disorders, but that the partner level effects would remain significant even after adjusting for these risk factors.

2. METHODS

2.1. Participants and procedures

Participants in this study were drawn from the Seattle Social Development Project (SSDP), a longitudinal study of the development of positive and antisocial behaviors. Sample members were recruited in the fall of 1985 from all fifth-grade students attending 18 Seattle elementary schools serving both high-crime and low-crime neighborhoods. The original study sample of 808 students was gender balanced and reflected the ethnic mix of the Seattle area at that time: 412 (51%) participants were male; 381 (47%) were White, 207 (26%) were African American, 177 (22%) were Asian American and 43 (5%) were Native American. A substantial proportion of participants were from low-income households – 52% of the participants qualified for the National School Lunch Program in fifth, sixth, or seventh grade.

Classroom assessments were conducted with students in the fall of fifth grade (age 10), and at age 11, and face-to-face interviews were conducted once a year from ages 12 to 16, at age 18, and at ages 21, 24, 27, 30, and 33. A parent, teacher, and child skills training nonrandomized intervention was conducted in elementary school (Hawkins et al., 1999). Parents provided written consent and youth provided assent for data collected before age 18. After age 18, youth provided consent at each wave. Data collection procedures were approved by the University of Washington Institutional Review Board. This analysis examined data collected from ages 14 through 18, and at ages 21, 24, 27, 30, and 33.

Despite high levels of retention through young adulthood (over 91% for each wave), diagnostic criteria for at least one substance were not available for 33 individuals (4.1%) at

any of the four waves due to attrition, refusal to answer, or death. One-hundred and thirty individuals who did not have a romantic partner at age 21 or 24, and an additional 17 who were missing data on at least one covariate were excluded from the analysis, leaving a sample size of 628. The analysis sample was not significantly different from the full sample with respect to demographics, disorder prevalence, or other measured variables.

2.2. Measures

2.2.1. Alcohol and tobacco use disorder types (ages 24–33)—DSM-IV tobacco (nicotine) dependence and alcohol abuse and dependence diagnostic criteria (American Psychiatric Association, 1994) for the previous year were assessed at ages 24, 27, 30, and 33 using the Diagnostic Interview Schedule (Robins et al., 1981). Classification of alcohol or tobacco dependence requires meeting three or more of seven criteria and classification of alcohol abuse requires meeting one of four criteria. Only five of the seven criteria for tobacco dependence were assessed at age 24 (tolerance and using larger amounts than intended were not assessed), so while tobacco dependence may be under-diagnosed, it is much more stable than alcohol use disorder in both our sample and national samples (Falk et al., 2006), and it is likely that those who would have been diagnosed if the other two criteria had been assessed at age 24 would have met criteria for diagnosis at later ages.

After coding individuals as meeting diagnosis for AUD, TD, or neither at each wave, we categorized participants into four groups based on meeting use disorder diagnosis at any of the waves at ages 24, 27, 30, or 33: TD only, AUD only, comorbid (both), and no disorder (neither). Individuals who met diagnosis for both AUD and TD but in different years were classified as comorbid because we were interested in any cumulative occurrence of either disorder in young adulthood.

2.2.2. Partner environment (ages 21–24)—Participants were asked at ages 21 and 24 whether they were currently in a relationship (married, cohabiting, engaged, or exclusively dating) or had been in a relationship in the past year. They were then asked about their partner's substance use at ages 21 and 24 and frequency of conflict with their partner at age 24.

General partner environmental influence was operationalized by partner conflict. Using an abbreviated version of the *Dyadic Adjustment Scale* (Spanier, 1976; Spanier and Thompson, 1982), partner conflict included five items regarding frequency of talk about separating, regrets, quarrels, and an overall assessment of the relationship. In addition, participants reported the frequency of disagreement with their partner on six issues including sex, goals, and philosophy of life. Response options ranged from 1 *always agree* to 6 *always disagree*, and responses were averaged into a single disagreement item. Participants also indicated from 1 *all the time* to 6 *never* how often they discussed separating from their partner, quarreled, regretted the relationship, or (reverse coded) got along. Responses were standardized, then averaged across waves for each item and across items (Cronbach's alpha across items = .82).

At ages 21 and 24, participants reported whether their partner regularly drank or smoked cigarettes (each coded as 0 *no* and 1 *yes*). For each substance, we took the maximum value

across the two waves to create a dichotomous measure indicating whether each participant had a partner who regularly drank or a partner who regularly smoked cigarettes in early young adulthood.

2.2.3. Previous substance use (ages 14–18)—At ages 14 to 16 and 18, participants reported their past-month drinking, binge drinking (five or more drinks in a row), and daily smoking frequency (none, less than 1 cigarette, 1–5 cigarettes, half a pack, a pack). Two dichotomous variables were created indicating any past-month binge drinking and daily smoking of at least one cigarette a day at each wave (each coded as 0 *no* and 1 *yes*), as we were most interested in controlling for heavy adolescent use. The sums of values across these four adolescent waves were used to indicate the number of years the participant reported heavy drinking or smoking in adolescence.

2.2.4. Dispositional characteristics: externalizing and internalizing (ages 14–18)—Behavioral Disinhibition was self-reported at ages 14–16 and 18. Five items assessed participants' frequency of sensation seeking and externalizing activities and impulses (adapted from Carver and White, 1994). Items included statements such as “How often do you shock people just for the fun of it?” and “How often do you do what feels good regardless of the consequences?” Responses ranged from 1 *never* to 6 *once a week or more* and were standardized and averaged across items and across waves (Cronbach's alpha for items averaged over four waves = .82, Cronbach's alpha at each wave = .76–.77). Inhibition/anxiety was self-reported between ages 14–16 using six items drawn from the Child Behavior Checklist-Self-Report (Lengua et al., 2001). Items included statements such as “I am nervous or tense” and “I am too fearful or anxious.” Response options ranged from 1 *not true* to 3 *often true*, and were standardized and averaged across items and across waves (Cronbach's alpha of items averaged over three waves = .76, Cronbach's alpha at each wave = .74–.77).

2.2.5. Depression (ages 24–33)—Participants were assessed at each adult wave for major depressive episode in the previous year using the Diagnostic Interview Schedule (Robins et al., 1981). Classification of depression required meeting five of nine DSM-IV criteria over a 2-week period (American Psychiatric Association, 1994). A count variable was created indicating the number of years the participant met DSM-IV criteria for depression at ages 24, 27, 30, or 33.

2.2.6. Demographics—Gender and ethnicity were self-reported. Participants self-reported their annual household income and household size at ages 24, 27, 30, and 33. Annual household income was then divided by the assessment year's weighted average poverty thresholds established by the U.S. Department of Health and Human Services for the given household size, resulting in an income-to-needs ratio. This ratio is used to determine eligibility for government assistance; a ratio below 1.0 indicates a household falling below the poverty line. The maximum income-to-needs ratio across the four waves was used, because average ratios or ratios at a particular age during young adulthood may have been influenced by college attendance and job mobility. A higher income-to-needs ratio indicated higher SES.

2.3. Analytic method

To test whether partner environment distinguished comorbid AUD and TD from non-comorbid AUD or TD, we conducted sequential multinomial logistic regressions, with comorbid for both disorders as the reference group, using SPSS version 16.0. Although the reference group would conventionally be the largest group (in this case, no disorder), we were more interested in what distinguished one disorder from both disorders than what distinguished no disorder from any disorder. Analyses were conducted in a sequence of four steps to assess unique influences of romantic partner environment and to control for potential adolescent and adult confounders. First, early adult partner influences on comorbidity were examined. In the second step, baseline substance use in adolescence was added to examine potential partner selection effects and to control for prior substance use. In the third step, gender, ethnicity, adolescent behavioral disinhibition, and adolescent inhibition/anxiety were added to control for potential adolescent confounds. Finally, income-to-needs ratio and depression were added in the fourth step to control for potential adult confounds.

3. RESULTS

3.1. Descriptive statistics

Out of the 628 participants in this analysis, 14.6% ($n = 92$) met diagnosis for both tobacco and alcohol use disorders, 9.9% ($n = 62$) met diagnosis for tobacco dependence only, 22.9% ($n = 144$) met diagnosis for alcohol use disorder only, and 52.5% ($n = 330$) met neither diagnosis. Table 1 displays descriptive information on key study variables. Of the comorbid individuals, the majority (75.0%) met diagnosis for both disorders in the same year, and the remaining 25.0% met both diagnoses in different years. About two thirds of the participants reported having a drinking partner and about two fifths reported having a smoking partner at age 21 and/or age 24. Participants reported low to moderate levels of partner conflict, with adequate variability. During adolescence, over a quarter of the participants engaged in past-month binge drinking and slightly fewer were ever daily smokers.

3.2. Zero-order correlations

Addressing the primary research question, as shown in Table 2, partner conflict, smoking, and drinking were all significantly correlated with comorbidity and negatively correlated with no disorder. Positive correlations between partner smoking and TD only and between partner drinking and AUD only support the substance-specific environmental relationship between partner substance use and disorder outcomes. Adolescent heavy smoking and drinking, however, were also correlated with partner drinking and smoking, and with all outcome groups (except adolescent daily smoking with AUD only) indicating relationships between both one's adolescent substance use and their partner's substance use, as well as their partner's substance use and later development of substance use disorders. Of the other potential confounding variables, all were significantly correlated with at least one outcome group and at least one partner variable. In particular, behavioral disinhibition was relatively strongly correlated with AUD only and comorbid outcome groups and with most covariates.

3.3. Sequential regression analyses

Our first hypothesis was that general and substance-specific partner influences would increase the likelihood of comorbidity for both alcohol and tobacco use disorders compared to having a single disorder. Accordingly, we regressed the nominal outcome variable consisting of four diagnostic outcome groups (no disorder, AUD only, TD only, comorbid) on partner conflict, smoking, and drinking. Consistent with hypothesis 1a, partner conflict significantly differentiated no disorder, TD only, and AUD only from the comorbid group. Participants with greater partner conflict had lower odds of having one disorder than being comorbid. Also supporting hypothesis 1b, partner smoking significantly differentiated no disorder and AUD only from the comorbid group. Participants who reported having a smoking partner were less likely to meet criteria for AUD than meet criteria for both alcohol and tobacco use disorders. Contrary to hypothesis 1b, partner drinking did not significantly differentiate these groups. Estimates from the sequential multinomial logistic regression are shown in Table 3.

Hypothesis 2 examined whether the effects of partner environment would remain after controlling for previous heavy substance use. By controlling for heavy use of alcohol and tobacco in high school, we sought to examine potential contagion effects of the partner's behavior on the occurrence of alcohol and tobacco use disorders, and to control for prior substance use. Accordingly, as the second step in the regression, we added binge drinking and daily smoking in adolescence to the previous analysis. The effects for general and substance-specific partner seen in Step 1 remained, with partner conflict associated with lower odds of a single disorder or no disorder compared to being comorbid. Partner smoking continued to be associated with lower odds of AUD only or no disorder compared to comorbidity. After controlling for previous substance use, the effect of partner conflict increased slightly, while the effect of partner smoking was slightly attenuated. Adolescent daily smoking significantly differentiated AUD only from the comorbid group and no disorder from the comorbid group, but adolescent binge drinking only differentiated no disorder from the comorbid group. These results indicate that a partner's own substance use contributes to one's later comorbid tobacco and alcohol use disorder and is not just a proxy for one's prior substance use and partner selection.

Hypothesis 3 examined whether the observed partner effects remained after additionally controlling for demographics, dispositional characteristics, and adult depression. The third step in the regression analyses included baseline dispositional characteristics (behavioral disinhibition and inhibition/anxiety) and demographics (gender and ethnicity) in addition to the variables tested in steps 1 and 2. Results indicate that the pattern of associations with partner influences remain largely the same after the inclusion of adolescent and demographic control variables. In addition, being male significantly differentiated TD only and no disorder from the comorbid group, and inhibition/anxiety significantly differentiated AUD only from the comorbid group. Ethnicity and behavioral disinhibition did not significantly distinguish these groups.

The fourth and final step included adult income and depression (Table 3) in order to test for adult confounds. The pattern of results concerning partner influences remained unchanged, except that the effect of partner conflict was reduced to marginal significance in

differentiating AUD only from comorbidity. Depression significantly differentiated being comorbid and having AUD only or no disorder, and income significantly differentiated being comorbid and having TD only. Inhibition/anxiety and adolescent daily smoking also significantly differentiated AUD only from being comorbid. Note that all partner and previous use variables of interest differentiate having no disorder from being comorbid, with the exception of partner drinking.

4. DISCUSSION

This study sought to examine general and substance-specific romantic partner environmental influences on the comorbidity of alcohol and tobacco use disorders. Findings demonstrated the utility of a person-centered approach and the influence of general and tobacco-specific, but not alcohol-specific, partner environments on comorbid alcohol and tobacco use disorders in young adulthood.

In this longitudinal panel we found 10 year cumulative prevalences of 22.9% with AUD only, 9.9% with TD only, and 14.6% who were comorbid for both disorders. Rates of these disorders in our sample were higher than reported past year prevalences and lower than reported lifetime prevalences from national studies (Falk et al., 2006; Kessler et al., 2005a, 2005b). This is likely because our sample is still fairly young, and our rates are based on repeated, one-year snapshots as opposed to true lifetime measures. The high rates of adolescent smoking may be because this was a high risk sample that went through high school in the 1990s, when U.S. rates of adolescent smoking peaked. Compared to the latent class typology of Jackson et al. (2000), we had higher rates of comorbidity (15% vs. 7%), but similar rates of TD (10% vs. 11%) and of AUD if the chronic and developmentally limited AUD groups are combined (23% vs. 22%). Our higher rates of alcohol use disorder and comorbidity may be attributed to our sample being a high-risk one, and the fact that we did not require AUD and TD to co-occur in the same year to be considered comorbid.

By using a typological person-centered approach, we could classify a priori a group of individuals with both AUD and TD who would not be otherwise identified in variable-centered approaches that predict only one disorder or behavior. This approach also allowed for better understanding of how specific risk factors differ for this comorbid group compared to the single-disorder groups. While partner smoking, a risk factor for developing TD, was a significant predictor for having TD in addition to AUD, partner regular drinking, a risk factor for developing AUD, was not a significant predictor for having AUD in addition to TD. Furthermore, although studies have shown that poor relationship quality is a risk factor for single substance use disorders, we found that it is also a significant predictor for having both of these substance use disorders compared to having only one disorder.

4.1. General and specific partner influences

The first major finding of this study provides evidence of general and substance-specific environmental influences in young adulthood. In accordance with study hypotheses, results indicated that both general and substance-specific adult partner environmental influences distinguished individuals who met criteria for both AUD and TD and those who only met

criteria for one disorder. Partner conflict and partner's regular use of tobacco increased the odds of being in the comorbid group over having one disorder, suggesting that relationship quality can influence the common or shared risk for multiple substance abuse and dependence. Although partner relationships are often thought of as protective against substance abuse, our results show that poor relationship quality, operationalized as partner conflict, increases the likelihood of being comorbid for both disorders over having a single disorder. Thus, future research should consider not only whether individuals are partnered, but also the quality of partnered relationships.

Our results also show evidence for substance-specific influences of partner environment, as partner smoking was a significant predictor of comorbid AUD and TD relative to having AUD only. These findings align with other research on common versus specific liabilities for substance use and abuse (Bailey et al., 2011; Jackson et al., 2009), pointing to a common vulnerability model, where underlying environmental and genetic risks, such as sensation-seeking personality and variation in biological substrates in the reward system, increase the likelihood of substance abuse and dependence. In this model, other substance-specific risks uniquely increase the likelihood of substance abuse and dependence on that substance alone.

There are several possible reasons why we did not find effects of partner regular drinking. First, because two thirds of participants reported having a partner who regularly drank alcohol at ages 21 or 24, when alcohol consumption is normative, this reduced variability in the partner alcohol measure may have lowered statistical power. Another reason may be the more isolated and discrete nature of alcohol use, compared with the more pervasive and perceptible nature of smoking, so a partner's drinking may not be as influential as a partner's smoking. It is also well known that tobacco use has different physiological effects that are more “addictive” and enduring. Finally, the social norms and policies surrounding use and heavy use vary by substance and developmental period (Hopkins et al., 2001; Jackson et al., 2009; Wagenaar et al., 2005).

4.2. Behavior contagion

The second major finding of this study adds to the body of research on contagion effects in partner relationships. By controlling for previous heavy substance use, our study presents evidence for the behavior contagion mechanism of partner substance use similarities (where an individual's partner's use influences their own use), in contrast to the assortative mating mechanism (where individuals select partners with similar behaviors). The significant associations between partner smoking and comorbid AUD and TD, after controlling for daily smoking in high school, show that the increased risk is not due just to smokers selecting other smokers as partners, though the slight attenuation in odds ratio shows that partner selection for similar behaviors may still be part of the story.

4.3. Adolescent daily smoking and dispositional characteristics

We found adult partner environment influences even after controlling for demographics, adolescent dispositional characteristics, and income and depression in adulthood. Of additional interest are the variables that had a significant association in the final step, when all variables were considered simultaneously. Daily smoking in adolescence was a highly

significant predictor of being comorbid compared to having AUD only. Though not surprising, this highlights the need to prevent initiation of smoking behaviors in adolescence, especially given the increased probability of becoming tobacco dependent for individuals who begin smoking early. Although behavioral disinhibition was correlated with the outcome groups at the zero order, it was not a significant predictor in the final model. It is likely that the effect of behavioral disinhibition was mediated through other model covariates, particularly adolescent binge drinking and daily smoking. On the other hand, inhibition/anxiety and adult depression were significantly greater for the AUD only group than the comorbid group, perhaps because anxious and depressed individuals with AUD are more likely to use smoking to moderate their internalizing traits. These findings contrast somewhat with other studies demonstrating stronger effects of externalizing behavior than internalizing behavior on the development of problem behaviors (National Institute on Alcohol Abuse and Alcoholism, 2004; Hill et al., 2010; Schuckit and Hesselbrock, 1994; Zucker, 2008).

4.4. Limitations

Some limitations should be considered when interpreting the findings. First, this analysis does not consider the course of substance use disorders or changes in partnering in young adulthood. Variation in ages of onset and possible desistence of disorder and simultaneous or alternating diagnoses were apparent in preliminary analyses and are likely to be of importance in prevention and gene-environment research (Conway et al., 2006; Jackson et al., 2009). Second, this study did not examine concurrent partner influences on alcohol and tobacco disorders or, because the partner use predictors and participant outcomes were measured at different time points, participant and partner concordance in substance use. Future analyses could model these transitions and heterogeneity by incorporating these time-varying predictors and outcomes. Third, assessments were taken every 3 years, so we may have missed some cases of disorder or partner environment. Lastly, as noted above, the limited variability of our participant reported measure of partner regular alcohol use may be contributing to the lack of significant alcohol-specific influences. Partner substance use measures that attempt to more objectively measure dosing or severity of use may provide more robust effects.

Despite these limitations, there are a number of strengths to this analysis. The data are from an ethnically diverse, gender-balanced, community sample with prospectively collected data and high retention. Four waves of adult assessment over 10 years allowed us to capture a higher cumulative prevalence of these substance use disorders. Furthermore, a longitudinal assessment starting in early adolescence allowed us to control for earlier substance use so that we could better understand the mechanisms of partner behavior similarity. Finally, information on relationship quality in addition to partner substance use allowed us to test both general and substance-specific partner environmental influences.

4.5. Implications for genetic research, prevention, and treatment

Studies examining the etiology of tobacco and alcohol use disorders may benefit from considering comorbid subtypes in addition to those based on single use disorders. Such comorbid phenotypes may drive more successful identification and understanding of genetic

and physiological mechanisms that act in concert with social environmental processes to lead to substance abuse (Conway et al., 2006; Jackson et al., 2009). Behavioral geneticists have identified several genetic markers associated with problem use of multiple substances (Li et al., 2008; Philibert et al., 2009), as well as genetic markers associated specifically with tobacco or alcohol problem use (Bierut et al., 2004). Person-centered typological approaches may aid in identifying individuals with a comorbid phenotype and in examining how variables influence this phenotype differently from how they influence single disorder phenotypes.

The distinction of behavior contagion versus assortative mating is important to recognize in gene-environment studies, because assortative mating may be considered a form of gene-environment correlation, where individuals actively seek, select, and modify environments that match their genetically influenced traits and dispositions, and should be adjusted for accordingly (Harden et al., 2008; Rose and Dick, 2004). Alternatively, partner behavior contagion may reflect causal effects of the environment on the individual, and may be less influenced by genetics or may involve different genetic processes compared to assortative mating.

Results from our study demonstrate general and specific partner environment influences on alcohol and tobacco use disorder comorbidity. These results suggest that interventions to prevent AUD and TD should focus on both general and substance-specific risk factors for these disorders. In young adulthood, relationship quality and partner conflict may be targets for general prevention, and partner smoking may be a target of tobacco-specific interventions. Furthermore, we found that adolescent daily smokers were more likely to meet criteria for TD and to be comorbid for both AUD and TD, emphasizing the need to prevent initiation and escalation of smoking in adolescence. Interventions to treat AUD and TD should also focus on their comorbid occurrence and simultaneous and alternating substance use (Drobes, 2002; Kalman et al., 2010), as well as improving relationship quality and engaging both partners to reduce the harms from alcohol and tobacco use on themselves and on each other.

Acknowledgments

The authors gratefully acknowledge SSDP panel participants for their continued contribution to the longitudinal study. We also acknowledge the SDRG Survey Research Division for their hard work maintaining high panel retention, and the SDRG editorial and administrative staff for their project and editorial support.

Portions of these findings were presented in June 2011 at the annual meeting of the Society for Prevention Research in Washington, DC.

Role of Funding Source

Funding for this study was provided by grants from the National Institute on Drug Abuse (NIDA; R01DA009679; R01DA024411), the National Institute on Alcohol Abuse and Alcoholism (NIAAA; R01AA016960) and by grant 21548 from the Robert Wood Johnson Foundation. The content of this paper is the sole responsibility of the authors and does not necessarily represent the official views of the funding agencies. NIAAA, NIDA, and RWJF had no role in determining study design; in the collection, analysis or interpretation of data; in the writing of the report; or in the decision to submit the paper for publication.

REFERENCES

- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed.. American Psychiatric Association; Washington, DC.: 1994.
- Babor TF, Dolinsky ZS, Meyer RE, Hesselbrock MM, Hofmann M, Tennen H. Types of alcoholics: concurrent and predictive validity of some common classification schemes. *Br. J. Addict.* 1992; 87:1415–1431. [PubMed: 1330126]
- Bachman, JG.; O'Malley, PM.; Schulenberg, JE.; Johnston, LD.; Bryant, AL.; Merline, AC. The Decline of Substance Use in Young Adulthood: Changes in Social Activities, Roles, and Beliefs. Lawrence Erlbaum Associates; Mahwah, NJ: 2002.
- Bachman, JG.; Wadsworth, KN.; O'Malley, PM.; Schulenberg, J.; Johnston, LD. Marriage, divorce, and parenthood during the transition to young adulthood: impacts on drug use and abuse.. In: Schulenberg, J.; Maggs, JL.; Hurrelmann, K., editors. *Health Risks and Developmental Transitions During Adolescence*. Cambridge University Press; Cambridge: 1997. p. 246-279.
- Bailey JA, Hill KG, Meacham MC, Young S, Hawkins JD. Strategies for characterizing complex phenotypes and environments: general and specific family environmental predictors of young adult tobacco dependence, alcohol use disorder, and co-occurring problems. *Drug Alcohol Depend.* 2011; 118:444–451. [PubMed: 21636226]
- Bierut LJ, Dinwiddie SH, Begleiter H, Crowe RR, Hesselbrock V, Nurnberger JI Jr, Porjesz B, Schuckit MA, Reich T. Familial transmission of substance dependence: alcohol, marijuana, cocaine, and habitual smoking: a report from the Collaborative Study on the Genetics of Alcoholism. *Arch. Gen. Psychiatry.* 1998; 55:982–988. [PubMed: 9819066]
- Bierut LJ, Rice JP, Goate A, Hinrichs AL, Saccone NL, Foroud T, Edenberg HJ, Cloninger CR, Begleiter H, Conneally PM, Crowe RR, Hesselbrock V, Li TK, Nurnberger JI Jr, Porjesz B, Schuckit MA, Reich T. A genomic scan for habitual smoking in families of alcoholics: common and specific genetic factors in substance dependence. *Am. J. Med. Genet.* 2004; 124A:19–27. [PubMed: 14679582]
- Carver CS, White TL. Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: the BIS/BAS Scales. *J. Pers. Soc. Psychol.* 1994; 67:319–333.
- Catalano, RF.; Hawkins, JD. The social development model: a theory of antisocial behavior.. In: Hawkins, JD., editor. *Delinquency and Crime: Current Theories*. Cambridge University Press; New York: 1996. p. 149-197.
- Centers for Disease Control and Prevention. Vital signs: current cigarette smoking among adults aged > 18 years – United States, 2009. *MMWR.* 2010; 59:1135–1140. [PubMed: 20829747]
- Chen K, Kandel DB. The natural history of drug use from adolescence to the mid-thirties in a general population sample. *Am. J. Public Health.* 1995; 85:41–47. [PubMed: 7832260]
- Clark DB, Sayette MA. Anxiety and the development of alcoholism. *Am. J. Addict.* 1993; 2:59–76.
- Conway KP, Compton WM, Miller PM. Novel approaches to phenotyping drug abuse. *Addict. Behav.* 2006; 31:923–928.
- Daley SE, Hammen C. Depressive symptoms and close relationships during the transition to adulthood: perspectives from dysphoric women, their best friends, and their romantic partners. *J. Consult. Clin Psychol.* 2002; 70:129–141. [PubMed: 11860039]
- Drobes DJ. Concurrent alcohol and tobacco dependence: mechanisms and treatment. *Alcohol Res. Health.* 2002; 26:136–142.
- Falk DE, Yi HY, Hiller-Sturmhofel S. An epidemiologic analysis of co-occurring alcohol and tobacco use and disorders: findings from the National Epidemiologic Survey on Alcohol and Related Conditions. *Alcohol Res. Health.* 2006; 29:162–171. [PubMed: 17373404]
- Fleming CB, White HR, Catalano RF. Romantic relationships and substance use in early adulthood: an examination of the influences of relationship type, partner substance use, and relationship quality. *J. Health Soc. Behav.* 2010; 51:153–167. [PubMed: 20617756]
- Grant BF, Harford TC. Comorbidity between DSM-IV alcohol use disorders and major depression: results of a national survey. *Drug Alcohol Depend.* 1995; 39:197–206. [PubMed: 8556968]

- Harden KP, Hill JE, Turkheimer E, Emery RE. Gene-environment correlation and interaction in peer effects on adolescent alcohol and tobacco use. *Behav. Genet.* 2008; 38:339–347. [PubMed: 18368474]
- Hasin DS, Stinson FS, Ogburn E, Grant BF. Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States. *Arch. Gen. Psychiatry.* 2007; 64:830–842. [PubMed: 17606817]
- Hawkins JD, Catalano RF, Kosterman R, Abbott R, Hill KG. Preventing adolescent health-risk behaviors by strengthening protection during childhood. *Arch. Pediatr. Adolesc. Med.* 1999; 153:226–234. [PubMed: 10086398]
- Hawkins JD, Catalano RF, Miller JY. Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: implications for substance-abuse prevention. *Psychol. Bull.* 1992; 112:64–105. [PubMed: 1529040]
- Hill KG, Hawkins JD, Bailey JA, Catalano RF, Abbott RD, Shapiro VB. Person-environment interaction in the prediction of alcohol abuse and alcohol dependence in adulthood. *Drug Alcohol Depend.* 2010; 110:62–69. [PubMed: 20299164]
- Hill KG, White HR, Chung I-J, Hawkins JD, Catalano RF. Early adult outcomes of adolescent binge drinking: Person- and variable-centered analyses of binge drinking trajectories. *Alcohol. Clin. Exp. Res.* 2000; 24:892–901. [PubMed: 10888080]
- Homish GG, Leonard KE. Spousal influence on smoking behaviors in a U.S. community sample of newly married couples. *Soc. Sci. Med.* 2005; 61:2557–2567. [PubMed: 15978712]
- Homish GG, Leonard KE, Kearns-Bodkin JN. Alcohol use, alcohol problems, and depressive symptomatology among newly married couples. *Drug Alcohol Depend.* 2006; 83:185–192. [PubMed: 16337752]
- Hopkins DP, Briss PA, Ricard CJ, Husten CG, Carande-Kulis VG, Fielding JE, Alao MO, McKenna JW, Sharp DJ, Harris JR, Woollery TA, Harris KW, Task Force on Community Preventive S. Reviews of evidence regarding interventions to reduce tobacco use and exposure to environmental tobacco smoke. *Am. J. Prev. Med.* 2001; 20:S16–S66.
- Horwitz AV, White HR. Becoming married, depression, and alcohol problems among young adults. *J. Health Soc. Behav.* 1991; 32:221–237. [PubMed: 1940207]
- Iacono WG, Carlson SR, Taylor J, Elkins IJ, McGue M. Behavioral disinhibition and the development of substance-use disorders: findings from the Minnesota Twin Family Study. *Dev. Psychopathol.* 1999; 11:869–900. [PubMed: 10624730]
- Iacono WG, Malone SM, McGue M. Behavioral disinhibition and the development of early-onset addiction: common and specific influences. *Annu. Rev. Clin. Psychol.* 2008; 4:325–348. [PubMed: 18370620]
- Jackson KM, Sher KJ, Rose RJ, Kaprio J. Trajectories of tobacco use from adolescence to adulthood: are the most informative phenotypes tobacco specific? Phenotypes and endophenotypes: foundations for genetic studies of nicotine use and dependence. *NCI Tobacco Control Monograph Series.* 2009:289–335.
- Jackson KM, Sher KJ, Wood PK. Trajectories of concurrent substance use disorders: a developmental, typological approach to comorbidity. *Alcohol. Clin. Exp. Res.* 2000; 24:902–913. [PubMed: 10888081]
- Jackson KM, Sher KJ, Wood PK, Bucholz KK. Alcohol and tobacco use disorders in a general population: short-term and long-term associations from the St. Louis Epidemiological Catchment Area Study. *Drug Alcohol Depend.* 2003; 71:239–253. [PubMed: 12957342]
- John U, Meyer C, Rumpf HJ, Schumann A, Thyrian JR, Hapke U. Strength of the relationship between tobacco smoking, nicotine dependence and the severity of alcohol dependence syndrome criteria in a population-based sample. *Alcohol Alcohol.* 2003; 38:606–612. [PubMed: 14633650]
- Kalman D, Kim S, DiGirolamo G, Smelson D, Ziedonis D. Addressing tobacco use disorder in smokers in early remission from alcohol dependence: the case for integrating smoking cessation services in substance use disorder treatment programs. *Clin. Psychol. Rev.* 2010; 30:12–24. [PubMed: 19748166]

- Kearns-Bodkin JN, Leonard KE. Alcohol involvement and marital quality in the early years of marriage: a longitudinal growth curve analysis. *Alcohol. Clin. Exp. Res.* 2005; 29:2123–2134. [PubMed: 16385182]
- Kessler RC, Berglund P, Demler O, Jin R, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Arch. Gen. Psychiatry.* 2005a; 62:593–602. [PubMed: 15939837]
- Kessler RC, Chiu WT, Demler O, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Arch. Gen. Psychiatry.* 2005b; 62:617–627. [PubMed: 15939839]
- Khantzian, EJ.; Albanese, MJ. *Understanding Addiction as Self Medication: Finding Hope Behind the Pain.* Rowman & Littlefield; Lanham, MD.: 2008.
- Kreek MJ, Nielsen DA, Butelman ER, LaForge KS. Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction. *Nat. Neurosci.* 2005; 8:1450–1457. [PubMed: 16251987]
- Laursen B, Hoff E. Person-centered and variable-centered approaches to longitudinal data. *Merrill Palmer Q.* 2006; 52:377–389.
- Le Strat Y, Ramoz N, Gorwood P. In alcohol-dependent drinkers, what does the presence of nicotine dependence tell us about psychiatric and addictive disorders comorbidity? *Alcohol Alcohol.* 2010; 45:167–172. [PubMed: 20089545]
- 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. *J. Consult. Clin. Psychol.* 2001; 69:683–698. [PubMed: 11550734]
- Leonard KE, Das Eiden R. Husband's and wife's drinking: unilateral or bilateral influences among newlyweds in a general population sample. *J. Stud. Alcohol.* 1999; 13:130–138.
- Leonard KE, Mudar P. Peer and partner drinking and the transition to marriage: a longitudinal examination of selection and influence processes. *Psychol. Addict. Behav.* 2003; 17:115–125. [PubMed: 12814275]
- Li CY, Mao X, Wei L. Genes and (common) pathways underlying drug addiction. *PLoS Comput. Biol.* 2008; 4:e2. [PubMed: 18179280]
- Maggs JL, Schulenberg JE. Trajectories of alcohol use during the transition to adulthood. *Alcohol Res. Health.* 2004; 28:195–201.
- Meier A, Allen G. Romantic relationships from adolescence to young adulthood: evidence from the National Longitudinal Study of Adolescent Health. *Sociol. Q.* 2009; 50:308–335.
- Merline AC, Schulenberg J, O'Malley PM, Bachman JG, Johnston LD. Substance use in marital dyads: premarital assortment and change over time. *J. Stud. Alcohol Drugs.* 2008; 69:352–361. [PubMed: 18432377]
- Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. *JAMA.* 2004; 291:1238–1245. [PubMed: 15010446]
- Naimi TS, Brewer RD, Mokdad A, Denny C, Serdula MK, Marks JS. Binge drinking among US adults. *JAMA.* 2003; 289:70–75. [PubMed: 12503979]
- National Institute on Alcohol Abuse and Alcoholism. *Alcohol and tobacco (Alcohol Alert No. 39).* Bethesda, MD: 1998.
- National Institute on Alcohol Abuse and Alcoholism. *Psychosocial processes and mechanisms of risk and protection.* *Alcohol Res. Health.* 2004; 28:143–154. [PubMed: 19093688]
- Philibert RA, Gunter TD, Beach SRH, Brody GH, Hollenbeck N, Andersen A, Adams W. Role of GABRA2 on risk for alcohol, nicotine, and cannabis dependence in the Iowa Adoption Studies. *Psychiatr. Genet.* 2009; 19:91–98. [PubMed: 19672139]
- Pombo S, Lesch OM. The alcoholic phenotypes among different multidimensional typologies: similarities and their classification procedures. *Alcohol Alcohol.* 2009; 44:46–54. [PubMed: 18832138]
- Rhule-Louie DM, McMahon RJ. Problem behavior and romantic relationships: assortative mating, behavior contagion, and desistance. *Clin. Child Fam. Psychol. Rev.* 2007; 10:53–100. [PubMed: 17318381]

- Robins, LN.; Helzer, JE.; Croughan, J.; Williams, JBW.; Spitzer, RL. NIMH Diagnostic Interview Schedule: Version III (May 1981). National Institute of Mental Health; Rockville, MD.: 1981.
- Rose RJ, Dick DM. Gene-environment interplay in adolescent drinking behavior. *Alcohol Res. Health.* 2004; 28:222–229.
- Rutter M, Moffitt TE, Caspi A. Gene-environment interplay and psychopathology: multiple varieties but real effects. *J. Child Psychol. Psychiatry.* 2006; 47:226–261. [PubMed: 16492258]
- Schuckit MA, Hesselbrock V. Alcohol dependence and anxiety disorders: what is the relationship? *Am. J. Psychiatry.* 1994; 151:1723–1734. [PubMed: 7977877]
- Sher KJ, Gotham HJ, Erickson DJ, Wood PK. A prospective, high-risk study of the relationship between tobacco dependence and alcohol use disorders. *Alcohol. Clin. Exp Res.* 1996; 20:485–492. [PubMed: 8727241]
- Sher KJ, Trull TJ. Personality and disinhibitory psychopathology: alcoholism and antisocial personality disorder. *J. Abnorm. Psychol.* 1994; 103:92–102. [PubMed: 8040486]
- Spanier GB. Measuring dyadic adjustment: new scales for assessing the quality of marriage and similar dyads. *J. Marriage Fam.* 1976; 38:15–28.
- Spanier GB, Thompson L. A confirmatory analysis of the Dyadic Adjustment Scale. *J. Marriage Fam.* 1982; 44:731–738.
- Substance Abuse and Mental Health Services Administration. Results from the 2003 National Survey on Drug Use and Health: National Findings. Office of Applied Statistics; Rockville, MD: 2004.
- Swendsen JD, Tennen H, Carney MA, Affleck G, Willard A, Hromi A. Mood and alcohol consumption: an experience sampling test of the self-medication hypothesis. *J. Abnorm. Psychol.* 2000; 109:198–204. [PubMed: 10895557]
- Tsuang MT, Lyons MJ, Meyer JM, Doyle T, Eisen SA, Goldberg J, True W, Lin N, Toomey R, Eaves L. Co-occurrence of abuse of different drugs in men: the role of drug-specific and shared vulnerabilities. *Arch. Gen. Psychiatry.* 1998; 55:967–972. [PubMed: 9819064]
- Tully EC, Iacono WG, McGuea M. Changes in genetic and environmental influences on the development of nicotine dependence and major depressive disorder from middle adolescence to early adulthood. *Dev. Psychopathol.* 2010; 22:831–848. [PubMed: 20883585]
- Wagenaar AC, Toomey TL, Lenk KM. Environmental influences on young adult drinking. *Alcohol Res. Health.* 2005; 28:230–235.
- Zucker RA. Anticipating problem alcohol use developmentally from childhood into middle adulthood: what have we learned? *Addiction.* 2008; 103:100–108. [PubMed: 18426543]

Table 1**Descriptive Statistics**

Continuous variable means and standard deviations are presented based on raw, unstandardized data to aid interpretation.

Study variables (response range)	n	%	mean	s.d.
Outcome				
Neither	330	52.5%	--	--
TD only	62	9.9%	--	--
AUD only	144	22.9%	--	--
Comorbid	92	14.6%	--	--
Total	628			
Partner environment				
Partner conflict (1–6)	--	--	2.12	.68
Partner drinking	408	65.0%	--	--
Partner smoking	263	41.9%	--	--
Demographics				
Gender				
Male	307	48.9%	--	--
Female	321	51.1%	--	--
Ethnicity				
Caucasian	305	48.6%	--	--
African American	155	24.7%	--	--
Asian American	134	21.3%	--	--
Native American	34	5.4%	--	--
Personality				
Disinhibition (1–5)	--	--	2.58	.94
Anxiety/inhibition (1–3)	--	--	1.67	.36
Adolescent substance use				
Years reported binge drinking 0	449	71.5%	--	--
1	124	19.7%	--	--
2	38	6.1%	--	--
3	16	2.5%	--	--
4	1	.2%	--	--
Years reported daily smoking 0	464	73.9%	--	--
1	75	11.9%	--	--
2	45	7.2%	--	--
3	28	4.5%	--	--
4	16	2.5%	--	--
Income-to-needs ratio	--	--	6.12	4.99
Depression (waves met criteria) 0	450	71.7%	--	--
1	109	17.4%	--	--
2	47	7.5%	--	--

Study variables (response range)	n	%	mean	s.d.
3	17	2.7%	--	--
4	5	.8%	--	--

Table 2

Pearson correlations between model variables (n = 628).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. No disorder																	
2. TD only	--																
3. AUD only	--	--															
4. Comorbid	--	--	--														
5. Partner conflict	-.12	-.02	.00	.18													
6. Partner smoking	-.27	.18	.01	.21	.15												
7. Partner drinking	-.15	.03	.08	.09	.08	.27											
8. Male	-.25	.01	.16	.15	.04	-.01	-.11										
9. Caucasian American	-.11	.05	.09	.00	-.07	.02	.19	.02									
10. African American	.04	.01	-.05	.00	.11	.06	-.03	-.01	--								
11. Asian American	.13	-.09	-.04	-.05	-.05	-.09	-.22	.03	--	--							
12. Native American	-.05	.04	-.03	.08	.05	.00	.03	-.08	--	--	--						
13. Behavioral disinhibition	-.30	.08	.14	.21	.18	.10	.17	.24	.13	.03	-.25	.11					
14. Inhibition/anxiety	.05	.00	-.13	.09	.16	.04	-.04	-.18	-.04	-.10	.13	.05	.03				
15. Adolescent daily smoking	-.31	.25	-.04	.27	.05	.23	.09	.02	.12	-.06	-.17	.17	.34	.08			
16. Adolescent binge drinking	-.29	.07	.01	.23	.02	.13	.12	.12	.10	-.05	-.15	.15	.41	.03	.39		
17. Adult income-to-needs ratio	-.03	-.12	.12	-.01	-.14	-.04	-.02	.16	.13	-.18	.09	-.09	.03	.01	-.15	.01	
18. Adult depression	-.16	.11	-.04	.18	.19	.13	.03	-.12	-.03	.03	-.07	.14	.07	.16	.16	.11	-.12

Bold = significant p < .05

Outcome variables (1-4) and ethnicity variables (9-12) are listed as dummy variables for computing correlations with other covariates. In the analysis, comorbid is the outcome reference group and Caucasian is the ethnicity reference group.

Table 3

Sequential multinomial logistic regressions differentiating the comorbid tobacco and alcohol disorder group from single disorder or none.

Step 1	NONE		TD		AUD	
	OR	95% CI	OR	95% CI	OR	95% CI
Partner conflict	0.57***	(0.43-0.78)	0.55**	(0.36-0.85)	0.65*	(0.46-0.90)
Partner drinking	0.73	(0.41-1.26)	0.77	(0.36-1.61)	1.20	(0.64-2.25)
Partner smoking	0.24***	(0.14-0.40)	1.31	(0.63-2.71)	0.38**	(0.22-0.68)
Step 2						
Partner conflict	0.53***	(0.38-0.74)	0.54**	(0.35-0.84)	0.61**	(0.43-0.87)
Partner drinking	0.85	(0.47-1.55)	0.84	(0.39-1.77)	1.29	(0.68-2.46)
Partner smoking	0.29***	(0.16-0.50)	1.21	(0.58-2.52)	0.44**	(0.24-0.78)
Adolescent binge drinking	0.43***	(0.30-0.62)	0.66+	(0.43-1.00)	0.87	(0.62-1.21)
Adolescent daily smoking	0.51***	(0.39-0.67)	1.15	(0.88-1.49)	0.60***	(0.45-.79)
Step 3						
Partner conflict	0.57**	(0.40-0.81)	0.57*	(0.36-0.90)	0.66*	(0.46-0.96)
Partner drinking	0.71	(0.38-1.35)	0.69	(0.30-1.44)	1.12	(0.58-2.27)
Partner smoking	0.26***	(0.15-0.47)	1.23	(0.59-2.64)	0.46**	(0.25-0.82)
Adolescent binge drinking	0.55***	(0.15-0.55)	0.74	(0.33-1.45)	0.88	(0.38-1.36)
Adolescent daily smoking	0.51***	(0.14-0.48)	1.10	(0.77-3.43)	0.58**	(0.19-0.68)
Male	0.23***	(0.14-0.45)	0.46*	(0.22-0.94)	0.62	(0.37-1.28)
African American	1.02	(0.51-1.96)	0.89	(0.40-2.04)	0.60	(0.30-1.23)
Asian American	0.97	(0.45-2.18)	0.49	(0.16-1.52)	0.81	(0.38-1.23)
Native American	0.67	(0.20-1.82)	0.84	(0.25-2.96)	0.49	(0.14-1.52)
Behavioral disinhibition	0.75	(0.53-1.12)	0.85	(0.50-1.28)	1.11	(0.73-1.58)
Inhibition/anxiety	0.80	(0.46-1.25)	0.71	(0.38-1.31)	0.50**	(0.29-0.84)
Step 4						
Partner conflict	0.62*	(0.43-0.89)	0.52***	(0.32-0.84)	0.72+	(0.49-1.05)

Step 1	NONE		TD		AUD	
	OR	95% CI	OR	95% CI	OR	95% CI
Partner drinking	0.66	(0.34-1.26)	0.71	(0.32-1.57)	1.04	(0.52-2.08)
Partner smoking	0.28***	(0.16-0.51)	1.22	(0.58-2.59)	0.48*	(0.26-0.88)
Adolescent binge drinking	0.57**	(0.39-0.84)	0.72	(0.46-1.12)	0.90	(0.63-1.29)
Adolescent daily smoking	0.52***	(0.38-0.70)	1.03	(0.77-1.38)	0.60**	(0.45-0.73)
Male	0.21***	(0.11-0.39)	0.51 ⁺	(0.24-1.09)	0.55 ⁺	(0.29-1.06)
African American	1.00	(0.51-2.00)	0.72	(0.31-1.67)	0.64	(0.31-1.32)
Asian American	0.90	(0.41-1.98)	0.46	(0.15-1.42)	0.76	(0.33-1.76)
Native American	0.77	(0.24-2.45)	0.76	(0.21-2.72)	0.59	(0.18-1.95)
Behavioral disinhibition	0.74	(0.51-1.09)	0.90	(0.55-1.41)	1.07	(0.72-1.59)
Inhibition/anxiety	0.87	(0.52-1.45)	0.74	(0.39-1.40)	0.53*	(0.30-0.91)
Income-to-needs ratio	0.86	(0.65-1.13)	0.55*	(0.33-0.92)	1.05	(0.83-1.33)
Adult depression	0.56**	(0.43-0.80)	0.87	(0.61-1.75)	0.70*	(0.50-0.98)

p<.001**
p<.01*
p<.05+
p<.10